1-1-2018

The Effect Of Treadmill Vs. Nustep Recumbent Cross Trainer On Gait And Lower Extremity Electromyography After Chronic Stroke

Nicholas Joseph Siekirk
Wayne State University,

THE EFFECT OF TREADMILL VS. NUSTEP RECUMBENT CROSS TRAINER ON GAIT AND LOWER EXTREMITY ELECTROMYOGRAPHY AFTER CHRONIC STROKE

by

NICHOLAS J. SIEKIRK

DISSERTATION

Submitted to the Graduate School

of Wayne State University,

Detroit, Michigan

in partial fulfillment of the requirements

for the degree of

DOCTOR OF PHILOSOPHY

2018

MAJOR: KINESIOLOGY

Approved By:

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Advisor   Date

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DEDICATION

To those who have inspired me...
_____

And to those who we have lost:

Tau Kappa Epsilon Theta Omicron

Justin C.Z. Rush (1989-2018)

Siekirk

John F. Siekirk (1924-2000)

Pulaski

Eugene R. Pulaski (1922-2009)
Anastasia S. Pulaski (1921-2012)
Alan D. Kirchoff (1974-2015)
James J. David (1976-2014)

Muhleck

Earl N. Muhleck (1931 – 2017)

Nave

Reno V. Nave (1938-2017)
_____

L'espoir ne meurt jamais

ACKNOWLEDGEMENTS

PREFACE

The basis for this research stemmed from a passionate and continued curiosity on how exercise can assist those with chronic disease and disability. Progression requires the continued validation and critical examination of methods and machinery.

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CHAPTER 1 INTRODUCTION

Humans will perform various rhythmic movements as part of their day's activities. This whole body movement is naturally multiplanar and requires a blend of acceleration, deceleration and dynamic stabilization (Wannier, Bastiaanse, Colombo & Dietz, 2001). Human locomotion is a rhythmic whole body movement that consists of alternating coupled patterns of flexion and extension. Rhythmic and coupled patterns of muscle activation (Jakobi & Chilibeck, 2001) and reflex responses (Cerri, Borroni & Baldissera., 2003; Zehr et al., 2007) have been observed across multijoint and multi-limb motor patterns such as walking, crawling and swimming (Huang & Ferris, 2009b). In populations that find whole body movement to be burdensome following injury or disease, an intermediary or assistive device is often deployed. Howbeit, targeted and purposeful exercise (with or without the assisted device) should also be considered as part of therapeutic programming. Ideally, this programming should promote muscle activation without compromising the exerciser's safety. Most individuals with a recent history of a cerebral vascular accident (CVA) are deconditioned; exhibiting a peak oxygen consumption that is about half of age-matched controls (Palmer-McLean & Harbst, 2003). This deconditioned state leaves tremendous room for improvement (Palmer-McLean & Harbst, 2003). Exercise not only can improve aerobic capacity but self-selected (SS) walking speed, increased mobility and reduced reliance on assisted devices (Palmer-McLean & Harbst, 2003). Rehabilitative exercise that targets locomotor pathways may improve quality of life (Stoloff, Zehr & Ferris, 2007). Improvements in gait provide a significant clinical marker of recovery (Yang et al., 2005). Therefore, it seems intuitive to investigate exercise modes that would improve gait.

The NuStep® (NuStep LLC Ann Arbor, MI, USA) is a widely available and commonly used recumbent cross-trainer that has been used both in clinical and research settings. NuStep

provides a coupled reciprocal leg extension-flexion with corresponding opposite arm flexion-extension (Stoloff et al., 2007). The motion simulates a reciprocal pattern similar to walking but in a seated and guided manner.

Statement of Problem – Muscle Activation – Pilot 1

Previous investigations have only studied the NuStep cross trainer during predetermined cadences. These predetermined cadences have varied between 30-120 steps per minute across several investigations (Huang & Ferris, 2004; Kao & Ferris, 2005; Billinger, Loudon & Gajewsk, 2008; Huang & Ferris, 2009a; Huang & Ferris, 2009b; Dalleck et al., 2011, Billinger et al., 2012; de Kam et al., 2013; Wilson et al., 2017). One previous investigation also matched stepping frequency on the NuStep Cross Trainer to the participant's stepping frequency while on the Treadmill (TM) (Stoloff, Zehr & Ferris, 2007). None of these investigations attempted to determine an individuals' self-selected cadence despite the clinical commonality of such use. That is, in clinical practice, patients are often asked to step at a comfortable pace as part of a warm-up. Moreover, despite this commonality of this cue, the muscular response is not clear. Therefore, the effect of a self-selected cadence, as determined by an individual's rating of perceived exertion on lower extremity muscle activation (as measured by EMG amplitude) on the NuStep cross trainer has yet to be determined. Furthermore, muscle activity of the lower extremity, in the absence of arm movement has not been investigated to date and therefore remains unknown.

For these reasons, our investigation sought answers to the following research questions: 1– What is the RPE based self-selected (SS) cadence of healthy exercisers on the NuStep? 2 - How does a deviation in SS cadence affect EMG recruitment on NuStep? 3 – How does an increase in resistance (while performing SS cadence) affect EMG recruitment on NuStep?

The purpose of this pilot was to study the electromyographic (EMG) activity of 12 lower extremity muscles during five different 5 minute stepping protocols; self-selected level 1 (SSL1), self-selected level 8 (SSL8), +20% self-selected (SS+20), -20% self-selected (SS-20), and 80 steps per minute at resistance level 1 (80SL1). Based on these research questions, we hypothesized that an increase in stepping cadence (above the participant's SS cadence) and an increase in resistance (at level 8) would result in higher mean EMG (mEMG) amplitudes. We also hypothesized that a decrease in stepping cadence (below the participant's SS cadence) would result in lowered mean EMG amplitudes.

Statement of the Problem Muscle Adaptation –Pilot Part II

Exercise has been shown to improve neural plasticity (Kandel et al., 2015). Previous research concluded that recumbent stepping relies on similar neural networks as walking, and therefore have suggested that the NuStep can promote neural plasticity and recovery of walking (Stoloff, Zehr & Ferris, 2007). It is also theorized that muscle recruitment becomes more efficient as a result of task-specific training (Kenny, Wilmore & Costil, 2015). Although muscle activity while stepping with a NuStep has been studied previously, the potential change in muscle activity to stepping over time has yet to be determined. Therefore the investigation sought to answer the following questions: 1 - Does the deviation from a self-selected stepping speed result in a delayed adaptation of muscle activation on the NuStep? 2 - Does an increase in stepping resistance at self-selected stepping speed result in a delayed adaptation of muscle activation on the NuStep?

The purpose of this investigation was to study the electromyographic (EMG) activity of 12 lower extremity muscles during five different stepping protocols; self-selected level 1 (SSL1), self-selected level 8 (SSL8), +20% self-selected (SS+20), -20% self-selected (SS-20), and 80

steps per minute at resistance level 1 (80SL1) at minute 2 and minute 4 of a 5 minute exercise protocol. Based on these research questions, we hypothesized that both elevated cadence (SS+20) and resistance (SSL8) would result in the highest adaptation in participant's EMG between minute 2 and minute 4 of the exercise protocol.

Statement of the Problem – NuStep Cross Trainer vs. Treadmill

Leg impairments continue to undermine the performance of valued activities long after formal rehabilitation has ended (Page, Levine, Teepen & Hartman, 2008). If locomotion is reliant on central pattern generators (CPG) – is it reasonable to investigate methods to activate such networks (Dietz, 2002). Therefore, the NuStep recumbent stepper can be a potential intervention to improve muscle activation and, perhaps, lower limb symmetry during gait with chronic stroke patients. Over time, these improvements in gait may improve functional mobility or quality of life. Stoloff, Zehr, and Ferris compared muscle activation and kinematics of 50% and 0% bodyweight walking vs. recumbent stepping (2007). The authors suggested that walking and recumbent stepping use similar muscular activation patterns despite substantial differences in joint kinematics (e.g., the range of motion and temporal differences in muscle EMG). Although these tasks differ in kinematics, recumbent stepping seems to rely on similar but simpler neural networks as walking (Stoloff, Zehr et Ferris, 2007). Neurologically impaired individuals may be able to improve walking ability from recumbent stepping (Huang & Ferris, 2009; Kao & Ferris, 2005; Stoloff, Zehr et Ferris, 2007; Zehr et al., 2007).

Spatiotemporal asymmetry is quite typical of poststroke gait. Hemiparetic gait is also characterized by slow and asymmetric steps (Yavuzer et al., 2006). This asymmetry leads to increased energy expenditure and risk of falls given equilibrium reaction is slowed (Sackley, Baguley, Gent & Hodgson, 1992). Impaired balance and increased fall risk are strongly

correlated with abnormal locomotive ability, functional deficits and length of stay in inpatient rehabilitation facilities (Pollock, Baer, Pomeroy and Langhorne, 2004). The restoration of hip, trunk and limb mechanics including improved weighting bearing on the paretic limb is of top priority in stroke rehabilitation. Since locomotion, balance and functional abilities (i.e., Activities of Daily Living, ADL) are dependent on the paretic limb's muscular activation - post-stroke, this dissertation focused on the effect of two exercise modalities on muscular activation (as measured by EMG) and joint excursion (ΔROM). The effect of a self-selected cadence (as determined by an individual's perceived exertion) on lower extremity muscle activation (as measured by EMG amplitude) on the NuStep Cross Trainer and Treadmill in a CVA population is yet to be adequately investigated. Therefore, this investigation sought answers to the following research questions: 1 - What is the mEMG/ ΔROM response on TM vs. NS in CVA population? 2 - What immediate effect does this modality have on gait in CVA? 3 - What is the mEMG/ΔROM response to TM vs. NS in age/sex-matched population? 4 - What immediate effect does this modality have on gait in CVA? The purpose of this investigation was, therefore, to compare the effects of treadmill walking vs. recumbent stepping on muscle mean EMG (mEMG) and joint excursion in chronic stroke survivors (i.e.,> 6 months post-CVA) vs. age (± 5 years) and sex-matched healthy participants. Secondly, the immediate effect of each exercise intervention on the participant's gait (over-ground 10m walk) was examined. We hypothesized that the TM would promote higher mEMG values below the knee as compared to the NuStep Cross Trainer (Soloff, Zehr, Ferris 2007). However, we expected to observe higher mEMG outputs in the thigh at a matched RPE based SS cadence on the NuStep. We expected to see similar joint excursions in the knee. However, we expected to see higher hip excursion but smaller ankle while on the

NuStep. Furthermore, it is hypothesized that following exercise on the NuStep Cross Trainer, gait parameters would improve in the CVA population.

CHAPTER 2 BACKGROUND

Cerebral Vascular Accident

The brain is highly vulnerable to disturbances of its blood supply (Barrett, Barman, Biotano, Brooks, 2012). CVAs are among the most frequent neurological disorders ranking fifth in the cause of death in the United States (Kochanket et al., 2014). About 795,000 people in the United States have a stroke each year (Mozzafarian et al., 2016). Stroke costs the United States an estimated $34 billion each year (Benjamin et al., 2017). This total includes the cost of health care services, medicines to treat stroke, and missed days of work (Benjamin et al., 2017). Approximately 66% will survive (Palmer-McLean & Harbst, 2003). The majority of CVA cases affect the elderly however 20% of strokes will occur in those less than 65 years of age (Palmer-McLean & Harbst, 2003).

A CVA is the result of vascular insufficiency in the brain through occlusion or hemorrhage of the brain's feeder vessels. This focal and acute disturbance not only affects nutrient delivery (i.e., oxygen and glucose) but metabolite removal (i.e., carbon dioxide). Due to this reduced blood supply (i.e., ischemia), CVA may breed both localized anoxia (i.e., the absence of oxygen) and hypoglycemia (i.e., low glucose). When ischemic conditions are severe and prolonged, neuronal infarction (i.e., neuron death) may occur. Atherosclerosis and thrombosis cause most occlusive strokes (Barrett, Barman, Biotano, Brooks, 2012). By comparison, hemorrhagic strokes are closely associated with advanced hypertension or an aneurysm (Barrett, Barman, Biotano, Brooks, 2012). Additionally, plaque can activate the body's clotting mechanism to reduce or worst-case block an artery. Strokes of either type may occur at any age from many other causes, including hypertension, diabetes mellitus, coronary arterial disease, smoking, alcoholism, trauma, infection, tumor, abnormal blood states (i.e., dyscrasia),

vascular malformation, immunological disorder, and exogenous toxins (Barrett, Barman, Biotano, Brooks, 2012; Palmer-McLean & Harbst, 2003).

The resulting neurological impairment depends on both the size and location of the ischemic area, as well as the availability of collateral blood flow (Palmer-McLean & Harbst, 2003). Following a CVA, persons may present with motor and sensory impairment, visual field deficits, impaired speech (i.e., expressive and receptive aphasia), mental confusion (Palmer-McLean & Harbst, 2003). Impairment of motor and sensory function may occur in the upper or lower extremity, or in both extremities, on the involved side (Palmer-McLean & Harbst, 2003). Cognitive and behavioral sequelae may influence exercise program retention and compliance (Palmer-McLean & Harbst, 2003). Frontal lobe involvement may reduce the drive for exercise initiation. Furthermore, apathy, frustration, loss of inhibition may occur as a result of impaired cognitive and executive functions (Palmer-McLean & Harbst, 2003). Temporal lobe involvement may limit future learning and interfere with memory recall (Palmer-McLean & Harbst, 2003). Finally, brain areas that mediate perception and arousal may lead to difficulty in maintaining attention (Palmer-McLean & Harbst, 2003).

Stroke is a leading cause of severe long-term disability. Stroke reduces mobility in more than half of stroke survivors age 65 and over (Benjamin et al., 2017). If the majority of strokes occur in the elderly, exercise prescription is further complicated by arthritis, orthopedic and cardiovascular ailments prevalent in the elderly (Palmer-McLean & Harbst, 2003).

CVAs may occur secondary to atherosclerotic lesions (Palmer-McLean & Harbst, 2003). Therefore many persons who experience a CVA have either coexisting coronary artery disease or are at risk for developing coronary artery disease. Therefore, exercise testing should be completed under the supervision of a qualified medical team complete with a 12-lead ECG

(Palmer-McLean & Harbst, 2003). The mode of exercise testing depends on the severity of neurological involvement. Keep in mind; exercise testing with focal neurological deficits can be more challenging than in the non-disabled persons (Palmer-McLean & Harbst, 2003). Exercise training programs can improve VO2 peak, endurance, and muscle strength. As a direct result, clients can elevate their independence and therefore become more employable (Palmer-McLean & Harbst, 2003).

Retraining of walking is a significant goal for persons with stroke (Olney & Richards, 1996). Only 23-37% of persons who have sustained a stroke can walk independently after one week (von Schroeder, Coutts, Lyden & Nickel, 1995) but 50-80% of survivors can ambulate unaided at 3 weeks or discharge (Burdett, Borello-France, Blatchly & Potter, 1988). At six months, <85% of survivors may walk unaided (Wade, Wood, Heller & Maggs, 1987). Reduced walking speeds and extended stance phases, longer on the unaffected side, are reported (Olney & Richards, 1996).

Locomotion

Humans utilize coordination patterns that maintain an integral frequency ratio between the upper limbs and lower limbs. This "coupling" is apparent during whole body rhythmic actions such as walking, crawling, and swimming. Muscle activation patterns and reflex responses during multijoint and multi-limb task have suggested that the "coupling" is driven by a neural component (Huang & Ferris 2009). Propriospinal connections between upper limb neural networks and lower limb neural networks have been implicated for this facilitation. Previous research on rhythmicity indicates that upper extremity activation (i.e., afferent feedback) may improve lower limb muscle recruitment (Huang & Ferris 2009).

It is hypothesized that basic neural signals are produced by a locomotor pattern generator and are shaped appropriately by cortical inputs and peripheral afferent feedback to regulate rhythmic movement (Stoloff, Zehr & Ferris, 2007). Locomotion is produced at seemingly low levels of the central nervous system (CNS) and is possible without intervention from higher centers (i.e., midbrain, cerebral cortex). However, because locomotion may occur in unfamiliar or unpredictable environments, higher center overwatch is often required (Pearson & Gordon, 2013). Real-time modification of the conventional- locomotive – motor program is necessary to adapt to changing environments. We must consider how neurons coordinate locomotion and how sensory input (e.g., visual, touch, or proprioceptive) may alter locomotion.

Modern research on the neural control of locomotion reached breakthrough by application of adrenergic drugs and later, the electrical brain stimulation of a de-cerebrate cat (Schmidt et al., 2018). Animal modeling (e.g., drug preparation, decerebrate preparation, deafferented preparation, immobilized preparation) of quadrupedal stepping have eluded that supraspinal commands are not necessary to produce the stepping motor pattern. The spinal cord neuron also houses the neural circuits responsible for locomotion. These spinal neurons are subject to supraspinal modulation. Lastly, these spinal pattern-generating networks do not require sensory input, however, are strongly influenced by the limb's sensory input (Pearson & Gordon, 2013).

Locomotion involves the coordinated contraction of several muscles. The analysis of gait reveals inherent complexities. However, gait may be broken into four distinct parts (Table 1). The stepping motor pattern is not merely an alteration of flexion and extension; instead, contractions are precisely timed and scaled to achieve a specific task (Pearson & Gordon, 2013). Contraction of the flexor muscles occurs during the early swing. Extensor muscles will contract

during the later phases. It should be understood, the timing and intensity of contraction are muscle dependent.

Movement	Stage	Anatomy
Flexion (F)	Early Swing	1. Flexion of hip, knee, and ankle
First extension (E_1)	Late Swing	1. Halfway through (F), the knee and ankle (plantarflexion) begin to extend while hip continues to flex. 2. Extension at the knee and ankle plantarflexion prepares extremity to accept weight at foot-contact.
Second extension (E_2)	Early Stance	1. The knee and ankle joints flex to produce co-activation of flexors-extensors. 2. An eccentric contraction of the plantar flexors and quadriceps occur due to weight acceptance. 3. A spring-like yield occurs at the eccentrically contracted muscles. 4. This yield allows the body to move forward over this foot.
Third extension (E_3)	Late Stance	1. The hip, knee, and ankle extend to provide a secondary propulsive force forward

Table 1: Stages of human gait (Pearson & Gordon, 2013).

Stroke Locomotion

Locomotion after stroke is slower with longer stance phase durations on both sides due to diminished strength and limited power (Olney & Richards, 1996). A failure to reach adequate speeds, in turn, results in the diminished energy-conserving exchanges between potential and kinetic energy of the upper body (Olney, Monga & Costigan, 1986). An increase in double support time improves postural control but is detrimental for energy conservation. The period of double support combines both a forward push and contralateral weight acceptance, which, over time is mechanically inefficient (Olney & Richards, 1996). A higher energy cost per unit traveled is the result (Olney & Richards, 1996). Early foot contact by the unaffected side is demonstrated as reduced hip flexor moment on the affected side struggles with reversing hip extension. An inability to generate sufficient push from the affected side reduces the swing phase of the

unaffected side (Olney and Richards, 1996). The affected side will have diminished knee flexion in the swing as the stroke survivor has the desire to keep the foot close to the ground. Furthermore, fear of lateral instability reinforces the need for double support (Olney and Richards, 1996). There is limited dorsiflexion at initial contact and during stance after stroke (Olney & Richards, 1996). Limited dorsiflexion stems from diminished strength and inadequate voluntary activation of the dorsiflexors. A lack of recruitment acuity in the shank may result in co-activation of the plantarflexors. Coupled with increased stiffness of the ankle plantarflexors (Dietz & Berger, 1984), ankle dorsiflexion is inadequate to clear the floor in swing (Olney & Richards, 1996).

The affected side knee may experience excessive knee flexion or hyperextension during stance. The person may seek stability and demonstrate hyperextension (compared to an able-bodied person) or excessively flex the knee because of reduced moment generation of the knee extensors, ankle plantarflexors and the hip extensors (Olney & Richards, 1996). Continued knee hyperextension into late stance prevents an effective push (Olney & Richards, 1996). In this case, failure to flex the knee causes the limb to stay extended through swing. To prevent dragging of the affected foot, the hip may hike or circumduct to clear the floor.

There is also evidence to suggest inappropriately timed and graded contraction on the affected side (Olney & Richards, 1996). A forward postural lean is coupled with continued activation of the hamstrings in the stance phase of the affected side (Olney & Richards, 1996). The hip and knee seem to compensate (i.e., extended activity) for diminished plantarflexion. Keep in mind; ankle plantarflexion is higher on the unaffected side. Overall, there is an excessive energy cost per unit walked (Olney & Richards, 1996).

Central Pattern Generators

The spinal cord is capable of producing rhythmic output from the motor neurons that are present even without input from higher centers and without feedback from the limbs (Schmidt et al., 2018). Muscle activity is accomplished through the work of interneurons that alternatively stimulate the flexor and extensor motor neurons in a pattern that resembles locomotion (Schmidt et al., 2018). According to the work of Graham Brown, activity alternates between circuits called half-centers (Brown, 1911). Half-center organization of the flexor and extensor interneurons likely mediates rhythmic stepping at the spinal level (Brown, 1911). Interneurons in this pathway will mediate long-latency reflexes from high threshold cutaneous muscle afferents. Ipsilateral and contralateral high threshold cutaneous muscle afferents mutually inhibit each other (See Scheme 1). Pearson &. Gordon gave the following example (2013):

For example, if two half-centers receive excitatory input, and the flexor half center receives the stronger input, the flexor muscle will contract while the extensor half center is inhibited. Then, as inhibitory output fatigues, the extensor half center's output will increase, causing inhibition of the flexor half center and contraction of the extensor muscles until inhibitory output fatigues.

Thus, the flexor and extensor muscles controlled by two half centers will alternatively contract and relax as long as the half centers receive tonic excitatory output. Graham Brown's theory is consistent with a system of interneurons generating flexor bursts that inhibit the system of interneurons generating the extensor burst, and vice versa (Brown, 1914; Brown, 1911). The interneurons mediating these burst patterns from flexor reflex afferents have not been fully identified, but interneurons housed at the intermediate region of the sixth lumbar segment's gray region is implicated (Pearson & Gordon, 2013).

The network capable of generating a rhythmic pattern of motor activity without phasic and peripheral input is a central pattern generator (CPG). CPGs have been analyzed and identified in 50+ motor systems that produce rhythmic behaviors such as walking, swimming, feeding, respiration and flying (Pearson & Gordon, 2013). Experimental induced CPGs, as compared to the naturally occurring phenomenon may differ. In nature, the shapes of these CPGs are, perhaps modulated by a sensorial input. The CPG's motor activity will depend on three factors (Table 2).

Cellular properties	Synaptic properties	Patterns of connections
Threshold	Sign	Reciprocal inhibition
Frequency-current relationship	Strength	Recurrent inhibition
Spike frequency adaptation	Time course	Parallel excitation and inhibition
Post-burst hyperpolarization	Transmission (electrical, chemical)	Mutual excitation
Delayed excitation	Release mechanisms (spike, graded signal)	
Post-inhibitory periods	Multi-component postsynaptic potentials	
Bursting (endogenous, conditional)	Facilitation/ depression (short term, long term).	

Table 2: Rhythmic motor activity generated by CPGs depends on three factors: (1): Cellular properties, (2): Synaptic properties between neurons and (3): Patterns of connections between neurons. Adopted from Pearson & Gordon (2013).

A simple network can generate rhythmic activity if a neurons firing rate can be inhibited or promoted per a timing pattern. For example, there is a brief increase in excitability of a neuron after an inhibitory tone has ended (i.e., post-inhibitory rebound). Two neurons that mutually inhibit each other (Scheme 1) can oscillate in an alternating fashion (i.e., each neuron has post-inhibitory rebound). Other time-dependent processes include synaptic depression, delayed excitation, and differences in time course of synaptic actions connecting two neurons (Pearson & Gordon, 2013).

The sequencing of motor neuron activity is regulated by diverse mechanisms (e.g., mutual inhibition, the rate of recovery from inhibition, mutual excitation) (See: Scheme 3). Mutual inhibition occurs when neurons firing in opposite phases are typically reciprocally coupled by inhibitory connections. Neurons may differ in the rate of inhibitory recovery. This rate will influence a different temporal onset of activity in two neurons that have been released from inhibition. Mutual excitation establishes synchronous firing in neuronal groupings. When a rapid, high-intensive burst of neurons is required, a mutual excitation can instigate the process (Pearson & Gordon, 2013).

Mammals constantly adjust to terrain and external conditions. These adjustments result in a motor pattern specific to the needs of the acute scenario. Input from the visual, vestibular and somatosensory systems may give precision to the foundational CPG. Proprioceptive input (i.e., via the Golgi tendon apparatus, muscle spindle, joint receptors) regulates the timing and amplitude of stepping.

This regulation is best shown in animal preparation (e.g., spinal and de-cerebrate cats) where intact proprioceptive input allows the animal to match the speed of a motorized treadmill. As speed has increased, the stepping rate increased via a reduction in time spent in stance phase (Pearson & Gordon, 2013). Proprioception regulates the timing and amplitude of stepping (Pearson & Gordon, 2013).

Sensory input, in part, regulates the length of stance and initiation of swing. During entrainment, a burst of activity in hip flexor motor neurons is initiated in synchrony with hip extension (Kriellaars, Brownstone, Noga, & Jordan, 1994). The afferent input that codes the correct hip angle at which swing initiation will arise is from the hip flexor's spindle (Pearson & Gordon, 2013). The stretching of the hip flexor inhibits the extensor's half center and will

facilitate burst activity in flexor motor neurons during gait (Hiebert, Whelan, Prochazka, & Pearson, 1996).
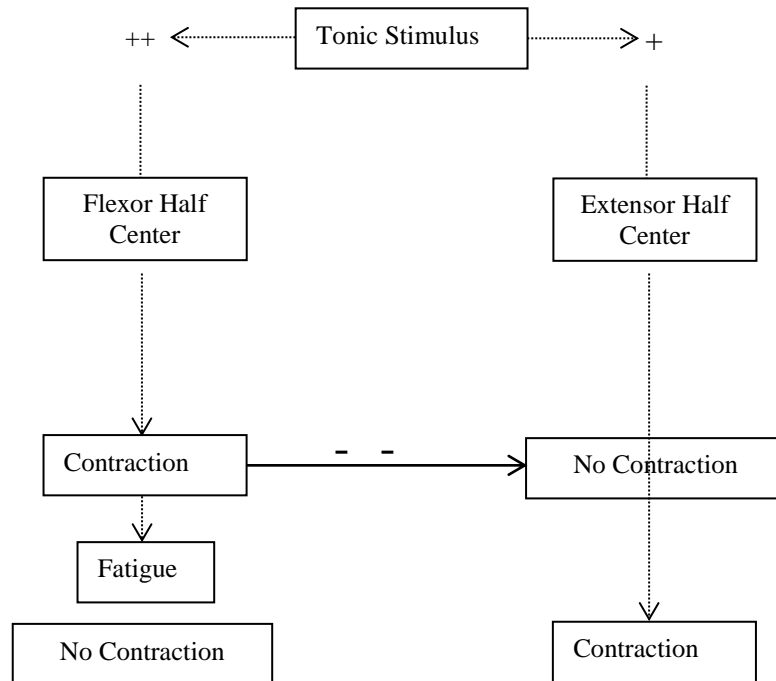
Scheme 1: CPG Networking: With tonic-excitatory input, inhibitory fatigue allows alternative contraction of flexor-extensors half centers. (++) stimulus strength > (+) stimulus strength; Scheme inspired by Pearson & Gordo (2013).
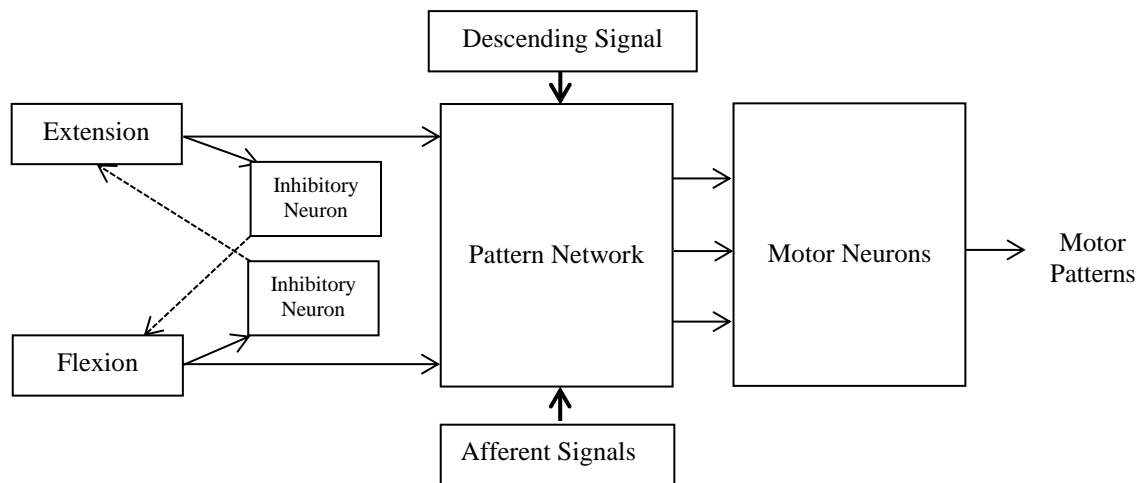
Scheme 2: Locomotor Pattern Generator: The primary rhythmic activity is produced by mutually inhibiting flexor and extensor half centers. The interneurons of these half centers drive motor neurons through an intermediate patterning network. This network controls the timing of activation of motor neurons across classes. Scheme inspired by Pearson & Gordo (2013).

Unloading of the extensor muscle occurs typically near end of stance. Extensor muscles must be unloaded to reduce GTO activity. Stimulation of the extensor's GTO and muscle spindle has prolonged stance phase as the GTO has an excitatory action on the ankle dorsiflexors during gait (Whelan, Hiebert, & Pearson, 1995). Other limbs accept the weight, and the extensor muscles are shortened which compromises the ability to produce high levels of force. Three excitatory pathways transmit sensory information from extensor muscles to extensor motor neurons:

1. Primary muscle spindles (group Ia afferent); mono-synaptic
2. Primary muscle spindles (group Ia afferent) and GTOs (group Ib afferent); disynaptic
3. Primary muscle spindles (group Ia afferent) and GTOs (group Ib afferent) + interneurons in the CPG; polysynaptic.

Afferent pathway from extensor muscle: Two mutually inhibiting groups of extensor and flexor interneurons constitute a CPG. Feedback from extensor muscles increase the activity in extensor motor neurons during stance and maintains activity while the extensor muscles are loaded (Pearson & Gordon, 2013). Ongoing and continuous regulation of extensor activity is completed through proprioceptive feedback. Feedback allows automatic adjustment of the force and length in extensor muscles in response to changing conditions (Pearson & Gordon, 2013). Additionally, cutaneous (i.e., exteroreceptors) receptors adjust stepping to external stimuli. Sensory input from the skin allows stepping to adjust to unexpected obstacles. This adjustment, however, is phase dependent. The same stimulus excites one group of motor neurons during one phase of locomotion may activate the antagonist motor neurons during another phase (Pearson & Gordon, 2013).

The CPG is thought to be activated or deactivated by supraspinal centers. In some cases, only a single pulse is required to initiate the CPG with no further higher level activity necessary for the oscillator to continue to operate (Schmidt et al., 2018). In other cases, a continuous input

but not necessarily rhythmic may be necessary (Schmidt et al., 2018). The activity may also be turned on by sensory input. Therefore, they can be turned on by a variety of stimulation sources, and they can continue until they are "run down" or are stopped by some other source of input (Schmidt et al., 2018). Although the prewired CPG evokes stereotyped action, modification of the basic pattern is possible in "higher" species such as cats (Schmidt et al., 2018). Examples of modification include speed and force of pattern. Additionally, lower feedback sources may serve to alter the particular pattern. Lastly, these pattern generators do not require conscious awareness to operate. Once initiated, they may continue without the involvement of the higher centers shall the environment not require high levels of attention (Schmidt et al., 2018).

Descending Signals

Stepping's basic motor pattern may be generated in the spinal cord. Fine control and modulation of stepping involve higher brain regions such as the motor cortex, cerebellum, and brainstem. Neurons in these regions are also rhythmically active during locomotion. Each region, however, plays a differing role in the regulation of normal locomotive function (Pearson & Gordon, 2013).

Visual information is relayed to motor cortex which enables guidance to movement. The visual cortex projects to the motor cortex. This pathway can also modify stepping movements according to visual input. Many neurons of the cortex project directly to the spinal cord and thus regulate the CPG's interneurons for locomotion. This projection helps the motor cortex adapt the timing and magnitude of motor activity to a specific task (Pearson & Gordon, 2013).

The cerebellum receives signals from both peripheral receptors (via the dorsal tracts) and spinal CPGs and adjusts locomotion via the brainstem's nuclei. The cerebellum modulates the

motor system. The cerebellum alters motor commands issued by the motor hierarchy to improve efficiency by three primary functions in motor control:

1. Comparison: The cerebellum compares intended movements to actual movements and corrects continuous movement in real time to minimize error.
2. Procedural Memory: The cerebellum plays a critical role in motor learning.
3. Integration: The cerebellum integrates information from entire motor hierarchy and proceeds to coordinate all aspects (from the spinal cord, brain stem, and cerebral cortex) leading to smooth and coordinated movement.

Most human CPG evidence comes from investigating human development. If an infant is held upright and moved over a horizontal surface; the baby can inadvertently mimic a stepping pattern. This mimicry suggests that basic neuronal circuits – characteristic of our species are in fact present at birth. Stepping has also been documented in infants with anencephaly (i.e., infants lacking cerebral and skull structure). Therefore, it is suggested that CPG circuits are located at or below the brain stem (Pearson & Gordon, 2013).

As automatic stepping turns to a functional walk, it is thought that supraspinal centers have begun regulation of the lower hierarchy. This voluntary control may be a result of the maturation of the reticulospinal pathway and regions of the brain stem (Pearson & Gordon, 2013). It is also plausible that descending brain systems have matured and modulation of this matured system has begun.

Currently, serotonin and norepinephrine are thought to be modulators of the human locomotor system. These modulators regulate the magnitude and timing of motor neuron activity in the spinal cord (Pearson et Gordon, 2013). NMDA-type receptors in the spinal cord are thought to initiate locomotor activity (Pearson & Gordon, 2013). Current evidence suggests that the signal to activate locomotion and later to control speed is transmitted to the spinal cord by glutamatergic neurons in the ventral reticulospinal pathway.

Evidence suggests that human walking relies on the same general principles of neuron organizations as quadrupedal walking (Pearson & Gordon, 2013). Intrinsic oscillatory networks are activated and modulated by higher brain centers and afferent input. However, a bipedal movement may place a higher demand on supraspinal centers. This demand may, in part, explain why human locomotion occurs later in life as compared to other species (Pearson & Gordon, 2013).

Motor Memory Consolidation

Shadmehr et. Holcomb (1997) demonstrated a structural shift in how the human brain consolidates motor memory. Using positron emission tomography (PET), the authors monitored regional cerebral blood flow, an indirect marker of neural activity. The investigation demonstrated that consolidation occurs through a shift from prefrontal regions of the cortex to the premotor, posterior parietal and cerebellar cortex structures. This shift was specific to the recalled and learned motor skill (rapid movements in a particular design against a robot induced resistance). With the passage of time, the devolvement of the prefrontal cortex suggests a change in the neural representation of the task's internal model. This change in neural representation may underlie the increased stability found in long-term memory.

Exercise and Neural Plasticity

Trained muscles generate a given amount of submaximal force with less EMG activity; suggesting a more efficient motor unit recruitment with practice (Kenney, Willmore & Costill, 2015). The benefits of physical activity on cognitive function have been previously linked. Physical activity can impact a wide variety of cognitive and learning processes including executive control, attention processing, and spatial memory. Exercise elicits structural plasticity in a wide variety of brain regions related to cognitive function. Neural plasticity is the change in

neural structure and function in response to experience based stimuli including hippocampal angiogenesis, changes in dendritic density/volume and neurogenesis (Kandel at al., 2015).

Long-term potentiation (LTP) and long-term depression (LTD) in the hippocampus are implicated in the generation of long-lasting changes in synaptic function (i.e., synaptic plasticity) (Pearson & Gordon, 2013). The bidirectional control of synaptic strength by LTP and LTD is believed to be essential for experience-dependent plasticity. LTP is NMDA receptor and experience dependent. LTP may work through transcription (receptor trafficking) or receptor function (phosphorylation). LTP is modifiable and changing often. LTP is thought to play a role in how motor patterns are learned and consolidated.

It seems that growth factors are implicated in mediating structural and cognitive plasticity post exercise. Growth factors include; Insulin-Like Growth Factor – 1 (IGF-1), Vascular Endothelial Growth Factor (VEG-F) and Brain-Derived Neurotrophic Factor (BDNF). These growth factors can influence the brain directly, and they have been shown to be important in neurogenesis and the antidepressant effects of exercise in animal models. Exercise can also alter the synthesis of these growth factors in the areas of the hippocampus, cortex, and amygdala. Blocking the function of BDNF has been reported to prevent the enhancement of cognitive function produced by exercise in rats (Kandell et al., 2015). BDNF could also contribute to the increase in synaptic plasticity and neurogenesis following exercise. In addition to enhancing cognitive function and learning and memory processes, physical activity is also well known to confer protection against deleterious effects of stress, a "stress-buffering effect."

One issue that arises when investigating the effects of exercise on brain and behavior is the ability to differentiate exercise effects from those of environmental enrichment. Recent work seems to suggest that long-term exercise by rodents have effects that are above and beyond those

demonstrated by enriched environments. Therefore, at least some of the benefits of exercise are independent of environmental enrichment.

Exercise's improvement of cognition (i.e., attention processing, executive function) and hippocampal-dependent memory have primarily been demonstrated primarily in aerobic exercise and yoga. Exercise has also demonstrated stress resilience effect reducing occurrences of anxiety and depression. The effects of exercise may have a top-down effect (i.e., brain to muscle) or a "bottom-up" effect (muscle to the brain). Areas of neural circuitry activated during exercise may provide signaling to induce change (i.e., motor systems, reward areas). Additionally, the periphery may signal the CNS via myokines or gut microbial resulting in brain adaptability and plasticity. Both these systems are thought to work through norepinephrine and serotonin pathways.

Recommendation for Exercise Programming with Stroke Survivors

Exercise training programs can improve mobility and independence post CVA. The ability to exercise will depend on the severity of neurological involvement and existing co-morbidities (Palmer-McLean & Harbst, 2003). Muscle weakness, limited range of motion and impaired sensation may preclude independent ambulation and or ability to exercise in the standing position. Lack of adequate balance may interfere with seated arm or leg ergometry. Muscular weakness and limited range of motion may also interfere with a person's ability to maintain crank rates (Palmer-McLean & Harbst, 2003). Aphasia, apraxia, and mental confusion may interfere with the ability to comprehend directions during exercise. The exercise professional should consider the client's motor abilities when selecting an appropriate exercise device. Common exercise modes are discussed in the following sections:

Treadmill.

Treadmill use may be appropriate for individuals with minimal motor impairment, who have stable standing balance and can ambulate independently without an assistive device (Palmer-McLean & Harbst, 2003). Previous trials suggest that task-specific training regimens increase affected leg movement (Macko et al., 2005; Smith, Silver, Golberg & Macko, 1999). Furthermore, the task-specific nature of this therapy increases plasticity in the spinal cord and improves functional mobility (Stoloff, Zehr & Ferris, 2007). Treadmill exercise should avoid abrupt changes in speed to reduce fall risk (Palmer-McLean & Harbst, 2003). Individuals with a sensorimotor impairment that result in weakness, loss of movement, or balance deficits may be unsafe on the treadmill (Palmer-McLean & Harbst, 2003). To improve safety and, in cases of severe weakness, bodyweight harnesses may be utilized to prevent a fall in the event of a misstep or loss of balance (Palmer-McLean & Harbst, 2003). Preferred walking speeds will be much slower, and energy expenditure at a specific work rate will be 55-64% greater in individuals with a CVA (Palmer-McLean & Harbst, 2003).

Ergometry.

Standard leg cycle ergometry may be utilized if the individual can safely maintain sitting balance (Palmer-McLean & Harbst, 2003). The affected extremities may require strapping to maintain machine contact if the individual cannot keep it secure independently. Exercise guidelines should be individualized; however, general testing guidelines have been suggested (i.e., 50 revolutions per minute with an output of 20 watts, with 20-watt increments per stage) (Palmer-McLean & Harbst, 2003).

If spasticity or muscle weakness in the affected extremity interferes with the ability to maintain pedal cadence, individuals could only use the unaffected side. However, it may be

difficult to achieve a work rate that can stress the heart. Therefore, combination ergometers (i.e., arms plus legs) are particularly useful. That is if spasticity or weakness of the affected side does not interfere with global, whole-body cadence (Palmer-McLean & Harbst, 2003). A hand/foot strap or mitt may be used to secure the hand of an individual whose extremity control is compromised (Palmer-McLean & Harbst, 2003). Situations that require the use of straps should be closely supervised (Palmer-McLean & Harbst, 2003).

NuStep Cross Trainer.

Exercise interventions that approximate the stepping motion could be useful for the neurological rehabilitation of gait (Stoloff, Zehr & Ferris, 2007). Bilateral, recumbent training devices offer a promising alternative to treadmill based approaches (Page, Levine, Teepen & Hartman, 2008). The NuStep combines both arm and leg exercise in a seated position (Palmer-McLean & Harbst, 2003). This device includes a seat with back support with the option of a seatbelt that produces additional trunk stability for the client with poor seated balance (Palmer-McLean & Harbst, 2003). For the client with significant mobility impairment, the seat can swivel to accommodate a transfer. Additionally, the armrests can hinge upward further facilitating transfer onto the device (Palmer-McLean & Harbst, 2003). Arm handles require a neutral-like position that is easier for clients with a limited range of motion. It is thought that this neutral-like position encourages a more upright trunk position (Palmer-McLean & Harbst, 2003). Finally, as opposed to tradition bike pedals, the NuStep's footplate contains raised lateral and posterior borders to maintain foot contact (Palmer-McLean & Harbst, 2003). Foot straps are optional. Older adults seem to prefer the recumbent position (Looney & Rimmer, 2003).

The NuStep Cross-trainer simulates the reciprocal motion of walking but in a seated and controlled manner. Differences between the NuStep and walking on joint kinematics (e.g.,

reduced the range of motion and shank temporal differences in muscle electromyography) have been documented (Stoloff, Zehr & Ferris, 2007). This research on the NuStep Cross Trainer has demonstrated that the quadriceps group (Vastus Medialis, Vastus Lateralis, and Rectus Femoris), medial hamstring, Soleus, and Gastrocnemius are primarily driving the pedals down phase (Huang & Ferris, 2004; Stoloff, Zehr & Ferris, 2007) (See Figure 2). Whereas, the anterior tibialis couples with the medial hamstring and gastrocnemius to drive the pedals' up phase (See Figure 3).

Movements of the handles are coupled to that of the foot pedals, so that extension of the right leg is associated with retraction of the left handle (Huang and Ferris, 2004). This mechanical coupling allows the arms to assist leg motion and vice versa (Zehr et al., 2007). Previous research on rhythmicity indicated that upper extremity activation and the consequential afferent feedback might improve lower limb muscle recruitment. As a result, researchers have demonstrated facilitation of leg muscles by simultaneous arm movements (Huang & Ferris, 2004; Billinger, Loudon & Gajewsk, 2008; Huang & Ferris, 2009). However, when legs are maximally activated, the combination of arm and leg movements did not provide additional facilitation to the already activated leg muscles (Huang & Ferris, 2004, Huang & Ferris, 2009). Ipsilateral coupling was also demonstrated when upper limb muscle activation increased muscle activation more in the same side lower limb as compared to the contralateral side. Reflex studies suggest that contralateral upper to lower limb coupling may be more prevalent during rhythmic movement compared to ipsilateral upper to lower limb coupling. Therefore, these data suggested that the supraspinal drive may be more critical compared to spinal mechanisms (i.e., contralateral reflexes) during maximal effort on the NuStep (Huang & Ferris, 2009a). In a subsequent investigation, arm movement also facilitated lower extremity electromyography (EMG) in

submaximal recumbent stepping (de Kam et al., 2013). When arm and leg movements were mechanically decoupled, maximal arm movement still facilitated muscle activity in passively moved legs (Billinger, Loudon & Gajewski, 2008).

Assumptions and Limitations

The subsequent investigation assumed that individuals studied put forth adequate effort. Geographic area was the City of Detroit and surrounding areas, which may limit conclusions to urban settings. The findings of this investigation are limited to the laboratory setting. Studied sample may not be representative of the larger population.

CHAPTER 3 METHODS MUSCLE ACTIVATION PILOT 1

Participants

Healthy males and females (n = 23) aged 23.52 ± 4.23 years were recruited to participate in the study. Participants had no neurologic conditions or acute orthopedic surgeries that impaired their ability to step. Furthermore, any cardiopulmonary diagnoses that reduced exercise capacity were excluded from this investigation. Participants had no known skin allergies to topical agents or adhesives. Participants signed an informed consent before testing. The investigation was approved by Wayne State University's institutional review board (Appendix A).

Measures

An instrumented version of the commercially available T5 NuStep Recumbent cross trainer (NuStep Inc., Ann Arbor, MI, USA) was utilized. The instrumented T5 NuStep Recumbent Cross Trainer can measure cadence of the participant (electronic step cadence meter and counter) while providing real-time visual feedback on pace against 15 levels of resistance (15 being the most challenging; at 1.0 increments of resistance). Both distal and proximal foot straps were utilized. The participant confirmed symmetrical tightness between each foot before all exercise bouts. Participants performed recumbent stepping without upper extremity assistance. The seat position was set so that the participant's right knee was near full extension (~15-20° of knee flexion when full knee extension = 0°) at the step's terminal range of motion. A goniometer was used to measure both the knee at full knee extension (i.e., pedal down) and right knee flexion (pedal up position). As the participants remained seated, the center of a handheld goniometer was placed over the lateral epicondyle of the femur. The proximal arm was placed at the lateral midline of the femur with reference to the greater trochanter whereas the distal arm

was aligned the lateral midline of the fibula with reference to the lateral malleolus. The degree of knee motion was quantified by both the participant's seat position and the degree of knee extension. Participants were instructed to keep their pelvis stable to reduce ipsilateral rotation and posterior tilting while stepping. Lastly, participants were instructed to step at a range of motion – that was as great as possible without using the pedal's end range bumper to propel the subsequent step and for which the seat position allowed one to remain seated.

Each electrode location was prepared by cleaning with rubbing alcohol and abrasive paper (Electrode Skin Prep Pads, Dynarex Corporation, Orangeburg, NY, USA). The electrodes (pre-gelled Ag/AgCl Noraxon Single Electrode, Noraxon USA Inc., AZ, USA) were placed over the muscle belly along the long axis and secured with paper tape based upon the Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM's) recommendations (Hermens et al., 1999). The alcohol was allowed to vaporize so that the skin was dry before electrode placement. The reference electrode was placed at a location in which the risk for disturbance signal was minimized. After the electrodes were placed, the electrode's (including the reference electrode) signal was assessed for contamination of movement artifacts and background noise. The electrode's final location was determined based on both initial palpation and assessment of signal quality.

We recorded muscle activity from 12 muscles (6 per lower extremity) using a surface electromyography system (16 channel wireless) with an EMG bandwidth of 5-500 Hz (Noraxon Inc. Scottsdale, AZ, USA). The Noraxon EMG system was synced with the instrumented NuStep cross trainer using a customized program written in Labview (National Instruments, Austin, TX, USA). This program collected EMG data in alternating 10-second epochs for the 3 minutes (minutes 2-4) of the 5-minute exercise protocol. EMG was processed with a second

order high pass filter (cut off frequency 80-250 Hz) with zero phase lag to attenuate low-frequency components such as mechanical artifact. EMG data were full wave rectified, smoothed at 300ms and normalized to the participant's maximum voluntary contraction (MVC). Mean EMG amplitude (mEMG) and peak EMG amplitude (pEMG) data were converted to a percentage of MVC. mEMG and pEMG of the rectus femoris (RF), vastus medialis oblique (VMO), semitendinosus (ST), tibialis anterior (TA), medial gastrocnemius (MG) and soleus (SOL) were recorded bilaterally.

Procedures

Before subsequent measurement blocks and to better acclimate to the task, participants performed two practice 5-second MVCs. The subsequent measured MVC blocks required 3 sets of 5 seconds each. Knee extension (i.e., VMO and RF MVC) was performed seated and at 60º and 15º of knee extension (0º = full knee extension). Knee flexion (i.e., ST MVC) was also performed seated and at 60º (0º = full extension) of knee flexion. SOL and TA MVCs were performed supine with hip and knee flexion of 90°. All MVCs were performed on the Humac Norm Machine (Computer Sports Medicine, Stoughton, MA, USA) except for bilateral plantarflexion (i.e., MG MVC). Bilateral MG MVC was performed standing and through active plantarflexion while full knee extension was maintained. Peak force and pEMG amplitude (uV) were recorded.

Initial cadence was first subjectively chosen by the participant in response to the statement "step at a pace in which you're comfortable." After each progressive minute, participants were asked to report their rating of perceived exertion (RPE) (Appendix C). If a participant reported below 12 or higher than 16 on the RPE scale during any one of the interval checks, the participant was instructed to speed up – or slow down accordingly. The perceived

exertion based SS cadence was then estimated as an average over the 10 min SS protocol. During this 10 min SS protocol, a clipboard covered the digital output so that no visual feedback on step rate was provided during the determination of SS cadence. The instrumented T5 NuStep cross trainer calculated SS cadence. This average (i.e., steps/min) was later rounded to meet the 5 step/min intervals of the system's pace partner. This pace partner would later establish cadence for the participant during each of the exercise protocols.

Participants performed all 5 min exercise protocols in a randomized order. Each 5 min protocol consisted of 1-min warm up, 3 mins of recorded exercise (at every other 10-second interval) and 1 min of cool down at the protocol's specified resistance level and cadence. NuStep's pace partner provided 100% visual feedback during each of the five protocols; SS cadence with level 1 resistance (SSL1), SS cadence with level 8 resistance (SSL8), +20% SS cadence (SS+20), -20% SS cadence (SS-20), and 80 steps per minute at resistance level 1 (80L1) (Figure 1a).

Figure 1a: NuStep Cross Trainer visual feedback during each exercise protocol.

Participants were instructed to keep their representative green circle (i.e., their current and real-time speed) inside the pace partner's white circle (i.e., pace partner) by stepping at their measured RPE based self-selected (SS) average steps per minute (aSPM). The pace partner progressed counterclockwise around the visual track displayed in front of the exercising participant.

Statistical Analysis

pEMG and mEMG were used to describe intramuscular signaling. pEMG and mEMG were evaluated by IBM SPSS Statistics 23. Twelve (i.e., 6 right, 6 left) one-way within subject ANOVAs were conducted to determine protocol effect on each muscle. All data were checked for one-way within-subject ANOVA assumptions including sphericity (Meyers, Gamst & Guarino, 2006, Vincent & Weir, 2012). Following a statistically different Mauchly's test of sphericity ($p < .05$), the sphericity corrected Greenhouse-Giesser F ratio was evaluated at $p < .05$. A pairwise t-test with a Bonferroni-corrected alpha was applied to the means of the 5 protocols post hoc, (i.e., exploratory $\alpha = 0.10/ 10$ comparisons $= 0.01$).

CHAPTER 4 RESULTS – MUSCULAR ACTIVATION – PILOT PART 1

Participant's mean height and weight were 1.68m ± 0.13m and 69.54kg ± 26.70kg, respectively (BMI = 24.32 ± 9.38). The most frequent seat position was NuStep setting #10 (n = 6, seat position ranged #5-13). Seat position resulted in a static mean right knee extension of 19.64 ± 6.25º and 78.82 ± 5.74º of right knee flexion at a terminal range of motion (full knee extension = 0º). Participant's RPE based SS cadence was 123.86 ± 18.12 steps per minute (spm). Mean cadence was calculated at 103.64spm ± 21.94spm for SS-20 and 142.73spm ± 25.25 spm for SS+20 respectively.

Participant's peak force did not differ between left and right extremities; $p > .05$. All muscle groups, regardless of the dependent variable (mEMG or pEMG) violated sphericity, $p < 0.05$. Therefore, F values were corrected by the Greenhouse Geisser adjustment. Protocol means ± standard deviation (Std) are listed in Table 3 (mEMG) and Table 4 (pEMG). Bonferroni-corrected t-test results are listed in Table 5 (mEMG) and Table 6 (pEMG).

Muscle	SSL8 %MVC mean ± Std	SS+20 %MVC mean ± Std	SS-20 %MVC mean ± Std	SSL1 %MVC mean ± Std	80L1 %MVC mean ± Std
LRF	13.61 ± 5.60	6.13 ± 4.14	2.49 ± 1.12	3.47 ± 1.31	2.33 ± 0.93
95% CI	11.48-15.87	4.64-7.95	2.05-2.97	2.97-4.00	1.97-2.70
RRF	20.71 ± 9.92	8.51 ± 4.63	3.41 ± 1.22	4.88 ± 2.79	4.35 ± 2.52
95% CI	16.64-25.01	6.64-10.54	2.93-3.95	3.74-6.17	3.41-5.45
LVMO	24.95 ± 10.54	12.70 ± 6.01	6.63 ± 2.42	8.28 ± 3.68	5.93 ± 2.27
95% CI	20.75-29.46	10.33-15.28	5.65-7.60	6.78-9.86	5.04-6.90
RVMO	22.89 ± 14.31	10.60 ± 6.48	5.48 ± 3.05	6.94 ± 4.07	4.36 ± 2.79
95% CI	17.35-28.81	7.81-13.39	4.25-6.83	5.20-8.80	3.27-5.66
LST	13.23 ± 8.90	7.83 ± 5.24	3.95 ± 2.92	4.91 ± 2.85	2.76 ± 1.65
95% CI	9.60-16.65	5.81-10.13	2.81-5.17	3.77-6.03	2.13-3.45
RST	9.03 ± 6.13	5.01 ± 3.71	2.87 ± 1.97	3.40 ± 2.10	1.73 ± 0.97
95% CI	6.80-11.59	3.61-6.58	2.05-3.75	2.63-4.31	1.36-2.12
LSOL	17.78 ± 12.46	7.82 ± 4.59	3.73 ± 2.99	6.35 ± 5.74	2.12 ± 1.36
95% CI	12.64-23.34	5.84-9.81	2.59-5.17	4.01-8.94	1.54-2.70
RSOL	19.68 ± 13.86	8.26 ± 4.34	4.45 ± 3.29	4.74 ± 3.22	3.43 ± 2.42
95% CI	14.13-26.03	6.48-10.14	3.08-5.98	3.45-6.10	2.45-4.57
LMG	18.97 ± 10.56	9.23 ± 5.98	3.07 ± 1.85	5.35 ± 4.06	2.73 ± 1.77
95% CI	14.88-23.16	6.89-11.86	2.38-3.81	3.90-7.09	2.06-3.47
RMG	20.44 ± 10.06	9.18 ± 5.47	4.54 ± 2.90	6.49 ± 4.63	4.03 ± 2.52
95% CI	16.57-24.57	7.08-11.47	3.52-5.82	4.83-8.58	3.08-5.08
LTA	12.41 ± 6.13	5.87 ± 4.03	2.82 ± 2.21	4.03 ± 2.95	2.26 ± 1.62
95% CI	10.03-14.89	4.28-7.55	1.98-3.74	2.86-5.26	1.64-3.00
RTA	15.76 ± 10.51	7.93 ± 5.54	3.75 ± 2.58	5.47 ± 3.97	3.47 ± 2.22
95% CI	11.54-20.28	5.78-10.40	2.71-4.91	3.97-7.12	2.63-4.37

Table 3: mEMG Mean ± Standard Deviation(Std) normalized to percentage of Maximum Voluntary Contraction (%MVC). 95% Confidence Interval (CI) is listed. Bonferroni-corrected alpha was applied to the means of the 5 protocols post hoc, (i.e. exploratory $\alpha = 0.10/10$ comparisons = 0.01).

Muscle	SSL8 %MVC mean ± Std	SS+20 %MVC mean ± Std	SS-20 %MVC mean ± Std	SSL1 %MVC mean ± Std	80L1 %MVC mean ± Std
LRF	20.44 ± 9.91	7.91 ± 5.18	3.51 ± 1.48	4.89 ± 1.78	3.38 ± 1.38
95% CI	16.26-25.13	5.77-10.26	2.88-4.15	4.11-5.63	2.76-3.93
RRF	30.22 ± 12.83	11.35 ± 5.65	5.56 ± 0.2.88	6.85 ± 3.92	6.68 ± 4.0
95% CI	24.86-35.59	8.94-14.03	5.12-8.70	5.27-8.73	5.12-8.27
LVMO	38.18 ± 15.96	18.74 ± 9.27	10.47 ± 4.24	12.45 ± 5.77	9.57 ± 3.78
95% CI	31.95-44.76	14.94-22.61	8.73-12.27	10.07-14.73	8.03-11.51
RVMO	30.61 ± 18.98	15.05 ± 9.56	8.13 ± 4.73	10.29 ± 6.33	6.28 ± 3.95
95% CI	22.43-38.92	11.16-19.31	6.21-10.22	7.74-13.05	4.69-8.13
LST	17.82 ± 10.68	11.72 ± 7.73	5.53 ± 4.01	7.49 ± 4.68	4.10 ± 2.56
95% CI	13.57-22.36	8.72-14.95	3.96-7.32	5.69-9.39	3.08-5.06
RST	12.80 ± 7.60	6.12 ± 4.26	4.02 ± 2.81	5.00 ± 3.45	2.42 ± 1.37
95% CI	9.68-15.99	4.50-8.09	2.87-5.27	3.62-6.49	1.87-3.00
LSOL	17.64 ± 7.30	11.47 ± 6.32	6.43 ± 5.41	9.61 ± 8.69	3.20 ± 1.76
95% CI	14.37-20.59	8.83-14.05	4.21-8.77	6.07-13.57	2.46-3.96
RSOL	28.49 ± 22.97	10.71 ± 5.10	6.33 ± 4.74	7.14 ± 5.04	4.22 ± 2.63
95% CI	19.50-38.43	8.61-12.92	4.32-8.52	5.09-9.33	3.11-5.33
LMG	24.28 ± 13.72	12.23 ± 8.17	4.07 ± 2.26	7.21 ± 5.41	4.40 ± 2.92
95% CI	18.61-30.17	9.04-16.66	3.18-5.12	5.11-9.62	3.28-5.67
RMG	26.69 ± 12.35	12.57 ± 7.16	6.32 ± 3.89	10.28 ± 7.84	5.86 ± 3.29
95% CI	21.67-32.11	9.60-15.76	4.81-7.96	7.24-13.69	4.50-7.25
LTA	17.04 ± 8.44	8.08 ± 5.47	3.94 ± 3.01	5.72 ± 4.17	3.43 ± 2.22
95% CI	13.30-20.93	6.00-10.51	2.76-5.30	4.05-7.48	2.56-4.38
RTA	19.97 ± 13.37	10.01 ± 6.78	5.14 ± 3.65	6.88 ± 4.11	4.80 ± 2.85
95% CI	14.53-25.87	7.34-13.00	3.74-6.62	5.21-8.66	3.63-5.99

Table 4: pEMG Mean ± Standard Deviation (Std) normalized to percentage of Maximum Voluntary Contraction (%MVC). 95% Confidence Interval (CI) is listed.

Muscle	Protocol mEMG Difference
LRF	5>1,2,3,4; 2>3,4; 2=1 ;1>4,3; 3=4 *(2 vs. 1, *p*=0.013);
RRF	5>1,2,3,4; 2>1,3,4; 1=3=4
LVMO	5>1,2,3,4; 2>1,2,3,4; 1=3=4 *(1 vs. 4, *p*=0.011, 1 vs. 3, *p*=0.035)
RVMO	5>1,2,3,4; 2>1,3,4; 1>3,4; 3=4
LST	5>1,2,3,4; 2>3,4; 2=1;); 1>4, 1=3 *(2 vs. 1, *p*=0.016)
RST	5>1,2,3,4; 2>4; 2=1=3; 1>4 *(2 vs. 1, *p*=0.022)
LMG	5>1,2,3,4; 2>1,3,4; 1=3=4
RMG	5>1,2,3,4; 2>1,3,4; 1>3;3=4
LSOL	5>1,2,3,4; 2>3,4; 2=1; 1=3=4 *(1 vs. 4, *p*=0.012; 1 vs. 3, *p*=0.049)
RSOL	5>1,2,3,4; 2>1,3,4; 1=3=4
LTA	5>1,2,3,4; 2>1,3,4; 1=3=4 *(1 vs. 4, *p*=0.017, 1 vs. 3, *p*=0.049)
RTA	5>1,2,3,4; 2>3,4: 2=1 *(1 vs. 2, *p*=0.024); 1=3=4

Table 5: Bonferroni Corrected Pairwise Comparison for mEMG across protocols: SSL8 (5), 80L1 (4), SS-20% (3), SS+20% (2), SSL1 (1). Significant is set at $p \leq .01$ (α=0.10/10 comparisons p ≤ 0.01). *****Comparisons with 0.05> p>0.01 are noted in parenthesis.

Muscle	Protocol pEMG Difference
LRF	5>1,2,3,4; 2>3,4; 2=1; 1>3,4; 3=4
	*(2 vs. 1, p=0.031)
RRF	5>1,2,3,4; 2>1,3,4; 1=3=4
LVMO	5>1,2,3,4; 2>1,3,4; 1=3=4
RVMO	5>1,2,3,4; 2>1,3,4; 1>4; 1=3; 3=4
	*(1 vs, 3, p=0.015)
LST	5>1,3,4; 5=2 *(5 vs. 2, p=.022); 2>1,3,4; 1>4: 1=3; 3=4
	*(1 vs, 3, p=0.032)
RST	5>1,2,3,4; 2>4; 2=1=3; 1>4; 3=4
	*(3 vs. 4, p=0.04)
LMG	5>1,2,3,4; 2>1,3,4; 1=3=4
RMG	5>1,2,3,4; 2>3,4; 2=1; 1=3=4
LSOL	5>2,3,4; 5=1;2> 3,4; 2=1; 1=3=4
	*(1 vs. 4, p=0.02; 5 vs. 1, p=0.013);
RSOL	5>1,3,4; 5=2 2>3,4; 2 =1; 1=3=4
	*(1 vs 4, p=0.037; 5 vs. 2, p=0.14; 2 vs. 1, p=0.031)
LTA	5> 1,2,3,4; 2> 1,3,4; 1=3=4
	*(1 vs. 3, p=.025; 1 vs. 4, p=0.036)
RTA	5>1,2,3,4; 2>3; 2=4=1
	*(2 vs. 4, p=0.011; 2 vs. 1, p=0.021; 2 vs. 4, p= 0.011); 3=4=1

Table 6. Bonferroni Corrected Pairwise Comparison for pEMG across protocols: SSL8 (5), 80L1 (4), SS-20% (3), SS+20% (2), SSL1 (1). Significant is set at p<0.01 (α=0.10/10 comparisons p ≤ 0.01). *****Comparisons with 0.05>p>0.01 are noted in parenthesis.

CHAPTER 5 METHODS – MUSCLE ADAPTATION –PILOT PART 2

Participants

Healthy males and females (n = 23) aged 23.52 ± 4.23 years were recruited to participate in the study. Participants had no neurologic conditions or acute orthopedic surgeries that impaired their ability to step. Furthermore, any cardiopulmonary diagnoses that reduced exercise capacity were excluded from this investigation. Participants had no known skin allergies to topical agents or adhesives. Participants signed an informed consent before testing. The investigation was approved by Wayne State University's institutional review board (Appendix A).

Measures

An instrumented version of the commercially available T5 NuStep Recumbent cross trainer (NuStep Inc., Ann Arbor, MI, USA) was utilized. The instrumented T5 NuStep Recumbent Cross Trainer can measure cadence of the participant (electronic step cadence meter and counter) while providing real-time visual feedback on pace against 15 levels of resistance (15 being the most challenging; at 1.0 increments of resistance). Both distal and proximal foot straps were utilized. The participant confirmed symmetrical tightness between each foot before all exercise bouts. Participants performed recumbent stepping without upper extremity assistance. The seat position was set so that the participant's right knee was near full extension (~15-20° of knee flexion when full knee extension = 0°) at the step's terminal range of motion. A goniometer measured full knee extension and right knee flexion. As the participants remained seated, the center of a handheld goniometer was placed over the lateral epicondyle of the femur. The proximal arm was placed at the lateral midline of the femur with reference to the greater trochanter whereas the distal arm was aligned the lateral midline of the fibula with reference to

the lateral malleolus. The degree of knee motion was quantified by both the participant's seat position and the degree of knee extension. Participants were instructed to keep their pelvis stable to reduce ipsilateral rotation and posterior tilting while stepping. Lastly, participants were instructed to step at a range of motion – that was as great as possible without using the pedal's end range bumper to propel the subsequent step and for which the seat position allowed one to remain seated.

Each electrode location was prepared by cleaning with rubbing alcohol and abrasive paper (Electrode Skin Prep Pads, Dynarex Corporation, Orangeburg, NY, USA). The electrodes (pre-gelled Ag/AgCl Noraxon Single Electrode, Noraxon USA Inc., AZ, USA) were placed over the muscle belly along the long axis and secured with paper tape based upon the Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM's) recommendations (Hermens et al., 1999). The alcohol was allowed to vaporize so that the skin was dry before electrode placement. The reference electrode was placed at a location in which the risk for disturbance signal was minimized. After the electrodes were placed, the electrode's (including the reference electrode) signal was assessed for contamination of movement artifacts and background noise. The electrode's final location was determined based on both initial palpation and assessment of signal quality.

We recorded muscle activity from 12 muscles (6 per lower extremity) using a surface electromyography system (16 channel wireless) with an EMG bandwidth of 5-500 Hz (Noraxon Inc. Scottsdale, AZ, USA). The Noraxon EMG system was synced with the instrumented NuStep cross trainer using a customized program written in Labview (National Instruments, Austin, TX, USA). This program collected EMG data in alternating 10-second epochs for the 3 minutes (minutes 2-4) of the 5-minute exercise protocol. EMG was processed with a second

order high pass filter (cut off frequency 80-250 Hz) with zero phase lag to attenuate low-frequency components such as mechanical artifact. EMG data were full wave rectified, smoothed at 300ms and normalized to the participant's maximum voluntary contraction (MVC). Mean EMG amplitude (mEMG) and peak EMG amplitude (pEMG) data were converted to a percentage of MVC. mEMG and pEMG of the rectus femoris (RF), vastus medialis oblique (VMO), semitendinosus (ST), tibialis anterior (TA), medial gastrocnemius (MG) and soleus (SOL) were recorded bilaterally.

Procedures

Before subsequent measurement blocks and to better acclimate to the task, participants performed two practice 5-second maximum voluntary contractions (MVC). The subsequent measured MVC blocks required 3 sets of 5 seconds each. Knee extension (i.e., VMO and RF MVC) was performed seated and at 60º and 15º of knee extension (0º = full knee extension). Knee flexion (i.e., ST MVC) was also performed seated and at 60º (0º = full extension) of knee flexion. SOL and TA MVCs were performed supine with hip and knee flexion of 90°. All MVCs were performed on the Humac Norm Machine (Computer Sports Medicine, Stoughton, MA, USA) except for bilateral plantarflexion (i.e., MG MVC). Bilateral MG MVC was performed standing and through active plantarflexion while full knee extension was maintained. Peak force and pEMG amplitude (uV) were recorded.

Initial cadence was first subjectively chosen by the participant in response to the statement "step at a pace in which you're comfortable." After each progressive minute, participants were asked to report their RPE (Appendix C). If a participant reported below 12 or higher than 16 on the RPE scale during any one of the interval checks, the participant was instructed to speed up – or slow down accordingly. The perceived exertion based SS cadence

was then estimated as an average over the 10 min SS protocol. During this 10 min SS protocol, a clipboard covered the digital output so that no visual feedback on step rate was provided during the determination of SS cadence. The instrumented T5 NuStep cross trainer calculated SS cadence. This average (i.e., steps/min) was later rounded to meet the 5 step/min intervals of the system's pace partner. This pace partner would later establish cadence for the participant during each of the exercise protocols. Participants performed all 5 min exercise protocols in a randomized order. Each 5 min protocol consisted of 1-min warm up, 3 mins of recorded exercise (at every other 10-second interval) and 1 min of cool down at the protocol's specified resistance level and cadence. NuStep's pace partner provided 100% visual feedback during each of the five protocols; (SS) cadence with level 1 resistance (SSL1), SS cadence with level 8 resistance (SSL8), +20% SS cadence (SS+20), -20% SS cadence (SS-20), and 80 steps per minute at resistance level 1 (80L1) (Figure 1a).

Statistical Analysis

EMG data were evaluated for parametric assumptions using IBM SPSS statistics 23. Numerous EMG data violated normality, homogeneity of variance and sphericity. Both data sets also contained high levels of skewness and kurtosis. Therefore, the EMG data were assessed with non-parametric measures. EMG during minute 2 and EMG during minute 4 were compared within protocols. 1x4 Friedman tests were conducted to determine a statistically significant difference ($p < .05$) in mEMG and pEMG between minute 2 and minute 4. Following a statistically significant Friedman test, a Wilcoxon Signed Rank test (WSRT) was conducted post hoc. P-values were adjusted using the Bonferroni correction ($\alpha = .10/ 12$ WSRT as determined a priori, $p < .0083$).

CHAPTER 6 RESULTS – MUSCLE ADAPTATION –PILOT PART 2

Mean Electromyography (mEMG)

Rectus Femoris.

Rectus Femoris (RF) mEMG was statistically different at the different time points during 80L1, $\chi^2(3) = 12.983$, $p = .004$; SS+20, $\chi^2(3) = 9.873$, $p = .018$; and SS-20, $\chi^2(3) = 9.873$, $p = .018$. SSL8 and SSL1 did not elicit statistically different results in RF, p > .05. A Wilcoxon signed-rank test determined that there was no statistical difference in LRF mEMG at minute 4 (*Mdn* = 2.060%) compared to minute 2 (*Mdn* = 2.630 %) in 80L1, $z = -2.2565$, $p = 0.022$, $r = -.33$. 19 participants demonstrated a lower RRF at minute 4 (*Mdn* = 2.550%) compared to minute 2 (*Mdn* = 3.010 %) in 80L1, $p = .0066$, $r = -.39$. In 18 participants, LRF at minute four (*Mdn* = 2.060 %) was lower than LRF at minute 2 (*Mdn* = 2.780 %) in SS-20, $z = -2.738$, p = .005, $r = -.40$. 20 participants demonstrated a reduction in median mEMG at minute 4 (*Mdn* = 5.820%) as compared to minute 2 (*Mdn* = 7.270%).

Vastus Medialis Oblique.

Vastus Medialis Oblique (VMO) was statistically different at the different time points during 80L1, $\chi^2(3) = 15.365$, $p = .001$; SS+20, $\chi^2(3) = 11.800$, $p = .007$; and SS-20, $\chi^2(3) = 15.470$, $p = .001$. SSL8 and SSL1 did not elicit statistically different results in VMO, $p > .05$. 21 participants saw a reduction in LVMO mEMG at minute 4 (*Mdn* = 5.480%) compared to minute 2 (*Mdn* = 6.810 %) in 80L1, $z = -3.5285$, $p < 0.005$, $r = -.52$. 20 participants saw a lower median RVMO mEMG at minute 4 (*Mdn* = 4.280 %) compared to minute 2 (*Mdn* = 6.170 %) in 80L1, $z = -3.1936$, $p = .0007$, $r = -.47$. 20 participants demonstrated a reduction in LVMO mEMG at minute 4 (*Mdn* = 9.685 %) compared to minute 2 (*Mdn* = 12.200 %) in SS+20, $z = -3.328$, $p < 0.005$, $r = -.50$.

Semitendinosus.

Semitendinosus (ST) was statistically different at the different time points during all protocols; SSL1, $\chi^2(3) = 14.486$, $p = .001$; SSL8, $\chi^2(3) = 7.825$, $p = .048$; 80L1, $\chi^2(3) = 15.991$, $p = .001$; SS+20, $\chi^2(3) = 10.543$, $p = .013$; and SS-20, $\chi^2(3) = 9.104$, $p = .026$. A Wilcoxon signed-rank test determined that there was no statistical difference in RST mEMG at minute 4 (*Mdn* = 3.015 %) compared to minute 2 (3.105 %), $z = -2.516$, $p = 0.010$ or LST at minute 4 (*Mdn* = 4.820) compared to minute 2 (*Mdn* = 4.670 %), $z = -.812$, $p = .429$ during SSL1. Post hoc Wilcoxon Signed Rank test demonstrated that SSL8 elicited no statistical difference between LST at minute 4 (*Mdn* = 10.500 %) and minute 2 in SSL8 (*Mdn* = 9.370 %), $p = .335$. Additionally, RST was not different at minute 4 (*Mdn* = 6.19 %) or minute 2 (*Mdn* = 8.010 %) in SSL8, $p = .123$. There was no statistical difference in LST mEMG at minute 4 (*Mdn* = 2.330 %) compared to minute 2 (*Mdn* = 2.900 %) in 80L1, $z = -2.2052$, $p = 0.026$, $r = -.33$. 19 participants saw a reduction in RST at minute 4 (*Mdn* = 1.480 %) as compared to minute 2 (*Mdn* = 1.850 %) in 80L1, $z = -2.6464$, $p = 0.007$, $r = -.39$.

Medial Gastrocnemius.

Friedman test revealed no statistical differences in mEMG between time points, $p > .05$. However, marginal statistical difference did occur in 80L1, $\chi^2(3) = 7.591$, $p = .054$.

Soleus.

Soleus (SOL) was statistically different at the different time points during SSL1, $\chi^2(3) = 14.048$, $p = .002$ and SS-20, $\chi^2(3) = 13.690$, $p = .003$. Friedman test revealed no statistical differences in SOL mEMG between time points in SSL8, 80L1 or SS+20, $p > .05$. A Wilcoxon signed-rank test determined that there was no statistical difference in LSOL mEMG at minute 4 (*Mdn* = 3.27 %) compared to minute 2 (*Mdn* = 3.690 %) during SSL1, $z = -2.451$, $p = 0.013$. 17

participants demonstrated a reduction in RSOL mEMG at minute 4 (*Mdn* = 3.440 %) compared to minute 2 (*Mdn* = 4.120 %) during SSL1, *z* = -2.808, *p* = 0.004.

Tibialis Anterior.

Tibialis Anterior (TA) was statistically different at the different time points during 80L1, $\chi^2(3)$ = 14.048, *p* = .002. There was no statistical difference in LTA mEMG at minute 4 (*Mdn* = 1.170 %) compared to minute 2 (*Mdn* = 1.610 %) in 80L1, *z* = -2.3420, *p* = 0.0179, *r* = -.35. 18 participants saw a reduction in the RTA at minute 4 (*Mdn* = 2.590 %) compared to minute 2 (*Mdn* = 3.250 %) in 80L1, *z* = -2.2052, *p* = 0.0062, *r* = -.39

Peak Electromyography (pEMG)

Rectus Femoris.

RF pEMG was statistically different at the different time points during 80L1, $\chi^2(3)$ = 12.965, *p* = .004. Friedman tests revealed no statistical differences in RF pEMG between time points in other protocols, *p* > .05. 17 participants saw a non-statistically significant reduction in LRF between minute 4 (*Mdn* = 6.280 %) and minute 2 (*Mdn* = 7.990 %) in 80L1, *z* = -2.540, *p* = .009, *r* = -.38. 17 participants also saw a non-statistically significant reduction in RRF between minute 4 (*Mdn* = 6.550 %) and minute 2 (*Mdn* = 14.100 %) during 80L1, *z* = -2.312, *p* = .010, *r* = -.34.

Vastus Medialis Oblique.

VMO pEMG was statistically different at the different time points during all protocols; SSL1, $\chi^2(3)$ = 22.429, *p* < .0005; SSL8, $\chi^2(3)$ = 22.943, *p* < .0005; 80L1, $\chi^2(3)$ = 32.217, *p* < .0005; SS+20, $\chi^2(3)$ = 21.057, *p* < .0005; and SS-20, $\chi^2(3)$ = 26.188, *p* < .0005. 17 participants demonstrated a statistically significant reduction in LVMO between minute 4 (*Mdn* = 16.500 %) and minute 2 (*Mdn* = 27.700 %) in SSL1, *z* = -2.868, *p* = .003, *r* = -.49. 19 participants

demonstrated a statistically significant reduction in RVMO between minute 4 (*Mdn* = 10.800 %) and minute 2 (*Mdn* = 26.300 %) in SSL1, z = -3.574, p < .0005, r = -.58.16 participants demonstrated a statistically significant reduction in LVMO between minute 4 (*Mdn* = 30.050 %) and minute 2 (*Mdn* = 62.800 %) in SSL8, z = -2.902, p = .002, r = -.51. 19 participants also demonstrated a statistically significant reduction in RVMO between minute 4 (*Mdn* = 35.000 %) and minute 2 (*Mdn* = 66.400 %) in SSL8, z = -3.263, p = .001, r = -.53.22 participants demonstrated a statistically significant reduction in LVMO between minute 4 (*Mdn* = 6.920 %) and minute 2 (*Mdn* = 23.200 %) in 80L1, z = -4.106, p < .0005, r = -.62. 19 participants also demonstrated a statistically significant reduction in RVMO between minute 4 (*Mdn* = 7.910 %) and minute 2 (*Mdn* = 18.700 %) in 80L1, z = -3.376, p < .0005, r = -.55. 22 participants demonstrated a statistically significant reduction in LVMO between minute 4 (*Mdn* = 13.600 %) and minute 2 (*Mdn* = 31.700 %) in SS+20, z = -4.107, p < .0005, r = -.62. 17 participants also demonstrated a statistically significant reduction in RVMO between minute 4 (*Mdn* = 18.650 %) and minute 2 (*Mdn* = 35.100 %) in SS+20, z = -2.906, p = .003, r = -.50. 19 participants demonstrated a statistically significant reduction in LVMO between minute 4 (*Mdn* = 8.950 %) and minute 2 (*Mdn* = 21.500 %) in SS-20, z = -4.107, p < .0005, r = -.62. 17 participants also demonstrated a statistically significant reduction in RVMO between minute 4 (*Mdn* = 8.960 %) and minute 2 (*Mdn* = 19.000 %) in SS-20, z = -2.312, p = .020, r = -.40.

Semitendinosus.

ST pEMG was statistically different at the different time points during SSL1, $\chi^2(3)$ = 14.782, p = .001 and SSL8, $\chi^2(3)$ = 22.527, p < .0005. Friedman test revealed no statistical differences in ST pEMG between time points in 80L1, SS+20 nor SS-20, p > .05. 16 participants demonstrated a non-statistically significant improvement in LST pEMG between minute 2 (*Mdn*

= 14.300 %) and minute 4 (*Mdn* = 25.050 %) in SSL1, z = -2.565, p = .00854, r = -.39. 18 participants also demonstrated a statistically significant improvement in RST between minute 2 (*Mdn* = 10.200 %) and minute 4 (*Mdn* = 25.050 %) in SSL1, z = -2.950, p = .002, r = -.43.17 participants demonstrated a non-statistically significant improvement in LST pEMG between minute 2 (*Mdn* = 24.900 %) and minute 4 (*Mdn* = 44.200 %) in SSL8, z = -1.737, p = .085, r = -.26. 20 participants also demonstrated a statistically significant improvement in RST between minute 2 (*Mdn* = 23.400 %) and minute 4 (*Mdn* = 50.000 %) in SSL8, z = -3.467, p < .0005, r = -.51.

Medial Gastrocnemius.

MG pEMG was statistically different at the different time points during SSL8, $\chi^2(3)$ = 13.200, p = .003. Friedman tests revealed no statistical differences in MG pEMG between time points in SSL1, 80L1, SS+20 nor SS-20, p > .05. However, post hoc analysis revealed no statistical difference in LMG between minute 2 (*Mdn* = 48.200) and minute 4 (*Mdn* = 71.800), z = -2.159, p = .030, r = -.33 or RMG between minute 2 (*Mdn* = 52.000) and minute 4 (*Mdn* = 75.900), z = -1.999, p = .046, r = -.31 during SSL8.

Soleus.

SOL pEMG was statistically different at the different time points during SSL1, $\chi^2(3)$ = 10.200, p = .016 and SS-20, $\chi^2(3)$ = 11.765, p = .007. Friedman tests revealed no statistical differences in SOL pEMG between time points in SSL8, 80L1 or SS+20, p > .05. 18 participants demonstrated a statistically significant improvement in LSOL pEMG between minute 2 (*Mdn* = 14.300 %) and minute 4 (*Mdn* = 17.500 %) in SSL1, z = -2.829, p = .003, r = -.42. 18 participants also demonstrated a non-statistically significant decrease in RSOL between minute 4 (*Mdn* = 7.770 %) and minute 2 (*Mdn* = 13.800 %) in SS-20, z = -2.555, p = .009, r = -.38.

Tibialis Anterior.

TA was statistically different at the different time points during SSL8, $\chi^2(3) = 12.055$, $p = .006$ and SS+20, $\chi^2(3) = 13.171$, $p = .003$. 17 participants demonstrated a statistically significant improvement in LTA pEMG between minute 2 (*Mdn* = 31.100 %) and minute 4 (*Mdn* = 56.000 %) in SSL8, $z = -2.585$, $p = .0082$, $r = -.38$. 18 participants demonstrated a non-statistically significant improvement in RTA pEMG between minute 2 (*Mdn* = 31.200 %) and minute 4 (*Mdn* = 47.450 %) in SSL8, $z = -2.484$, $p = .011$, $r = -.37$. 17 participants demonstrated a statistically significant improvement in LTA pEMG between minute 2 (*Mdn* = 15.000 %) and minute 4 (*Mdn* = 32.600 %) in SS+20, $z = -2.776$, $p = .004$, $r = -.41$.

CHAPTER 7 METHODS – NUSTEP CROSS TRAINER VS. TREADMILL

Participants

Both healthy (n=19) and chronic stroke (≥6 months post CVA, n = 15) participants (aged 18-80) were recruited. Any participant that was diagnosed with a musculoskeletal, neurological, cardiopulmonary or respiratory condition that limited their ability to perform the investigation was excluded. Participants had no skin allergies to topical agents or adhesives. All participants signed an informed consent before testing. The investigation was approved by Wayne State University's institutional review board (Appendix B). All testing was conducted in the Neurotech laboratory in the Eugene Applebaum College of Pharmacy and Health Sciences at Wayne State University. This study was supported by a grant from the National Institutes of Health, P30 AG015281 and the Michigan Center for Urban African American Aging Research. Leg preference data were collected after the initial lab visit. Participants answered the following questions:

1- Which leg would you balance on for an extended period?
2- Which leg would you choose to kick with if you had to kick hard or far?

Baseline Measures

Upon the participant's arrival, researchers verbally explained the testing proceedings to the participant. The participant was then free to read the informed consent. Participants initialed and signed the informed consent when they had no more questions or concerns. Researchers then verbally acquired their date of birth, age, and shoe size. Stroke hemisphere (i.e., side affected), and date of CVA were obtained for the chronic stroke participants. Resting blood pressure was taken on the participant's preferred side; in response to the inquiry; "Which side is your blood pressure is usually taken at the doctor's office?' The subject was seated with the preferred arm passively stabilized on a medical table with the arm around heart level. Baseline blood pressure

and pulse were measured and recorded (Omron 10 Series BP785N Upper Arm Blood Pressure Monitor, Omron, Kyoto, Japan). Height and weight were measured. The order of testing procedures is outlined in Table 7.

Step	Element of Lab Visit
1	Informed Consent
2	Baseline (DOB, Shoe size, BP_{pre}, HR_{pre})
3	WiGAT[1]
4	$HR_1 + BP_1$
5	RPE based Self Selected Protocol-1 (10 Minutes)
6	$HR_2 + BP_2$
7	RPE based Self Selected Protocol-2 (10 Minutes)
8	EMG setup
9	MVCs
10	Goniometer setup
11	$HR_3 + BP_3$
12	Exercise Protocol $_1$ (5 minutes)
13	WiGAT[2]
14	HR4 + BP4
15	Exercise Protocol $_2$ (5 minutes)
16	WIGAT[3]

Table 7: Order of data collection during NuStep vs. Treadmill (NVT)

Procedures

Wireless gait assessment (WiGAT).

Previous researchers described the need to quantify gait (Page, Levine, Teepen & Hartman, 2008). The WiGAT system is an electronically infused shoe sole. The sole measured various spatial and temporal gait parameters including, but not limited to walking speed (m/sec), stride length, double support time, bilateral asymmetry, and stance-swing phase percentages. Electrodes are located on the 1st, 5th metatarsal heads, anterior toe and posterior heel (Scheme 3). WiGAT has been previously validated (Macleod, Conway, Allan & Galen, 2014). Three 10 meter walks were conducted pre-exercise intervention to establish baseline parameters. Three 10 meter walks were conducted immediately post exercise interventions.
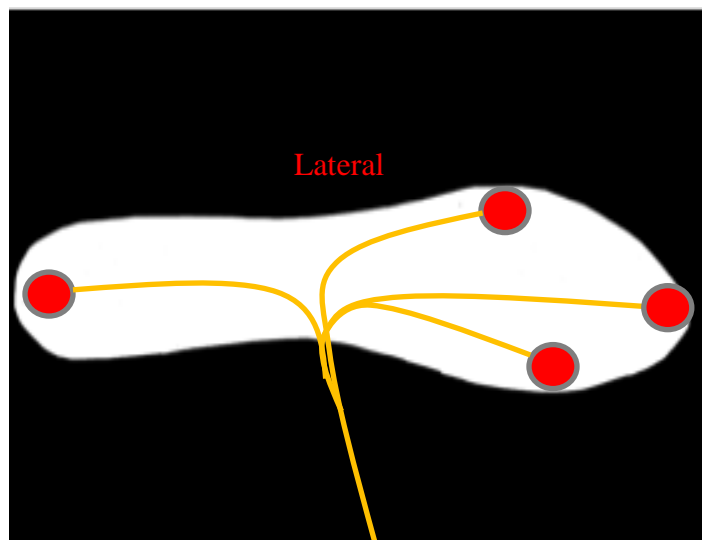
Scheme 3: Schematic representation of the WiGAT setup on the right foot. The red circles represent the location of sensors whereas the yellow lines represent the hardwiring.

Self-selected (SS) protocols.

A random number generator was utilized to dictate the order of the RPE based SS protocols. An even number (parameters, 1-100) resulted in the NuStep being performed first. RPE was collected in the last 10-15 seconds of each minute during the ten-minute protocol.

RPE SS protocol – NuStep Cross Trainer.

An instrumented version of the commercially available T5 NuStep Recumbent cross trainer (NuStep Inc., Ann Arbor, MI, USA) was utilized. The instrumented T5 NuStep Recumbent Cross Trainer can measure cadence of the participant (electronic step cadence meter and counter) while providing real-time visual feedback on pace against 15 levels of resistance (15 being the most challenging; at 1.0 increments of resistance). Both the RPE based SS protocol and the corresponding exercise bout were performed at level 1 of resistance. Both distal and proximal foot straps were utilized. Before exercise, the participant confirmed symmetrical tightness between each foot and between the distal and proximal foot straps. Participants performed recumbent stepping without upper extremity assistance. The seat position was set so

that the participant's right knee was near full extension (~15-20° of knee flexion when full knee extension = 0°) at the right step's terminal down position (See Figure 2). Participants confirmed a comfortable and safe position as previously described (Page, Levine, Teepen & Hartman, 2008). A handheld goniometer measured full knee extension and right knee flexion (See Figures 2 & 3). As the participants remained seated, the center of a handheld goniometer was placed over the lateral epicondyle of the femur.

Figure 2: NuStep Cross Trainer – R pedal down position

Figure 3: NuStep Cross Trainer – R pedal up position

The proximal arm was placed at the lateral midline of the femur with reference to the greater trochanter whereas the distal arm was aligned the lateral midline of the fibula with reference to the lateral malleolus. The participant's seat position influenced the degree of knee motion.

Participants were instructed to step at a full range of motion 1- without hitting bumper's end range and 2- for which the pedal position allows one to remain seated. The participant subjectively chooses a self-selected cadence in response to the instruction "step at a pace in which you're comfortable." After each minute, subjects are asked to report their Borg rating of perceived exertion (RPE) (Borg, 1982) (Appendix C). Therefore, if a participant falls below a 12 or higher than a 16 on the RPE scale during any one of the interval checks, the subject was instructed to speed up – or slow down accordingly. The preferred stepping rate (aSPM) was an average calculated over the 10-minute protocol.

RPE SS Protocol – Treadmill.

Participants were instructed that our goal was to find a comfortable walking pace that they could maintain for 10 minutes. Initial cadence was between 2.0-3.0 kph for healthy participants and 1.0 kph-2.0 kph for chronic stroke. After each minute, subjects were asked to report their RPE. If the participant fell below a 12 or higher than a 16 on the RPE scale during any one of the interval checks, the treadmill speed was adjusted accordingly ($\pm$0.2-0.4 kph). The preferred stepping rate was an average taken over the 10-minute protocol. If it was possible, participants were instructed not to hold handrails.

Electromyography.

Each electrode location was prepared by cleaning with rubbing alcohol and abrasive paper (Electrode Skin Prep Pads, Dynarex Corporation, Orangeburg, NY, USA). The surface electrodes (pre-gelled Ag/AgCl Noraxon Single Electrode, Noraxon USA Inc., AZ, USA) were placed over the muscle belly along the long axis and secured with paper tape based upon the Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM's) recommendations (Hermens et al., 1999). The reference electrode was placed at a location in

which the risk for disturbance signal was minimized (See Scheme 4). After the electrodes were placed, the electrode's (including the reference electrode) signal was assessed for contamination of movement artifacts and background noise.

Scheme 4: Example of Left Rectus Femoris Box placement

The electrode's final location was determined on both initial muscle palpation and the visual assessment of signal quality (Scheme 6). We recorded muscle activity from 12 muscles (6 per lower extremity) using a surface electromyography system (16 channel wireless) with an EMG bandwidth of 5-500 Hz (Noraxon Inc. Scottsdale, AZ, USA). The Noraxon EMG system was synced with a customized program written in Labview (National Instruments, Austin, TX, USA). This program collected EMG data in alternating 10-second epochs for the 3 minutes (minutes 2-4) of the 5-minute exercise protocol. EMG was processed with a second order high pass filter (cut off frequency 80-250 Hz) with zero phase lag to attenuate low-frequency components such as mechanical artifact. EMG data were full wave rectified, smoothed at 300ms and normalized to the participant's maximum voluntary contraction (MVC). Mean EMG amplitude (mEMG) were converted to a percentage of MVC. mEMG of the rectus femoris (RF),

vastus medialis oblique (VMO), semitendinosus (ST), tibialis anterior (TA), medial gastrocnemius (MG) and soleus (SOL) were recorded bilaterally. To minimize mechanical artifact, we secured the electrodes with tape and medical wrap as previously described (Stoloff, Zehr & Ferris, 2007).

Scheme 6: Left: Anterior view. Right: Posterior view. The electrode's final location was determined on both initial muscle palpation and the visual assessment of signal quality.

Maximum Voluntary Contractions.

Before subsequently measured blocks and to acclimate, participants performed one to two practice 5-seconds MVCs. Handheld dynamometer recorded both peak force and time to peak force (sec) (Lafayette Instrument 01165 Manual Muscle Testing Device). pEMG amplitude (uV) was measured. EMG signal processing and analysis is performed as per the International Society of Electrophysiology and Kinesiology guidelines (Hermens et al., 1999). The subsequent

measured MVC blocks required 3 repetitions of 5 seconds each. Limited rest (i.e., 1-5 seconds) separated each repetition. Participants were encouraged by the researcher yelling "push-push-push" (i.e., knee extension, plantarflexion) or "pull-pull-pull" (i.e., knee flexion, dorsiflexion). Participants were instructed to cross their arms and to breathe out during the 5-second repetition slowly.

All MVCs were performed seated. All MVCS followed a preplanned order and were performed on the right side before the left. Knee extension was performed first and later followed by knee flexion. Dorsiflexion superseded ankle plantarflexion. One to two minutes rest was given between joints. Isometric knee extension was performed at 90º (0º = full extension) of hip and knee flexion. Whereas, isometric knee flexion was performed at 90º hip flexion and 70º (0º = full extension) of knee flexion. Both plantarflexion and dorsiflexion were performed at 90° of hip and knee flexion in ankle neutral (i.e., 0° dorsiflexion and 0° plantarflexion).

Exercise Protocol.

Subjects performed both exercise protocols in a randomized order. A second random number generator determined this order. An even number (parameters: 1-100) determined that the NuStep would be performed first. Each protocol is 5 minutes was duration. Each 5-minute protocols consisted of a 1-minute acclimation, 3 minutes of recorded exercise (EMG and wireless goniometer data) and 1 minute of non-recorded exercise. The researcher adjusted TM pace whereas the participant maintained the NuStep pace through real-time feedback (See Figure 1b).
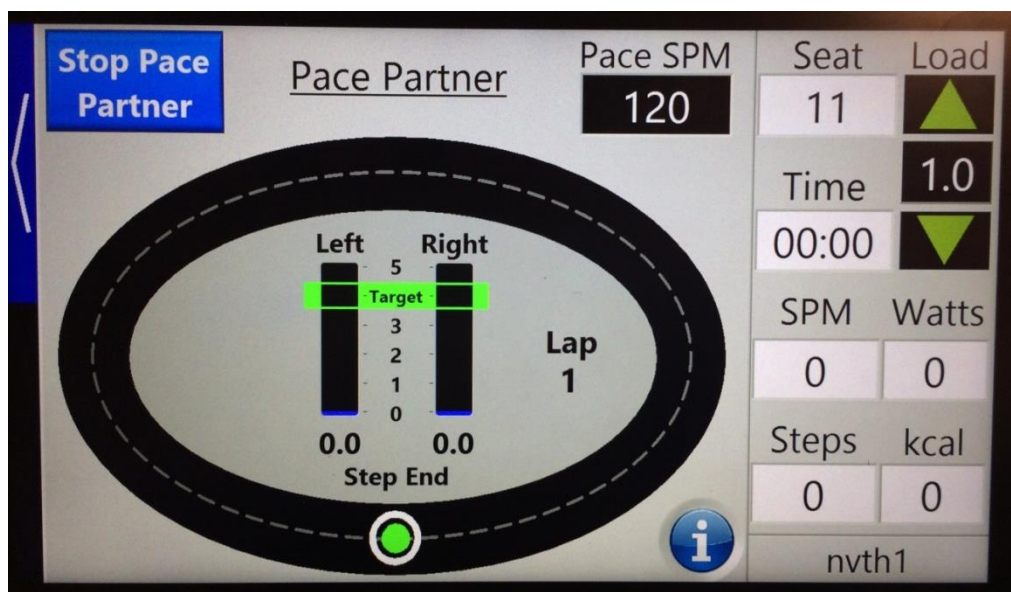
Figure 1b: NuStep Cross Trainer visual display during 5-minute exercise protocols.

Wireless Goniometry.

150mm twin axis goniometers were utilized at the hip and knee. 110 mm sensors were mounted to the ankle, bilaterally. End blocks were placed with the strain gauge reaching across the joint where the least movement occurred between the skin and underlying skeletal structure. A medical grade double-sided adhesive tape was utilized for attachment of all goniometers to the subject. Goniometer range of motion (ROM) was calculated as a difference score (i.e., maximum minus minimum) during the 5-minute exercise bouts on both the NuStep (NS) and treadmill (TM). The difference score represents the degrees of ROM that the joint experienced as a result of the exercise mode.

Blood Pressure (BP) and Heart Rate (HR).

González-Camarena et al. (2000) demonstrated the need to reestablish baseline levels of heart rate and blood pressure before a second exercise test. Therefore, heart rate (HR) and blood pressure (BP) were collected 3-5 minutes post-exercise bout. HR and BP were collected after SS

protocol 1 and SS protocol 2. HR and BP were collected before and after Exercise Protocol 1 (Figure #). Participants were given additional rest if HR and BP remained elevated.

Statistical Analysis

Statistical analysis was conducted using SPSS (Version 25). SPSS classified outliers into two categories. Extreme outliers were removed from data sets. Mild outliers were retained across all data sets. SPSS makes a distinction between mild outliers that are more than 1.5 box lengths from one hinge of the box (using a circle) and extreme outliers that are more than 3 box lengths from a hinge (using an asterisk). If data sets met parametric assumptions, an independent t-test or a Welch t-test (i.e., when the two samples have unequal variances and unequal sample size) was utilized for planned comparisons between conditions (i.e., healthy vs. stroke). If parametric assumptions were not met, the non-parametric Mann Whitney U test was utilized. Paired samples t-test was utilized to compare within conditions (i.e., Treadmill stroke vs. NuStep stroke). If parametric assumptions were not met, a Wilcoxon signed rank test or sign test was performed. The Wilcoxon signed-rank test is used to determine whether there is a median difference between paired or matched observations. The sign test is used to determine whether there is a median difference between paired or matched observations. This test can be considered as an alternative to the paired-samples t-test or Wilcoxon signed-rank test when the distribution of differences between paired observations is neither normal nor symmetrical, respectively.

One way repeated measures ANOVA was used to compare gait parameters at baseline, post-NuStep, and post TM. The one-way repeated measures analysis of variance (ANOVA) is an extension of the paired-samples t-test and is used to determine whether there are any statistically significant differences between the means of three or more levels of a within-subjects factor (i.e., independent variable). If parametric assumptions were not met, a non-parametric Friedman test

was employed. The Friedman test is the non-parametric alternative to the one-way repeated measures ANOVA test and is used to determine whether there are any statistically significant differences between the distributions of three or more related groups.

CHAPTER 8 RESULTS – NUSTEP CROSS TRAINER VS. TREADMILL

34 total participants completed the study. Participants were divided into chronic stroke (n = 15) and healthy age and sex-matched conditions (n = 19). The healthy condition consisted of 13 women and 6 men; whereas, the stroke condition consisted of 9 women and 6 men, respectively. The chronic stroke group was, on average, 10 ± 5 years post CVA. Among the stroke participants, 14 were hemiplegic (n = 8 right, n = 6 left) whereas one subject experienced a global and bilateral CVA. Task leg preference is listed in Table 8.

	Balance		Kick	
	Right	Left	Right	Left
Stroke	3	10	6	7
Healthy	15	2	15	2

Table 8: Task leg preference: Number of participants. Two subjects in both conditions were unresponsive.

	Age	BMI	Height	Weight
	(*Mdn*)	(*Mean ± Stdev*)	(*Mdn*)	(*Mdn*)
Stroke	66	27.02 ± 4.57	1.70m	77.00kg
Healthy	57	26.46 ± 4.63	1.74m	82.55kg

Table 9: Subject Demographics. Stdev: Standard Deviation

Age

Statistical difference was set at $p < .05$. Age data were first visual inspected by box plot. Due to two outliers, a Mann-Whitney U test determined if there were differences in age between healthy and stroke. Distributions of the ages were not similar, as assessed by visual inspection. Healthy (mean rank = 15.97) and stroke (mean rank = 19.43) ages were not significantly different, $U = 171.50$, $z = 1.007$, $p = .319$, $\eta^2 = .03$ (Table 9).

BMI

Statistical difference was set at $p < .05$. There were no outliers in the BMI data, as assessed by inspection of a boxplot. BMI data were normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). There was homogeneity of variances for both healthy and stroke, as assessed by Levene's test for equality of variances ($p = .803$). Therefore, an independent-samples t-test determined if there was a statistical difference between the BMI of healthy and stroke subjects. No statistical differences was observed between healthy BMI ($M = 26.46$, $SD = 4.63$) and the stroke ($M = 27.02$, $SD = 4.57$) BMI, $p > .05$, $d = .12$ (Table 9).

Height

There were mild outliers in the healthy (n = 2) and stroke (n = 2) height data, as assessed by inspection of a boxplot. Therefore, a Mann Whitney U test was used to compare the conditions. Distribution shapes between the two groups were similar. Median height was not statistically significantly different between healthy ($Mdn = 1.70$m) and stroke ($Mdn = 1.74$m), $U = 159.000$, $z = .574$, $p = .584$ (Table 9), using an exact sampling distribution for U (Dinneen & Blakesley, 1973).

Weight

There were mild outliers in the healthy (n = 4) and stroke (n = 0) height data, as assessed by inspection of a boxplot. Therefore, a Mann Whitney U test was used to compare the conditions. Distribution shapes between the two groups were similar. Median weight was not statistically significantly different between healthy ($Mdn = 77.00$ kg) and stroke ($Mdn = 82.55$ kg), $U = 173.000$, $z = 1.058$, $p = .302$, using an exact sampling distribution for U (Dinneen & Blakesley, 1973) (Table 9).

Rating of Perceived Exertion (RPE) - Between Conditions

Statistical difference was set at $p < .025$ unless otherwise noted. If parametric assumptions were met, an independent t-test was utilized. If parametric assumptions were not met, a Mann Whitney U test was utilized. If the two distributions have a different shape, the Mann-Whitney U test was used to determine whether there were differences in the distributions of the two compared groups. However, if the two distributions were the same shape, the Mann-Whitney U test was used to determine whether there were differences in the medians of the two compared groups.

Treadmill – healthy vs. stroke conditions.

Treadmill TM RPE contained mild outliers (n = 3). Therefore, a Mann-Whitney U test was run to determine if there were differences in the 10 minute RPE based self-selected (SS) protocol on TM between healthy and stroke conditions. Distributions of RPE for healthy and stroke were not similar, as assessed by visual inspection. RPE for stroke (mean rank = 15.83) was not statistically different to healthy RPE (mean rank = 18.82), $U = 117$, $z = -.868$, $p = .391$, $\eta^2 = .02$.

NuStep – healthy vs. stroke conditions.

NuStep (NS) RPE did not contain outliers, as assessed by box plot. NS RPE was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). There was homogeneity of variances, as assessed by Levene's test for equality of variances ($p = .446$). There was no statistical difference in NS RPE between healthy ($M = 12$, $SD = 2$) and stroke ($M = 11$, $SD = 2$), $t(32) = 1.026$, $p = .313$, $d = .35$.

Rating of Perceived Exertion (RPE) - Within Conditions

When parametric assumptions were met, a paired t-test was utilized. Statistical difference was set at p < .025 unless otherwise noted.

Healthy – treadmill vs. NuStep.

Healthy Treadmill RPE contained two outliers, as assessed by box plot. Wilcoxon Signed Rank Test was utilized to describe the median differences between the 10 minute RPE based SS protocol on TM and NS in healthy participants. The distribution of differences was symmetrically shaped. Healthy RPE contained two mild outliers as assessed by inspection of a boxplot. A Wilcoxon signed-rank test determined that there was no statistical difference in RPE medians between TM (*Mdn* = 12.70) and the NS (*Mdn* = 11.90) in healthy subjects, z = -1.525, p = .127, r = - .25.

Stroke – treadmill vs. NuStep.

Stroke TM RPE contained one mild outlier as assessed by inspection of a boxplot. Wilcoxon Signed Rank Test was first utilized to describe the median differences between the 10 minute RPE based SS protocol on TM and NS. However, the distribution of differences was not symmetrically shaped. Therefore, A sign test determined that there was no statistical difference in RPE medians between TM (*Mdn* = 11.90) and the NS (*Mdn* = 11.40) in healthy subjects, z = - .866, p = .388, r = - .16.

Self-Selected (SS) Protocol – By Exercise Mode

Statistical difference was set at p<.05. When parametric assumptions were met, an independent t-test was utilized.

SS TM speed: – healthy vs. stroke conditions.

There were no outliers, as assessed by a boxplot. However, healthy SS TM Speed violated normality, as described by Shapiro-Wilk, ($p < .05$). A Mann-Whitney U test was run to determine if there were differences in SS TM speeds between healthy and stroke. Distributions of SS TM speed were not similar, as assessed by visual inspection. Stroke SS TM speed (mean rank = 9.20) was statistically lower than the healthy condition (mean rank = 24.05), $U = 18.000$, $z = -4.319$, $p < .0005$, $\eta^2 = 3.25$.

SS NS average steps per minute (SPM).

Box plot inspected revealed mild outliers in the stroke condition (n=2). A Mann-Whitney U test was run to determine if there were differences SS NS aSPM between healthy and stroke on NuStep. Distributions of the aSPM were similar, as assessed by visual inspection. Stroke SS NS speed (*Mdn* = 108 aSPM) was not statistically different than the healthy condition (*Mdn* = 121 aSPM), $U = 91.000$, $z = -1.787$, $p = .078$, $\eta^2 = -.11$.

10 minute SS exercise bout vs. 5 minute SS exercise bout pace.

Statistical difference was set at $p < .05$ unless otherwise noted. When parametric assumptions were met, a paired t-test was utilized.

There was one mild outlier in the data (n = 1), as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. The difference scores for the TM average speed between the SS protocol and the 5 min exercise bout on TM bout were not distributed normally; Shapiro-Wilk's test ($p < .05$). The distribution of the differences between the two related groups was asymmetrical in shape. Therefore, an exact sign test was used to compare the differences in TM speed (kph) between the 10 minute SS bout and the 5-minute

exercise bout. The TM calculated SS pace (*Mdn* = 3.550 kph), and TM set pace (*Mdn* = 3.500 kph) did not differ, *p* = 1.00.

There were no outliers in the NS data, as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. The difference scores violated normality, as assessed by Shapiro-Wilk's test (*p* < .05). Furthermore, the distribution of the differences between the two related groups was asymmetrical in shape. Therefore, an exact sign test was used to compare the steps per minute (spm) medians of the 10 minute SS bout and the 5-minute exercise bout. 10 minute SS (*Mdn* = 112.50 spm) and the set 5 minute pace partner (*Mdn* = 113.50 spm) did not differ, *p* = .089.

Force: Between Conditions

Statistical difference was set at *p* < .025 unless otherwise noted. When parametric assumptions were met, an independent samples t-test was utilized.

Knee – healthy vs. stroke conditions.

An independent t-test was administered to detect a difference between right (R) knee extension (Ext) between healthy and stroke conditions. There were no outliers in the data, as assessed by inspection of a boxplot. R Knee Ext force was normally distributed in both conditions, as assessed by Shapiro-Wilk's test (*p* > .05). There was homogeneity of variances for force for healthy and stroke conditions, as assessed by Levene's test for equality of variances (*p* = .750). R knee Ext force was higher in healthy (*M* = 25.08kg, *SD* = 6.31kg) than stroke (*M* = 16.23kg, *SD* = 5.93kg). Independent t-test revealed a mean difference of 8.86kg between the healthy and stroke participants; 95% CI [4.53 kg, 13.18 kg], *t*(32) = 4.175, *p* < .0005, *d* = 1.45.

There was one mild outlier in the healthy L knee Ext data, as assessed by inspection of a boxplot. Therefore, a Mann Whitney U test was run to determine force differences in left (L)

knee Ext between conditions. Distributions of the force values were not similar, as assessed by visual inspection. Distributions of the force values were not similar, as assessed by visual inspection. Healthy force values (mean rank = 21.11) were statistically significantly higher than for stroke (mean rank = 12.93), $U = 74$, $z = -2.376$, $p = .017$, $\eta^2 = .17$.

R knee flx force did not contain any outliers. Data were normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). However, the assumption of homogeneity of variances was violated, as assessed by Levene's test for equality of variances ($p = .009$). A Welch t-test determined that healthy R knee Flx force was 12.70kg higher in the healthy condition ($M = 21.71$ kg, $SD = 8.13$kg) than stroke ($M = 9.01$kg, $SD = 3.46$kg), 95% CI [8.42 to 16.97kg], $t(25.795) = 6.101$, $p < .0005$.

An independent t-test was administered to detect a difference between L knee Flx between healthy and stroke conditions. There were no outliers in the data, as assessed by inspection of a boxplot. L Knee Flx force was normally distributed in both conditions, as assessed by Shapiro-Wilk's test ($p > .05$). There was homogeneity of variances for force for healthy and stroke, as assessed by Levene's test for equality of variances ($p = .159$). L knee Flx force was higher in healthy ($M = 21.30$kg, $SD = 8.21$kg) than stroke ($M = 14.63$kg, $SD = 6.29$kg), a statistically significant mean difference; $M = 5.77$kg, 95% CI [0.54, 10.99], $t(32) = 2.247$, $p = .032$, $d = .79$.

Ankle – healthy vs. stroke conditions.

There were two mild outliers in the R dorsiflexion (dflex) data, as assessed by inspection of a boxplot. A Mann-Whitney U test was run to determine if there were differences in R dflex between healthy and stroke. Distributions of the force values were not similar, as assessed by

visual inspection. Healthy force values (mean rank = 24.00) were statistically and significantly higher than for the stroke condition (mean rank = 9.27), $U = 19$, $z = -4.284$, $p < .0005$, $\eta^2 = .56$.

There were no outliers in the L dflex data, as assessed by inspection of a boxplot. L dflex force was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). The assumption of homogeneity of variances was violated, as assessed by Levene's test for equality of variances ($p = .043$). Therefore, a Welch t-test was run to determine if there were differences in L dflex force between healthy and stroke. There was a statistically significant difference in L dflex between conditions, with healthy ($M = 15.88$kg, $SD = 4.60$kg) scoring higher than stroke ($M = 11.51$kg, $SD = 2.64$kg), $M = 4.36$kg, 95% CI [1.64, 7.08], $t(32) = 3.268$, $p = .002$.

One mild Outlier existed in the R plantarflexion (pflex) data, as assessed by inspection of the boxplot. A Mann-Whitney U test was run to determine if there were differences in R pflex between healthy and stroke. Distributions of the force values were not similar, as assessed by visual inspection. Healthy force values (mean rank = 24.47) were statistically significantly higher than for stroke (mean rank = 8.67), $U = 10$, $z = -4.596$, $p < .0005$, $\eta^2 = .64$.

One mild Outlier also existed in the L plantarflexion (pflex) data, as assessed by inspection of the boxplot. A Mann-Whitney U test was run to determine if there were differences in L pflex between the conditions. Distributions of the force values were not similar, as assessed by visual inspection. Healthy force values (mean rank = 22.53) were statistically significantly higher than for stroke (mean rank = 11.13), $U = 47$, $z = -3.313$, $p = .001$, $\eta^2 = .33$.

Force: Within Conditions – Bilateral Comparison

Statistical difference was set at p<.025 unless otherwise noted. When parametric assumptions were met, a paired samples t-test was utilized.

Healthy knee force – right vs. left.

There were two mild outliers in the healthy condition knee ext difference data. The distribution of the differences between the two related groups was asymmetrical in shape. Therefore, an exact sign test was used to compare the extremity force differences. Healthy participants were stronger in L knee extension (*Mdn* = 28.321 kg) than R knee extension (*Mdn* = 26.323 kg); a median difference of 1.5574 kg, *p* = .004, *r* = -.52. Of the 19 healthy participants recruited to the study, the L knee was stronger in 16 healthy participants, whereas three participants demonstrated a higher median force in the R knee.

There were no outliers in the healthy knee flx difference data. The difference scores were normally distributed, as assessed by Shapiro-Wilk's test (*p* > .05). Healthy subjects did not elicit a difference in knee flx force between the L (*M* = 21.30 kg, *SD* = 8.21 kg) and the R (*M* = 21.71kg, *SD* = 8.13 kg), *t*(18) = -.492, *p* = .629, *d* = .05.

Healthy ankle force – right vs. left.

Dflex (n = 2) and pflex (n = 1) peak force contained outliers in the healthy condition. A Wilcoxon signed-rank test determined that there was no statistically difference in healthy R dflex (*Mdn* = 16.587kg) and L DorsiFlex (*Mdn* = 15.468kg), *z* = 1.529, *p* = .126, *r* = .25. A Wilcoxon signed-rank test determined that there was no statistically significant median difference in force production in L PlantarFlx (*Mdn* = 14.94kg) compared to R PlantarFlx (*Mdn* = 19.28kg) in the healthy condition, *z* = 1.912, *p* = .056, *r* = .31.

Stroke knee force – right vs. left.

As assessed by inspection of a boxplot, there were no outliers in the knee ext difference score within the stroke condition. The difference scores for stroke R knee ext and L knee ext force were normally distributed, as assessed by Shapiro-Wilk's test (*p* > .05). A paired sample t-

test was run to test bilateral differences in knee extension. Stroke subjects produced a higher level of force on the L side (M = 20.74 kg, SD = 6.637) as opposed to the R side (M = 16.23 kg, SD = 5.925kg). L knee ext elicited a mean increase of 4.516 kg, 95% CI [2.125, 6.908] in force compared to R side, $t(14)$ = 4.050, p = .001, d = 1.05.

There were no outliers in the knee flx difference score within the stroke condition. The difference scores for Stroke R knee flx and L knee flex force were normally distributed, as assessed by Shapiro-Wilk's test (p > .05). Stroke participants produced a higher level of L knee flex (M = 15.534kg, SD = 6.285kg) as opposed to R KneeFlx (M = 10.285kg, SD = 5.960kg), a statistically significant mean difference of 5.248 kg, 95% CI [1.084, 9.413], $t(14)$ = 2.703, p = .017, d = 0.70.

Stroke ankle force – right vs. left.

Boxplot inspection revealed the existence of one mild outlier in stroke dorsiflexor bilateral comparison. The distribution of the differences between the two related groups was asymmetrical in shape. Therefore, an exact sign test was used to compare the extremity force differences. Stroke participants exhibited no statistical difference between L DorsiFlx (Mdn = 11.401kg) than R DorsiFlex (Mdn = 6.079kg). However, a median difference of 4.824 kg was found, p = .035, r = -.38. Of the 15 stroke participants recruited to the study, the L ankle elicited an improvement in force production in 12 participants compared to the R ankle, whereas three participants demonstrated a higher median force in the R ankle.

There were no outliers in the stroke pflex data. The differences between the PlantarFlx forces were normally distributed, as assessed by Shapiro-Wilk's test (p > .05). Paired samples t-test revealed no statistical difference between the right (M = 7.76kg, SD = 3.35kg) and left (M = 9.39kg, SD = 3.56kg) extremities, $t(14)$ = 1.079, p = .299, d = .47.

NuStep Cross Trainer Static Knee Positions

There were no outliers in the data, as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. Static R knee flx was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). Variances were homogeneous, as assessed by Levene's test for equality of variances ($p = .885$). Right knee flexion was 5°, 95% CI [2°, 8°] greater in the healthy condition (M = 79°, SD = 4°) than stroke (M = *74°*, SD = 4°), $t(32) = 3.515$, $p = .001$, $d = 1.21$.

There were two mild outliers in the knee ext data, as assessed by inspection of a boxplot. Therefore, a Mann-Whitney U test was run to determine if there were differences in knee ext between conditions. Condition distributions were not similar, as assessed by visual inspection. There was no statistical difference existed in knee extension between healthy (mean rank = 18.03) and stroke (mean rank = 16.83), $U = 132.50$, $z = -.349$, $p = .732$, $\eta^2 = -.02$, using an exact sampling distribution for U (Dinneen & Blakesley, 1973).

NuStep Seat Positions

Inspection of the boxplot identified one mild outlier. A Mann-Whitney U test was run to determine if there were differences in seat position between conditions. Condition distributions were not similar, as assessed by visual inspection. There was a statistically significantly difference in seat position between healthy (mean rank = 14.16) and stroke (mean rank = 21.73), $U = 206.000$, $z = 2.246$, $p = .027$, $\eta^2 = .15$.

Heart Rate (HR)

There were no outliers in the HR data, as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. HR was normally distributed at each time point, as assessed by Shapiro-Wilk's test ($p > .05$). Mauchly test of sphericity indicated that the

assumption of sphericity was violated, $\chi^2(9) = 18.270$, $p = .032$. Epsilon (ε) was 0.741, as calculated according to Greenhouse & Geisser (1959), and was used to correct the one-way repeated measures ANOVA. HR was statistically significantly different at the different time points during the exercise intervention, $F(2.964, 82.996) = 14.562$, $p < .001$, partial $\eta^2 = .34$. Post-hoc paired comparisons are outlined in Figure 4.
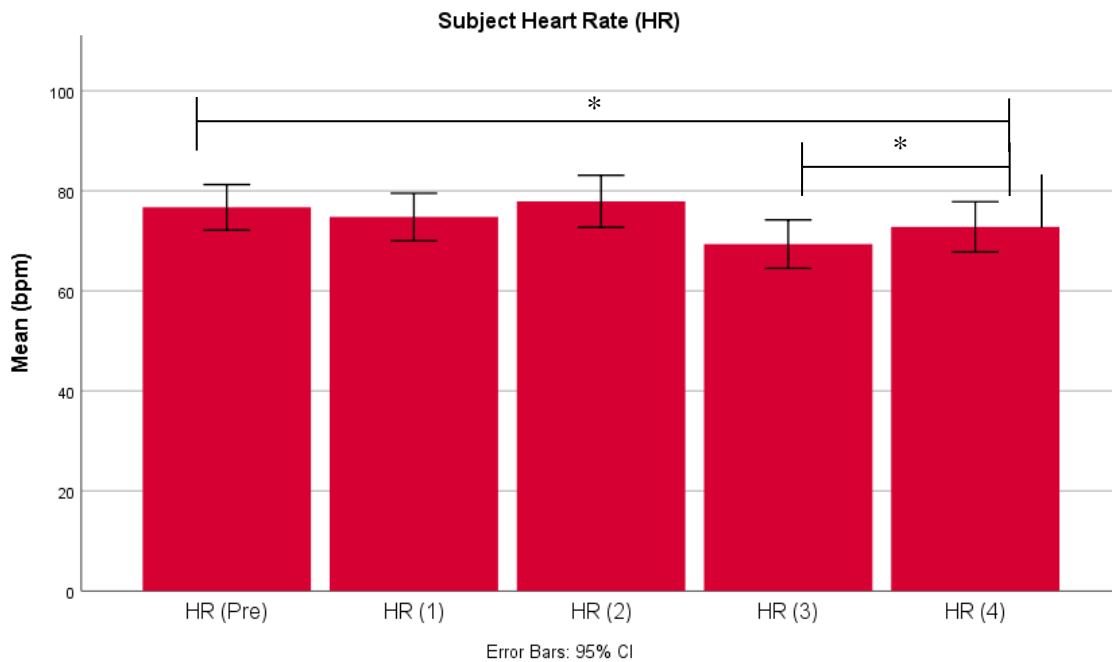
Figure 4: Subject Heart Rate (HR). HR pre: Resting HR, HR (1): Post WiGAT 1, HR (2): Post SS Exercise Protocol 1, HR (3): After MVC, HR (4): After Exercise Bout 1. *$p < .05$

The highest HRs were recorded at baseline (Figure 4: HR (Pre)) ($M = 76.59$ bpm, $SD = 11.97$ bpm) and post 10 minute SS protocol 1; (Figure 4: HR (2)) ($M = 77.90$ bpm, $SD = 13.67$), p<0.05. There was no statistical difference between HR (1) and HR (2), $p = .478$. A mean difference of 3.45 beats occurred post MVC; (Figure 4: HR (3)) and prior to the last 5 minute exercise bout (Figure 4: HR (4)), $p = .01$. HR (4) ($M = 72.79$ bpm, $SD = 13.20$bpm) was statistically lower than HR (Pre) ($M = 76.69$bpm, $SD = 11.97$bpm).

Systolic Blood Pressure

There were two mild outliers in the systolic blood pressure (SBP) data, as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. Therefore, the non-parametric Friedman test was used to compare SBP mean ranks across time. SBP was not statistically different at the different time points during the intervention, χ^2 (4) = 3.665, p = .453.

Diastolic Blood Pressure

There were mild outliers in the diastolic blood pressure (DBP) data (n = 2). A non-parametric Friedman test was used to compare DBP mean ranks across time. DBP was not statistically different at the different time points during the intervention, χ^2(4) = 1.662, p = .798.

Goniometers

TM ∆ROM – between conditions.

Right hip ∆ROM.

There were no R hip outliers in the TM ∆ROM data, as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. R Hip ∆ROM values were normally distributed for both conditions as assessed by Shapiro-Wilk's test (p > .05). Variances were homogeneous, as assessed by Levene's test for equality of variances (p = .083). There were 11 total healthy (M = 33.72º, SD = 14.01°) and 3 stroke participants (M = 22.23∘, SD = 0.64°). There was no statistical difference between conditions on TM, t(12) = 1.379, p = .193, d = 1.16.

Left hip ∆ROM.

There was one mild L hip outlier in the TM data. A Mann-Whitney U test was run to determine if there were differences in L Hip ∆ROM between the stroke and healthy conditions

on the TM. There was no statistically significantly difference in L Hip ΔROM mean rank between the 6 stroke participants (mean rank = 18.54) and 13 healthy participants (mean rank = 21.58), $U = 181$, $z = .785$, $p = .447$, $\eta^2 = .02$.

Right knee ΔROM.

TM R knee ΔROM data did not contain outliers. However, R knee data violated normality, as assessed by Shapiro Wilk, ($p = .044$). Therefore, A Mann-Whitney U test was run to determine if there were differences in R Knee ΔROM between stroke and healthy conditions on the TM. Distributions of R knee ΔROM for healthy and stroke were not similar, as assessed by visual inspection. There was no statistically significantly difference in R Knee ΔROM mean rank between the 17 healthy participants (mean rank = 17.06) and 15 stroke (mean rank = 15.87) participants, $U = 181$, $z = .785$, $p = .447$, $\eta^2 = .02$.

Left knee ΔROM.

L Knee ΔROM contained one mild outlier. A Mann-Whitney U test was run to determine if there were differences in L Knee ROM between stroke and healthy conditions on the TM. There was no statistically significantly difference in L Knee ROM between the 14 healthy participants (mean rank = 11.07) and 9 stroke (mean rank = 13.44) participants, $U = 76$, $z = .820$, $p = .439$, $\eta^2 = .03$.

Right ankle ΔROM.

Six extreme outliers were removed from R ankle (ank) TM ΔROM data. Two mild outliers were retained. R ank data violated normality, $p = .015$. A Mann-Whitney U test was run to determine if there were differences in R ank ΔROM between stroke (n=10) and healthy (n=14) conditions on the TM. There was no statistically significantly difference in R ank ΔROM between the conditions on the TM, $U = 64$, $z = -.351$, $p = .752$, $\eta^2 < .01$.

Left ankle ΔROM.

Three extreme outliers were removed from the L ank TM ΔROM data. One mild outlier was retained. A Mann-Whitney U test was run to determine if there were differences in L ank ΔROM between stroke (n=12) and healthy (n=17) conditions on the TM. There was statistical increase in L ank ROM in healthy (*Mdn* = 33.08°) as compared to the stroke (*Mdn* = 27.14°), *U* = 48.000, *z* = -2.391, *p* = .016, η^2 = .20.

NS ΔROM – between conditions.

Right hip ΔROM.

There were no outliers in the R hip data, as assessed by inspection of a boxplot. NS R hip ΔROM was normally distributed, as assessed by Shapiro-Wilk's test (*p* > .05). Variances were homogeneous, as assessed by Levene's test for equality of variances (*p* = .755). An independent-samples t-test was run to determine if there were differences in R hip ΔROM between the healthy (n = 10) and stroke (n = 3) conditions. ΔROM did not differ between healthy (*M* = 25.35, *SD* = 10.59) than stroke participants (*M* = 19.84, *SD* = 9.04), *t*(11) = .810, *p* = .435, *d* = .56.

Left hip ΔROM.

There were no outliers in the L hip data. Hip L was normally distributed, as assessed by Shapiro-Wilk's test (*p* > .05). Homogeneity of variances, as assessed by Levene's test for equality of variances was violated (*p* = .026). A Welch t-test was, therefore, run to determine if there were differences in L hip ΔROM between the healthy (n=12) and stroke (n=6) conditions. ΔROM did not differ between healthy (*M* = 28.75, *SD* = 11.36) than stroke conditions (*M* = 48.31, *SD* = 48.31), *t*(5.280) = -.981, *p* = .370, *d* = 0.56.

Right knee ΔROM.

There were no outliers in the R knee ΔROM data. R knee was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). Variances were homogeneous, as assessed by Levene's test ($p = .218$). An independent-sample t-test was run to determine if there were differences in R Knee ΔROM between the healthy (n = 19) and stroke (n = 15) conditions. ROM did not differ between healthy ($M = 60.89°$, $SD = 11.45°$) and stroke participants ($M = 67.55º$, $SD = 27.58º$), $t(32) = -.218$, $p = .346$, $d = 0.32$.

Left knee ΔROM.

There was one mild outlier in the L knee ΔROM data. A Mann-Whitney U test was run to determine if there were differences in L knee ΔROM between healthy (n=14) and stroke (n = 10). ΔROM distributions were not similar, as assessed by visual inspection. There was no statistically significantly difference in mean rank ΔROM between healthy (mean rank = 10.93) and stroke (mean rank = 14.70), conditions $U = 92$, $z = 1.288$, $p = .212$, $\eta^2 = .07$.

Right ankle ΔROM.

Two extreme outliers were removed from the R ank ΔROM data. Stroke R ank data violated normality, $p < 0005$. A Mann-Whitney U test was run to determine if there were differences in ΔROM between healthy (n = 16) and stroke (n = 10). R ank ΔROM distributions were not similar, as assessed by visual inspection. There was no statistically significantly difference in R ank ΔROM between healthy (mean rank = 13.88) and stroke (mean rank = 12.90) conditions, $U = 74.000$, $z = -.316$, $p = .776$, $\eta^2 < .01$.

L Ankle ΔROM.

Three extreme outliers were removed from the L ank ΔROM data. L ank was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). Variances were homogeneous, as

assessed by Levene's test, was violated ($p = .138$). A Welch t-test was, therefore, run to determine if there were differences in L ank between the healthy (n = 18) and stroke (n = 12) conditions. ROM did not differ between healthy ($M = 22.28°$, $SD = 6.64°$) and stroke participants ($M = 26.23º$, $SD = 22.13º$), $t(28) = -.717$, $p = .479$, $d = .24$.

Healthy – Within Condition.

Statistical difference was set at $p<.025$ unless otherwise noted. When parameter assumptions were met, a paired t-test was utilized. The ΔROM difference (i.e., ROM TM – ROM NS) was calculated and evaluated for outliers.

Right hip ΔROM.

Healthy R (n = 1) and L hip (n = 1) both contained mild outliers. Distributions were not symmetrical. Therefore, a sign test determined that there was no statistical difference in R hip ΔROM between TM ($Mdn = 33.61$) and the NS ($Mdn = 27.09$) in healthy subjects, $z = 1.206$, $p = .277$, $r = .26$.

Left hip ΔROM.

A sign test also determined that there was no statistical difference in L hip ΔROM between TM ($Mdn = 32.58$) and the NS ($Mdn = 25.15$) in healthy subjects, $z = 1.109$, $p = .267$, $r = .22$.

Right knee ΔROM.

R knee ΔROM did not contain outliers across the healthy condition. R knee difference was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). A paired t-test was conducted to compare the TM (n = 18) to NS (n = 18). Healthy subjects produced a higher level of R knee ΔROM on TM ($M = 74.78°$, $SD = 21.95°$) as opposed to the NS ($M = 61.00°$, $SD =$

11.76°). TM R knee ΔROM demonstrated a higher mean ROM of 13.79°, 95% CI [3.78°, 23.80°] compared to NS, $t(17) = 2.91$, $p = .010$, $d = .78$.

Left knee ΔROM.

L knee ΔROM contained one mild outlier. Distributions were not symmetrical. Therefore, a sign test determined that there was no statistical difference in L knee ΔROM between TM ($Mdn = 69.86$) and the NS ($Mdn = 61.74$) in healthy subjects, $z = .802$, $p = .424$, $r = .12$.

Right ankle ΔROM.

Healthy R ank ΔROM contained three mild outliers. Therefore, an exact sign test determined that there was a statistical difference in R ank ΔROM between TM ($Mdn = 39.33$) and the NS ($Mdn = 25.43$) in healthy subjects, $z = 2.750$, $p = .004$, $r = .49$.

Left ankle ΔROM.

Two extreme outliers were removed in the L ank ΔROM. One mild outlier was retained. The distribution of differences was not symmetrically shaped. Therefore, an exact sign test was used to compare the L ank differences between the NS (n = 18) and TM (n = 17). Healthy participants demonstrated greater median ROM on TM ($Mdn = 33.08°$) than NS ($Mdn = 21.76°$), $z = 3.250$, $p = .001$, $r = .59$.

Stroke – Within Condition.

Statistical difference was set at p<.025 unless otherwise noted. When parameter assumptions were met, a paired t-test was utilized. The ΔROM difference (i.e., ROM TM – ROM NS) was calculated and evaluated for outliers.

Right hip ΔROM.

R hip ΔROM differences did not contain outliers in the stroke condition. R hip difference was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). Paired t-test was

conducted to compare R hip ΔROM between the NS and TM (n = 3). Stroke participant's R hip ΔROM did not differ between the TM (M = 22.23º, SD = .64º) compared to NS (M = 19.84º, SD = 9.04º), $t(2)$ = -.430, p = .709, d = .37.

L Hip ΔROM.

L hip ROM differences contained two extreme outliers in the stroke condition. L hip difference was normally distributed, as assessed by Shapiro-Wilk's test (p > .05). Paired t-test was conducted to compare L hip ΔROM between the NS and TM (n = 4). The paired t-test revealed no statistical differences between TM (M = 29.10º, SD = 8.72°) and NS (M = 61.31º, SD = 56.43º), $t(2)$ = 1.327, p = .316, d = .79.

Right knee ΔROM.

R knee ΔROM did not contain outliers in the stroke condition. R knee difference was normally distributed, as assessed by Shapiro-Wilk's test (p > .05). Paired t-test was conducted to compare R knee ΔROM between the NS and TM (n = 15). Stroke participant's R knee ΔROM did not differ between the TM (M = 72.02º, SD = 35.27º) compared to NS (M = 67.55º, SD = 27.58º), $t(14)$ = -.360, p = .724, d = .14.

Left knee ΔROM.

L knee ROM differences did not contain outliers in the stroke condition. L hip ΔROM was normally distributed, as assessed by Shapiro-Wilk's test (p > .05). Paired t-test was conducted to compare L knee ΔROM between the NS and TM (n=9). No statistical differences in ΔROM existed between TM (M = 86.96º, SD = 40.22) and NS (M = 81.78º, SD = 31.45º), $t(8)$ = .281, p = .786, d = .11.

Right ankle ΔROM.

The stroke condition's R ank ΔROM did not contain outliers. R ank ΔROM was normally distributed, as assessed by Shapiro-Wilk's test (*p* > .05). Paired t-test was conducted to compare R ank ΔROM between the NS and TM (n = 10). R ank ROM did not differ between the TM (*M* = 31.95º, *SD* = 15.37º) compared to NS (*M* = 52.63º, *SD* = 50.19º), *t*(9) = 1.388, *p* = .199, *d* = .56.

Left ankle ΔROM.

Stroke L ank ROM differences did not contain outliers. L hip difference was normally distributed, as assessed by Shapiro-Wilk's test (*p* > .05). Paired t-test was conducted to compare L ank ΔROM between the NS and TM (n = 15). The paired t-test revealed no statistical differences between TM (*M* = 60.80º, *SD* = 61.49º) and NS (*M* = 54.63º, *SD* = 62.22º), *t*(14) = -.275, *p* = .787, *d* = .09.

Mean Electromyography

mEMG NuStep Cross Trainer -- between conditions.

Statistical difference was set at *p* < .025 unless otherwise noted. When parameter assumptions were met, an independent t-test was utilized. Mean electromyography (mEMG) was reported as a percentage of maximum voluntary contraction (%). mEMG was evaluated for outliers by box plot.

Rectus femoris.

One extreme outlier was removed from the right Rectus Femoris (RF) data set. RRF mEMG was normally distributed for each condition, as assessed by Shapiro-Wilk's test (*p* > .05). Variances were homogeneous, as assessed by Levene's test for equality of variances (*p* = .173). Independent t-test demonstrated a statistically significant difference in RRF %MVC between healthy (*M* = 6.24%, *SD* = 3.86%) and stroke (*M* = 12.17%, *SD* = 6.24%, *t*(31) = -3.552, *p* =

.001, *d* = 1.22. Stroke RRF %MVC was 5.924 degrees 95% CI [2.52, 9.33 %] higher than the healthy condition.

One extreme outlier was removed from mean mEMG left Rectus Femoris (LRF) data set. Mild outliers were retained (n=3). A Mann-Whitney U test was run to determine if there were differences in LRF %MVC in healthy and stroke on NuStep. Distributions for healthy and stroke were not similar, as assessed by visual inspection. LRF %MVC for stroke (mean rank = 19.93) were statistically higher than for healthy (mean rank = 11.62), $U = 174$, $z = 2.579$, $p = .009$, $\eta^2 = .23$.

Vastus medialis oblique.

One extreme outlier was removed for RVMO %MVC. Mild outliers were retained (n=3). A Mann-Whitney U test was run to determine if there were differences in RMVO %MVC in healthy and stroke on NuStep. Distributions for healthy and stroke were not similar, as assessed by visual inspection. RVMO %MVC for stroke (mean rank = 16.93) was not statistically different than RVMO %MVC healthy (mean rank = 17.05) on NuStep, $U = 132$, $z = -.036$, $p = .986$, $\eta^2 < .01$.

One extreme outlier was removed for LVMO. Mild outliers were retained for LVMO (n = 2). A Mann-Whitney U test was run to determine if there were differences in LVMO %MVC in healthy and stroke on NuStep. Distributions for healthy and stroke were not similar, as assessed by visual inspection. LVMO %MVC for stroke (mean rank = 20.96) was not statistically different as compared to healthy (mean rank = 14.08), $U = 188.500$, $z = 2.022$, $p = .042$, $\eta^2 = 12.39$.

Semitendinosus.

Extreme outliers were removed from mean mEMG right Semitendinosus (RST) dataset (n = 2). Mild outliers were retained (n = 3). A Mann-Whitney U test was run to determine if there were differences in RST %MVC between healthy and stroke on NuStep. Distributions were not similar, as assessed by visual inspection. RST %MVC for stroke (mean rank = 22.77) was statistically higher as compared to healthy (mean rank = 12.21), $U = 205.000$, $z = 3.124$, $p = .001$, $\eta^2 = .31$.

Extreme outliers were removed from mean mEMG left Semitendinosus (LST) dataset (n=2). Mild outliers of the LST were retained (n=2). A Mann-Whitney U test was run to determine if there were differences in LST %MVC in healthy and stroke on NuStep. No statistical difference between stroke (mean rank = 17.86) and healthy (mean rank = 15.44) conditions was detected, $U = 145.000$, $z = .722$, $p = .488$, $\eta^2 = .02$.

Soleus.

Two extreme outliers were removed from the right Soleus (RSOL) data. The mild outlier of the RSOL was retained (n = 1). A Mann-Whitney U test was run to determine if there were differences in RSOL %MVC between healthy and stroke on NuStep. Distributions were not similar, as assessed by visual inspection. RSOL %MVC for stroke (mean rank = 21.57) was statistically higher as to the healthy condition (mean rank = 12.56), $U = 197.000$, $z = 2.697$, $p = .007$, $\eta^2 = .23$.

Left Soleus (LSOL) contained two extreme outliers. These data points were removed. LSOL did not contain any further outliers. LSOL mEMG was normally distributed for each condition, as assessed by Shapiro-Wilk's test ($p > .05$). Variances were homogeneous, as assessed by Levene's test for equality of variances ($p = .284$). An independent t-test revealed a

statistically significant difference in LSOL %MVC between healthy and stroke, $t(30) = -4.307$, $p < .0005$, $d = 1.49$. Stroke LSOL mEMG was 6.847 %MVC, 95% CI [3.60, 10.09 %MVC) higher than the healthy condition.

Gastrocnemius.

The mild outlier of the right medial gastrocnemius (RMG) was retained (n=1). A Mann-Whitney U test was run to determine if there were differences in RMG %MVC between healthy and stroke on NuStep. Distributions of the %MVCs for healthy and stroke were not similar, as assessed by visual inspection. RMG %MVC for stroke (mean rank = 22.93) were statistically and significantly higher as compared to healthy (mean rank = 13.21), $U = 224.000$, $z = 2.827$, $p = .004$, $\eta^2 = .24$.

One extreme outlier was removed from left medial gastrocnemius (LMG). The mild outliers of the LMG were retained (n = 2). A Mann-Whitney U test was run to determine if there were differences in LMG %MVC between healthy and stroke on NuStep. Distributions of the %MVCs for healthy and stroke were not similar, as assessed by visual inspection. LMG %MVC for stroke (mean rank = 22.82) was statistically and significantly higher as compared to healthy (mean rank = 12.71), $U = 214.000$, $z = 2.969$, $p = .002$, $\eta^2 = .27$.

Tibialis anterior

The mild outliers of the right tibialis anterior (RTA) were retained (n = 2). Distributions of the %MVCs for healthy and stroke were not similar, as assessed by visual inspection. A Mann-Whitney U test was run to determine if there were differences in RTA %MVC between healthy and stroke on NuStep. RTA %MVC for stroke (mean rank = 20.83) did not differ to healthy (mean rank = 14.87), $U = 192.500$, $z = 1.734$, $p = .083$.

No outliers were present in the left tibialis anterior (LTA) data. LTA mEMG was normally distributed for each condition, as assessed by Shapiro-Wilk's test ($p > .05$). Variances were homogeneous, as assessed by Levene's test for equality of variances ($p = .448$). Independent t-test revealed no statistically difference in LTA %max between healthy and stroke, $t(32) = -1.668$, $p = .105$.

mEMG treadmill -- betwen conditions.

Statistical difference was set at $p < .025$. When parameter assumptions were met, an independent t-test was utilized. Mean electromyography (mEMG) is reported as a percentage of maximum voluntary contraction (%MVC). mEMG was evaluated for outliers by box plot.

Rectus femoris.

One extreme outlier was removed for RRF. One mild outlier was retained for RRF. A Mann-Whitney U test was run to determine if there were differences in RRF %MVC between healthy and stroke on the TM. Distributions of the %MVCs for healthy and stroke were not similar, as assessed by visual inspection. RRF %MVC for stroke (mean rank = 22.93) were statistically and significantly higher as compared to healthy (mean rank = 12.63), $U = 216.000$, $z = 3.024$, $p = .002$, $\eta^2 = .28$.

Three extreme outliers were removed from the LRF data set. LRF mild outliers were retained (n = 3). A Mann-Whitney U test was run to determine if there were differences in LRF %MVC in healthy and stroke on TM. Distributions for healthy and stroke were not similar, as assessed by visual inspection. LRF %MVC for stroke (mean rank = 18.53) did not differ to healthy (mean rank = 13.62) on TM, $U = 158.000$, $z = 1.502$, $p = .140$, $\eta^2 = .08$.

Vastus medialis oblique.

Mild outliers were retained for RVMO (n = 2). A Mann-Whitney U test was run to determine if there were differences in RMVO %MVC in healthy and stroke on TM. Distributions for healthy and stroke were not similar, as assessed by visual inspection. RVMO %MVC for stroke (mean rank = 21.23) did not differ from healthy (mean rank = 14.55), $U = 198.500$, $z = 1.942$, $p = .051$, $\eta^2 = .24$.

One extreme outlier was removed from LVMO. A mild outlier was retained for LVMO (n = 1). A Mann-Whitney U test was run to determine if there were differences in LVMO %MVC in healthy and stroke on NuStep. Distributions for healthy and stroke were not similar, as assessed by visual inspection. LVMO %MVC for stroke (mean rank = 20.67) were not statistically different as compared to healthy (mean rank = 13.94), $U = 190.000$, $z = 1.989$, $p = .048$, $\eta^2 = .12$.

Semitendinosus.

Seven extreme outliers were removed from the RST data set. Mild outliers were retained (n = 2). A Mann-Whitney U test was run to determine if there were differences in RST %MVC between healthy and stroke on TM. Distributions were not similar, as assessed by visual inspection. RST %MVC for stroke (mean rank = 19.22) were statistically and significantly higher as compared to healthy (mean rank = 11.39), $U = 128.000$, $z = 2.418$, $p = .015$, $\eta^2 = .22$. Four extreme outliers were removed from the LST dataset. One mild outlier of the LST was retained (n = 1). A Mann-Whitney U test was run to determine if there were differences in LST %MVC in healthy and stroke on TM. LST %MVC for stroke (mean rank = 13.83) did not differ from healthy (mean rank = 16.61), $U = 88.000$, $z = -.847$, $p = .415$, $\eta^2 = .06$.

Soleus

Four extreme outliers were removed from the RSOL mEMG data. The mild outliers of the RSOL were retained (n = 3). A Mann-Whitney U test was run to determine if there were differences in RSOL %MVC between healthy and stroke on TM. RSOL %MVC for stroke (mean rank = 18.92) did not differ from healthy (mean rank = 13.22), U = 149.000, z = 1.736, p = .087, η^2 = .10.

LSOL data contained one extreme outlier. LSOL did not contain any mild outliers. LSOL mEMG was normally distributed for each condition, as assessed by Shapiro-Wilk's test (p > .05). Homogeneity of variances was violated, as assessed by Levene's test for equality of variances (p = .020). An independent t-test, with equal variance not assumed revealed no statistical difference in LSOL %MVC between healthy (M = 25.15%, SD = 7.78%) and stroke (M = 31.80%, SD = 12.70%), $t(20.067)$ = -1.735, p = .098.

Gastrocnemius.

The mild outlier of the RMG was retained (n = 1). Two extreme outliers were removed. A Mann-Whitney U test was run to determine if there were differences in RMG %MVC between healthy and stroke on TM. Distributions of the %MVCs for healthy and stroke were not similar, as assessed by visual inspection. RMG %MVC for stroke (mean rank = 19.23) did not differ from healthy (mean rank = 14.63), U = 159.000, z = 1.362, p = .182, η^2 = .06.

One mild outlier of the LMG was retained (n = 1). One extreme outlier was removed. A Mann-Whitney U test was run to determine if there were differences in LMG %MVC between healthy and stroke on TM. Distributions of the %MVCs for healthy and stroke were not similar, as assessed by visual inspection. LMG %MVC for stroke (mean rank = 18.57) did not differ from healthy (mean rank = 15.84), U = 159.000, z = .801, p = .439, η^2 = .02.

Tibialis Anterior.

RTA mEMG did not contain any outliers LSOL data did not contain any outliers. RTA mEMG was normally distributed for each condition, as assessed by Shapiro-Wilk's test ($p > .05$). Homogeneity of variances was violated, as assessed by Levene's test for equality of variances ($p = .001$). A Welch t-test revealed no statistical difference in RTA %MVC between healthy ($M = 29.34\%$, $SD = 12.84\%$) and stroke ($M = 38.06\%$, $SD = 31.31\%$) on TM, $t(17.720) = -1.014$, $p = .324$.

One mild outlier of the LTA was retained (n=1). Two extreme outliers were removed. A Mann-Whitney U test was run to determine if there were differences in LTA %MVC between healthy and stroke on TM. Distributions of the %MVCs for healthy and stroke were not similar, as assessed by visual inspection. LMG %MVC for stroke (mean rank = 13.08) did not differ from healthy (mean rank = 18.84), $U = 79.000$, $z = -1.708$, $p = .092$, $\eta^2 = .09$.

Stroke mEMG within condition.

Statistical difference was set at p<.025. When parameter assumptions were met, a paired t-test was utilized. Muscle difference (i.e., Muscle TM – Muscle NS) was calculated and evaluated for outliers. Mean electromyography (mEMG) is reported as a percentage of maximum voluntary contraction (%MVC). mEMG was evaluated for outliers by box plot.

Rectus femoris.

One extreme outlier was removed from RRF. RRF contained one mild outlier. A Wilcoxon Signed Rank test was utilized to test for a difference between exercise modes in the stroke participants. Of the 15 participants recruited to the study, the TM (*Mdn* = 19.8 %) elicited a higher mEMG in 12 participants compared to the NuStep (*Mdn* = 13.00 %). The TM elicited a statistically significant median increase in mEMG, $z = -2.840$, $p = .005$, $r = -0.52$.

One extreme outlier was removed from LRF. LRF contained no outliers in the data, as assessed by inspection of a boxplot. The difference scores were not distributed normally, as assessed by Shapiro-Wilk's test ($p = .042$). A Wilcoxon signed-rank test determined that there was no statistical median difference between TM ($Mdn = 25.30$ %) and NuStep ($Mdn = 27.50$ %), $z = .795$, $p = .427$, $r = .15$.

Vastus medialis oblique.

One extreme outlier was removed from RVMO. RVMO difference contained no outliers in the data. The difference scores were normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). Paired t-test was utilized. TM produced higher mEMG in RVMO ($M = 30.30\%$, $SD = 25.76\%$) as opposed to the NuStep ($M = 19.92\%$, $SD = 17.40\%$). TM elicited a mean increase of 12.98 %, 95% CI [3.418, 22.538] in the 5 minute exercise protocol at the RPE based SS pace, $t(14) = 2.912$, $p = .011$, $d = 1.12$.

LVMO difference contained no outliers in the data, as assessed by inspection of a boxplot. The difference scores were normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). A paired t-test demonstrated no statistical difference in LVMO between TM ($M = 27.46$ %, $SD = 15.95\%$) and NS ($M = 24.26\%$, $SD = 15.16\%$) in the stroke participants, $t(14) = .764$, $p = .458$, $d = .20$.

Semitendinosus.

An extreme outlier was removed from the RST data set. RST difference contained no further outliers. The difference scores were normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). There was no statistical difference in RST between TM ($M = 60.88\%$, $SD = 75.19\%$) and NS ($M = 67.05\%$, $SD = 167.58\%$) in the stroke participants, $t(14) = -.264$, $p = .813$, $d = -.06$.

An extreme outlier was removed from the LST dataset. One mild outlier was retained. The distribution of differences was not symmetrically shaped. Therefore, an exact sign test was used to compare the differences in %MVC between the two exercise modes. The TM (*Mdn* = 20.30%) elicited a statistically significant 7.155% *Mdn* increase compared to the NuStep (*Mdn* = 6.65%), *p* = .007, *r* = -.46.

Gastrocnemius.

Extreme outliers were removed from the RMG data set (n = 2). As a result, there were no further outliers in the data. The RMG difference scores were normally distributed, as assessed by Shapiro-Wilk's test (*p* = .558). No statistical differences existed between TM (*M* = 101.07%, *SD* = 107.36%) and NuStep (*M* = 60.62%, *SD* = 167.58%) for RMG, *t*(14) = 2.775, *p* = .043, *d* = .57.

One extreme outlier was removed from the LMG data set. The LMG difference score were normally distributed, as assessed by Shapiro-Wilk's test (*p* > .05). No statistical differences existed between TM (*M* = 102.07%, *SD* = 215.13%) and NuStep (*M* = 33.71%, *SD* = 167.58%) for LMG in the stroke population, *t*(14) = 1.463, *p* = .166, *d* = .37.

Soleus.

Two extreme outliers were removed from the RSOL data set. The distribution of differences was not symmetrically shaped. An exact sign test was used to compare the median differences in RSOL %MVC between the two exercise modes. The TM (*Mdn* = 44.50%) produced higher RSOL mEMG as compared to the NuStep (*Mdn* = 31.90 %), *p* = .001, *r* = -.57.

One extreme outlier was removed from the LSOL data set. The LSOL difference score were normally distributed, as assessed by Shapiro-Wilk's test (*p* > .05). No statistical differences

existed between TM (*M* = 101.07%, *SD* = 107.36%) and NuStep (*M* = 60.62%, *SD* = 167.58%) for RMG, *t*(14) = 2.775, *p* = .043, *d* = .57.

Tibialis anterior.

One mild RTA difference outlier was retained. The distribution of differences was not symmetrically shaped. An exact sign test was used to compare the differences in %MVC between the two exercise modes. There was no statistical difference between the TM (*Mdn* = 23.50%) and NuStep (*Mdn* = 19.20%) in stroke participants, *p* = .035, *r* = -.46.

LTA difference contained no outliers. The LTA difference score were normally distributed, as assessed by Shapiro-Wilk's test (*p* >.05). LTA between was higher in TM (*M* = 26.51%, *SD* = 10.27%) as compared to the NuStep (*M* = 18.31%, *SD* = 167.58%), *t*(14) = 2.775, *p* = .015, *d* = .72.

Healthy mEMG within condition.

Statistical difference was set at p < .025. When parameter assumptions were met, a paired t-test was utilized. Muscle differences (Δ%MVC) (i.e., Muscle TM – Muscle NS) were calculated and evaluated for outliers.

Rectus femoris.

RRF difference score did not contain any outliers. The difference scores for RRF were normally distributed, as assessed by Shapiro-Wilk's test (*p* >.05). TM produced higher mEMG in RRF (*M* = 9.67 %MVC, *SD* = 5.67) as opposed to the NS (*M* = 6.25%, *SD* = 3.86%). TM elicited a mean increase of 3.34%, 95% CI [1.557%, 5.309%] in the 5 minute exercise protocol at the RPE based SS pace, *t*(18) = 3.845, *p* = .001, *d* = .88.

Multiple extreme outliers were removed from the LRF dataset (n = 4). LRF contained one mild outlier as assessed by inspection of a boxplot. The distribution of differences was

symmetrically shaped. A Wilcoxon signed-rank test determined that there was no statistical difference in mEMG medians between TM (*Mdn* = 13.200%) and the NuStep (*Mdn* = 6.91%) in healthy subjects, z = -1.891, p = .059, r = - .46.

Vastus medialis oblique.

RVMO difference contained no outliers in the data. The difference scores were normally distributed, as assessed by Shapiro-Wilk's test (p = .971). There was no statistical difference in RVMO between the TM (*M* = 16.04%, *SD* = 6.24%) and the NuStep (*M* = 14.03 %, *SD* = 6.11%), t(18) = 1.608, p = .125, d = .37.

LVMO difference contained one mild outlier in the data, as assessed by inspection of a boxplot. The distribution of differences was symmetrically shaped. A Wilcoxon signed-rank test determined that there was no statistical difference in LVMO medians between TM (*Mdn* = 17.10 %) and the NS (Mdn = 15.60%) in healthy subjects, z = -1.730, p = .084. r = - .41.

Semitendinosus.

Two extreme outliers were removed from the RST data set. RST difference contained no further outliers in the data. The difference scores were normally distributed, as assessed by Shapiro-Wilk's test (p > .05). There was no statistical difference in RST between TM (*M* = 33.39%, *SD* = 68.68%) and NS (*M* = 7.16%, *SD* = 3.71%) in the healthy participants, t(18) = 1.670, p = .112, d = 38.

Extreme outliers were removed from the LST data set (n = 3). LST difference contained no further outliers in the data. The difference scores were normally distributed, as assessed by Shapiro-Wilk's test (p > .05). TM produced higher mEMG in LST (*M* = 20.60%, *SD* = 10.44%) as opposed to the NS (*M* = 10.28%, *SD* = 14.27%). TM elicited a mean increase of 10.32%, 95%

CI [1.952, 18.684] in the 5 minute exercise protocol at the RPE based SS pace, $t(17) = 2.602$, $p = .019$, $d = .61$.

Medial gastrocnemius.

RMG difference did not contain any extreme outliers. As a result, there were no further outliers in the data. The RMG difference scores were normally distributed, as assessed by Shapiro-Wilk's test ($p = .098$). TM produced higher mEMG in RMG ($M = 40.33\%$, $SD = 21.85\%$) as opposed to the NuStep ($M = 16.98\%$, $SD = 13.38\%$). TM elicited a mean increase of 23.35%, 95% CI [15.603%, 31.102%] in the 5 minute exercise protocol at the RPE based SS pace, $t(18) = 6.331$, $p < .0005$, $d = 1.45$.

LMG difference did not contain any extreme outliers. The LMG difference score were normally distributed, as assessed by Shapiro-Wilk's test ($p = .533$). TM produced higher mEMG in LMG ($M = 34.93\%$, $SD = 15.26\%$) as opposed to the NuStep ($M = 13.02\%$, $SD = 9.48\%$). TM elicited a mean increase of 21.91%, 95% CI [16.381%, 27.433%] in the 5 minute exercise protocol at the RPE based SS pace, $t(18) = 8.329$, $p < .0005$, $d = 1.91$.

Soleus.

One extreme outlier was removed from the RSOL data set. RSOL difference score did not contain any further outliers. The RSOL difference score were normally distributed, as assessed by Shapiro-Wilk's test ($p = .629$). TM produced higher mEMG in RSOL (M = 37.41%, SD = 31.82%) as opposed to the NuStep (M = 9.76%, SD = 6.88%). TM elicited a mean increase of 27.64%, 95% CI [12.089%, 43.194%] in the 5 minute exercise protocol at the RPE based SS pace, $t(18) = 3.734$, $p = .002$, $d = .86$.

The LSOL difference score did not contain any outliers and was normally distributed, $p = .596$. TM produced higher mEMG in LSOL ($M = 25.15\%$, SD = 7.78%) as opposed to the

NuStep (M = 5.79%, SD = 3.57%). TM elicited a mean increase of 19.35%, 95% CI [16.117%, 22.600%] in the 5 minute exercise protocol at the RPE based SS pace, $t(18)$ = 12.563, p < .0005, d = 2.88.

Tibialis anterior.

One extreme RTA outlier was removed from the dataset. RTA difference score contained one mild outlier. The difference scores were symmetrically distributed, as assessed by a histogram. A Wilcoxon signed-rank test determined that there was a statistically significant increase in mEMG (Mdn = 13.19%) when subjects walked on the TM (Mdn = 28.60%) compared to the recumbent cross trainer (Mdn = 12.80%), z = -3.783, p < .0005, r = -.72.

LTA difference contained no extreme outliers. However, the set contained one mild outlier. The difference scores were symmetrically distributed, as assessed by a histogram. A Wilcoxon signed-rank test determined that there was a statistically significant increase in mEMG (Mdn = 15.67%) when healthy subjects walked on the TM (Mdn = 28.70%) compared to the NS (Mdn = 11.500%), z = -3.300, p = .001, r = -.62.

CVA Condition – affected side vs. non-affected side.

Unilateral stroke participants (n=14) were divided into left affected size (n = 6) and right affected side (n = 8). One participant was removed from consideration due to the global nature of their CVA. Alpha was set at $p \leq .0167$.

Affected side.

Three extreme outliers were removed from the NS affected RF data. Data did not contain any mild outliers. However, NS affected RF data violated normality, as assessed by Shapiro-Wilk's test (p < .001). The difference scores were symmetrically distributed, as assessed by a histogram. A Wilcoxon signed-rank test determined that there was a statistically significant

increase in mEMG on the TM (*Mdn* = 19.80%) compared to the NS (*Mdn* = 13.00%), *z* = -2.701, *p* = .007, *r* = -.60.

Affected VMO did not contain any outliers. VMO difference scores were normally distributed, as assessed by Shapiro-Wilk's test (*p* = .200). TM produced higher mEMG in affected VMO (*M* = 30.23 %MVC, *SD* = 19.67) as opposed to the NuStep (*M* = 19.20 %MVC, *SD* = 6.88). TM elicited a mean increase of 11.03 %MVC, 95% CI [4.58, 17.88] in the 5 minute exercise protocol at the RPE based SS pace, *t*(13) = 3.691, *p* = .003, *d* = .59.

Affected ST contained two extreme outliers that were removed. One mild outlier was retained. The difference scores were symmetrically distributed, as assessed by a histogram. A Wilcoxon signed-rank test determined that there was no statistically significant difference in affected ST mEMG on the TM (*Mdn* = 30.80 %) compared to the NS (*Mdn* = 26.45 %), *z* = -1.647, *p* = .099, *r* = -.34.

Affected TA data contained one mild outlier. The difference scores were symmetrically distributed, as assessed by a histogram. A Wilcoxon signed-rank test determined that there was a statistical increase of affected TA mEMG on the TM (*Mdn* = 25.35%) compared to the NS (*Mdn* = 17.55 %), *z* = -2.417, *p* = .016, *r* = -.46.

Affected MG data contained three extreme outliers which were removed. Difference scores were normally distributed, as assessed by Shapiro-Wilk's test (*p* = .20). TM produced higher mEMG in affected MG (*M* = 63.84 %, *SD* = 43.75%) as opposed to the NuStep (*M* = 42.91%, *SD* = 39.66%). TM elicited a mean increase of 20.92%, 95% CI [5.72, 36.13] in the 5 minute exercise protocol at the RPE based SS pace, *t*(10) = 3.066, *p* = .012, *d* = .50.

Affected SOL data contained four extreme outliers which were removed. One mild outlier was retained. A Wilcoxon signed-rank test determined that there was a statistical increase

of affected SOL mEMG on the TM (*Mdn* = 44.45%) compared to the NS (*Mdn* = 16.25%), z = -2.803, p = .005, r = -.63.

Non-affected side.

Non affected RF Data contained three extreme outliers. RF data also contained 1 mild outlier. The difference scores were not symmetrically distributed, as assessed by a histogram. However, the distribution of differences was not symmetrically shaped. Therefore, A sign test determined that there was no statistical difference in non-affected RF mEMG between TM (*Mdn* = 25.90%) and the NS (*Mdn* = 20.65%) in CVA subjects, z = .000, p = 1.000.

Non affected VMO data contained one mild outlier. The difference scores were symmetrically distributed, as assessed by a histogram. A Wilcoxon signed-rank test determined that there was no statistically significant difference in mEMG on the TM (*Mdn* = 19.70%) compared to the NS (*Mdn* = 20.30%), z = -.175, p = .861, r = -.03.

Non-affected ST contained one extreme outlier that was removed. One mild outlier was retained. Wilcoxon signed-rank test determined that there was a statistically significant increase in non-affected ST mEMG on the TM (*Mdn* = 23.55 %) compared to the NS (*Mdn* = 10.45 %), z = -3.059, p = .002, r = -.62.

One extreme outlier was removed from non-affected TA data. Non-affected TA difference scores were normally distributed, as assessed by Shapiro-Wilk's test (p = .136). TM produced higher mEMG in non-affected TA (M = 26.65 %, SD = 11.25) as opposed to the NuStep (M = 18.48 %, SD = 9.53). TM elicited a mean increase of 8.17 %, 95% CI [3.02, 13.32] in the 5 minute exercise protocol at the RPE based SS pace, $t(12)$ = 3.455, p = .005, d = .78.

Non-affected MG contained two extreme outliers which were removed. Difference scores were normally distributed, as assessed by Shapiro-Wilk's test (p = .200). TM did not produce a

statistically different mEMG in non-affected MG (M = 35.25 %, SD = 21.32) as opposed to the NuStep (M = 27.96 %, SD = 20.09), $t(11)$ = 2.368, p = .037, d = .35.

Non-affected SOL data contained one extreme outlier which was removed. Two mild outliers were retained. A Wilcoxon signed-rank test determined that there was a statistical increase of non-affected SOL mEMG on the TM (Mdn = 24.30 %) compared to the NS (Mdn = 11.20 %), z = -3.040, p = .002, r = -.60.

Wireless Gait Assessment (WiGAT) - right vs. left.

Left stride length.

Six extreme outliers were moved from the raw data. Mild outliers were kept in the L stride length data (n = 3). L stride length was normally distributed in both conditions, as assessed by Shapiro-Wilk's test (p > .05). Mauchly's test of sphericity indicated that the assumption of sphericity was violated, $\chi^2(2)$ = 10.982, p = .004. Epsilon (ε) was 0.755, as calculated according to Greenhouse & Geisser (1959), and was used to correct the one-way repeated measures ANOVA. L stride length was statistically significantly different for treatment time during this exercise intervention, $F(1.510, 43.792)$ = 3.719, p =.044, partial η^2 = .114 (Figure 5). The interaction between exercise mode and condition was not statistically significant, $F(1.510, 43.792)$ = .646, p =0.484, partial η^2 = .022. Data are mean ± standard deviation unless otherwise stated. Pairwise comparisons were adjusted for multiple comparisons with the Bonferroni correction. There was an increase in L stride length from 1.25 ± 0.25 m at baseline to 1.28 ± 0.27 m post-TM, an increase of 0.33m, 95% CI [0.04m to .063], which was statistically significant, p = .023. There was an increase in L stride length from 1.25m ± 0.25m at baseline to 1.29m ± 0.27m post-NS, an increase of 0.047m, 95% CI [-0.04m to .098m], which was not

statistically significant, $p = .078$. Post TM (1.28m ± 0.27m) and post NS (1.29m ± 0.27m) did not differ in L stride length, $p = 1.00$ (See Figure 6).
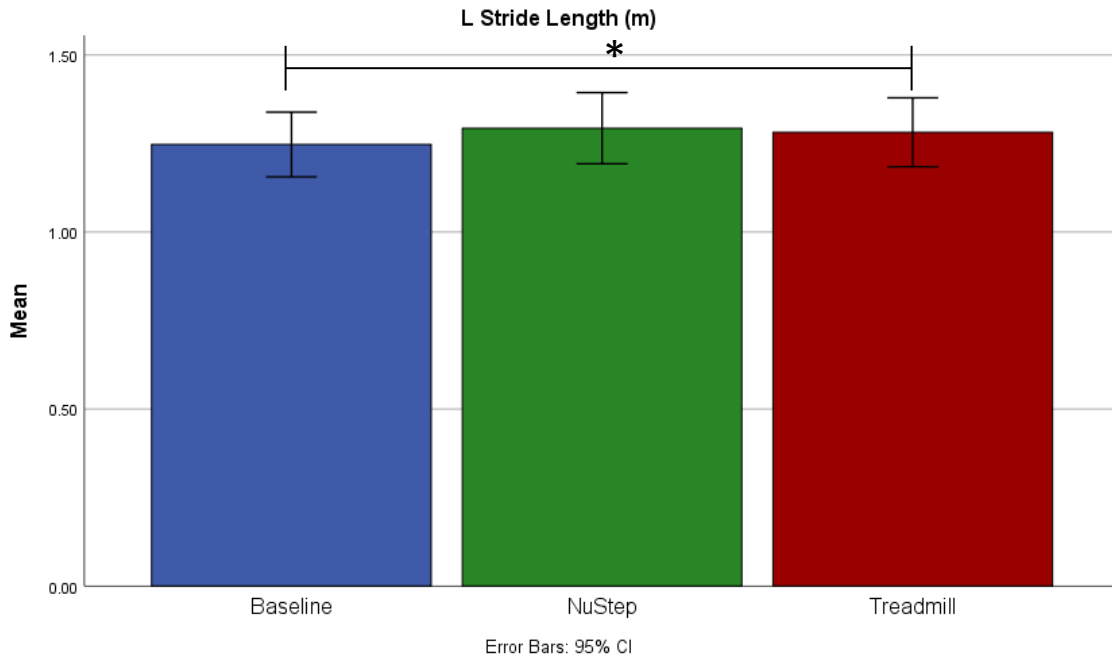

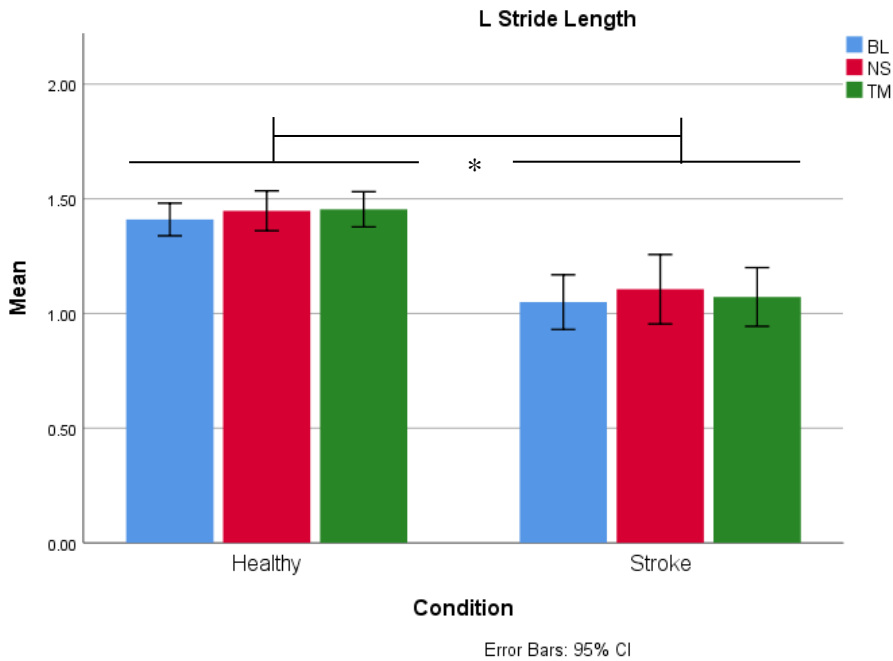
Figure 5: Left stride length (m) by treatment.

Figure 6: WiGAT Left (L) stride length post-exercise mode between conditions compared to baseline.

L stride length between conditions was statistically different, $F(1, 29) = 29.82$, $p < .0005$, partial $\eta^2 = .507$. Post hoc pairwise comparison demonstrated decreased L stride length in stroke participants. This 0.362m decrement, 95% CI [0.226m to .497m] was statistically significant, $p < .0005$ (Figure 6).

R stride length.

Outliers were kept in the R stride length data. R stride length was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). Mauchly's test of sphericity indicated that the assumption of sphericity had been violated, $\chi 2(2) = 17.086$, $p < .0005$. Epsilon (ε) was 0.686, as calculated according to Greenhouse & Geisser (1959), and was used to correct the one-way repeated measures ANOVA. The exercise treatment did not lead to any statistically significant changes in R stride length, $F(1.373, 39.814) = 2.734$, $p = .095$, $\eta^2 = .086$.
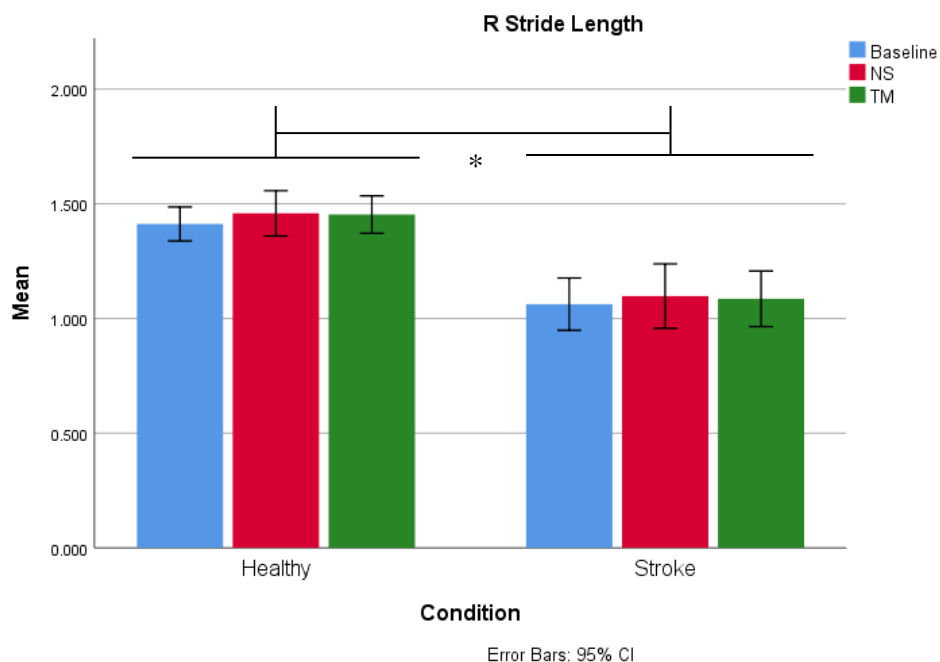

Figure 7: WiGAT Right (R) Stride Length post-exercise mode between conditions compared to baseline.

The interaction between exercise mode and condition (i.e. stroke vs. healthy) was not statistically significant, $F(1.373, 39.814) = .116$, $p = .813$, partial $\eta^2 = .004$. R stride length between conditions was statistically different, $F(1, 29) = 28.813$, $p < .0005$, partial $\eta^2 = .498$. Post hoc pairwise comparison demonstrated decreased R stride length in stroke participants. This 0.359m decrement, 95% CI (0.222m to .496m) was statistically significant, $p < .0005$ (Figure 7).

Walking speed.

One mild outlier was retained in the R stride length data. Gait speed for each condition and exercise mode was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). Mauchly's test of sphericity indicated that the assumption of sphericity was not violated, $\chi^2(2) = 1.642$, $p = .440$. The exercise treatments lead to statistically significant changes in gait speed, $F(2, 64) = 3.157$, $p = .049$, $\eta^2 = .049$. However, the interaction between treatment and condition was not statistically significant, $F(2,64) = 1.083$, $p = .342$, partial $\eta^2 = .033$. Data are mean ± standard deviation unless otherwise stated. Post hoc pairwise comparison demonstrated that baseline gait speed (1.26 ± 0.35 mps), TM (1.30 ± 0.38 mps) and NS (1.30 ± 0.36 mps) did not differ, $p > 0.05$.

Gait speed between conditions were statistically different, $F(1, 32) = 33.769$, $p < .0005$, partial $\eta^2 = .513$. Post hoc pairwise comparison demonstrated enhanced gait speed (m/sec) in healthy participants. This 0.505 m/sec increase, 95% CI [0.328m/sec to 0.682m/sec] was statistically significant, $p < .0005$ (See Figure 8).

Double support time.

One mild outlier was retained in the R stride length data. Double support time (DST) was normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$). Mauchly's test of sphericity indicated that the assumption of sphericity was not violated, $\chi^2(2) = 1.813$, $p = .404$. The exercise

treatment did not lead to any statistically significant changes in double support time, $F(2, 56) = 2.421$, $p = .098$, $\eta^2 = .080$.
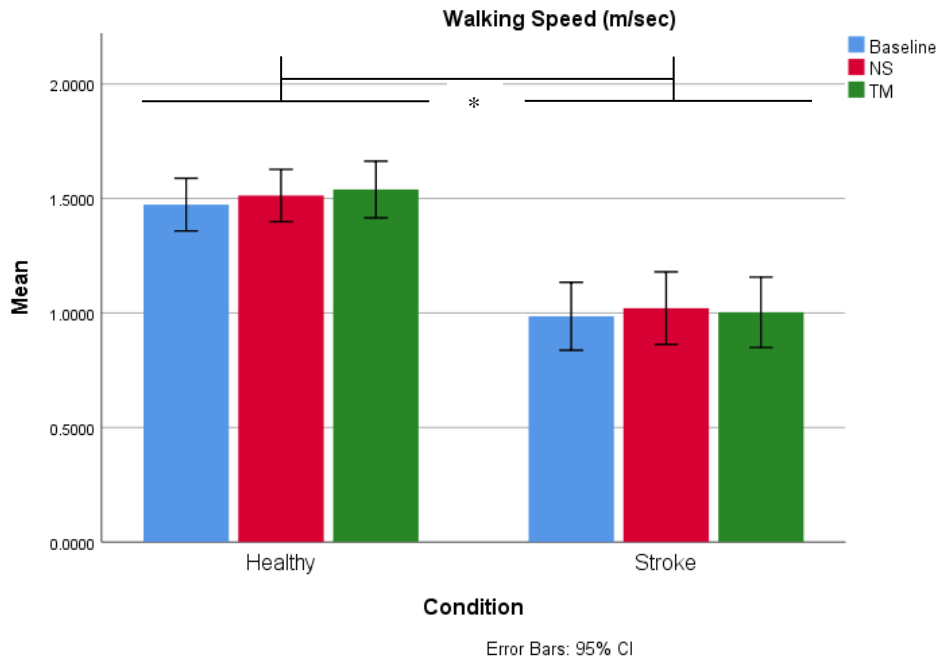

Figure 8: WiGAT Walking Speed post-exercise mode between conditions as compared to baseline.
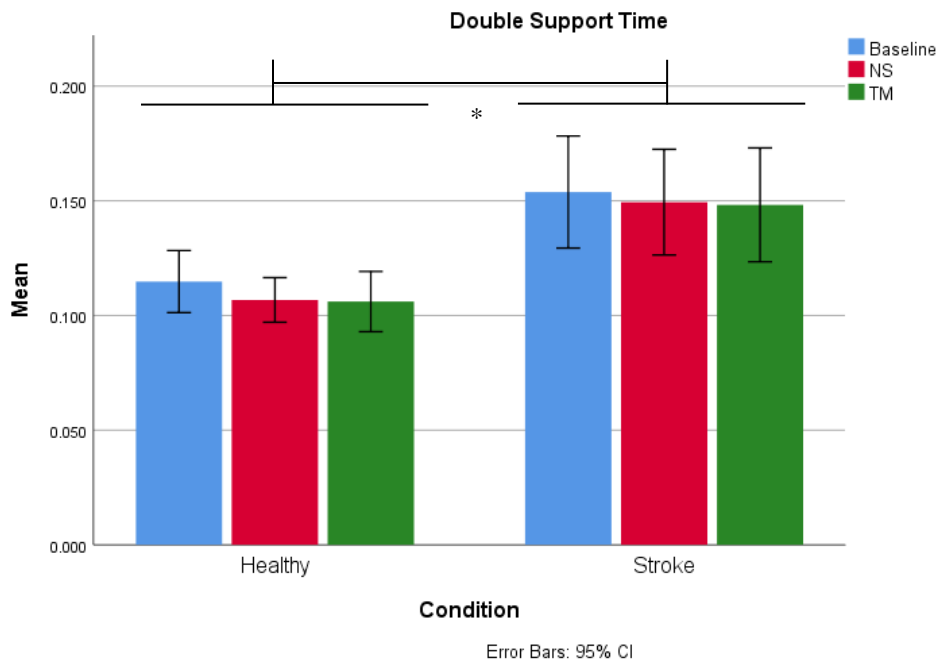

Figure 9: WiGAT Double Support Time (DST) post-exercise mode between conditions as compared to baseline.

The interaction between exercise mode and condition (i.e. stroke vs. healthy) was also not statistically significant, $F(2,56) = .155$, $p = .857$, partial $\eta^2 = .005$. However, F test demonstrated increased DST in stroke participants, $F(1,28) = 13.130$, $p = .001$, partial $\eta^2 = .319$. This 0.041s increase, 95% CI [0.018s to .065s] was statistically significant, $p = .001$ (See Figure 9).

Asymmetry index.

Extreme outliers were removed from asymmetry index (AI) data set. Mild outliers were retained during analysis.

	Condition	Mean	Standard Deviation	N
Baseline	Healthy	-7.14	19.89	11
	Stroke	47.25	51.35	14
	Total	**23.32**	**48.50**	**25**
NuStep	Healthy	-1.17	8.19	11
	Stroke	46.39	56.55	14
	Total	**25.47**	**48.38**	**25**
Treadmill	Healthy	-7.94	16.23	11
	Stroke	54.45	52.55	14
	Total	**27.00**	**51.03**	**25**

Table 10: Asymmetry Index post exercise mode across conditions as compared to baseline; positive = right > left, negative = left > right.

AI violated normal distribution, as assessed by Shapiro-Wilk's test ($p > .05$). Mauchly's test of sphericity indicated that the assumption of sphericity was not violated, $\chi^2(2) = 4.587$, $p = .101$. The exercise treatment did not lead to any statistically significant changes in AI, $F(2, 46) = .427$, $p = .655$, $\eta^2 = .018$ (Table 10). The interaction between exercise mode and condition was also not statistically significant, $F(2,56) = 2.056$, $p = .148$, partial $\eta^2 = .082$. AI between conditions was statistically different, $F(1, 23) = 11.449$, $p = .003$, partial $\eta^2 = .332$. Post hoc pairwise comparison demonstrated increased AI in stroke participants. This 54.779 increase (95% CI, 21.288 to 88.269) was statistically significant, $p = .003$ (Figure 10).
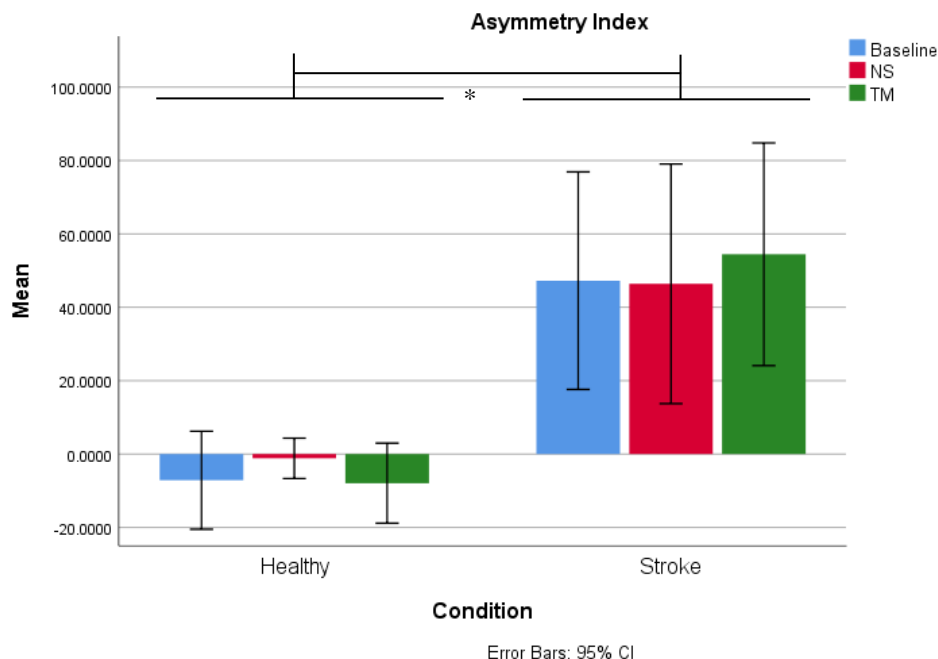
Figure 10: WiGAT: Asymmetry Index: post exercise mode between conditions as compared to baseline; positive = right > left, negative = left > right.

Left stance percentage (%).

Extreme outliers were removed (n=3). There were mild outliers in the data (n=3), as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. L stance % was normally distributed at each time point, as assessed by Shapiro-Wilk's test ($p > .05$). Mauchly's test of sphericity indicated that the assumption of sphericity had not been violated, $\chi^2(2) = .980$, $p = .756$. The exercise treatments did not lead to statistically significant changes in L stance %, $F(2, 56) = .852$, $p = .852$, $\eta^2 = .030$. The interaction between treatment and condition was statistically significant, $F(2,56) = 5.054$, $p = .010$, partial $\eta^2 = .153$. L stance % between conditions were statistically different, $F(1, 28) = 19.70$, p < .0005, partial $\eta^2 = .413$. Pairwise comparison demonstrated enhanced stance % in stroke participants. This 5.04% increase, 95% CI [2.705% to 7.343%] was statistically significant, p < .0005.

Right stance percentage (%).

There were mild outliers in the data (n=2), as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. R stance % was normally distributed at each time point, as assessed by Shapiro-Wilk's test ($p > .05$). Mauchly's test of sphericity indicated that the assumption of sphericity had not been violated, $\chi^2(2) = 4.197$, p = .123. The exercise treatments did not lead to statistically significant changes in R stance %, $F(2, 60) = 2.164$, $p = .124$, $\eta^2 = .067$. The interaction between treatment and condition was not statistically different, $F(2,60) = .231$, p =.794, partial $\eta^2 = .008$. Furthermore, F test for the effect of condition demonstrated no statistical difference between healthy and stroke, $F(1, 30) = .898$, $p = .351$, partial $\eta^2 = .029$.

Left swing percentage (%).

Extreme outliers were removed (n=3). There were mild outliers in the data (n=5), as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. L swing % was normally distributed at each time point, as assessed by Shapiro-Wilk's test ($p > .05$). Mauchly's test of sphericity indicated that the assumption of sphericity was violated, $\chi^2(2) = 7.192$, p = .027. The exercise treatments did not lead to statistically significant changes in L swing %, $F(2, 54) = .552$, p = .579, η2 = .020. The interaction between treatment and condition was not statistically significant, $F(2,54) = 1.171$, p =.311, partial η2 = .042. F test for the effect of condition demonstrated statistical difference between healthy and stroke, $F(1, 27) = 20.076$, $p < .0005$, partial $\eta^2 = .426$. Post hoc pairwise comparison demonstrated enhanced L swing % in healthy participants. This 5.258 % increase, 95% CI [2.850% to 7.666%] was statistically significant, $p < .0005$.

Right swing percentage (%).

There was one mild outlier in the data (n=1), as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. R swing % was normally distributed at each time point, as assessed by Shapiro-Wilk's test ($p > .05$). Mauchly's test of sphericity indicated that the assumption of sphericity had not been violated, $\chi^2(2) = 2.614$, $p = .271$. The exercise treatments did not lead to statistically significant changes in R stance %, $F(2, 60) = 1.991$, $p = .145$, $\eta^2 = .062$. The interaction between treatment and condition was not statistically significant, $F(2,60) = .231$, $p = .935$, partial $\eta^2 = .002$. F test for the effect of condition demonstrated no statistical difference between healthy and stroke, $F(1, 30) = 1.271$, $p = .269$, partial $\eta^2 = .041$.

Affected side stance (%).

There were no outliers in the data, as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box.
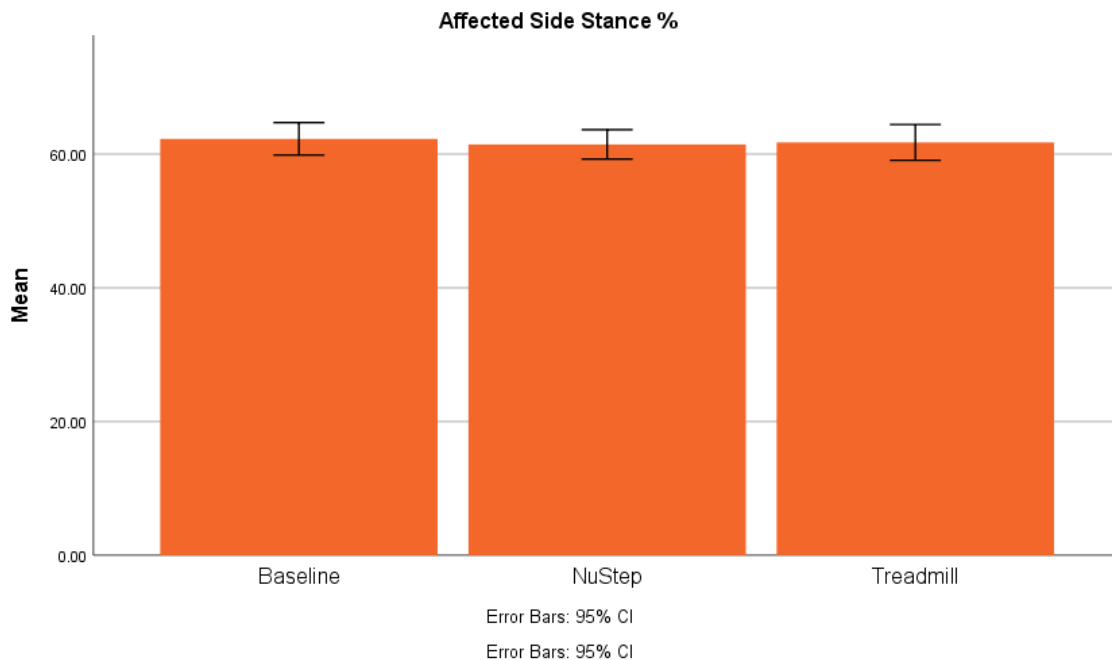

Figure 11: Affected Side Stance % Mean Data.

Affected side stance % was normally distributed at each time point (i.e. baseline, post NuStep, post treadmill), as assessed by Shapiro-Wilk's test ($p > .05$). Mauchly's test of sphericity indicated that the assumption of sphericity had not been violated, $\chi^2(2) = .124$, $p = .940$. The exercise intervention did not lead to any statistically significant changes in affected side stance %, $F(2, 26) = 1.032$, $p = .370$, $\eta^2 = .074$ (Figure 11).

Non-affected side stance (%).

An extreme outlier was removed from the data, as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box.

Figure 12: Non-Affected Side Stance % Median Data. * $p < .05$.

One mild outlier was retained. Non-affected side stance % was statistically significantly different at the different time points during the exercise intervention, $\chi^2(2) = 6.500$, $p = .039$. Pairwise comparisons were performed with a Bonferroni correction for multiple comparisons. Non-affected side stance % was statistically significantly different between baseline ($Mdn = 67.89$)

and NuStep (*Mdn* = 67.58), *p* = .043. Treadmill (*Mdn* = 67.89) did not differ from baseline, *p* = 1.00. Treadmill did not statistically differ from NuStep, *p* = .199 (Figure 12).

Affected side swing (%).

There were no outliers in the data, as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. Affected side stance % was normally distributed at each time point (i.e. baseline, post NuStep, post treadmill), as assessed by Shapiro-Wilk's test (*p* > .05). Mauchly's test of sphericity indicated that the assumption of sphericity had not been violated, $\chi^2(2)$ = .124, *p* = .940. The exercise intervention did not lead to any statistically significant changes in affected side swing %, $F(2, 26)$ = 1.032, *p* = .370, η^2= .074 (Figure 13).

Figure 13: Affected Side Swing % Mean Data.

Non-affected side swing (%).

An extreme outlier was removed from the data, as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. A mild outlier were retained.
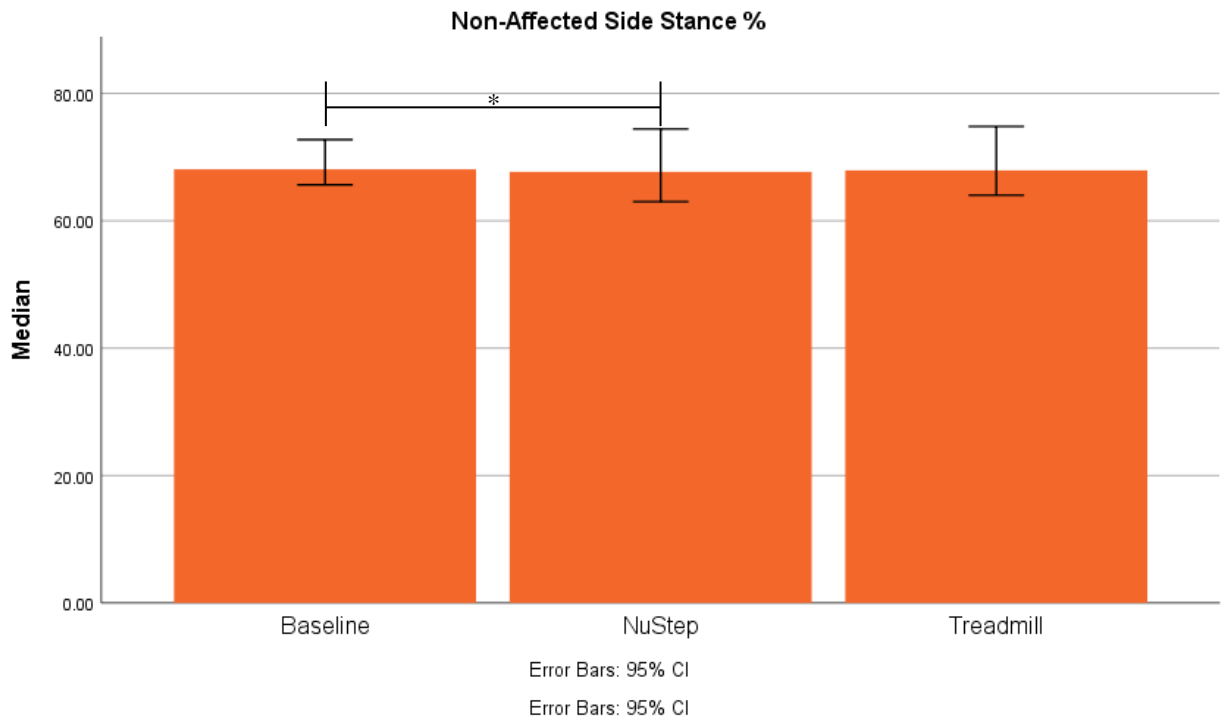
Non-affected side swing % was statistically significantly different at the different time points during the exercise intervention, $\chi^2(2) = 8.000$, $p = .018$. Pairwise comparisons were performed with a Bonferroni correction for multiple comparisons. Non-affected side swing % was statistically significantly different between baseline (*Mdn* = 32.11) and NuStep (*Mdn* = 32.42), $p = .043$. Treadmill (*Mdn* = 32.11) did not differ from baseline, $p = 1.00$. Treadmill was statistically different from NuStep, $p = .043$ (Figure 14).
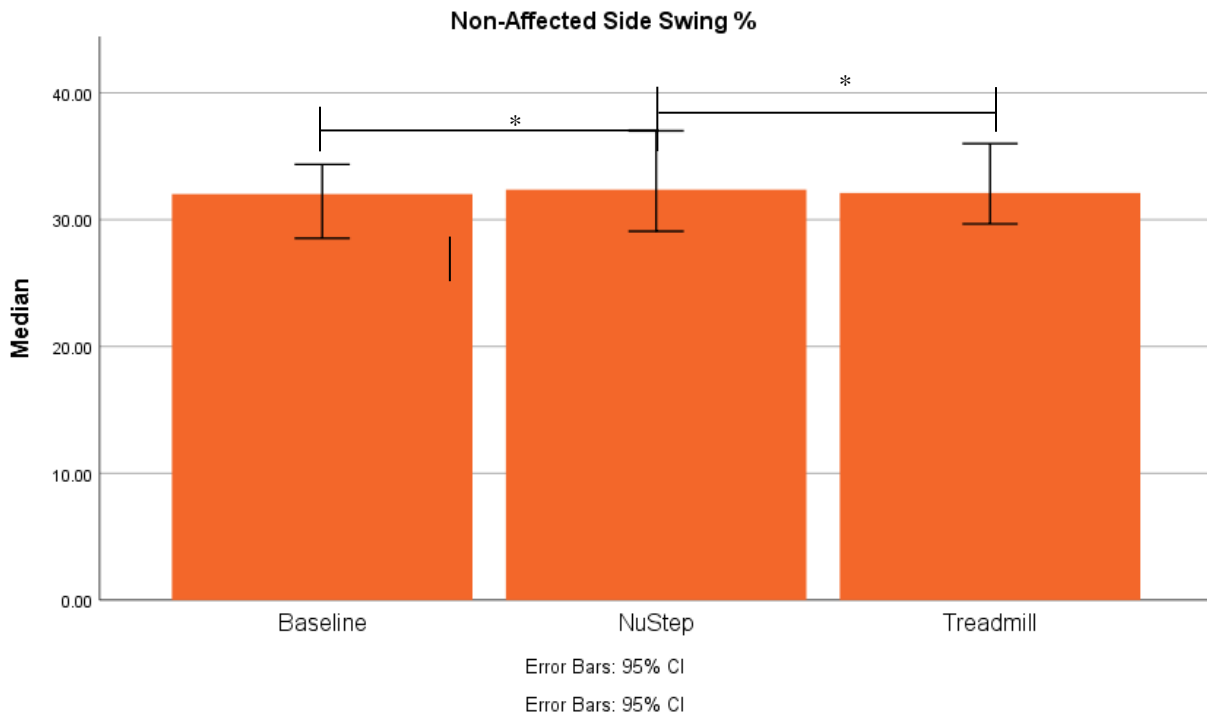
Figure 14: Non-Affected Side Swing % Median Data. * $p < .05$.

Affected side stride length (m).

There were no outliers in the data, as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. Affected side stride length was normally distributed at all time points, as assessed by Shapiro-Wilk's test ($p < .05$). Mauchly's test of sphericity indicated that the assumption of sphericity had been violated, $\chi^2(2) = 7.284$, $p = .026$.
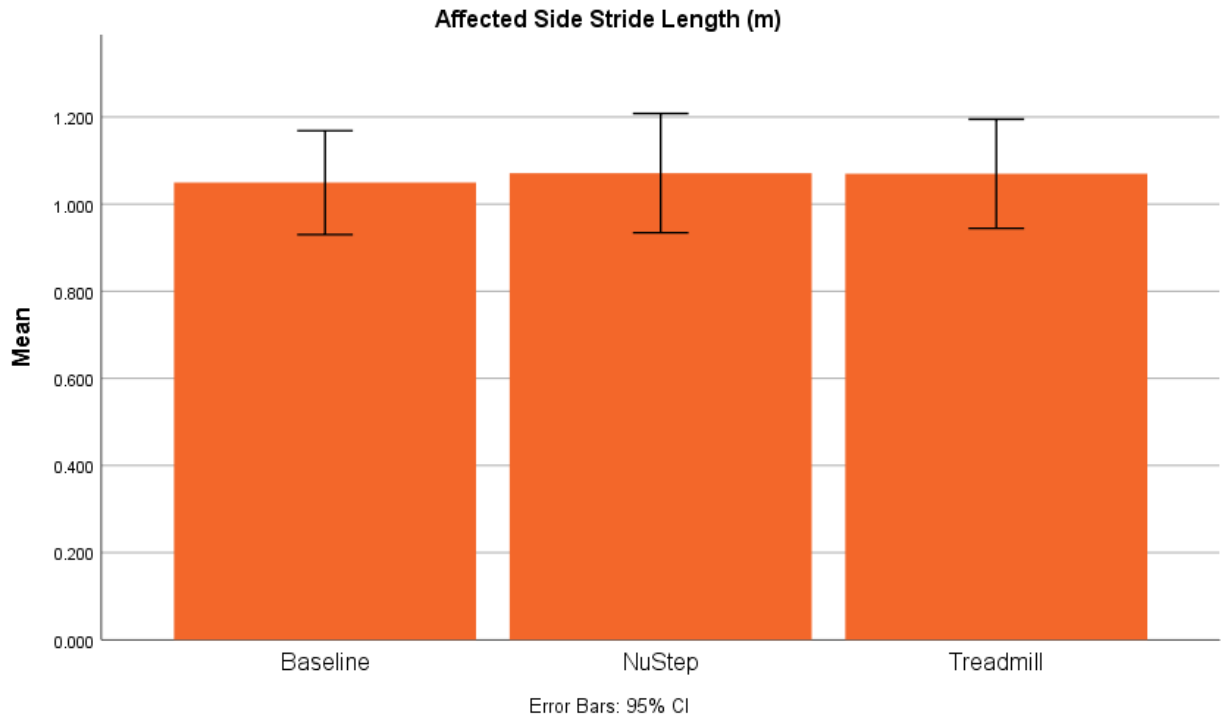
Figure 15: Affected Side Stride Length (m) Mean Data.
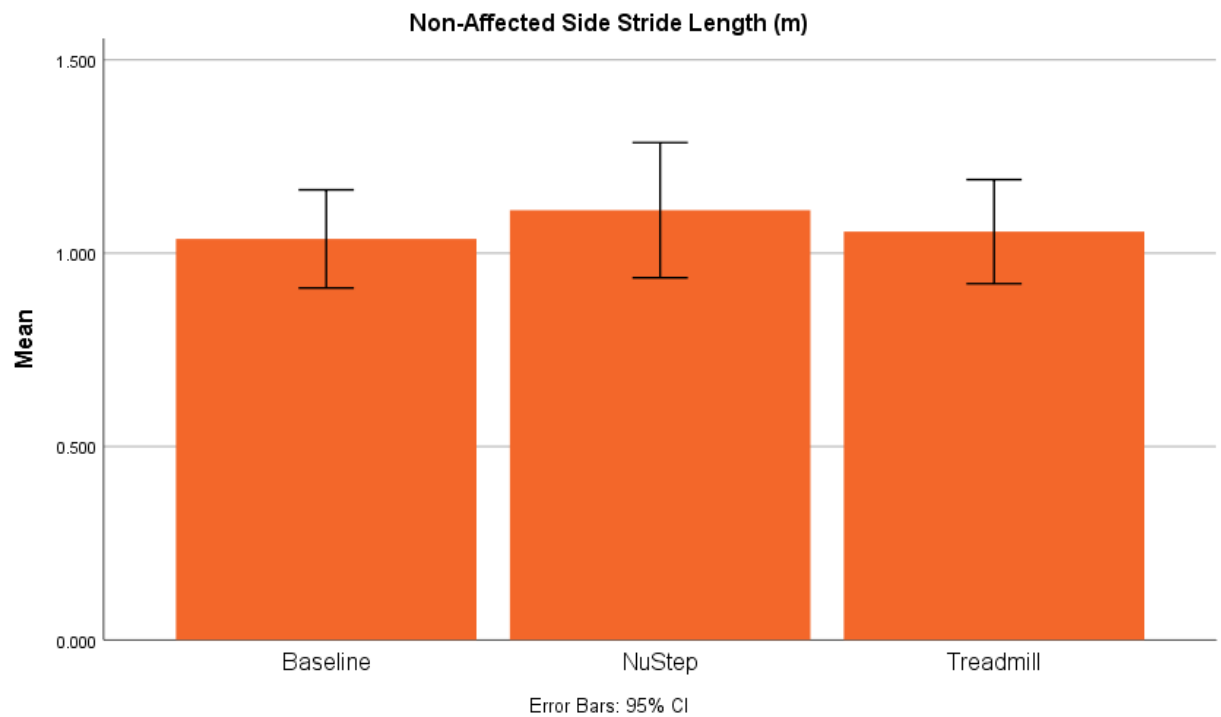
Figure 16: Non-Affected Side Stride Length (m) Mean Data.

Epsilon (ε) was 0.687, as calculated according to Greenhouse & Geisser (1959), and was used to correct the one-way repeated measures ANOVA. The exercise interventions did not lead to any statistically significant changes in affected side stride length $F(1.375, 17.869) = .686$, $p = .464$, $\eta^2 = .050$ (Figure 15).

Non-affected side stride length (m).

There were no outliers in the data, as assessed by inspection of a boxplot for values greater than 1.5 box-lengths from the edge of the box. Affected side stride length was normally distributed at all time points, as assessed by Shapiro-Wilk's test ($p < .05$). Mauchly's test of sphericity indicated that the assumption of sphericity had been violated, $\chi^2(2) = 7.284$, $p = .026$. Epsilon (ε) was 0.674, as calculated according to Greenhouse & Geisser (1959), and was used to correct the one-way repeated measures ANOVA. The exercise interventions did not lead to any statistically significant changes in non-affected side stride length $F(1.348, 16.172) = 3.096$, $p = .088$, $\eta^2 = .205$ (Figure 16).

CHAPTER 9 DISCUSSION

Muscle Activation – Pilot 1

To our knowledge, this is the first investigation to determine SS cadence based upon participant's perceived exertion (i.e., RPE) using the NuStep Cross Trainer. All participants completed all 5 protocols despite, anecdotally, participants being most challenged to meet their SS cadence at resistance level 8. The results of this investigation are most likely dependent on two factors: 1- An increase in speed and resistance causes an increase in global muscular activation and 2- the researcher's specific directions to the participant.

The findings indicated that increased resistance (SSL8) and step cadence (SS+20%) resulted in increased muscular activation as measured by mEMG and pEMG. Increased step cadence (SS+20%) did not lead to enhanced neuromuscular recruitment of lower extremity musculature over the SSL8 protocol. Rather, SSL8 produced the highest mEMG and pEMG measurements for all muscles under all protocols. SSL8 elicited the highest pEMG measures of $38.18 \pm 15.96\%$ (95% CI = 31.95 – 44.7%) in LVMO (Table 6). SSL8 also resulted in the highest mEMG of $24.95 \pm 10.54\%$ (95% CI = 20.75 -29.46 %) in LVMO. These percentages of VMO activation are consistent with previous walking protocols (Powers, Landel & Perry, 1996). Additionally, the protocol differences were consistent across both the right and left legs.

However, future research should identify whether a leg preference exists in NuStep propulsion (e.g., higher mEMG and pEMG values in preferential legs across protocols). Our findings are consistent with Huang & Ferris (2004) and Kao & Ferris (2005) where an increase in resistance (i.e., highest resistance level in which participant could step for 20 seconds) and frequency (30-120 steps/min) improved self-driven EMG amplitude. However, this investigation's SS cadence (123.86 ± 18.12 steps per minute) was similar to a 1.5 m/sec

frequency matched protocol in a previous investigation (i.e., 123 ± 3 steps per minute) (Stoloff, Zehr & Ferris, 2007). However, unlike that investigation, our exercise bout was longer (i.e., 5 minutes vs. 10s). Current EMG analysis demonstrated that improving cadence or resistance increased muscular activation in comparison to SS cadence without resistance.

When learning a motor task, the novice individual aims to limit task complexity. According to Bernstein, the learning process requires the initial "freezing" of limbs (Schmidt & Lee, 2011). This freezing reduces the number of degrees of freedom across multiple joints and thus, a reduction of task complexity. Despite constrained kinematics, the NuStep is an exercise modality that, when compared to walking has reduced degrees of freedom, and thereby, is a less complicated task. An individual's stepping is constrained by the seat and pedal positions (Stoloff, Zehr & Ferris, 2007) As a result; recumbent stepping generally has smaller limb forces (Huang and Ferris, 2004). However, recumbent stepping does not provide variability in step to step kinematic motor pattern (Stoloff, Zehr & Ferris, 2007). Lastly, the need for the participant to weight bear through the lower extremities is absent while stepping on a recumbent cross trainer. The foot remains in contact with the pedal.

The seated posture and guided coordination (i.e., fixed footpath) is thought to reduce cognitive demand. A reduction in cognitive demand is coupled with high levels of muscular activation can be a potent recipe for neurological recovery. Therefore, the NuStep cross trainer may provide an intermediate progression between supine and weight-bearing exercise.

Limitations and Future Direction

Peripheral factors may affect EMG amplitude, including but not limited to muscle fiber composition, blood flow, fiber diameter, electrode location, intracellular action potential change (i.e., calcium) and the quantity of subcutaneous tissue (De Luca, 1997; Reaz, Hussain, & Mohd-

Yasin, 2006; Arabadzhiev, Dimitrov, & Dimitrov, 2014). To improve internal validity and to eliminate day to day variations in EMG measures, all testing was conducted within a single session. Therefore, the location of EMG electrodes did not change. Nonetheless, caution should be used when interpreting changes in EMG amplitude over time. Furthermore, the large number of analyses may have risked Type I error. In contrast, the consequential alpha adjustments may have reduced the investigation's ability to detect change (i.e., Type II error).

Muscle Adaptation – Pilot 2

The purpose of this investigation was to study the potential change in EMG activity of 12 lower extremity muscles over a 5-minute exercise bout and during five different stepping protocols. mEMG was compared between the second and fourth minute of this 5-minute exercise bout. Results indicated a higher level of muscle adaptation, as measured by the reduction of mEMG during minute 4 at protocols below the subject's self-selected pace. Despite the changes in mEMG, the subject's work output was held constant. These statistically significant reductions in muscle activation are interpreted as an acute response. At a significantly lower cadence, it is presumed that a new motor pattern was acquired to adapt to the stepping demands. It appears that each muscle may contain differing temporal costs to achieve exercise efficiency. Lastly, a reduction in muscle adaptation occurred at the higher cadence (i.e., SS+20%) and resistance (i.e., SSL8). Trained muscles generate a given amount of submaximal force with less EMG activity; suggesting a more efficient motor unit recruitment with practice (Kenney, Willmore & Costill, 2015).

We speculate that the self-selected speed is the cadence to which the participant operated at the highest efficiency. A protocol that requires the exerciser to operate at speed significantly below this self-selected cadence required the most significant change in muscle adaptation, and

perhaps, learning. In theory, as the participant's motor pattern adapted, we would expect to see reduced cortical activity coupled with enhanced dendrite gyrus, basal ganglia, and cerebellum activity. However, we only measured an acute performance variable and therefore can only speculate at this time. Previous investigators also speculated that cortical reorganization, brought about by repeated leg use in a functional manner, (i.e., simulation of walking), was at least partially responsible for the improvement in balance and impairment (Page, Levine, Teepen, Hartman, 2008).

Limitations and Future Directions.

This investigation validated the presence of an acute adaptation in muscle while on the NuStep Cross Trainer. Future investigations should measure a potential learning effect at a retention test (i.e. 24-48 hours after last practice trial). Additionally, the ability to decipher whether leg dominance influences muscle adaptation during the exercise bout or learning (e.g., at retention) is needed. This research demonstrated that recumbent stepping is altered as a consequence of exercising below a self-selected cadence. Previously, it was assumed that recumbent stepping patterns do not change as a function of time.

NuStep Cross Trainer vs. Treadmill

The purpose of this investigation was to compare the effects of treadmill walking vs. recumbent stepping on muscle mEMG in chronic stroke survivors (i.e.,> 6 months post stroke) vs. age and sex-matched healthy participants. More specifically, a self-selected cadence (as determined by an individual's RPE) on lower extremity muscle activation (as measured by EMG amplitude) between the NuStep Cross Trainer and Treadmill. Their subsequent effect on gait was also investigated. BMI, age, height, and weight did not differ between the conditions. RPE based 10 minute SS protocol was not statistically different between the participant's conditions on TM

or NS. Stroke RPE did not statistically differ on the TM ($Mdn = 11.9$) as compared to the NS ($Mdn = 11.4$). Healthy subjects also did not differ in the RPE based SS protocol across exercise modes. It seems that physiological state (as measured by HR and BP) did not influence our data. HR was below 80 beats per minute at all points of measurement.

TM speed during the RPE based 10 minute SS protocol was higher in the healthy condition despite no statistical difference in RPE. This is in agreement with previous observations that individuals with stroke demonstrate excessive energy cost per distance walked (Olney & Richards, 1996). Preferred walking speeds will be much slower, and energy expenditure at a specific work rate will be 55-64% greater in individuals with a CVA (Palmer-McLean & Harbst, 2003). NS Average steps per minute did not differ between the stroke and healthy conditions during the RPE based 10 minute SS protocol.

Machine setting constraints ($\pm$ 5 steps on NS, 0.2 kph on TM) did not create any statistical differences between the RPE based 10 minute SS protocol and the 5-minute exercise protocol. Chronic stroke subjects sat in a farther seat position from pedals. This seat position perhaps led to the lessened degree of right knee flexion measured in the pedal up position (See Figure 3). Height and weight were equated between the two groups. Therefore, height did not account for this relationship. Perhaps, an extensor pattern (i.e., hip extension, knee extension, hip adduction, hip internal rotation, plantarflexion and ankle inversion) was demonstrated in the chronic stroke condition and consequently influenced a chosen comfortable seat position and thus a pedal up knee flexion.

The healthy condition was stronger than the chronic strokes in all joint actions. Both groups, however, demonstrated stronger L knee extension as compared to R knee extension. R knee ext was performed first for both groups. This may be the result of an ordered learning

effect. Researchers verbally expressed both force and time to force to be recorded. This may have led to a subconscious change in body position (i.e., hip extension) during L knee extension that would be biomechanically more advantageous for the biarticulate rectus femoris . Otherwise, the healthy condition was bilaterally symmetrical in knee flx, ankle dflx, and ankle plantarflx. The stroke condition was comprised of 8 individuals with right side hemiplegia. Therefore, the stroke condition demonstrated higher in strength in both L knee ext and L knee flx. However, no strength deficits were observed in the ankle (i.e., plantar or dorsiflexion). Both leg preference questions elicited a higher majority of CVA participants selecting the left leg to balance (n=10) and kick (n=7).

NS resulted in higher mEMG of the RF (RRF and LRF), RST, SOL (RSOL and LSOL), MG (RMG and LMG) in the stroke condition as compared to the healthy condition, $p < .025$. The TM provoked a higher mEMG in RRF and RST in the stroke condition as compared to the healthy, $p < .025$. Healthy condition saw higher mEMG on the TM in RRF, LST, MG (RMG and LMG), SOL (RSOL and LSOL), and TA (RTA and LTA).However, when the NS is compared to the TM within the stroke condition, mEMG is higher in RRF, RMVO, LST, RSOL and LTA on TM as compared to NS, $p < .025$. RMG, LMG, and RTA saw a non-statistically different mean averages favor the TM. Lofty standard deviations, perhaps due to a mechanical artifact, may have influenced this statistical assessment. 5 of 12 (42%) measured muscles (i.e. left vs. right) demonstrated higher mEMG outputs on the TM in the CVA population. 8 of 12 (67%) measured muscles (i.e. left vs. right) demonstrated higher mEMG outputs on the TM in the healthy population. Therefore, it seems the extent of this normative relationship (TM > NS) was diminished in the CVA population.

mEMG was also evaluated by comparing the affected side (i.e., hemiplegic) to unaffected lower extremity in the CVA population. All muscles on affected lower extremity saw a greater mEMG on TM except ST. TM also demonstrated higher mEMG values of the non-affected lower extremity in the ST, TA, and SOL. RF, VMO, and MG of the non-affected leg were not statistically different between the exercise modes. Therefore, when comparing affected vs. non-affected lower extremity in the CVA population, 8 of 12 muscles elicited higher mEMG on the TM.

No difference existed between conditions on TM ΔROM (i.e., total ROM excursion) except for L ank where healthy ROM was higher than the stroke condition, $p < .025$. No difference existed between conditions on NS across all joints between conditions, $p < .025$. Healthy participants demonstrated higher ROM in R knee, R ank and L ank on TM compared to NS, $p < .025$. No difference in the stroke condition between TM and NS suggesting that the ROM excursion (i.e., max-min) experienced is similar between the modalities for chronic CVA. However, this result should be taken with extreme caution as a result of goniometer malfunction that reduced total n.

L stride and R stride lengths were longer in healthy participants. Both exercise modes improved L stride length. However, TM elicited a statistically significant effect on L stride length ($M = 1.28m$, $SD = .27$) in both conditions compared to baseline ($M = 1.25$, $SD = .25m$). NS's increased in L stride length was no statistically different from baseline. However, NS and TM were also not statistically different. R stride length was unaffected by exercise. Longer stance phases, greater on the unaffected side, are reported in a CVA population (Olney & Richards, 1996). L stance % was higher in the stroke population. This also led to a decreased swing percentage % on the L side. However, non-affected side stance % was increased with the

NS (Figure 12). Furthermore, the non-affected side swing % was statistically higher after NuStep (Figure 14). The NuStep was an effective method for improving stance-swing parameters in the non-affected leg despite the chronic nature of the stroke population. Exercise interventions did not lead to changes in affected side stance %, swing %, and stride length. Lastly, exercise interventions did not affect stride length on the non-affected lower extremity. Exercise treatment proved to be ineffective in modulating double support time in either group. However, as expected, double support time was longer in the chronic stroke condition (Olney & Richards, 1996).

The chronic stroke suffered from a higher asymmetry index, as calculated by the WiGAT system. There is currently limited evidence on the effect of visual feedback (VF) while exercising on the NS. Previous investigations saw that stepping on the NS caused specific muscles (i.e., VMO and Soleus) in particular to be activated preferentially in chronic stroke (Pardo et al., 2018). Participants who generated more force on their non-affected side without VF had a more balanced force production with VF (n = 5, the others were closer to the optimal 50:50 stepping without visual feedback) (Pardo et al., 2018). Gait indices showed a trend towards improved swing to stance ratio after training (Pardo et al., 2018). Clinically, the ability to improve the symmetry of stepping in the stroke population by using visual feedback could be of interest to clinicians, who may want to include the NuStep as an intervention to encourage the forced use of the affected side. This preliminary investigation gives reasoning to perform a longitudinal investigation whether the improvement of stepping symmetry after extended (i.e.,>1 day) NuStep training. In the current investigation, no cueing or instructions were given to the participant in regards to exercise symmetry. This could, in part, explain why exercise treatment did not elicit an effect on asymmetry.

Healthy individuals walked faster during the 3 x 10 m hallway walks. Exercise treatments did not elicit change to participants walking speed compared to baseline. Normal gait mechanics outline that a limb will spend 60% of the gait cycle in stance. L stance phase was higher in stroke ($M = 67.27\%$) as compared to healthy ($M = 62.24\%$). L swing phase was lower in stroke ($M = 32.73\%$) as compared to healthy ($M = 37.99\%$). Exercise treatment did not affect L or R stance. R stance phase was equal between the two conditions.

A reduction in cognitive demand is coupled with high levels of muscular activation can be a potent recipe for promoting neurological recovery but maintaining patient safety. Both TM and NS improved mEMG output. A TM, however, requires a higher level of postural control and thus explains why TM had higher mEMG in the majority of muscles across both conditions. Treadmill ambulation training can also require exceptional resources or additional personnel to administer (Page, Levine, Teepen & Hartman, 2008). In the current investigation, the TM protocols required a minimum of 2-3 researchers (1 to spot the participant, 1 to run the EMG software, 1 to run TM). The NuStep only required one researcher. Clinically, a therapist acting alone (i.e., without assistance) may choose the NS over TM to reduce perception or chances of fall while maintaining high percentages of their MVC during the exercise bout. This reduces accessibility to patients (Stoloff, Zehr & Ferris, 2007). Several sophisticated devices have been developed to overcome these limitations, but their cost and size may limit their use. Less expensive alternatives that facilitate muscle activity through a modified stepping pattern could improve accessibility (Stoloff, Zehr & Ferris, 2007).

In a randomized, controlled, single-blinded crossover study, NuStep participation (3x per week for 30 minutes) showed impairment reductions (i.e., increased score on Fugl-Meyer Assessment of Motor Recovery after Stroke) and improved balance (i.e. Berg Balance Scale)

over 8 weeks (Page, Levine, Teepen & Hartman, 2008). Impairment changes included new and isolated ankle movement and reduced dysmetria (i.e., lack of coordination of movement typified by an undershooting or overshooting of intended position) and improved speed of affected heel to opposite knee task. Berg Balance Scale assessment saw positive changes in sit to stand ability, increased time in an unsupported stance, and transferability. However, due to the small sample sizes, statistical analysis was not applied (Page, Levine, Teepen & Hartman, 2008). A 2007 study was the first to compare muscle activation and kinematics of treadmill (TM) walking to the NuStep Cross Trainer (NS) (Stoloff, Zehr & Ferris, 2007). The authors studied subjects that walked with bodyweight support (i.e. 50% body weight) and without bodyweight (i.e. 0% bodyweight) support at 0.5 meters/second (m/s), 1.0 m/s, and 1.5 m/s. (Robomedica, Inc. Irvine, CA). Both arms and legs propelled the NuStep Cross Trainer to three different frequencies corresponding to their preferred stride frequency at the speeds as mentioned above. The average corresponding step frequencies for 0.5 m/s, 1.0 m/s and 1.5 m/s were 71 ± 3 steps/min, 101 ± 3 steps per min (steps/min) and 123 ± 3 steps/min (mean $\pm$ standard error of the mean). The 1.5 m/s stepping frequency was similar to our 5-minute exercise protocols during Pilot 1 (i.e., 123.86 ± 18.12 steps/min). Resistance was increased to maximize EMG amplitude during the 20 seconds (stepping frequency of 0.5 m/s and 1.0 m/s) and 10 seconds (stepping frequency of 1.5 m/s) bouts. Ten healthy subjects (aged 18-27) participated in the investigation. Both goniometer (ankle, knee, hip, elbow and shoulder) and EMG data (lower extremity: soleus, tibialis anterior, medial and lateral gastrocnemius, vastus medialis, vastus lateralis, medial hamstring, rectus femoris; upper extremity: biceps brachii, triceps brachii, anterior deltoid, posterior deltoid) was recorded unilaterally on the subject's left side (Appendices D & F). The current investigations measured muscular activation bilaterally. Left heel strike to left heel strike defined the step cycle

while on TM. Whereas, left leg extension to left leg extension defined the step cycle on NS.

EMG data were normalized to the value calculated for walking at 1.5 m/s. Lower extremity

EMG results are outlined in Figure 11. During both stance and swing phases (corresponding to

limb extension and flexion for NuStep), thigh and upper limb muscles were lower during

walking than during the NuStep (Appendix D). That is, the NuStep cross trainer saw elevated

upper limb and thigh (vastus medialis, vastus lateralis, rectus femoris and medial hamstring)

EMG (Stoloff, Zehr & Ferris, 2007). Whereas, both healthy and stroke populations saw higher

RRF mEMG on the TM as compared to the NS. During the stance phase (i.e., extension phase

on NuStep), tibialis anterior and medial gastrocnemius EMG were lower during the NS as

compared to the TM, $p < .01$ (Stoloff, Zehr & Ferris, 2007). During the swing phase (i.e., flexion

on NuStep), soleus, medial gastrocnemius and lateral gastrocnemius were higher on NuStep, $p < .01$(Stoloff, Zehr & Ferris, 2007). Medial hamstring activation was out of phase for the NuStep

as compared to walking (Appendix D) (Stoloff, Zehr & Ferris, 2007). Soleus activation was

shifted earlier in the step cycle on NuStep (Appendix D). The upper limb seemed to display

fundamentally different muscle activation patterns (Appendix E). Cross-correlation analysis of

individual muscle EMG between conditions showed a high correlation ($r > 0.70$) in 7 of 12

muscles (Appendix G). The soleus, tibialis anterior, medial hamstring, and gastrocnemius

(medial and lateral) demonstrated a low correlation between ($r < 0.70$) the conditions. The

correlation coefficient comparing walking and recumbent stepping were lower at faster speeds

for lateral gastrocnemius, rectus femoris, medial hamstring, biceps brachii and triceps brachii, $p < .05$. All joints except the shoulder had significantly different minimum and maximum joint

angles for TM and NS (Stoloff, Zehr & Ferris, 2007). Minimum shoulder joints angles were

different, but maximum joint angles were comparable (Stoloff, Zehr & Ferris, 2007). Excursions

of the hip, elbow, and shoulder were significantly less for walking than for the NS. At some

speeds, the excursion of the knee and ankle were greater for the NS than it was for 50%

bodyweight support, $p < .05$. However, there was no significant difference between the knee and

ankle excursion for NS and 0% bodyweight support. There were no differences in minimum,

maximum or excursion across stepping speeds (Stoloff, Zehr & Ferris, 2007). These authors

concluded that stepping on the NuStep Cross trainer activates similar motor pathways as

walking, despite temporal differences in individual muscle EMG (Stoloff, Zehr & Ferris, 2007).

Authors speculated that the lower correlations observed in the leg might depend on afferent

feedback for activation (Stoloff, Zehr & Ferris, 2007).

Conclusions

The exercise professional should consider the client's motor abilities when selecting an

appropriate exercise device. For example, impaired sitting balance may limit the use of upright

ergometers that do not have torso support. Elevated exercise modes may also require a step stool

to perform an independent or assisted transfer (Palmer-McLean & Harbst, 2003). A recumbent

device may be more appropriate (Palmer-McLean & Harbst, 2003). Furthermore, the NuStep

was among the preferred exercise modalities in the elderly (Looney & Rimmer, 2003).

The NuStep Cross Trainer may be a useful adjunct to physical therapy requiring

minimum supervision with lasting effects after discharge (Teepen, Baltzer, Dunning & Levine,

2005). It also offers promise to patients discharged from therapy as it combines aspects of

strength and cardiovascular training (Teepen, Baltzer, Dunning & Levine, 2005). However, the

potential drawback to exercise machines for stepping practice is that they sacrifice some task

specificity and lowered mEMG (Stoloff, Zehr & Ferris, 2007). To our knowledge, this is the first

investigation to examine the effect of a NS and TM on mEMG in a CVA and healthy (age and

sex matched) population. We demonstrated that the NuStep Cross Trainer immediately improved gait parameters on the non-affected leg following a 5-minute stepping protocol.

Limitations and Future Directions.

Muscle strength tests can include computerized dynamometers (e.g., CybexTM, BiodexTM) or manually by handheld dynamometers (Lafayette Instrument 01165 Manual Muscle Testing Device). It should be noted that strength testing can be problematic in populations with brain injury (Palmer-McLean & Harbst, 2003). Strength can only be reliably tested when an individual can isolate joint movements.

Exercise programs for individuals with CVA should be aimed at not only at increasing the levels of physical fitness but also at reducing risk factors (Palmer-McLean & Harbst, 2003). Theoretically, a reduction in risk factors should decrease the incidence of secondary strokes An aerobic conditioning program can alter several of the risk factors associated with CVA, including reduced hypertension, enhanced glucose regulation, improved blood lipid profiles and improved body composition (Palmer-McLean & Harbst, 2003). Associative depression may improve as a result of exercise. Exercise is associated with a more significant reduction in depression symptoms compared with no treatment, placebo, or active control interventions, such as relaxation or meditation (Cooney, Dwan & Meed, 2014). However, analysis of high-quality studies alone suggests only small benefits (Cooney, Dwan & Meed, 2014). This study did not measure spasticity, passive range of motion, or motor recovery as outlined by previous investigations (Page, Levine, Teepen & Hartman, 2008). However, the current population accrued more time between CVA and testing (120 months ± 60 months) compared to 44.43 ± 24.48 months (Page, Levine, Teepen & Hartman, 2008). The current investigation did not include exclusion criteria for prescribed medication that may improve spasticity. However, with

a repeated measures design, each participant served as their own control mitigating this limitation. The current investigation lost goniometric data due to malfunctioning equipment. This problem also occurred in prior investigations Stoloff, Zehr & Ferris, 2007). Future research should examine the effects of NS practice over time. How the results of the current investigation compare to a sub-chronic CVA population also remains unknown. Future research should examine whether there are changes in CVA vs. healthy EMG according to pedal position or stepping phase. Lastly, the effect of whole body NS exercise on gait should be investigated.

APPENDIX A

WAYNE STATE UNIVERSITY

NOTICE OF EXPEDITED APPROVAL

To: Sujay Galen
Physical Therapy Program
Eugene Applebaum College of Ph

From: Lawrence R. Crane, M.D. or designee _D. Bielawski /2.2._
Chairperson, Medical Institutional Review Board (M1)

Date: May 15, 2015

RE: **IRB #:** 046515M1E

Protocol Title: Muscle Activity Profile During Recumbent Stepping

Funding Source:

Protocol #: 1504013972

Expiration Date: May 14, 2016

Risk Level / Category: Research not involving greater than minimal risk

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The above-referenced protocol and items listed below (if applicable) were **APPROVED** following *Expedited Review* (Category (4)*) by the Chairperson/designee *for* the Wayne State University Institutional Review Board (M1) for the period of 05/15/2015 through 05/14/2016. This approval does not replace any departmental or other approvals that may be required.

- Revised Protocol Summary Form (revision received in the IRB office 5/8/15).
- Protocol (received in the IRB office 4/27/15)
- Medical Research Informed Consent (revision dated 5/8/2015)
- Study Participant Checklist.
- Study Flyer

° Federal regulations require that all research be reviewed at least annually. You *may* receive a *"Continuation Renewal Reminder"* approximately two months prior to the expiration date; however, it is the Principal Investigator's responsibility to obtain review and continued approval *before* the expiration date. Data collected during a period of lapsed approval is unapproved research and can never be reported or published as research data.

° All changes or amendments to the above-referenced protocol require review and approval by the IRB **BEFORE** implementation.

° Adverse Reactions/Unexpected Events (AR/UE) must be submitted on the appropriate form within the timeframe specified in the IRB Administration Office Policy (http://www.irb.wayne.edu//policies-human-research.php).

NOTE:

1. Upon notification of an impending regulatory site visit, hold notification, and/or external audit the IRB Administration Office must be contacted immediately.
2. Forms should be downloaded from the IRB website at **each** use.

*Based on the Expedited Review List, revised November 1998

Muscle Activity Profile During Recumbent Stepping

Research Informed Consent
Title of Study: Muscle Activity Profile During Recumbent Stepping

Principal Investigator (PI): Sujay Galen, PT, PhD, FHEA
Physical Therapy Department
Wayne State University
1 (313) 577-5531

Purpose

You are being asked to be in a research study investigating muscle activity during recumbent stepping because you are within the ages of 18-65 and have no known orthopedic or neurological conditions that affect the way you can perform stepping. This study is being conducted at Wayne State University. The estimated number of study participants to be enrolled at Wayne State University is about 40. **Please read this form and ask any questions you may have before agreeing to be in the study.**

In this research study, we will be recording the electrical activity in your lower extremity muscles using a non-invasive technique known as electromyography (EMG). This is a very commonly used investigative procedure in exercise testing studies. The electrical activity is recorded using sensors placed on your skin. The electrical activity is recorded and wirelessly transmitted to a computer using a commercially available EMG system that has been used in previous studies in our laboratory. You will be performing stepping using a commercially available stepper that is widely used in gymnasium. Physical rehabilitation centers and Physical Therapy offices. The name of this stepper is NuStep. The primary purpose of this study is to identify how muscles of the lower extremity are engaged while you perform stepping using the NuStep. We hypothesize that altering the stepping speed and the level of resistance during stepping will alter how the lower extremity muscles are engaged.

Study Procedures

If you agree to take part in this research study, you will be asked to complete a history questionnaire and to complete several trials of stepping using the NuStep. The following points listed below help to clarify and highlight the key points:

1. Provide us a signed copy of this form, after we have established your eligibility to participate in the study.
2. If, after filling out the form, you are no longer interested in continuing the study, you are free to quit at any time.
3. During the first part of the study you will be asked to step for 10 minutes using the NuStep using a self-selected stepping speed. During this time of stepping we will ask you tell us your perceived exertion using a scale known as the Borg rating of perceived exertion scale. You will be asked to slow down if you report a value of 16 on this scale which equates to you perceiving your exertions to be hard.
4. We will then clean your skin over some of the lower extremity muscles located on the front and back of your thigh and lower leg using alcohol swabs. Once the skin is clean, we will stick the sensors to the skin over these muscle locations and secure them using paper tape. You will then be asked to perform a forceful contraction of the key lower extremity muscles,

Submission/Revision Date: [5/08/2015]   Page 1 of 4   _____
Protocol Version #: [1]   Participant's Initials
                                                          Form Date 10/2013

Muscle Activity Profile During Recumbent Stepping

by pushing hard against a device known as the dynamometer, which will provide a known resistance. During this contraction, your peak muscle activity will be recorded. All muscle activity recorded during stepping will be expressed as a percentage of this peak muscle activity.

5. The second part of the study will involve stepping using 3 different speeds based on your self-selected speed. You will also be asked to step at a speed of 80steps/min. While stepping at your self-selected speed the resistance on one of the exercise stepping trial will be kept at the minimum(level 1) and on another exercise stepping trial it will be increased to a mid-level resistance (level 8). Therefore there will be a total of 5 exercise stepping trials. Each exercise stepping trial will last exactly for 5 minutes. Your stepping speed will be kept constant using a metronome. Between each of the 5 exercise stepping trials you will be provided a 5 minute rest break.

6. The study involves just a single laboratory visit, that will last for no more than 90 minutes

Benefits

As a participant in this research study, there may be no direct benefit for you; however, information from this study may benefit other people now or in the future.

Risks

By taking part in this study, you may experience the following risks:

Physical Risks: There is a small risk of experiencing muscle fatigue while performing stepping. You will be asked to report your perceived exertion and you will also be provided with 5 minutes of rest between each exercise stepping trial.

Social Risk: There is also a small risk of loss of confidentiality about your participation in the study. The only place your name will appear is on the informed consent. These forms will be stored in a locked cabinet in the principal investigators office.

Alternatives

You may choose not to participate.

Study Costs

Participation in this study will be of no cost to you.

Compensation

You will not be paid for taking part in this study.

Research Related Injuries

Muscle Activity Profile During Recumbent Stepping

In the event that this research related activity results in an injury, treatment will be made available including first aid, emergency treatment, and follow-up care as needed. Care for such will be billed in the ordinary manner to you or your insurance company. No reimbursement, compensation, or free medical care is offered by Wayne State University. If you think that you have suffered a research related injury, contact the PI right away at (313) 577-5531.

Confidentiality

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. You will be identified in the research records by a code name or number. Information that identifies you personally will not be released without your written permission. However, the study sponsor, the Institutional Review Board (IRB) at Wayne State University, or federal agencies with appropriate regulatory oversight [e.g., Food and Drug Administration (FDA), Office for Human Research Protections (OHRP), Office of Civil Rights (OCR), etc.) may review your records.

When the results of this research are published or discussed in conferences, no information will be included that would reveal your identity.

Voluntary Participation/Withdrawal

Taking part in this study is voluntary. You have the right to choose not to take part in this study. If you decide to take part in the study you can later change your mind and withdraw from the study. You are free to only answer questions that you want to answer. You are free to withdraw from participation in this study at any time. Your decisions will not change any present or future relationship with Wayne State University or its affiliates, or other services you are entitled to receive.

The PI may stop your participation in this study without your consent. The PI will make the decision and let you know if it is not possible for you to continue. The decision that is made is to protect your health and safety, or because you did not follow the instructions to take part in the study.

Questions

If you have any questions about this study now or in the future, you may contact Sujay Galen or one of his research team members at the following phone number (313) 577-5531. If you have questions or concerns about your rights as a research participant, the Chair of the Institutional Review Board can be contacted at (313) 577-1628. If you are unable to contact the research staff, or if you want to talk to someone other than the research staff, you may also call (313) 577-1628 to ask questions or voice concerns or complaints.

Muscle Activity Profile During Recumbent Stepping

Consent to Participate in a Research Study

To voluntarily agree to take part in this study, you must sign on the line below. If you choose to take part in this study you may withdraw at any time. You are not giving up any of your legal rights by signing this form. Your signature below indicates that you have read, or had read to you, this entire consent form, including the risks and benefits, and have had all of your questions answered. You will be given a copy of this consent form.

_____   _____
Signature of participant   Date
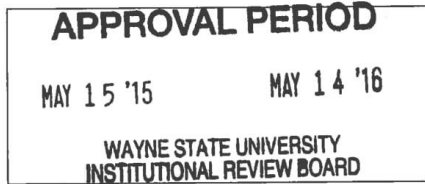
_____   _____
Printed name of participant   Time

_____   _____
Signature of person obtaining consent   Date

_____   _____
Printed name of person obtaining consent   Time

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┌─────────────────────────────────────────┐
│  APPROVAL PERIOD                         │
│                                          │
│  MAY 15 '15          MAY 14 '16          │
│                                          │
│     WAYNE STATE UNIVERSITY               │
│   INSTITUTIONAL REVIEW BOARD             │
└─────────────────────────────────────────┘
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Muscle Activity Profile During Recumbent Stepping

Study Participant Checklist

☐ No history of orthopedic conditions of the lower extremity
☐ No history of neurologic conditions of the lower extremity
☐ Obtained Informed Consent
☐ Recorded Participant Measurements:
    ☐ Height
    ☐ Weight
    ☐ BMI

Equipment/Procedure Checklist

☐ Perform 10 minutes of stepping to determine self-selected speed
☐ Prepare the skin using alcohol skin prep wipes over key lower extremity muscles
☐ Apply the EMG sensors over the skin locations
☐ Ensure a stable and noise free EMG signals in all muscles
☐ Perform a maximum voluntary contraction using the dynamometer for the following muscles

    ☐ Rectus Femoris
    ☐ Vastus Medialis
    ☐ Medial Hamstrings
    ☐ Tibialis Anterior
    ☐ Medial Gastrocnemius
    ☐ soleus

☐ Perform the 5 exercise stepping protocols and note down the randomized order in the box provided
    ☐ 20% below Self Selected speed with resistance level 1
    ☐ Self Selected speed with resistance level 1
    ☐ Self Selected speed with resistance level 8
    ☐ Pre-set speed of 80steps/minute with resistance level 1
    ☐ 20% above Self Selected speed with resistance level 1

☐ Trials completed: Remove all sensors from participants

Version 1   04/27/2015

Participants Needed for a Research Study of Muscle Activity Profile During Recumbent Stepping

Eligibility

- Age 18-65 years
- No neurological and orthopedic conditions affecting mobility
- No known allergy to adhesive paper tapes

The Research Study

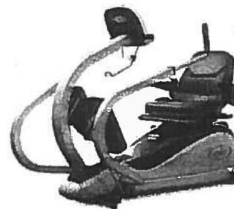Measures the electrical activity in your muscle using surface skin sensors while you perform stepping on a recumbent stepper known as the NuStep.

When and Where

- During 2015-16 at the Wayne State University Physical Therapy labs
- At the corner of John R. and Mack Avenue
- Approximately 90 minutes will be needed to complete all tests

Contact: **Nick Siekirk by email Nicholas.siekirk@wayne.edu**

Principal Investigator:
Sujay Galen, PT, PhD
Physical Therapy Program
259 Mack Ave. Detroit

APPROVAL PERIOD

AY 1 5 '15   MAY 1 4 '16

WAYNE STATE UNIVERSITY
INSTITUTIONAL REVIEW BOARD
Recumbent Stepper

Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu	Wayne State NuStep Research Nicholas.siekirk@wayne.edu

WAYNE STATE UNIVERSITY

NOTICE OF EXPEDITED AMENDMENT APPROVAL

To: Sujay Galen
Physical Therapy Program
Eugene Applebaum College of Ph

From: Lawrence R. Crane, M.D. or designee _N. Bellanmi PhD/lDA_
Chairperson, Medical Institutional Review Board (M1)

Date: July 01, 2015

RE: IRB #: 046515M1E

Protocol Title: Muscle Activity Profile During Recumbent Stepping

Funding Source:

Protocol #: 1504013972

Expiration Date: May 14, 2016

Risk Level / Category: Research not involving greater than minimal risk

The above-referenced protocol amendment, as itemized below, was reviewed by the Chairperson/designee of the Wayne State University Institutional Review Board (M1) and is APPROVED effective immediately.

- Key Personnel Additions: Amanda Rohkohl (Student Investigator), Jamie Plumb (Student Investigator), Austin Swadling (Student Investigator), Salvatore Deangelo (Student Investigator)

APPENDIX B

WAYNE STATE UNIVERSITY

IRB Administration Office
87 East Canfield, Second Floor
Detroit, Michigan 48201
Phone: (313) 577-1628
FAX: (313) 993-7122
http://irb.wayne.edu

NOTICE OF EXPEDITED APPROVAL

To: Sujay Galen
Physical Therapy Program
Eugene Applebaum College of Ph

From: Lawrence R. Crane, M.D. or designee
Chairperson, Medical Institutional Review Board (M1)

Date: June 01, 2017

RE: **IRB #:** 035517M1E

Protocol Title: Exploratory studies on muscle activation using NuStep

Funding Source: Sponsor: State of Michigan

Protocol #: 1703000417

Expiration Date: May 31, 2020

Risk Level / Category: Research not involving greater than minimal risk

The above-referenced protocol and items listed below (if applicable) were **APPROVED** following *Expedited Review* (Category (3)* (4)*) by the Chairperson/designee *for* the Wayne State University Institutional Review Board (M1) for the period of 06/01/2017 through 05/31/2020. This approval does not replace any departmental or other approvals that may be required.

- Revised Protocol Summary Form (revisions received in the IRB Office 05/23/2017
- Protocol (received in the IRB Office 03/13/2017)
- Medical records are not being accessed, therefore HIPAA does not apply.
- Research Informed Consent – Study 1 – Healthy Subject
- Research Informed Consent – Study 1 – Stroke Subject
- Research Informed Consent – Study 2 – Healthy Subject
- Research Informed Consent – Study 2 – Stroke Subject
- Flyer
- Data Collection Tools: Nustep Data Collection – Study 1 and Nustep Data Collection – Study 2
- Please note: This submission was reviewed under the IRB Administration Office Flexible Review and Oversight Policy. The expiration date is May 31, 2020.

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* Federal regulations require that all research be reviewed at least annually. You *may* receive a *"Continuation Renewal Reminder"* approximately two months prior to the expiration date; however, it is the Principal Investigator's responsibility to obtain review and continued approval *before* the expiration date. Data collected during a period of lapsed approval is unapproved research and can never be reported or published as research data.

* All changes or amendments to the above-referenced protocol require review and approval by the IRB **BEFORE** implementation.

* Adverse Reactions/Unexpected Events (AR/UE) must be submitted on the appropriate form within the timeframe specified in the IRB Administration Office Policy (http://www.irb.wayne.edu//policies-human-research.php).

NOTE:

1. Upon notification of an impending regulatory site visit, hold notification, and/or external audit the IRB Administration Office must be contacted immediately.
2. Forms should be downloaded from the IRB website at **each** use.

*Based on the Expedited Review List, revised November 1998

WAYNE STATE UNIVERSITY

NOTICE OF EXPEDITED AMENDMENT APPROVAL

To: Sujay Galen
Physical Therapy Program
Eugene Applebaum College of Ph

From: Lawrence R. Crane, M.D. or designee
Chairperson, Medical Institutional Review Board (M1)

Date: March 26, 2018

RE: IRB #: 035517M1E

Protocol Title: Exploratory studies on muscle activation using NuStep

Funding Source: Sponsor: State of Michigan

Protocol #: 1703000417

Expiration Date: May 31, 2020

Risk Level / Category: Research not involving greater than minimal risk

The above-referenced protocol amendment, as itemized below, was reviewed by the Chairperson/designee of the Wayne State University Institutional Review Board (M1) and is APPROVED effective immediately.

- Coordinating Center Application – Receipt of Coordinating Center Application dated 03/09/2018 to reflect the IRB approval for University of Michigan - Flint (approval dated 01/10/2018) and the Wayne State IRB is the IRB of Record using the SMART IRB agreement.

  Notify the IRB of any changes to the funding status of the above-referenced protocol.

Effect of visual feedback on lower extremity muscle activation during recumbent stepping

Research Informed Consent
Title of Study: Exploratory studies on muscle activation – Study 2 – Stroke Subject.

Principal Investigator (PI):   Sujay Galen, PT, PhD, FHEA
Physical Therapy Department
Wayne State University
(313) 577-5531

Purpose

You are being asked to be in a research study of investigating lower extremity muscle activation during recumbent stepping versus over ground walking because you are between the ages of 18-80, and you have had a stroke which does not limit your ability to perform stepping on the Nustep. This study is being conducted at Wayne State University. The estimated number of study participants to be enrolled will be 25 participants who have not had a stroke, and 25 participants with stroke. **Please read this form and ask any questions you may have before agreeing to be in the study.**

In this research study, we will be recording the electrical activity in your lower extremity muscles using a non-invasive technique known as electromyography (EMG). This is a very commonly used investigative procedure in exercise testing studies. The electrical activity is recorded using sensors placed on your skin. The electrical activity is recorded and wirelessly transmitted to a computer using a commercially available EMG system that has been used in previous studies in our laboratory. You will be performing stepping using a commercially available stepper that is widely used in gymnasiums, physical rehabilitation centers and Physical Therapy offices. The name of this stepper is NuStep. The primary purpose of this study is to identify how muscles of the lower extremity are engaged while you perform stepping using the NuStep. We hypothesize that stepping in the recumbent stepper will result in greater muscle activity in the lower extremities compared to walking overground.

Study Procedures

If you agree to take part in this research study, you will be asked to complete a single trial of stepping using the NuStep, and 3 10 meter walks over ground. The following points listed below help to clarify and highlight the key points:

1. Provide us a signed copy of this form, after we have established your eligibility to participate in the study.
2. If, after filling out the form, you are no longer interested in continuing the study, you are free to quit at any time.
3. After filling out the consent form, you will be asked to provide a 2 ml sample of your saliva by spitting into a clear plastic tube. The tube will be sent for genetic testing, and the saliva

Submission/Revision Date: 5/31/2017   Page 1 of 5
Protocol Version #: 1

_____
Participant's Initials
Form Date 04/2015

Effect of visual feedback on lower extremity muscle activation during recumbent stepping

sample will be analyzed for a particular gene variation known as VAL66MET. A gene is the basic physical and functional unit of heredity and it contains DNA which has instructions to make proteins. One such protein is called the brain derived neurotrophic factor (BDNF). The presence of this protein promotes neuronal connections in the brain, and therefore presence of certain gene variation (eg. VAL66MET) has been shown to affect the production BDNF. These gene variations are commonly known as polymorphism (ability of a gene to be expressed in more than one form). Presence or absence of this genetic polymorphism affects how neurons can make connections with one another, when individuals try to learn new movement patters. No other genetic testing will be performed. The genetic testing lab will not be provided with your name, just a study code will be attached to the plastic tube before sending for analysis. The Val66MET genetic variation as explained previously, has been shown to have some association with how we learn new movement patterns, such as stepping on the NuStep. Therefore this will help us explain some of the variability in our collected data, and provide us some insight into how muscle recruitment happens when an individual learns to perform stepping using the NuStep.

4. We will then clean your skin over some of the lower extremity muscles located on the front and back of your thigh and lower leg using alcohol swabs. Once the skin is clean, we will stick the sensors to the skin over these muscle locations and secure them using paper tape. You will then be asked to perform a forceful contraction of the key lower extremity muscles, by pushing hard against a device known as the dynamometer, which will provide a known resistance. During this contraction, your peak muscle activity will be recorded. All muscle activity recorded during stepping will be expressed as a percentage of this peak muscle activity.

5. Your walking pattern will be assessed using a wireless gait assessment tool (Wi-GAT). This will involve placing an insole with sensors inside your shoe. Once the insoles are in place you will be asked to walk 10 meters 3 times to assess your walking. EMG data will also be collected while walking.

6. The stepping trial will last exactly for 5 minutes. you will be provided a 5 minute rest break between the over ground walking trials and stepping .

7. The order of the stepping and walking trials will be randomized.

8. The study involves just a single laboratory visit, that will last for no more than 90 minutes.

Benefits

As a participant in this research study, there may be no direct benefit for you; however, information from this study may benefit other people now or in the future.

Risks

By taking part in this study, you may experience the following risks:

Submission/Revision Date: 5/31/2017   Page 2 of 5
Protocol Version #: 1   _____
                                                                  Participant's Initials
                                                                       Form Date 04/2015

Effect of visual feedback on lower extremity muscle activation during recumbent stepping

Physical Risks: There is a small risk of experiencing muscle fatigue while performing stepping. You will be asked to report your perceived exertion and you will also be provided with 5 minutes of rest between each exercise stepping trial.

Social Risk: There is also a small risk of loss of confidentiality about your participation in the study. The only place your name will appear is on the informed consent. These forms will be stored in a locked cabinet in the principal investigator's office.

Alternatives

You may choose not to participate.

Study Costs

Participation in this study will be of no cost to you.

Compensation

You will be provided $30 compensation for participating in this study

Research Related Injuries

In the event that this research related activity results in an injury, treatment will be made available including first aid, emergency treatment, and follow-up care as needed. Care for such will be billed in the ordinary manner to you or your insurance company. No reimbursement, compensation, or free medical care is offered by Wayne State University. If you think that you have suffered a research related injury, contact the PI right away at (313) 577-5531.

Confidentiality

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. You will be identified in the research records by a code name or number. Information that identifies you personally will not be released without your written permission. However, the study sponsor, the Institutional Review Board (IRB) at Wayne State University, or federal agencies with appropriate regulatory oversight [e.g., Food and Drug Administration (FDA), Office for Human Research Protections (OHRP), Office of Civil Rights (OCR), etc.) may review your records.

When the results of this research are published or discussed in conferences, no information will be included that would reveal your identity.

Voluntary Participation/Withdrawal

Effect of visual feedback on lower extremity muscle activation during recumbent stepping

Taking part in this study is voluntary. You have the right to choose not to take part in this study. If you decide to take part in the study you can later change your mind and withdraw from the study. You are free to only answer questions that you want to answer. You are free to withdraw from participation in this study at any time. Your decisions will not change any present or future relationship with Wayne State University or its affiliates, or other services you are entitled to receive.

The PI may stop your participation in this study without your consent. The PI will make the decision and let you know if it is not possible for you to continue. The decision that is made is to protect your health and safety, or because you did not follow the instructions to take part in the study.

Questions

If you have any questions about this study now or in the future, you may contact Sujay Galen, Vicky Pardo or one of their research team members at the following phone number (313) 577-5531. If you have questions or concerns about your rights as a research participant, the Chair of the Institutional Review Board can be contacted at (313) 577-1628. If you are unable to contact the research staff, or if you want to talk to someone other than the research staff, you may also call (313) 577-1628 to ask questions or voice concerns or complaints.

Consent to Participate in a Research Study
To voluntarily agree to take part in this study, you must sign on the line below. If you choose to take part in this study you may withdraw at any time. You are not giving up any of your legal rights by signing this form. Your signature below indicates that you have read, or had read to you, this entire consent form, including the risks and benefits, and have had all of your questions answered. You will be given a copy of this consent form.

Effect of visual feedback on lower extremity muscle activation during recumbent stepping

_____   _____
Signature of participant   Date

_____   _____
Printed name of participant   Time

_____   _____
Signature of person obtaining consent   Date

_____   _____
Printed name of person obtaining consent   Time

APPROVAL PERIOD

JUN 0 1 2017   MAY 3 1 2020

WAYNE STATE UNIVERSITY
INSTITUTIONAL REVIEW BOARD

Effect of visual feedback on lower extremity muscle activation during recumbent stepping

Research Informed Consent
Title of Study: Exploratory studies on muscle activation – Study 2 – Healthy Subject.

Principal Investigator (PI):   Sujay Galen, PT, PhD, FHEA
Physical Therapy Department
Wayne State University
(313) 577-5531

Purpose

You are being asked to be in a research study of investigating lower extremity muscle activation during recumbent stepping versus over ground walking because you are between the ages of 18-80 and either do not have any musculoskeletal, neurological, cardiopulmonary, respiratory, or other medical condition that limits your ability to perform stepping on the NuStep. This study is being conducted at Wayne State University. The estimated number of study participants to be enrolled will be 25 participants who have not had a stroke, and 25 participants with stroke. **Please read this form and ask any questions you may have before agreeing to be in the study.**

In this research study, we will be recording the electrical activity in your lower extremity muscles using a non-invasive technique known as electromyography (EMG). This is a very commonly used investigative procedure in exercise testing studies. The electrical activity is recorded using sensors placed on your skin. The electrical activity is recorded and wirelessly transmitted to a computer using a commercially available EMG system that has been used in previous studies in our laboratory. You will be performing stepping using a commercially available stepper that is widely used in gymnasiums, physical rehabilitation centers and Physical Therapy offices. The name of this stepper is NuStep. The primary purpose of this study is to identify how muscles of the lower extremity are engaged while you perform stepping using the NuStep. We hypothesize that stepping in the recumbent stepper will result in greater muscle activity in the lower extremities compared to walking overground.

Study Procedures

If you agree to take part in this research study, you will be asked to complete a single trial of stepping using the NuStep, and 3 10 meter walks over ground. The following points listed below help to clarify and highlight the key points:

1. Provide us a signed copy of this form, after we have established your eligibility to participate in the study.
2. If, after filling out the form, you are no longer interested in continuing the study, you are free to quit at any time.

Submission/Revision Date: 5/31/2017   Page 1 of 5
Protocol Version #: 1

_____
Participant's Initials
Form Date 04/2015

Effect of visual feedback on lower extremity muscle activation during recumbent stepping

3. After filling out the consent form, you will be asked to provide a 2 ml sample of your saliva by spitting into a clear plastic tube. The tube will be sent for genetic testing, and the saliva sample will be analyzed for a particular gene variation known as VAL66MET. A gene is the basic physical and functional unit of heredity and it contains DNA which has instructions to make proteins. One such protein is called the brain derived neurotrophic factor (BDNF). The presence of this protein promotes neuronal connections in the brain, and therefore presence of certain gene variation (eg. VAL66MET) has been shown to affect the production BDNF. These gene variations are commonly known as polymorphism (ability of a gene to be expressed in more than one form). Presence or absence of this genetic polymorphism affects how neurons can make connections with one another, when individuals try to learn new movement patters. No other genetic testing will be performed. The genetic testing lab will not be provided with your name, just a study code will be attached to the plastic tube before sending for analysis. The Val66MET genetic variation as explained previously, has been shown to have some association with how we learn new movement patterns, such as stepping on the NuStep. Therefore this will help us explain some of the variability in our collected data, and provide us some insight into how muscle recruitment happens when an individual learns to perform stepping using the NuStep.
4. We will then clean your skin over some of the lower extremity muscles located on the front and back of your thigh and lower leg using alcohol swabs. Once the skin is clean, we will stick the sensors to the skin over these muscle locations and secure them using paper tape. You will then be asked to perform a forceful contraction of the key lower extremity muscles, by pushing hard against a device known as the dynamometer, which will provide a known resistance. During this contraction, your peak muscle activity will be recorded. All muscle activity recorded during stepping will be expressed as a percentage of this peak muscle activity.
5. Your walking pattern will be assessed using a wireless gait assessment tool (Wi-GAT). This will involve placing an insole with sensors inside your shoe. Once the insoles are in place you will be asked to walk 10 meters 3 times to assess your walking. EMG data will also be collected while walking.
6. The stepping trial will last exactly for 5 minutes. you will be provided a 5 minute rest break between the over ground walking trials and stepping .
7. The order of the stepping and walking trials will be randomized.
8. The study involves just a single laboratory visit, that will last for no more than 90 minutes.

Benefits

As a participant in this research study, there may be no direct benefit for you; however, information from this study may benefit other people now or in the future.

Effect of visual feedback on lower extremity muscle activation during recumbent stepping

Risks

By taking part in this study, you may experience the following risks:

Physical Risks: There is a small risk of experiencing muscle fatigue while performing stepping. You will be asked to report your perceived exertion and you will also be provided with 5 minutes of rest between each exercise stepping trial.

Social Risk: There is also a small risk of loss of confidentiality about your participation in the study. The only place your name will appear is on the informed consent. These forms will be stored in a locked cabinet in the principal investigator's office.

Alternatives

You may choose not to participate.

Study Costs

Participation in this study will be of no cost to you.

Compensation

You will not be paid for taking part in this study,

Research Related Injuries

In the event that this research related activity results in an injury, treatment will be made available including first aid, emergency treatment, and follow-up care as needed. Care for such will be billed in the ordinary manner to you or your insurance company. No reimbursement, compensation, or free medical care is offered by Wayne State University. If you think that you have suffered a research related injury, contact the PI right away at (313) 577-5531.

Confidentiality

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. You will be identified in the research records by a code name or number. Information that identifies you personally will not be released without your written permission. However, the study sponsor, the Institutional Review Board (IRB) at Wayne State University, or federal agencies with appropriate regulatory oversight [e.g., Food and Drug Administration (FDA), Office for Human Research Protections (OHRP), Office of Civil Rights (OCR), etc.) may review your records.

Effect of visual feedback on lower extremity muscle activation during recumbent stepping

When the results of this research are published or discussed in conferences, no information will be included that would reveal your identity.

Voluntary Participation/Withdrawal

Taking part in this study is voluntary. You have the right to choose not to take part in this study. If you decide to take part in the study you can later change your mind and withdraw from the study. You are free to only answer questions that you want to answer. You are free to withdraw from participation in this study at any time. Your decisions will not change any present or future relationship with Wayne State University or its affiliates, or other services you are entitled to receive.

The PI may stop your participation in this study without your consent. The PI will make the decision and let you know if it is not possible for you to continue. The decision that is made is to protect your health and safety, or because you did not follow the instructions to take part in the study.

Questions

If you have any questions about this study now or in the future, you may contact Sujay Galen, Vicky Pardo or one of their research team members at the following phone number (313) 577-5531. If you have questions or concerns about your rights as a research participant, the Chair of the Institutional Review Board can be contacted at (313) 577-1628. If you are unable to contact the research staff, or if you want to talk to someone other than the research staff, you may also call (313) 577-1628 to ask questions or voice concerns or complaints.

Consent to Participate in a Research Study
To voluntarily agree to take part in this study, you must sign on the line below. If you choose to take part in this study you may withdraw at any time. You are not giving up any of your legal rights by signing this form. Your signature below indicates that you have read, or had read to you, this entire consent form, including the risks and benefits, and have had all of your questions answered. You will be given a copy of this consent form.

Effect of visual feedback on lower extremity muscle activation during recumbent stepping

_____	_____
Signature of participant	Date
_____	_____
Printed name of participant	Time
_____	_____
Signature of person obtaining consent	Date
_____	_____
Printed name of person obtaining consent	Time

APPROVAL PERIOD

JUN 0 1 2017   MAY 3 1 2020

WAYNE STATE UNIVERSITY
INSTITUTIONAL REVIEW BOARD

APPENDIX C

RPE Scale

6	
7	Very, Very Light
8	
9	Very Light
10	
11	Fairly Light
12	
13	Somewhat Hard
14	
15	Hard
16	
17	Very Hard
18	
19	Very, Very Hard
20	

APPENDIX D
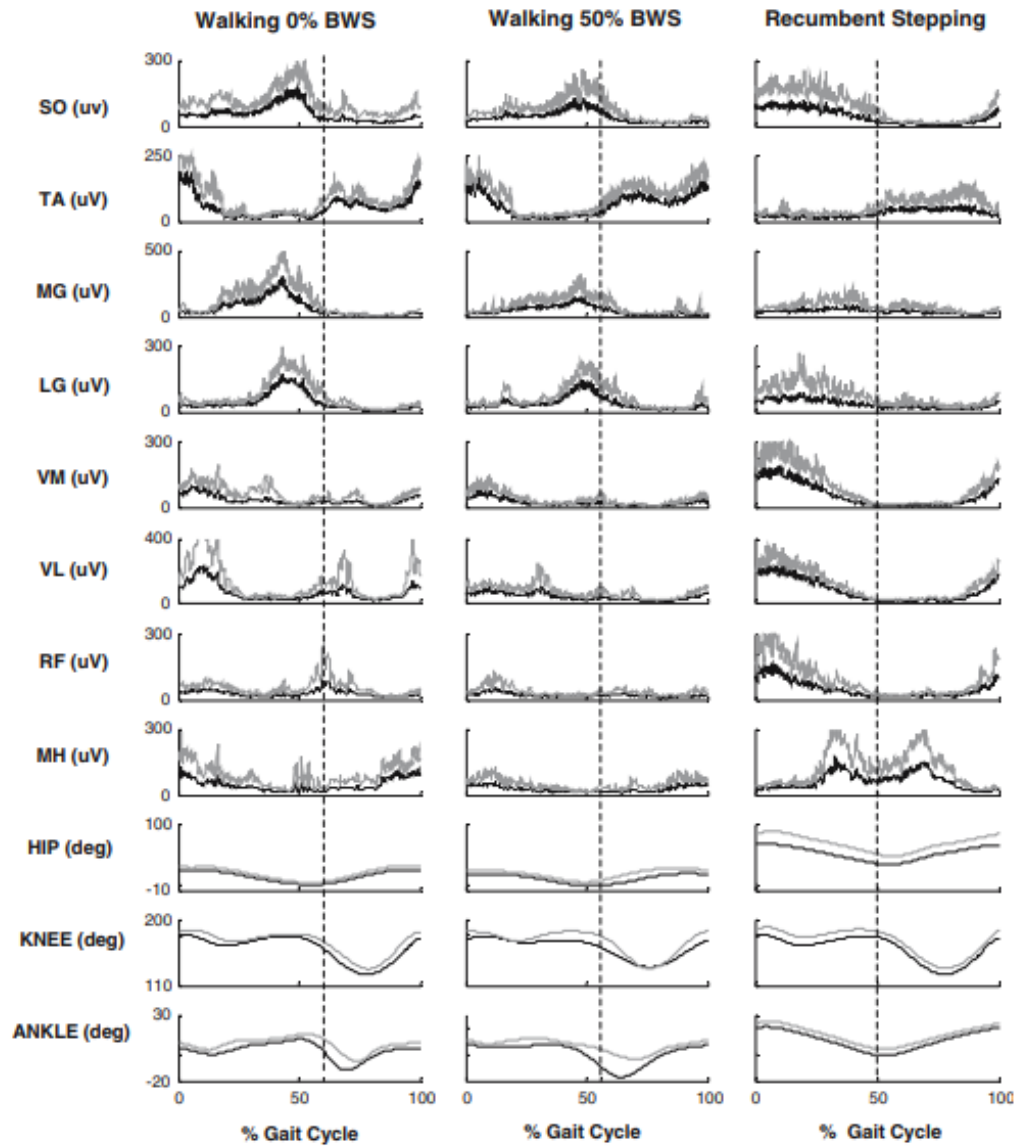
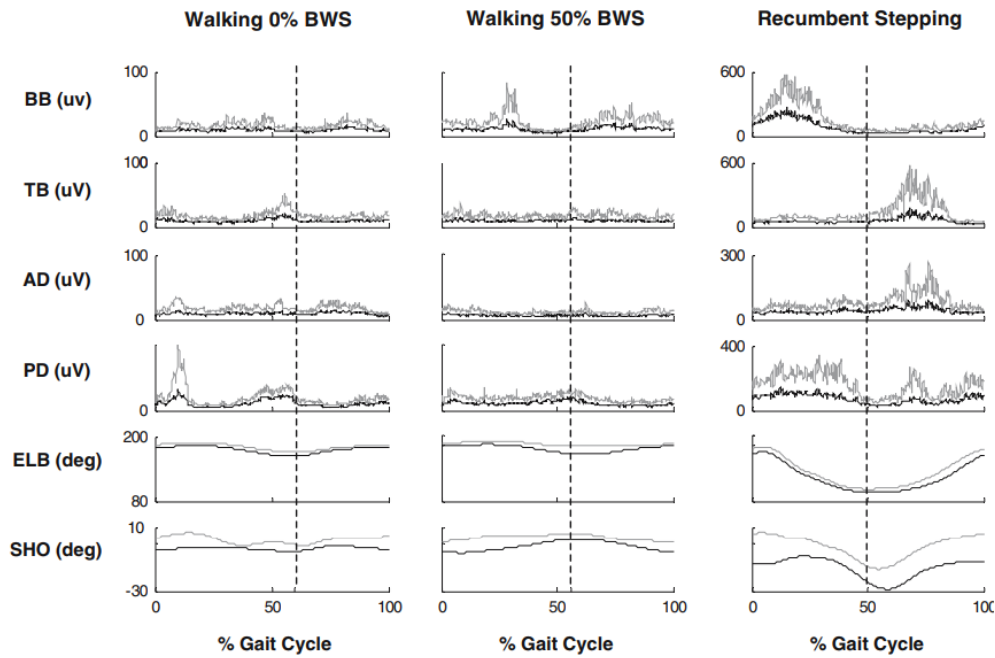Adopted from Stoloff, Zehr & Ferris, 2007. Averaged rectified lower limb EMG and joint angle averaged over 5 step cycles: (SO: soleus, TA: tibialis anterior, MG: medial gastrocnemius, LG: lateral gastrocnemius, VM: vastus medialis, VL: vastus lateralis, MH: medial hamstring, RF: rectus femoris). Grey traces indicate one standard deviation. The dashed line represents the split between stance (extension) and swing (flexion).

APPENDIX E
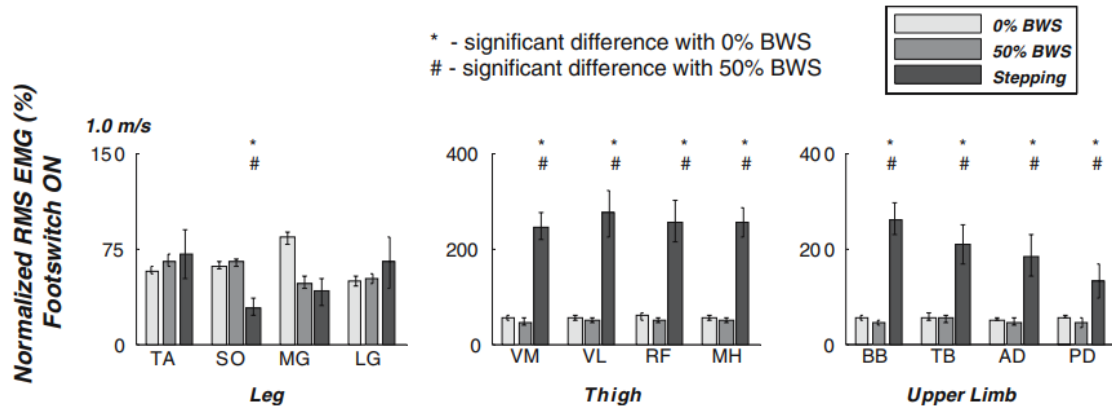
Averaged root-mean-square (RMS) EMG during stance (extension) with standard error bars for walking at 1.0 m/s and stepping at the corresponding frequency.

APPENDIX F

Adopted from Stoloff, Zehr & Ferris, 2007. Averaged rectified lower limb EMG and joint angle averaged over 5 step cycles: (BB: biceps brachii, TB: triceps brachii, AD: anterior deltoid, PD: posterior deltloid. Grey traces indicate one standard deviation. The dashed line represents the split between stance (extension) and swing (flexion).
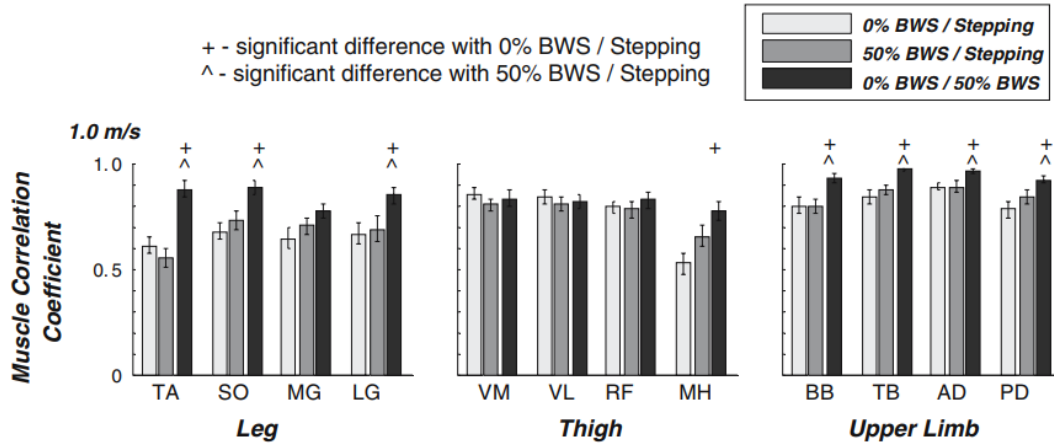
APPENDIX G

Adopted from Stoloff, Zehr & Ferris, 2007. Averaged (n = 10) correlation coefficient for muscle EMG for walking at 1.0 m/s and stepping frequency.

REFERENCES

Arabadzhiev, T. I., Dimitrov, V. G., & Dimitrov, G. V. (2014). The increase in surface EMG could be a misleading measure of neural adaptation during the early gains in strength. *European journal of applied physiology, 114*(8), 1645-1655.

Barrett, K. E., Barman, Susan M., Biotano, Scott., Brooks, Heddwen L. (2012). *Ganong's Review of Medical Physiology* (24 ed.): Tata McGraw-Hill

Billinger, S., Loudon, J., & Gajewski, B. (2008). Validity of a total body recumbent stepper exercise test to assess cardiorespiratory fitness. *The Journal of Strength & Conditioning Research, 22*(5), 1556-1562.

Benjamin, E. J., Blaha, M. J., Chiuve, S. E., Cushman, M., Das, S. R., Deo, R., Floyd, J., Fornage, M., Gillespie, C., Isasi, C. (2017). Heart disease and stroke statistics-2017 update: a report from the American Heart Association. *Circulation, 135*(10).

Billinger, S., Van Swearingen, E., McClain, M., Lentz, A., & Good, M. (2012). Recumbent stepper submaximal exercise test to predict peak oxygen uptake. *Medicine and science in sports and exercise, 44*(8), 1539.

Borg, G. A. (1982). Psychophysical bases of perceived exertion. *Med sci sports exerc, 14*(5), 377-381.

Brown, T. G. (1911). The intrinsic factors in the act of progression in the mammal. *Proceedings of the Royal Society of London. Series B, containing papers of a biological character, 84*(572), 308-319.

Brown, T. G. (1914). On the nature of the fundamental activity of the nervous centres; together with an analysis of the conditioning of rhythmic activity in progression, and a theory of the evolution of function in the nervous system. *The Journal of Physiology, 48*(1), 18-46.

Burdett, R. G., Borello-France, D., Blatchly, C., & Potter, C. (1988). Gait comparison of subjects with hemiplegia walking unbraced, with ankle-foot orthosis, and with Air-Stirrup® brace. *Physical Therapy, 68*(8), 1197-1203.

Cerri, G., Borroni, P., & Baldissera, F. (2003). Cyclic H-reflex modulation in resting forearm related to contractions of foot movers, not to foot movement. *Journal of Neurophysiology, 90*(1), 81-88.

Cooney, G., Dwan, K., & Mead, G. (2014). Exercise for depression. *Jama, 311*(23), 2432-2433.

Dalleck, L. C., Borresen, E. C., Parker, A. L., Abel, K. M., Habermann, L. A., McLaughlin, S. J., & Tischendorf, J. S. (2011). Development of a metabolic equation for the NuStep recumbent stepper in older adults. *Perceptual and Motor Skills, 112*(1), 183-192.

de Kam, D., Rijken, H., Manintveld, T., Nienhuis, B., Dietz, V., & Duysens, J. (2013). Arm movements can increase leg muscle activity during submaximal recumbent stepping in neurologically intact individuals. *Journal of Applied Physiology, 115*(1), 34-42.

De Luca, C. J. (1997). The use of surface electromyography in biomechanics. *Journal of applied biomechanics, 13*(2), 135-163.

Dietz, V. (2002). Do human bipeds use quadrupedal coordination? *Trends in neurosciences, 25*(9), 462-467.

Dietz, V., & Berger, W. (1984). Interlimb coordination of posture in patients with spastic paresis: impaired function of spinal reflexes. *Brain, 107*(3), 965-978.

Dinneen, L., Blakesley, B. (1973). Algorithm AS 62: a generator for the sampling distribution of the Mann-Whitney U statistic. *Journal of the Royal Statistical Society. Series C (Applied Statistics), 22*(2), 269-273.

González-Camarena, R., Carrasco-Sosa, S., Román-Ramos, R., Gaitán-González, M. J., Medina-baÑuelos, V., & Azpiroz-Leehan, J. (2000). Effect of static and dynamic exercise on heart rate and blood pressure variabilities. *Medicine and science in sports and exercise, 32*(10), 1719-1728.

Greenhouse, S. W., & Geisser, S. (1959). On methods in the analysis of profile data. *Psychometrika, 24*(2), 95-112.

Hermens, H. J., Freriks, B., Merletti, R., Stegeman, D., Blok, J., Rau, G., Disselhorst-Klug, C., Hägg, G. (1999). European recommendations for surface electromyography. *Roessingh Research and Development, 8*(2), 13-54.

Hiebert, G., Whelan, P., Prochazka, A., & Pearson, K. (1996). Contribution of hind limb flexor muscle afferents to the timing of phase transitions in the cat step cycle. *Journal of Neurophysiology, 75*(3), 1126-1137.

Huang, H. J., & Ferris, D. P. (2004). Neural coupling between upper and lower limbs during recumbent stepping. *Journal of Applied Physiology, 97*(4), 1299-1308.

Huang, H. J., & Ferris, D. P. (2009a). Upper and lower limb muscle activation is bidirectionally and ipsilaterally coupled. *Medicine and science in sports and exercise, 41*(9), 1778.

Huang, H. J., & Ferris, D. P. (2009b). Upper limb effort does not increase maximal voluntary muscle activation in individuals with incomplete spinal cord injury. *Clinical Neurophysiology, 120*(9), 1741-1749.

Jakobi, J. M., & Chilibeck, P. D. (2001). Bilateral and unilateral contractions: possible differences in maximal voluntary force. *Canadian journal of applied physiology, 26*(1), 12-33.

Kandel, E. R., Schwartz, James, H., Jessell, Thomas, M., Siegelbaum, Steven, A., Hudspeth, AJ.
. (2013). *Principles of Neural Science* (5th ed.): McGraw Hill Medical.

Kao, P.-C., & Ferris, D. P. (2005). The effect of movement frequency on interlimb coupling
during recumbent stepping. *Motor control, 9*(2), 144-163.

Kenney, W. L., Wilmore, J. H., & Costill, D. L. (2015). *Physiology of Sport and Exercise* (6th
ed.): Human Kinetics.

Kochanek, K. D., Murphy, S. L., Xu, J., & Arias, E. (2014). Mortality in the united states, 2013.
In: US Department of Health and Human Services, Centers for Disease Control and
Prevention, National Center for Health Statistics.

Kriellaars, D., Brownstone, R., Noga, B. R., & Jordan, L. (1994). Mechanical entrainment of
fictive locomotion in the decerebrate cat. *Journal of Neurophysiology, 71*(6), 2074-2086.

Looney, M. A., & Rimmer, J. H. (2003). Aerobic exercise equipment preferences among older
adults: a preliminary investigation. *Journal of applied measurement, 4*(1), 43-58.

Macko, R. F., Ivey, F. M., Forrester, L. W., Hanley, D., Sorkin, J. D., Katzel, L. I., Silver, K.H.,
Goldberg, A. P. (2005). Treadmill exercise rehabilitation improves ambulatory function
and cardiovascular fitness in patients with chronic stroke: a randomized, controlled trial.
Stroke, 36(10), 2206-2211.

Macleod, C. A., Conway, B. A., Allan, D. B., & Galen, S. S. (2014). Development and validation
of a low-cost, portable and wireless gait assessment tool. *Medical Engineering and
Physics, 36*(4), 541-546.

Meyers, L. S., Gamst, G., & Guarino, A. J. (2006). *Applied multivariate research: Design and
interpretation*: Sage.

Mozaffarian, D., Benjamin, E. J., Go, A. S., Arnett, D. K., Blaha, M. J., Cushman, M., . . .

Fullerton, H. J. (2015). Heart disease and stroke statistics—2016 update: a report from

the American Heart Association. *Circulation*, CIR. 0000000000000350.

Olney, S. J., Monga, T. N., & Costigan, P. A. (1986). Mechanical energy of walking of stroke

patients. *Archives of physical medicine and rehabilitation, 67*(2), 92-98.

Olney, S. J., & Richards, C. (1996). Hemiparetic gait following stroke. Part I: Characteristics.

Gait & posture, 4(2), 136-148.

Palmer-McLean, K., & Harbst, K. B. (2003). Stroke and Brain Injury. In J. L. Durstine, Moore,

G.E. (Ed.), *ACSM's Exercise Management for Persons with Chronic Diseases and

Disabilities* (pg. 238-246): Human Kinetics.

Page, S. J., Levine, P., Teepen, J., & Hartman, E. C. (2008). Resistance-based, reciprocal upper

and lower limb locomotor training in chronic stroke: a randomized, controlled crossover

study. *Clinical rehabilitation, 22*(7), 610-617.

Pardo, V., Caldwell, J., DeAngelo, S., Rohkohl, A., Swadling, A.Galen, S.. (2018, Feb.). *Effects

of Visual Feedback on Muscle Recruitment and Retention of Force Production During

Recumbent Stepping in Individuals With Stroke.* APTA – Combined Sections Meeting.

New Orleans, LA.

Pearson, K., Gordon, E. (2013). Locomotion. In E. R. Kandel, Schwartz, James, H., Jessell,

Thomas, M., Siegelbaum, Steven, A., Hudspeth, A. (Ed.), *Principles of Neural Science*

(5th ed., pp. 813-834). United States of America: The McGraw-Hill Companies.

Pollock, A., Baer, G., Pomeroy, V., & Langhorne, P. (2004). Physiotherapy treatment approaches

for the recovery of postural control and lower limb function following stroke. Cochrane

Database of Systematic Reviews, 1, 1-31.

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Powers, C. M., Landel, R., & Perry, J. (1996). Timing and intensity of vastus muscle activity during functional activities in subjects with and without patellofemoral pain. *Physical Therapy, 76*(9), 946-955.

Reaz, M. B., Hussain, M., & Mohd-Yasin, F. (2006). Techniques of EMG signal analysis: detection, processing, classification and applications. *Biological procedures online, 8*(1), 11-35.

Sackley, C., Baguley, B., Gent, S., & Hodgson, P. (1992). The use of a balance performance monitor in the treatment of weight-bearing and weight-transference problems after stroke. *Physiotherapy, 78*(12), 907-913.

Schmidt, R. A., & Lee, T. D. (2011). *Motor control and learning : a behavioral emphasis* (5th ed.). Champaign, IL: Human Kinetics.

Schmidt, R. A., Lee, T., Winstein, C., Wulf, G., & Zelaznik, H. (2018). *Motor Control and Learning,* (6th ed). Champaign, IL: Human kinetics.

Stoloff, R., Zehr, E., & Ferris, D. (2007). Recumbent stepping has similar but simpler neural control compared to walking. *Experimental brain research, 178*(4), 427-438.

Teepen, J., Baltzer, K., Dunning, K., Levine, P., & Page, S. (2005). Stepping Out: Improving Community Ambulation and Function After Stroke. *Journal of Neurologic Physical Therapy, 29*(4), 215.

Vincent, W., & Weir, J. (2012). *Statistics in kinesiology*. Champaign, IL: Human Kinetics.

von Schroeder, H. P., Coutts, R. D., Lyden, P. D., & Nickel, V. L. (1995). Gait parameters following stroke: a practical assessment. *Journal of rehabilitation research and development, 32*(1), 25.

Wannier, T., Bastiaanse, C., Colombo, G., & Dietz, V. (2001). Arm to leg coordination in humans during walking, creeping and swimming activities. *Experimental brain research, 141*(3).

Whelan, P., Hiebert, G., & Pearson, K. (1995). Stimulation of the group I extensor afferents prolongs the stance phase in walking cats. *Experimental brain research, 103*(1), 20-30.

Wilson, D. R., Mattlage, A. E., Seier, N. M., Todd, J. D., Price, B. G., Kwapiszeski, S. J., Vardey, R., Billinger, S. A. (2017). Recumbent Stepper Submaximal Test response is reliable in adults with and without stroke.

Yang, Y. R., Yen, J. G., Wang, R. Y., Yen, L. L., & Lieu, F. K. (2005). Gait outcomes after additional backward walking training in patients with stroke: a randomized controlled trial. *Clinical rehabilitation*, *19*(3), 264-273.

Yavuzer, G., Eser, F., Karakus, D., Karaoglan, B., & Stam, H. J. (2006). The effects of balance training on gait late after stroke: a randomized controlled trial. *Clinical rehabilitation*, *20*(11), 960-969.

Zehr, E. P., Balter, J. E., Ferris, D. P., Hundza, S. R., Loadman, P. M., & Stoloff, R. H. (2007). Neural regulation of rhythmic arm and leg movement is conserved across human locomotor tasks. *The Journal of Physiology, 582*(1), 209-227.

ABSTRACT

THE EFFECT OF TREADMILL VS. NUSTEP RECUMBENT CROSS TRAINER ON GAIT AND LOWER EXTREMITY ELECTROMYOGRAPHY AFTER CHRONIC STROKE

by

NICHOLAS J. SIEKIRK

August 2018

Advisor: Dr. Qin Lai

Major: Kinesiology

Degree: Doctor of Philosophy

Pilot Part 1: The purpose was to investigate the effect of a perceived exertion based cadence on lower extremity muscle activation, as measured by surface electromyography (EMG) on a recumbent cross trainer. The purpose of this investigation was to study the EMG activity of 12 lower extremity muscles during five different stepping protocols; perceived exertion based self-selected (SS) cadence with level 1 resistance (SSL1), SS cadence with level 8 resistance (SSL8), +20% SS cadence (SS+20), -20% SS cadence (SS-20), and at a set 80 steps per minute at resistance level 1 (80L1). In order to determine SS cadence each participant performed 10 minutes (min) pretest of stepping with a rate of perceived exertion of 12 to16. Participants then performed all five protocols in randomized order with 5 mins of rest between each protocol. Both mean (mEMG) and peak (pEMG) normalized amplitudes were recorded from the rectus femoris (RF), vastus medialis oblique (VMO), semitendinosus (ST), tibialis anterior (TA), medial gastrocnemius (MG) and soleus (SOL) bilaterally. Healthy participant's (n = 22, aged 23.52 ± 4.23 years) SS cadence was 123.86 ± 18.12 steps/min. SSL8 and SS+20 produced the highest mEMG and pEMG in all muscle groups ($p < .05$). Generally, SSL1, SS-20 and 80L1 did not

differentially activate muscles based on mEMG and pEMG. The present findings indicated that increased resistance (SSL8) and increased step cadence (SS+20) resulted in the greatest activation of lower extremity muscles during recumbent stepping.

Pilot Part 2: Muscle recruitment becomes more efficient as a result of task-specific training. Although the muscle activity of recumbent stepping has been studied previously, it remains unclear if an individual alters recruitment as they acclimate to the stepping motion. The purpose of this study was to measure the change in EMG activity between minute (min) 2 and min 4 of a 5 min stepping bout. EMG was measured bilaterally at 6 separate lower extremity muscles during five different stepping protocols (self-selected level 1 [SSL1], self-selected level 8 [SSL8], +20% self-selected [SS+20], -20% self-selected [SS-20], and 80 steps per min resistance level 1 [80SL1]). 22 healthy male and female adults (aged = 23.52 ± 4.23 years) signed an informed consent prior to the study. Self-selected cadence was established during 10 mins of stepping with a RPE between 12 and 16. Participants then performed all 5-min protocols in randomized order with 5 min of rest between each. Due to parametric violations, mean EMG (mEMG) and peak EMG (pEMG) were analyzed with non-parametric tests. A 1 x 4 Friedman test was conducted to determine statistical significant difference in mEMG and pEMG between min 2 and min 4 of stepping in each muscle. Following a statistically significant Friedman test (p<.05), a post hoc Wilcoxon Signed Rank test (WSRT) was conducted. Participants' self-selected cadence was 126.80 ± 17.87 steps/min. WSRT showed a significant reduction in mEMG activation at min 4 in 5 muscles (rectus femoris [RF], vastus medialis oblique [VMO], semitendinosus [ST], tibialis anterior [TA]) at 80SL1, VMO at SS+20% and RF and VMO at SS-20, (p<.01). WSRT showed a significant reduction in pEMG activation of VMO at min 4 in all protocols, but higher pEMG at min 4 in ST in SSL1 and SSL8, soleus in SSL1 and TA in SS+20.

Results indicate a higher level of learning, as measured by the reduction of mEMG during min 4 at protocols below the subject's self-selected pace. At a significantly lower cadence, it is presumed that a new motor pattern was acquired to adapt to the stepping demands.

NuStep Cross Trainer vs. Treadmill: The NuStep Recumbent Cross Trainer relies on similar neural networks as gait. Therefore, neurologically impaired individuals may improve walking ability after exercise on the NuStep. The purpose of this investigation was to measure the effect of two exercise mode (NuStep Recumbent Cross Trainer vs. Treadmill) on intra-exercise muscle activity (as measured by mean electromyography) and post exercise spatial and temporal gait parameters during a 3 x 10m hallway walk. 34 participants were divided into two groups; chronic stroke (10 ± 5 years post cerebral vascular accident) and an age and sex matched control. In order to determine SS cadence each participant performed 10 minutes (min) pretest of stepping with a rate of perceived exertion (RPE) of 12 to16. Participants then performed two 5 minute exercise bouts on each mode. Mean electromyography (mEMG) values were normalized to maximum voluntary contraction and were recorded from the rectus femoris (RF), vastus medialis oblique (VMO), semitendinosus (ST), tibialis anterior (TA), medial gastrocnemius (MG) and soleus (SOL) bilaterally. Stroke (n = 15) and healthy (n = 19) did not differ in age (*Mdn*: 66 vs. 57, respectively) or BMI (Stroke: $M = 27.02$, $SD = 4.57$ vs. Healthy: $M = 26.46$, $SD = 4.63$), $p<.05$. Healthy participants were stronger at all joints, $p<.025$. Goniometer data was measured at the hip, knee and ankle. Range of motion change (ΔROM) was calculated (maximum-minimum degree; ΔROM). There was no statistical differences between the TM and NS in ΔROM. The TM elicited a higher mEMG on a majority of the observed muscles. The NuStep Cross Trainer immediately improved gait parameters (i.e. decreased stance % and increased swing %) on the non-affected leg following a 5-minute stepping protocol.

AUTOBIOGRAPHICAL STATEMENT

Nicholas (Nick) J. Siekirk completed his Ph.D. at Wayne State University (Detroit, MI, USA) under the direction of Dr. Qin Lai (Texas A&M University) as part of the Motor Behavior Lab in the Division of Kinesiology Healthy and Sports Sciences. Nick was awarded the prestigious Rumble Fellowship from the College of Education in 2014. Nick later taught coursework in motor behavior, biomechanics and anatomy/physiology. During his tenure, he had the privileged opportunity to become a Research Assistant in the Neurotech Laboratory (Physical Therapy Program, Eugene Applebaum College of Pharmacy and Health Sciences) under Drs. Sujay Galen (University of Strathclyde, Glasgow, UK) and Victoria Pardo (University of Indianapolis). Furthermore, in 2012, Nick received his Masters of Science in Exercise Science from Oakland University (Rochester, MI, USA) under Drs. Brian Goslin (University of Cape Town, South Africa), Tamara Hew-Butler (University of Cape Town in South Africa) and Charles Marks (University of Michigan). As an undergraduate Nick attended Adrian College (Adrian, MI, USA) under Dr. Adam Coughlin (Michigan State University) and completed his Bachelors of Arts in Exercise Science in 2010. Nick was hired as an Assistant Professor at Georgia Southern University (Statesboro, GA, USA) in 2018.