Dropped Foot Impairment Post Stroke: Gait Deviations and the Immediate Effects of Ankle-Foot Orthotics and Functional Electrical Stimulation

by

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Abstract

Individuals with stroke often demonstrate impaired ankle-foot function, commonly termed dropped foot that affects their ability to walk safely at home and within their community. While interventions are available to improve gait function, they have inconsistency demonstrated positive effects due to the lack of evidence-based practice guidelines and a limited understanding of the mechanisms leading to dropped foot. The aim of this dissertation was to 1) determine the relationship between dropped foot gait deviations and impaired sensorimotor control, 2) compare gait biomechanics between stroke survivors with and without dropped foot impairment, and 3) evaluate the immediate effects of an ankle-foot orthotic (AFO) and functional electrical stimulation (FES) device among stroke survivors with dropped foot impairment. Our evaluation combined standardized clinical measures of ankle-foot function (i.e. sensorimotor control, strength, spasticity and range of motion) and gait analysis using advanced laboratory techniques (i.e. electromyography and electrical goniometers) to quantify mechanisms of dropped foot impairment. Fifty-five stroke survivors completed the assessment prior to discharge from inpatient rehabilitation. Individuals with poor generation of isometric dorsiflexor force and reduced passive ankle range of motion were likely to demonstrate greater plantarflexion in swing and limited stance phase ankle joint excursion, respectively. Results from the gait analysis revealed a delayed onset and reduced activation time of the ankle dorsiflexors, and decreased co-activation time in the stance phase as possible mechanisms leading to dropped foot. A detailed case series was performed with four stroke survivors with dropped foot currently using an AFO. Application of an AFO immediately improved peak dorsiflexion in the swing phase and limited ankle range of motion during stance. When walking with the FES device, individuals with moderate dorsiflexor muscle weakness improved their ankle position at initial contact and increased peak dorsiflexion during stance, while no significant changes were observed among individuals with greater impairment. Overall, the results highlighted individual differences in response to interventions aimed at improving dropped foot gait deviations. These findings contribute to a greater understanding of gait dysfunction post stroke, and may lead to the development of a more effective clinical assessment and intervention strategies to improve dropped foot impairment.

Keywords: Dropped foot, ankle-foot orthotic, function electrical stimulation, gait and stroke

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AFO	Ankle-foot orthotic
ANOVA	Analysis of variance
AROM	Active range of motion
AT	Articulating
BBS	Berg Balance Scale
CMSA	Chedoke McMaster Stroke Assessment Scale
COP	Centre of pressure
COVS	Clinical Outcomes Variable Scale
CRT	Current amplitude
DF	Dorsiflexion
EMG	Electromyography
FES	Functional electrical stimulation
fMRI	Functional magnetic resonance imaging
GC	Gait cycle
MAS	Modified Ashworth Scale
MG	Medial gastrocnemius
MRC	Medical Research Council - manual muscle test
MVC	Maximum voluntary contraction
NA	Non-articulating
ODFS	Odstock Dropped Foot Stimulator
PF	Plantarflexion
PLS	Posterior spring leaf
PROM	Passive range of motion
ROM	Range of motion
SD	Standard deviation
TA	Tibialis Anterior
TENS	Transcutaneous electrical stimulation
TMS	Transcranial magnetic stimulation

CHAPTER 1

Literature Review

1.1 Introduction

Walking is one of the most important physical activities performed by humans in terms of efficiently moving within our environment and helping maintain healthy body structures and functions. When walking ability is compromised by disease or injury, health-related quality of life may decline due to reduced activity and participation (Mayo et al., 1996). Although approximately 85% of individuals survive a stroke (Heart & Stroke Foundation, 2002), a large proportion has problems with mobility that limit their ability to ambulate in the community. Given the importance of walking, it is not surprising that recovery of walking function is a goal most often stated by stroke survivors and represents a primary focus of rehabilitation programs (Brandstater, de Bruin, Gowland, & Clark, 1983). Many evaluations are performed to understand sensorimotor impairments underlying gait disorders post stroke and to develop effective evidence-based interventions. Information gained from physiological and biomechanical measures provide insight on the pathology and resultant functional outcomes. While motor impairments are thought to be the primary cause of poor walking ability (Lamontagne, Stephenson, & Fung, 2007), other factors such as sensation, perception and cognition have also been shown to influence functional mobility (Bowen et al., 2001; Lamontagne, De Serres, Fung, & Paquet, 2005; Niam, Cheung, Sullivan, Kent, & Gu, 1999).

Walking after a stroke is often characterized by slow speed, asymmetrical pattern, poor endurance (Dean, Richards, & Malouin, 2001), and difficulty adapting to task and environmental demands (Said et al., 2008). Of specific interest to this work are sensorimotor impairments at the ankle-foot complex that contribute to gait dysfunction. Impaired ankle-foot function, commonly termed dropped foot, may result in the inability to lift the foot during the swing phase of gait and reduced stability during stance. Other related gait deviations may include difficulty pushing-off to propel the leg forward and reduced knee flexion in swing (Lamontagne, Malouin, & Richards, 2001; Lamontagne, Malouin, Richards, & Dumas, 2002,). The combination of these factors leads to an increased risk of tripping and falling; therefore requires a greater amount of cognitive attention to ambulate at home and within the community. Current technological interventions to assist stroke survivors with foot clearance issues include ankle-foot orthotics (AFO) and dropped foot functional electrical stimulators (FES).

Despite the impact of ankle-foot impairments, there are no standardized guidelines for identifying individuals with dropped foot issues, and subsequently how to determine the appropriate prescription of technological interventions. This work aims to evaluate locomotor disorders of stroke survivors with dropped foot impairment, and to provide evidence on the immediate effects of dropped foot technology to assist with the development of prescription guidelines in the future.

1.2 Stroke population

In Canada, there are approximately 40,000 to 50,000 strokes per year and more than 300,000 Canadians are living with the effects of stroke (Heart & Stroke Foundation, 2002; Mayo et al., 1996). While the mortality rates of stroke have been declining, the prevalence of stroke continues to remain the same or increase over time. Of every 100 people who have a stroke, approximately 75% are left with mild to severe impairments affecting motor, sensory, cognitive, emotional and perceptual functions (Heart & Stroke Foundation, 2002). Theses stroke-related impairments impact performance and limit participation of daily physical activities. Research on hospital inpatients and community-dwelling stroke survivors has reported low levels of walking activity during everyday life (Prajapati, Gage, Brooks, & McIlroy, 2008). This finding has important implications on functional recovery and overall health status of individuals with stroke (Ivey, Macko, Ryan, & Hafer-Macko, 2005). With the low rate of stroke survivors able to ambulate safely within their community (Lord, McPherson, McNaughton, Rochester, & Weatherall, 2004), it is important to continue research directed at understanding and developing strategies to reduce the sequelae of post-stroke impairments.

1.3 Neurophysiological evaluation of gait dysfunction post stroke

The basic motor pattern for walking is generated in the spinal cord, while fine motor control involves various brain regions including motor cortex, cerebellum and brainstem (Dietz, 1996). Although it is difficult to assess the contribution of supraspinal control mechanisms involved in walking, a better understanding may provide insight on the underlying causes of gait deficits post stroke. Generally, descending pathways from the brainstem and motor cortex initiate locomotion and modify the motor pattern based on feedback from the limbs, while the cerebellum regulates speed, range and coupling of movements (Kandell, Schwartz, & Jessell, 2000). Additionally, the motor cortex is involved in visual-motor coordination (Kandell et al., 2000). Results from transcranial magnetic stimulation (TMS) studies show excitation in motor cortex areas involved in controlling ankle function during walking (Capaday, Lavoie, Barbeau, Schneider, & Bonnard, 1999). These findings are consistent with impaired ankle control in the swing phase of gait after injury to the motor cortex (Knutsson & Richards, 1979). Previous research has established a relationship between lesion locations and gait asymmetry among chronic stroke survivors (Alexander et al., 2009). A subtraction lesion analysis was used to highlight damaged areas, specifically the inferior portion of the posterolateral putamen, more frequently observed in patients with asymmetrical gait (Alexander et al., 2009). These findings are supported by other research linking the putamen to poor functional outcomes among chronic stroke survivors (C. L. Chen, Tang, Chen, Chung, & Wong, 2000; Miyai, Blau, Reding, & Volpe, 1997). Dobkin and colleagues reported activation of the putamen region during isolated active ankle dorsiflexion with functional magnetic resonance imaging (fMRI) of healthy adults (Dobkin, 2004). This movement is an important component of the flexion synergy during the swing phase of gait to achieve successful foot clearance. Therefore, the putamen may play an important role in locomotor movements, as well as functional reorganization during recovery post stroke.

Conversely, a bottom-top approach utilizing peripheral electrophysiological measures of muscle activation patterns, afferent feedback and cutaneous responses can provide information on impaired gait function. Electromyography (EMG) studies have revealed altered motor patterns of the lower extremity muscles in hemiparetic gait. Observations show an overall decreased in EMG amplitude and abnormal timing of muscle activation throughout the gait cycle (Lamontagne et al., 2007). Reduced EMG amplitudes may be attributed to changes at the muscle fiber level; studies have reported a decrease in the number of functioning motor units, change in fiber typing and reduced motor firing rate (Arene & Hidler, 2009). Knutsson and Richards classified chronic stroke survivors into three types of disturbed motor control patterns observed on the paretic side during gait; 1) premature activation of the ankle plantarflexors muscles in the stance phase, 2) abnormally low activation for most muscle groups, and 3) coactivation of several muscle groups (Knutsson & Richards, 1979). This classification system is limited as EMG profiles are complex and more than one type may occur in a single patient. Abnormal EMG patterns have also been observed on the non-paretic side, possibly due neurological injury and/or biomechanical compensations. Coactivation of opposing muscles is a functionally important mechanism that contributes to joint stiffness and postural stability in stance. Lamontagne et al. 2000 reported greater coactivation time of agonist-antagonists ankle muscles on the non-paretic limb during double support phases of gait and less coactivation time of the paretic ankle muscles in single support phase compared to healthy controls (Lamontagne, Richards, & Malouin, 2000). This pattern of coactivation was related to slow gait speed, poor postural stability and decreased ankle strength (Lamontagne et al., 2000). A cause-effect relationship has not been established as other sensorimotor impairments at the ankle joint, such as spasticity and muscle paresis, have also been correlated with poor gait performance (Lamontagne, Malouin, & Richards, 2001; Lamontagne, Malouin, Richards, & Dumas, 2002). Changes in EMG patterns with recovery of gait performance may not be dependent on improved temporal activation towards a 'normal' profile (Den Otter, Geurts, Mulder, & Duysens, 2007). Exploring EMG profiles during gait is important to understand and monitor changes in locomotor performance due to impaired and compensatory neuromuscular strategies.

Afferent feedback generated during a continuous walking bout contributes to ongoing modulation of muscle activation. After stroke, changes in the excitatory and/or inhibitory input from group Ia, Ib, II and cutaneous afferent signals may underlie disturbances to movement patterns during gait. Several studies have demonstrated modulation of the soleus H-reflex during the gait cycle of healthy adults; increased during stance and inhibited during the swing phase (Capaday & Stein, 1986; Capaday & Stein, 1987; Ferris, Aagaard, Simonsen, Farley, & Dyhre-Poulsen, 2001; Schneider, Lavoie, & Capaday, 2000). Mechanical perturbations to the stretch reflex pathway during treadmill walking have demonstrated impaired inhibition of the soleus short latency reflex after stroke (Berger, Horstmann, & Dietz, 1984). This facilitated input from group Ia monosynaptic pathways may combine responses from group II and Ib mediated reflex pathways (Maupas, Marque, Roques, & Simonetta-Moreau, 2004; Nardone & Schieppati, 2005). Specifically, facilitation has been observed in the Ib inhibitory pathways from the medial gastrocnemius to soleus on the paretic side during rest (Delwaide & Oliver, 1988), along with lack of reciprocal inhibition of the peroneal nerve to soleus (Crone, Johnsen, Biering-Sorensen, & Nielsen, 2003). Likewise, facilitation of group II pathways among persons with stroke was increased on the paretic side during rest (Marque, Simonetta-Moreau, Maupas, & Roques, 2001). Since these pathways are modified at rest due to spasticity, it is hypothesized that altered feedback may occur during gait performance (Marque et al., 2001). Evidence has demonstrated a flexor withdrawal reflex in response to cutaneous afferent stimulation of the superficial peroneal and sural nerves in healthy adults and other neurological populations (Knikou, 2007; Knikou, 2010). Persons with stroke exhibit a similar reflex response to stimulation during the swing phase with smaller changes in joint kinematics; however suppression of extensor muscle activation was observed in stance (Zehr, Fujita, & Stein, 1998). The impact of ankle plantarflexor spasticity on gait performance post stroke appears to involve different afferent feedback pathways and identifying specific mechanisms remains challenging due to considerable variation within the population.

1.4 Biomechanical evaluation of gait dysfunction post stroke

Several studies have reported altered spatial and temporal characteristics of hemiparetic gait relative to healthy controls (G. Chen, Patten, Kothari, & Zajac, 2005; Hsu, Tang, & Jan, 2003; Kim & Eng, 2003; Olney & Richards, 1996; Patterson et al., 2008). Preferred or self-selected gait velocity of stroke survivors ranges from approximately 0.18 to 1.03 m/s depending on severity of impairments, whereas healthy adults of similar ages average about 1.4 m/s (Bohannon, 1997; Brandstater et al., 1983). Temporal asymmetry (i.e. stance and/or swing time) appears to be more prevalent compared to spatial asymmetry (i.e. step length), and has been correlated to reduced paretic limb single support time to return to a more stable double support phase (Patterson et al., 2008). Step length asymmetry is more likely to appear among stroke survivors who exhibit severe temporal asymmetry (Patterson et al., 2008) and tends to vary in direction (Kim & Eng, 2003). Step width was greater compared to healthy adults at matched walking speeds to possibly compensate for poor balance control (G. Chen et al., 2005). Stroke survivors with severe temporal asymmetry were likely to have a slower gait velocity and greater lower limb motor impairment (Kim & Eng, 2003; Patterson et al., 2008). When compared to healthy controls walking at a matched slow speed, a longer proportion of time spent in the stance phase was observed for both limbs (G. Chen et al., 2005). Gait velocity and temporal-spatial symmetry are frequently used as indicators of locomotor function to monitor gait performance and evaluate the effects of rehabilitation strategies.

Among healthy individuals, the magnitude of joint excursions in the sagittal plane is positively related to gait velocity (D. A. Winter, 1991). Therefore, stroke survivors who walk at a slower speed will show reduced sagittal peak joint excursions for both lower limbs. Additional kinematic deviations of hemiparetic gait have been reported to provide insight on underlying impairments and related compensatory strategies not due to slow walking speeds. Typical characteristics on the paretic side may include increased ankle plantarflexion at initial contact, knee hyperextension at mid-stance, less hip extension at toe-off, reduced knee flexion and ankle dorsiflexion during the swing phase (G. Chen et al., 2005; Kim & Eng, 2004; Olney & Richards, 1996). In the frontal plane, deviations consist of increased pelvic hiking, hip abduction and ankle inversion during the swing phase for the paretic limb (Kuan, Tsou, & Su, 1999). Additionally, the paretic hip and ankle may display increased external rotation throughout the gait cycle (Kuan et al., 1999). These kinematic deviations are consistent with two main impairments on the paretic side: 1) difficulty with swing initiation and forward propulsion, and 2) poor single limb balance control. A considerable amount of variation in lower limb joint kinematics profiles has been identified across persons with stroke, along with different kinetic strategies to achieve similar movement outcomes (Kim & Eng, 2004).

Kinetic measures of moments and forces generated in the lower limb joints provide information to understand outcome movement patterns. Generally the amplitude of lower limb moment and power profiles are reduced on the paretic side compared to the non-paretic side and healthy controls (Olney, Griffin, Monga, & McBride, 1991). However, joint kinetic profiles in the sagittal plane are strongly related to walking speed and functional capacity. Specifically slow walkers display reduced power for the hip flexors at pull-off, ankle plantarflexors during push-off and knee extensors in the stance phase (Kim & Eng, 2004; Olney et al., 1991). These factors result in a lower kinetic energy on the paretic side consistent with inadequate leg propulsion and increased energy cost during the swing phase (G. Chen et al., 2005). The mechanical energy cost continues to increase when the trunk is raised during the swing phase due to pelvic hiking of the paretic limb to compensate for reduce knee flexion (G. Chen et al., 2005). In contrast, increased propulsion observed on the non-paretic side is the result of greater kinetic energy at toe-off, possibly to reduce time spent in single limb support on the paretic side (G. Chen et al., 2005). These findings are consistent with low amplitude impulse anterior-posterior ground reaction forces on the paretic side at push-off (Bowden, Balasubramanian, Neptune, & Kautz, 2006). Stroke survivors who walk faster demonstrate large positive power bursts bilaterally for the hip extensors in early stance and paretic hip flexors in pre-swing compared to healthy controls (Olney et al., 1991). More detailed study is required to examine changes in kinetic profiles between limbs with motor recovery and improved gait velocity.

Centre of pressure (COP) patterns under the feet can provide information on forward progression and lateral stability during the stance phase. Stroke survivors walking at their comfortable speed displayed an asymmetrical displacement in the COP trajectory between limbs correlated to severity of motor impairment (Chisholm, Perry, & McIlroy, 2011). As well, greater COP variability was reported for the non-paretic limb possibly indicating difficulty with forward propulsion of the paretic limb (Chisholm et al., 2011). Mizelle and colleagues, reported bilateral COP variables as good predictors of gait velocity post stroke, especially variability of COP displacement (Mizelle, Rodgers, & Forrester, 2006). Other observations from studies examining pressure distribution patterns include less pressure under the lateral forefoot compared to healthy controls (Meyring, Diehl, Milani, Hennig, & Berlit, 1997) and shorter peak pressure time on the paretic side among slower walkers (Titianova, Mateev, Peurala, Sivenius, & Tarkka, 2005).

Biomechanical measures of gait performance are useful for describing changes in joint motions and understanding how forces generated by muscles produce the outcome movement patterns. In combination with electrophysiological measures, information can be interpreted to understand pathological mechanisms of gait disorders post stroke and develop effective rehabilitation strategies to improve function.

1.5 Dropped Foot Impairment

Dropped foot is described clinically as poor ankle dorsiflexion during the swing phase along with a forefoot or flat-foot initial contact in stance (Burridge, Taylor, Hagan, Wood, & Swain, 1997). Stroke related impairments causing dropped foot include weakness of the ankle dorsiflexor muscles and increased spasticity of the ankle plantarflexor muscles (Burridge, Taylor, Hagan, Wood, & Swain, 1997). The challenges with understanding how dropped foot impairs gait function are two-fold; 1) there is no standardized method to assess dropped foot, and 2) different underlying impairments may result in various joint kinematic deviations and EMG profiles. This makes it difficult for clinicians to develop targeted and effective intervention strategies. Stroke survivors with dropped foot may be prescribed an ankle-foot orthotic (AFO) and/or functional electrical stimulation (FES) device to improve gait kinematics in the swing phase.

1.6 Ankle-foot orthotics

An AFO is often recommended to minimize kinematic gait deviations caused by altered sensorimotor function at the ankle and knee joints post stroke. In particular, AFOs manipulate the foot's position to provide medial-lateral stability at the ankle during the stance phase, and limit plantarflexion to assist with heel strike at initial contact and toe clearance in the swing phase, thus potentially reducing the risk of falling. Previous work reported that approximately 22% of stroke survivors required an AFO at discharge from inpatient rehabilitation (Teasell, McRae, Foley, & Bhardwaj, 2001). Additionally, these individuals scored lower on clinical indices of motor control and balance compared to non-users at admission and discharge (Teasell et al., 2001). Typical clinical indications for an AFO prescription include ankle dorsiflexion weakness and/or plantarflexion spasticity that contribute to significant gait deviations compromising walking safety. A challenge in prescribing an AFO is finding a balance between providing maximum stability and assistance to various joints, while minimizing restrictions or deviations imposed by the device.

Previous research has reported positive effects of different AFO types on various gait parameters, with improvements in stride length and walking speed most frequently stated (Abe, Michimata, Sugawara, Sugaya, & Izumi, 2009; Bleyenheuft, Caty, Lejeune, & Detrembleur, 2008; Danielsson & Sunnerhagen, 2004; de Wit, Buurke, Nijlant, Ijzerman, & Hermens, 2004; Erel, Uygur, Engin Simsek, & Yakut, 2011; Hesse, Luecke, Jahnke, & Mauritz, 1996; Hesse, Werner, Matthias, Stephen, & Berteanu, 1999; Iwata et al., 2003; Nolan, Savalia, Lequerica, & Elovic, 2009; Nolan, Savalia, Yarossi, & Elovic, 2010; Nolan & Yarossi, 2011; Rao et al., 2008; Simons, van Asseldonk, Kooij, Geurts, & Buurke, 2009; Tyson & Thornton, 2001; Wang, Lin, Lee, & Yang, 2007). As well, there have been reports of improvement in functional walking test scores when using an AFO (Simons et al., 2009; Tyson & Thornton, 2001). In contrast, other studies found no difference in gait performance when using an AFO with respect to walking speed and cadence (Churchill, Halligan, & Wade, 2003; Gok, Kucukdeveci, Altinkaynak, Yavuzer, & Ergin, 2003; Lairamore, Garrison, Bandy, & Zabel, 2011). Many studies observed increased ankle dorsiflexion during the swing phase and at initial contact, along with reduced plantarflexion moment at terminal stance limiting push-off power (Bleyenheuft et al., 2008; Fatone & Hansen, 2007; Gok et al., 2003; Mulroy, Eberly, Gronely, Weiss, & Newsam, 2010). In the stance phase, ankle plantarflexion is usually limited and peak dorsiflexion will depends on flexibility of the AFO design. Restricting ankle plantarflexion may increase the knee flexion moment and vastus lateralis activity early in the stance phase to control forward rotation (Hesse et al., 1996; Lehmann, 1993). It is hypothesized that non-articulating AFOs can be used to control mild hyperextension in mid-stance and/or instability during loading at the knee joint through manipulation of external moments created by the ground reaction force vector (Fatone, Gard, & Malas, 2009). Fatone et al reported increased peak knee extensor moment in early stance with three different hinged AFOs, while reduced knee hyperextension and a tendency to delay onset of hyperextension with an AFO only occurred in stroke patients who displayed hyperextension during stance without an AFO (Fatone et al., 2009).

Other types of orthotics (i.e. 'dynamic' or posterior leaf spring) focus on energy return systems intended to improve terminal stance by allowing ankle dorsiflexion in mid-stance to absorb energy like a spring for an improved push-off power (Abe et al., 2009; Cakar, Durmus, Tekin, Dincer, & Kiralp, 2010; C. C. Chen et al., 2010; Simons et al., 2009; Wang et al., 2007). While studies with stroke survivors have been limited to spatial-temporal outcomes, evidence from the cerebral palsy literature indicates varied amplitudes for peak ankle dorsiflexion during mid-stance and reduced power generation was observed at terminal stance by restricting ankle plantarflexion (Buckon, Thomas, Jakobson-Huston, Sussman, & Aiona, 2001; Buckon et al., 2004; Van Gestel, Molenaers, Huenaerts, Seyler, & Desloovere, 2008). Therefore, the posterior leaf spring design may not improve terminal stance and the amount of ankle dorsiflexion in mid-stance may be linked to other lower limb impairments controlling forward progression.

There is an anecdotal view that continued AFO use will lead to prolonged dependence on the device and learned disuse of ankle muscles, therefore decreasing the opportunity for motor relearning. Hesse and colleagues reported reduced tibialis anterior (TA) activity and increased vastus lateralis activity after using a rigid double-stopped AFO for less than 1 week compared to barefoot walking (Hesse et al., 1999). Mulroy et al found reduced TA activity with application of a hinged AFO with plantarflexion stop compared to shoes only, while other AFO types did not change TA activation (Mulroy et al., 2010). These findings support the concern for learned disuse with AFO application among stroke survivors; however mechanical characteristics of the AFO device and variation across the population may influence the results. The effect of AFO use on muscle activation patterns require further investigation and interpretation relative to underlying pathological mechanisms.

The biomechanical effect of the AFO will depend on properties of the design, such as articulation type, force system, stiffness and range of motion permitted. Technology of AFO designs are continually evolving to address current issues with the goal of improving ankle kinematic and kinetic profiles towards 'normal'. While the majority of studies demonstrated improved ankle dorsiflexion during the swing phase with AFO application, other biomechanical outcomes that varied across studies may be explained by differences among stroke participants and AFO design. Therefore, specific investigation is required to determine clinical characteristics of stroke survivors who would benefit from each type of AFO device.

1.7 Functional electrical stimulation

FES is another intervention used to improve gait deviations due to dropped foot impairments. Dropped foot FES devices stimulate the common peroneal nerve to elicit ankle dorsiflexion with some eversion during the swing phase. In addition, the stimulation may decrease the antagonist ankle plantarflexors via reciprocal inhibition, and assist with a flexion withdrawal reflex at the knee and hip joints. Current FES systems use a heel switch or tilt sensor to trigger stimulation by detecting stance and swing phases of the gait cycle, along with surface or implanted electrodes. Systems with implanted electrodes offer more precise muscle selectivity and reduce set up time, while surface electrodes do not require surgical intervention and can be applied in early stages of rehabilitation (Popovic, Curt, Keller, & Dietz, 2001). The latter is exceptionally important as patients recover functional movements; the stimulation program and electrode placement can be continuously adjusted to meet their needs. The immediate effect observed when the stimulation is turned on is referred to as the orthotic effect, while improvement that is maintained after the stimulation is turned off is the therapeutic or carry-over effect (Thrasher & Popovic, 2008).

Application of dropped foot FES systems has demonstrated positive orthotic effects on many gait parameters, such as increased walking speed, improved symmetry index and reduced energy cost (Burridge, Taylor, Hagan, Wood, & Swain, 1997; Hausdorff & Ring, 2008; Stein et al., 2010; Taylor et al., 1999). Additionally, Kesar et al. reported that chronic stroke survivors demonstrated immediate improvement in ankle dorsiflexion during the swing phase with a surface FES system, along with reduced knee flexion during the swing phase and reduced ankle plantarflexion at toe-off (Kesar et al., 2009). FES applied to both the ankle dorsiflexors during the swing phase and ankle plantarflexors during terminal stance immediately resulted in greater swing phase knee flexion, greater ankle plantarflexion angle at toe-off and increased forward propulsion compared to stimulation of only the dorsiflexors (Kesar et al., 2009). Kesar et al also observed greater peak ankle dorsiflexion during the swing phase with a variable-frequency train compared to a constant-frequency train stimulation pattern typically used by FES systems (Kesar et al., 2010). Variable-frequency stimulation programs may produce greater muscle force rates and joint excursions during dynamic contractions versus constant-frequency, although the therapeutic effects have not been determined (Kesar et al., 2010).

Previous studies examining the long-term influence of FES systems on gait performance have demonstrated similar positive effects on walking speed and energy cost after 1-3 months (Embrey, Holtz, Alon, Brandsma, & McCoy, 2010; Hausdorff & Ring, 2008; Sabut, Lenka, Kumar, & Mahadevappa, 2010; Sabut, Sikdar, Mondal, Kumar, & Mahadevappa, 2010; Stein et al., 2010). Research by Stein and colleagues examined the long-term therapeutic effects of FES among individuals with progressive and non-progressive neurological conditions (Stein et al., 2010). Walking speed of the non-progressive group increased over the 3 month follow-up with the FES stimulation turned off and on, indicating a therapeutic and long-term effect over time (Stein et al., 2010). In contrast, Kottink et al. found no change in walking speed with the FES turned off at 4, 8, 12 and 26 weeks follow up compared to the control group receiving conventional therapy; however improvement was maintained when stimulation was turned on (Kottink et al., 2008). Chronic stroke survivors with spastic dropped foot demonstrated a similar improvement in walking speed, step length, cadence and energy cost with a 12 week rehabilitation program (60 min/day, 5 day/week) with or without 30 minutes of FES training (Sabut et al., 2010). A meta-analysis on the therapeutic effect of FES and transcutaneous electrical stimulation (TENS) post-stroke reported an improved gait speed from combining eight studies with intervention periods ranging 3-12 weeks (Robbins, Houghton, Woodbury, & Brown, 2006). The pooled results should be interpreted with caution due to different application methods and non-significant results from two FES studies. Therapeutic effect of FES on gait function remains inconclusive and may depend on specific underlying pathology, training intensity and application method.

It is proposed that the active muscle contraction from FES application results in central and peripheral changes facilitated by afferent feedback pathways for motor relearning. Findings from upper extremity TMS studies suggest that FES may have an important role in stimulating cortical sensory areas, enhancing cortical hyperexcitability and reorganization to improve motor function (Ring & Weingarden, 2007). Chronic stroke survivors demonstrated improved isometric ankle dorsiflexors strength after a 6-month trial wearing FES for daily activities (6-8 hours/day) plus walking training (1hr/day for 6 days/week) (Embrey et al., 2010). Sabut and colleagues reported improved manual muscle test scores, lower spasticity rating and greater functional recovery with 20-30 minutes of FES therapy in addition to a conventional rehabilitation program for 12 weeks (1hr/day for 5days/week) (Sabut, Sikdar, Kumar, & Mahadevappa, 2011). These findings indicate improved motor function possibly due to enhance cortical activation and reorganization of lower extremity areas.

FES offers many advantages compared to AFOs, such as active muscle contraction, improved muscle strength (Embrey et al., 2010), reduces muscle tone (Gerrits, de Haan, Sargeant, Dallmeijer, & Hopman, 2000), greater energy efficient use of proximal lower limb muscles (Winchester, Carollo, & Habasevich, 1994), and assists with motor relearning (de Kroon, van der Lee, IJzerman, & Lankhorst, 2002). However, there are a few limitations contributing to uptake of commercially available systems for long-term daily use, for example rapid muscle fatigue, greater physical effort, application training and financial cost. FES can be used to successfully improve gait function for stroke survivors who meet the clinical requirements and who are motivated to do ambulatory training with the device.

1.8 Current prescription guidelines

Prescription recommendations for AFO devices are primarily based on best practice points of leading researchers, physicians, orthotists and clinicians in the field. The International Society for Prosthetics and Orthotics (ISPO) recommendations for stroke survivors provides clinical indications for different types of orthotic devices (Appendix 1; Condie, Campbell, & Martina, 2004). The report summarized a poor level of evidence in the quality and quantity of research, and remained inconclusive towards specific prescription guidelines for orthotic treatment. Many research studies failed to report adequate details on the participant's functional status and/or design parameters of the AFO tested. This document presents other barriers to implementation in clinical practice, such as the large size and lack of general terms for non-orthotists; thus limiting knowledge translation of research findings. A recent paper by Bowers and Ross (2010) developed best practice statements intended to guide practice and promote consistency in orthotic management (Bowers & Ross, 2010). This document was designed to be a quick reference tool specifically for non-orthotics specialist that addresses issues such as service planning, screening and referral, patient assessment, biomechanical effects on gait function, and monitoring/follow-up. The screening tool outlines specific criteria to evaluate ankle joint movement in the swing and stance phases of gait via yes/no response. The challenges to implementing these guidelines include increasing awareness of orthotic information available and providing education to front line clinical staff to identify patients that may benefit from an AFO device.

General criteria for application of FES for individuals with neuromuscular disorders are available (Popovic et al., 2001). For dropped foot stimulators, the patient must be able to stand and walk either alone or with minimal assistance, and have intact peripheral nervous system (Kottink et al., 2004). Function of proximal lower limb muscles should be preserved to facilitate balance and posture during walking. Typically participants recruited for FES research are high functioning defined by their ability to independently walk a certain distance or time, and presence of mild-moderate of ankle-foot impairment (i.e. no severe plantarflexion contracture). Contraindications to FES include communication disorders, pacemakers, epilepsy, painful irritation of the skin/tissue, and limited passive range of movement. Patient selection, guided training and effective follow-up are important factors for successful FES intervention (Burridge et al., 1997; Taylor et al., 1999).

In addition to these guidelines, a successful AFO or FES prescription will depend on contextual factors, such as compliance and environmental demands. This information is essential for clinicians to give instructions to their patients and set up a review schedule. Many barriers to compliance have been identified for orthotics, such as appearance, weight, and difficulty donning/doffing independently (Beckerman, Becher, Lankhorst, & Verbeek, 1996; Tyson & Thornton, 2001). Similar issues have been identified with FES devices, for example difficulty with electrode placement and skin irritation. The effect of theses devices are commonly evaluated in a clinical setting, which typically does not capture the complexity of environmental factors encountered at home and within the community (i.e. uneven terrain, stairs, ramps and traffic conditions). It would be beneficial to include different environmental demands in the evaluation of gait and functional mobility. Additionally the regular daily activity level of patient may impact their choice of dropped foot device depending on comfort level over long durations. Most studies with a long-term follow-up period did not provided specific details on daily activities performed when using a dropped foot device. It seems likely that participants who regularly engage in physical activities, such as walking, may perform better on follow-up measures. A dose-response relationship has not been established between duration and frequency of AFO or FES use, and improved gait performance. Likewise, whether using a dropped foot device facilitates higher levels of daily walking activity remains unknown.

Walking activity may be a significant confounding factor that has not been addressed in the literature to date.

1.9 Summary

Overall there is compelling evidence that ankle-foot impairments greatly contribute to post stroke gait dysfunction. Whereas the quality of evidence supporting application of dropped foot assistive technology (i.e. AFO and FES) to improve gait function is lacking well designed controlled trials with in-depth analyses on specific impairments. Importantly, while studies have highlighted mechanisms of dropped foot impairments and positive effects of AFO and FES devices, there are a few key issues not addressed by research to date. Many studies provided insufficient details on characteristics of stroke survivors with dropped foot impairments, and it remains unknown how they compare to individuals with good sensorimotor recovery on standardized functional measures. A standardized definition and objective measures have not been developed for dropped foot due to various kinematic and EMG profiles across patients. As well, a limited number of studies have focused on comparing the effects of both technologies on ankle joint kinematics and muscle activation patterns during gait. A better understanding of how AFO/FES devices modify gait performance of individuals with different underlying dropped foot mechanisms will advance the success of the AFO/FES prescription. A novel element of the present work involves understanding the relationship between measures of ankle-foot impairments and gait deviations to develop clinical indications of dropped foot among stroke survivors. In addition, specific case studies will explore the immediate effects of AFO and FES technology on gait biomechanics among individuals with various mechanisms of dropped foot impairment.

The main focus of this work is on understanding mechanisms of dropped foot impairments and related deviations in gait function post stroke. It will focus on developing a clinical assessment that may help identify individuals who could benefit from an AFO/FES prescription. Specifically, the first research paper aims to determine the relationship between dropped foot gait deviations and impaired sensorimotor function. We hypothesized those kinematic deviations indicative of dropped foot would be related to quantitative and objective measures of lower limb sensorimotor impairment. This work will inform physiotherapists on measurement tools that may be used to screen patients for an advanced gait assessment of dropped foot. The second research paper aims to compare gait biomechanics between stroke survivors with and without dropped foot impairments. We hypothesized that stroke survivors with dropped foot would demonstrated greater ankle plantarflexion in the swing phase along with impaired timing of ankle dorsiflexor activation. This work will contribute to our understanding of impaired muscle activation patterns underlying dropped foot. The third research paper aims to explore the immediate effects of AFO and FES devices on gait performance among stroke survivors with different impairments resulting in dropped foot. Findings from this work will provide insight on positive and negative responses to AFO and FES devices for individuals with different dropped foot impairments.

CHAPTER 2

Clinical correlates of standardized measures of ankle-foot impairments and dropped foot gait deviations among stroke survivors

2.1 Introduction

Although previous research has reported that 60% to 80% of stroke survivors are able to ambulate independently on discharge from rehabilitation (Jorgensen, Nakayama, Raaschou, & Olsen, 1995), many individuals still exhibit gait deviations contributing to limited functional mobility (Olney & Richards, 1996). Post stroke gait is often characterized by slow speed, spatial-temporal asymmetry and increased energy demands (Brouwer, Parvataneni, & Olney, 2009; Kim & Eng, 2003; Olney, Monga, & Costigan, 1986; Olney & Richards, 1996; Patterson et al., 2008). Underlying factors contributing to these changes include muscle weakness, spasticity, and poor coordination (Olney & Richards, 1996). Ankle muscle weakness among stroke survivors has been associated with low plantarflexor power for push-off and reduced swing phase dorsiflexion during gait (Olney et al., 1991; Olney & Richards, 1996). Previous research has demonstrated positive correlations between ankle plantarflexors strength and self-selected preferred gait velocity (Hsu et al., 2003). Ankle plantarflexor spasticity was the most important determinant of both spatial and temporal asymmetry among stroke survivors with mild to moderate hemiparetic gait (Hsu et al., 2003). Some studies reported increased plantarflexor spasticity contributed to a slower gait velocity (Hsu et al., 2003; Lin, Yang, Cheng, & Wang, 2006), while others found no significant relationship (Nadeau, Gravel, Arsenault, & Bourbonnais, 1999a; Bohannon RW et al., 1987). Although these studies provide evidence that ankle impairments play an important role in limiting walking ability for stroke survivors, it remains unclear how clinical measures of ankle impairments relate to dropped foot gait deviations requiring intervention. Selecting appropriate therapeutic interventions to improve ankle function is essential to facilitate recovery of gait.

Stroke survivors presenting with dropped foot gait deviations may benefit from application of an ankle-foot orthotic (AFO) and/or functional electrical stimulator (FES) to improve functional mobility. Specific indications of dropped foot include reduced ankle dorsiflexion during swing, lack of heel strike at initial contact and limited ankle range of motion during the stance phase of gait (Olney & Richards, 1996; Kinsella & Moran, 2008). As a result, individuals may engage compensatory movements to improve toe clearance, such as hip hiking and limb circumduction during the swing phase. While these deviations are commonly identified in an observational gait analysis, there is no gold standard measure of dropped foot to indicate whether further evaluation of underlying impairments is required to determine whether an AFO or FES prescription is appropriate. Identifying individuals with dropped foot impairment before discharge from rehabilitation may facilitate earlier prescription of an AFO or FES device to improve mobility at home and within the community.

Clinical evaluation of ankle impairments post stroke usually involves standardized non-parametric functional tests to measure sensorimotor control, muscle weakness, spasticity, balance and sensation. These tools provide clinicians with information to identify functional limitations, develop treatment plans and determine response to rehabilitation. The Chedoke McMaster Stroke Assessment (CMSA) Impairment Inventory quantifies recovery of sensorimotor control based on performance of selected motor tasks with increasing complexity (Gowland et al., 1995). In the stroke literature, the CMSA lower limb components have demonstrated moderate correlations with indices of impaired gait function, such as velocity and temporal symmetry. These relationships appear to be more evident among those with severe sensorimotor impairment (Patterson et al., 2008). Medical Research Council (MRC) scale is used frequently to qualitatively describe manual muscle test strength due to its easy of application. This measure is criticized for lack of sensitivity to change and has demonstrated a clear ceiling effect. As well, this scale neither considers the range of motion or defines the strength of resistance to which the movement is performed (Bohannon, 2007). In contrast, a similar test evaluating maximum isometric ankle dorsiflexors strength via hand-held dynamometry has been positively related to gait velocity and temporal symmetry among stroke survivors (Lin et al., 2006). Ankle plantarflexor spasticity is commonly measured with the Modified Ashworth Scale (MAS) at rest and has demonstrated negative correlations to walking speed (Hsu et al., 2003; Lamontagne et al., 2001). While these measures provide an indication of the quality of movement during isolated motor tasks, most are ordinal scales with broad functional descriptors that may lack specificity to identify significant functional deficits during gait performance.

Previous work reported that approximately 22% of stroke survivors required an AFO at discharge from inpatient rehabilitation (Teasell et al., 2001). Stroke survivors using an AFO had lower admission and discharge scores on the CMSA upper and lower limb components, Functional Independence Measure and Berg Balance Scale (BBS) (Teasell et al., 2001). It is unknown how these scores are related to specific dropped foot gait deviations that lead to an AFO prescription. As well, individuals with dropped foot gait may not receive a prescription at discharge due to difficulty donning/doffing a device independently, patient and clinician perceptions, financial cost, and other stroke-related impairments. A current systematic review highlights the need for more specific investigation into clinical indications of stroke survivors that may benefit from an AFO prescription (Leung & Moseley, 2003).

The purpose of this study is to 1) determine the relationship between sagittal ankle kinematics during gait and standardized functional outcome measures among sub-acute stroke survivors at discharge from inpatient rehabilitation, and 2) compare standardized functional outcome measures between individuals with and without dropped foot gait deviations. We hypothesize that sagittal ankle kinematics will be correlated to lower limb sensorimotor control, and not related to non-parametric standardized indices for ankle plantarflexor spasticity (MAS) and ankle strength (MRC). Additionally, the standardized clinical outcome measures will indicate greater impairment among individuals with dropped foot gait.

2.2 Methods

Participants

Fifty-five individuals who sustained a stroke (ischemic or hemorrhagic) and were inpatients at the Toronto Rehabilitation Institute participated. Inclusion criteria were ability to walk 5m independently with or without a gait aid and understand instructions. Participants were excluded if they had other neurological or musculoskeletal disorders limiting their walking function. This study was approved by the local university and hospital research ethics boards. All participants provided informed consent.

Clinical Assessment

All hospital inpatients have a standardized clinical assessment performed with a trained physiotherapist at admission and discharge from rehabilitation. The following information was obtained, when available, from their medical charts: demographic data, medical history, AFO prescription, CMSA leg and foot score, Berg Balance Scale (BBS) and Clinical Outcome Variables Scale (COVS). The CMSA measures severity of sensorimotor impairments at the leg and foot on a 7 point scale, with higher score indicating better motor recovery. The CMSA has been reported to have a high inter- ($r = 0.85$-0.96) and intra-rater ($r = 0.94$-0.98) reliability (Gowland et al., 1993). The BBS evaluates 14 tasks on a scale of 0 to 4 with a higher score indicating complete ability to perform the task. This scale has been validated in the stroke population and has high inter- (r = 0.95-0.98) and intra-rater (r = 0.97) reliability (Berg, Wood-Dauphinee, & Williams, 1995).

Task Protocol

Participants completed additional standardized tests to evaluate ankle joint range of motion (ROM), spasticity and muscle strength. Passive and active ROM at the ankle joint was assessed for the dorsiflexors and plantarflexors using electrical goniometers following a standardized procedure (Clarkson, 2005). Participants were in a seated position with the lower leg supported and knee extended. Each movement was performed twice to obtain an average score. The MAS was utilized to measure ankle plantarflexor spasticity at rest on a 5 point scale (Blackburn, van Vliet, & Mockett, 2002). The movement was performed two or three times obtain a score. The participant was seated with their hip flexed to approximately 90°, knee extended and lower leg supported. The participants were allowed to rest when needed. Maximal voluntary contractions (MVC) were used to determine isometric muscle strength of the ankle dorsiflexors and plantarflexors muscles for both limbs. Participants were in a seated position with their knee at 90° and foot strapped to a load cell positioned at the metatarsal heads (Figure 2.1). Instructions were to push on the load cell and hold for 1 second to measure plantarflexor strength, followed by rest for 1 minute then pull against the load cell for dorsiflexor strength. This procedure was repeated twice for each muscle for both limbs. Participants were also scored on the MRC Manual Muscle Test scale for both limbs.

Participants were asked to walk a distance of 7 m over a pressure sensitive mat (GaitRite® system; Appendix 2) with their regular footwear under two conditions: (1) at their preferred speed and (2) at the fastest speed at which they felt safe. Three trials were performed per condition. Participants were instructed to stand upright with equal weight between limbs starting 1 m before the mat and continued to walk 1 m after the mat. Gait aids permitted include wheeled walkers and single or quad point canes. Electrical goniometers were placed bilaterally with the axis inferior to the lateral malleoli to record sagittal ankle kinematics.

Measurements

Custom made electrical goniometers were designed using a 10 kΩ potentiometer, 9V battery and 5 mm thermoplastic (weight = 105g, Figure 2.2) to measure sagittal ankle movements. A positive value indicates ankle plantarflexion and a negative value indicates ankle dorsiflexion. A load cell (Appendix 2) was mounted to a platform at a 10° angle with a strap fixed to measure muscle force. These signals were sampled at 1000 Hz and stored for offline analysis (Noraxon Telemyo System; Appendix 2). The devices were calibrated with a mechanical goniometer and weights, respectively.

Spatial-temporal gait parameters recorded using a pressure sensitive mat (GaitRite Systems, Appendix 2), which is 5.25 m in length, 0.88 m in width and contains a grid pattern of 48 by 288 sensors arranged 1.27 cm on center. Data were sampled at a frequency of 120 Hz. The GaitRite mat signal was synced to the Noraxon system to calculate contact times relative to the electrical goniometers.

Data and Statistical Analysis

Data analysis software included with the GaitRite system automatically outputs gait velocity, spatial-temporal parameters and foot contact times for each trial. A custom software program was designed in Microsoft Visual Basic (version 6.0) to merge data from each measurement tool to calculate muscle force and sagittal ankle kinematics. A calibration slope of 0.1519 was applied to the load cell signal from the MVC tests to convert uV to N. A second-order low-pass Butterworth filter was applied to electrical goniometer signal with a cutoff frequency of 3 Hz (D. A. Winter, 2005). The participant's ankle position during stance before the walking trial was considered to be neutral (90°) and the goniometer signal was compared to the output at 90° during the manual calibration. Relative ankle dorsiflexion and plantarflexion motions were calculated from neutral by applying the slope (17.2 uV/degree) calculated from the manual calibration. Initial and last foot contact times from the GaitRite data were applied to determine stance and swing phases of the gait cycle. The number of foot contacts analyzed range from 4-10 per trial. Outcome measures include peak ankle dorsiflexion (minimum value) during the swing phase and ankle ROM during the stance phase (difference between minimum and maximum joint motion from initial contact to toe-off).

Descriptive statistics, mean and standard deviation (SD), were calculated for standardized functional outcome scores, ankle ROM at rest, muscle force and sagittal ankle kinematics. Pearson's correlation coefficients were performed to evaluate the relationships between sagittal ankle kinematics and ankle ROM at rest. Spearman's correlations were utilized to relate sagittal ankle kinematics to standardized functional outcomes (CMSA-foot, MAS and MRC). Participants were divided into two groups based on the presence of dropped foot during gait. Criteria for dropped foot included 1) an AFO prescription at discharge or 2) peak ankle dorsiflexion during the swing phase and/or stance ROM less than normative values from healthy adults walking at a slow cadence (mean ± 1SD), along with ankle plantarflexion position at initial contact (D. A. Winter, 1991). Toe drag along the mat in the swing phase of gait was noted during testing. A three-way analysis of variance (ANOVA) was used to compare sagittal ankle kinematics between groups (dropped foot vs. non-dropped foot), limbs (paretic vs. non-paretic) and walking conditions (preferred vs. fast). Post hoc Tukey analysis was performed on pair-wise comparisons. Independent t-tests were performed between dropped foot and non-dropped foot participants to determine differences in discharge CMSA-leg, CMSA-foot, BBS, COVS, MRC, active ROM, passive ROM and ankle dorsiflexors/plantarflexors isometric force. Statistical significance was set at alpha 0.05.

Figure 2.1: Picture of the set up position for testing maximal voluntary isometric contractions of the ankle dorsiflexor and plantarflexor muscles.

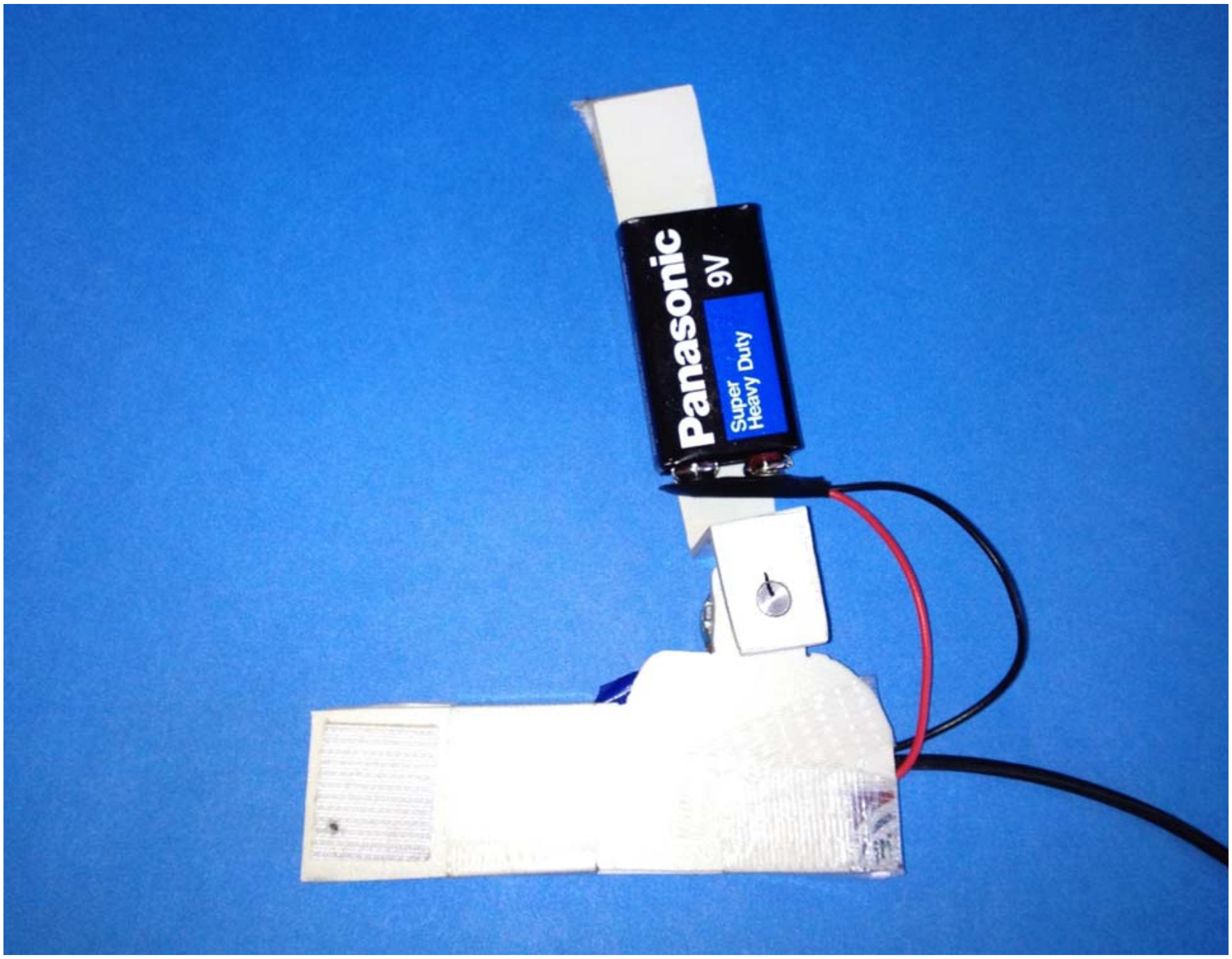


Figure 2.2: Picture of the custom made electrical goniometers used to measure sagittal ankle movements.

2.3 Results

Participants

Table 2.1 presents a summary of participant demographic, stroke information, clinical scores and gait data. Participants (n=55) average length of stay for inpatient rehabilitation was 47.7 ± 20.4 days. Eleven participants did not perform the MVC tests with the load cell due to technical issues. One participant did not complete the fast walking trials due to low tolerance and muscle fatigue. For individuals who were bilaterally affected, paretic limb was the side with the lower CMSA leg and foot score.

Objective 1

Peak ankle dorsiflexion during swing and ROM during stance on the paretic side was not significantly related to the CMSA leg and foot scores (Table 2.2, Figure 2.3). Similarly, the correlations between ankle spasticity (MAS) and dorsiflexor strength (MRC) scores with peak dorsiflexion during swing and ROM during stance were not significant on the paretic side ($p>0.05$). Plantarflexor strength (MRC) was negatively correlated to paretic peak dorsiflexion during swing ($df=54$, $r= -0.27$, $p=0.044$). Paretic ankle dorsiflexors isometric MVC force was positively related to peak dorsiflexion in the swing phase (Figure 2.3, $df=42$, $r=0.32$, $p=0.039$). Paired t-test revealed poor ankle dorsiflexors MVC force on the paretic compared to non-paretic side (7.0 ± 3.5 N vs. 8.3 ± 3.7 N, $p=0.022$). Passive and active ankle ROM on the paretic side revealed positive correlations to stance phase ROM ($df=54$, $r=0.48$ and $r=0.45$, $p<0.001$ respectively).

Secondary analysis was conducted with the removal of 4 participants who displayed dropped foot, as possible confounders for correlations with peak dorsiflexion during swing. These participants slid their paretic limb across the floor during the swing phase producing relatively good peak ankle dorsiflexion, despite their poor sensorimotor control. MRC plantarflexors score revealed a significant negative correlation to peak ankle dorsiflexion ($df=40$, $r= -0.37$, $p=0.008$). MAS and AROM displayed a trend towards a relationship with peak ankle dorsiflexion during swing ($df=50$, $r= -0.18$, $p=0.19$ and $r= -0.21$, $p=0.152$, respectively).

Objective 2

Twelve participants fulfilled our criteria for dropped foot during gait; 3 participants with both reduced swing phase peak ankle dorsiflexion and stance phase ankle ROM, 6 participants with only reduce peak dorsiflexion in swing, and 3 participants with only reduced stance ROM. Peak dorsiflexion in the swing phase was 2.5° non-paretic and 0.7° paretic for the non-dropped foot group compared to 3.7° non-paretic and 3.9° paretic for the dropped foot group in the preferred condition (main group effect p=0.01, Figure 2.4). Peak ankle dorsiflexion during the swing phase was not significantly different between limbs and walking conditions (p=0.37 and p=0.776, respectively, Figure 2.4). Stance phase ankle ROM was 19.5° non-paretic and 18.0° paretic for the non-dropped foot group compared to 17.8° non-paretic and 18.6° paretic for the dropped foot group in the preferred condition (main group effect p=0.927, Figure 2.4). Stance phase ankle ROM was slightly greater for preferred by 1.2° compared to fast walking conditions (p=0.015), while no difference was found between limbs (p=0.173, Figure 2.4). Self-selected preferred and fast gait velocity was significantly slower in the dropped foot group compared to non-dropped foot (p<0.001, Table 2.1). Statistical tests revealed significant differences between groups for CMSA leg and foot scores, MRC dorsiflexors and plantarflexors, MAS and COVS (Table 2.1). Ankle isometric plantarflexor force on the paretic side was significantly lower in the dropped foot group compared to non-dropped foot (4.5 ± 2.5 N vs. 8.0 ± 3.9 N, p=0.01), while dorsiflexors force revealed a similar trend (6.0 ± 3.0 N vs. 7.3 ± 3.6 N, p=0.288 respectively). No difference was found between groups for MVC isometric dorsiflexors and plantarflexors force on the non-paretic side (p>0.05). Participants without dropped foot demonstrated a greater active ROM on the paretic side at rest compared to those with dropped foot (p=0.017), while passive ROM was equal between groups (p=0.543, Figure 2.5). Peak dorsiflexion during the MAS test for plantarflexors spasticity was significantly different between groups (non-dropped foot: -16.6° ± 4.8, dropped foot: -12.2° ± 4.2, p=0.01). At discharge, 4 participants from the dropped foot group were using a standard AFO and provided with an orthotic referral for a custom device.

Table 2.1: Participant demographic, stroke information and clinical data

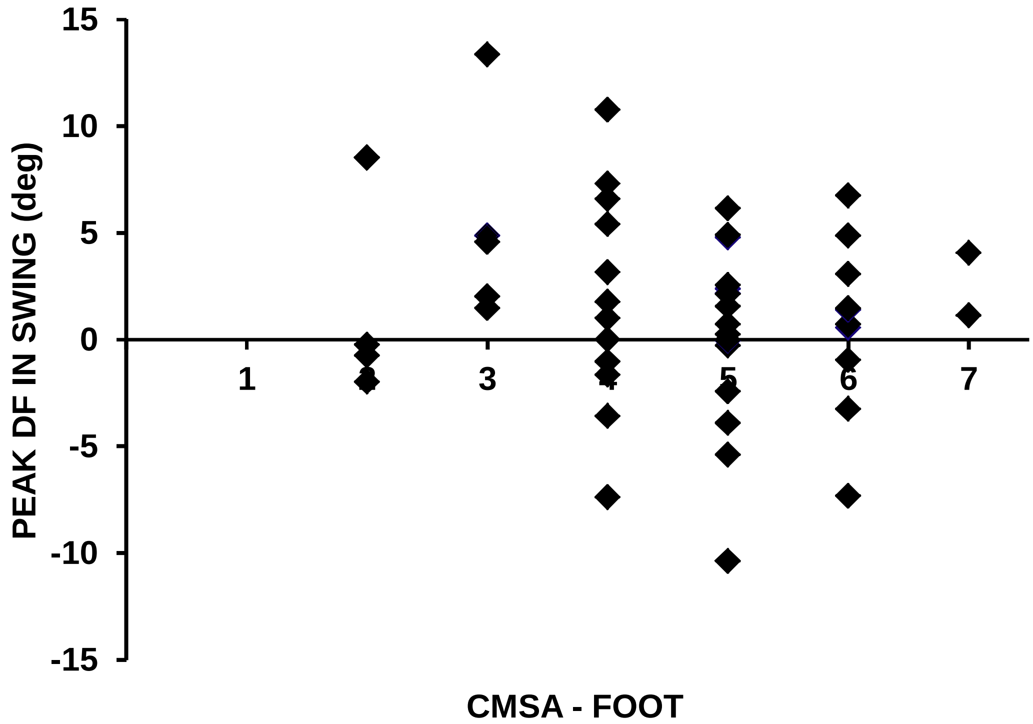
	All Participants	Non-Dropped Foot	Dropped Foot
Demographic Data			
n	55	43	12
Age (yrs)	68.9 ± 12.3	69.5 ± 12.2	66.8 ± 13.2
Gender (Female : Male)	18 : 37	15 : 28	3 : 9
Stroke Information			
Time post-stroke (days)	40.0 ± 17.8	35.0 ± 11.5	57.9 ± 24.7**
Type of stroke			
Ischemic	48	36	12
Hemorrhagic	7	7	0
Hemisphere affected			
Left	28	19	9
Right	21	19	2
Bilateral	6	5	1
Assistive Devices			
Gait aid (Yes: No)	28 : 28	19 : 25	9 : 3
AFO (Yes : No)	4 : 51	0 : 43	4 : 8
Clinical Data			
CMSA leg score	4.9 ± 1.0	5.2 ± 1.1	4.3 ± 1.1*
CMSA foot score	4.6 ± 1.2	5.0 ± 1.0	3.4 ± 1.2**
BBS	45.0 ± 8.6	45.7 ± 8.8	41.5 ± 8.3
COVS	76.7 ± 8.5	78.6 ± 7.7	69.7 ± 6.8**
MAS**			
0	34	34	0
1	11	8	3
1+	4	1	3
2	4	0	4
3	2	0	2
4	0	0	0
MRC - Strength			
Dorsiflexors	4.2 ± 0.9	4.4 ± 0.7	3.3 ± 1.0**
Plantarflexors	4.3 ± 0.9	4.5 ± 0.5	3.3 ± 1.2**
Gait Data			
Velocity (cm/s)			
Preferred	59.6 ± 25.6	64.9 ± 25.7	40.8 ± 14.6**
Fast	95.5 ± 38.1	103.6 ± 37.6	67.1 ± 24.6**

Note: Data represents mean ± standard deviation for continuous data. The number of individual in each category is presented for count variables. Time post-stroke is the number of days between stroke onset and study visit. Significance of independent t-test comparing clinical scores between groups are indicated by *p>0.05 and **p>0.01.

Table 2.2: Correlations between sagittal paretic ankle kinematics and clinical measures

	Swing Peak DF (r)	p value	Stance ROM (r)	p value
CMSA				
Leg	-0.14	0.315	0.05	0.714
Foot	-0.17	0.247	0.05	0.735
MRC				
Dorsiflexors	-0.21	0.122	-0.04	0.788
Plantarflexors	**-0.27**	**0.044**	0.00	0.976
MVC				
Dorsiflexors	**0.32**	**0.039**	-0.04	0.820
Plantarflexors	-0.03	0.827	0.09	0.583
MAS	-0.11	0.408	0.09	0.503
PROM	0.05	0.737	**0.48**	**<0.001**
AROM	-0.13	0.355	**0.45**	**<0.001**

Note: Values presented are Spearman and Pearson correlations with associated p values for ordinal and continuous data, respectively. Bold values indicate statistical significance at $p<0.05$. All correlations have a df=54, except for MVC dorsiflexors and plantarflexors (df=42).



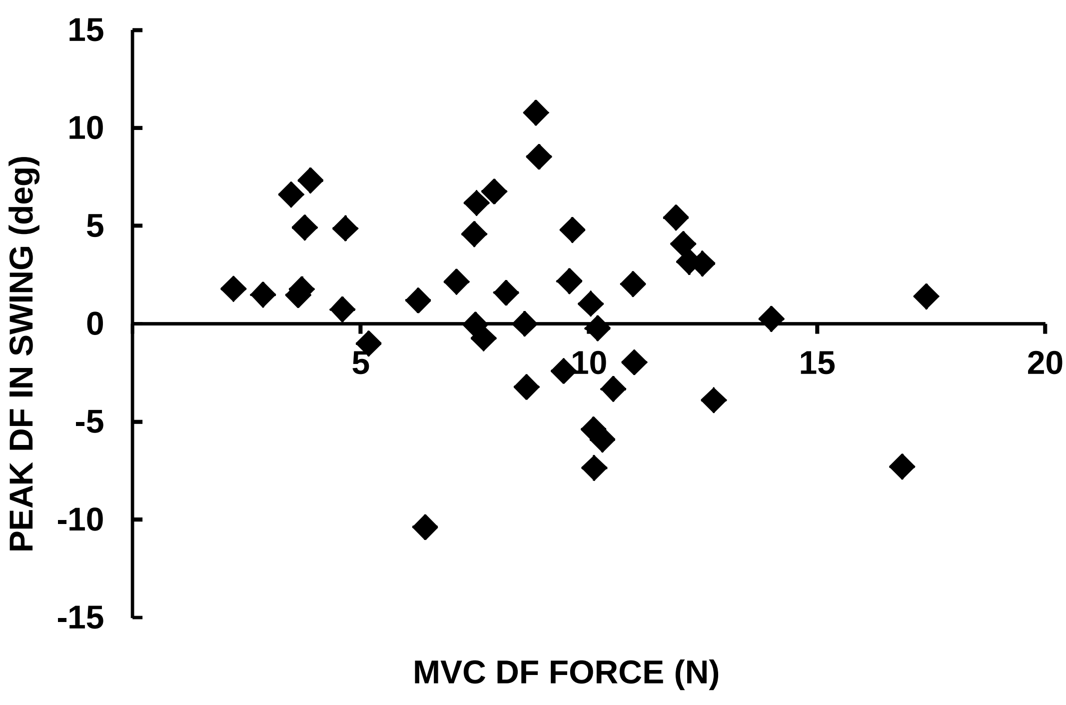


Figure 2.3: Illustration of the relationships between peak ankle dorsiflexion during the swing phase of gait and a) CMSA foot score ($r = -0.17$, $p = 0.247$) and b) MVC dorsiflexor muscle force ($r = 0.32$, $p = 0.039$).

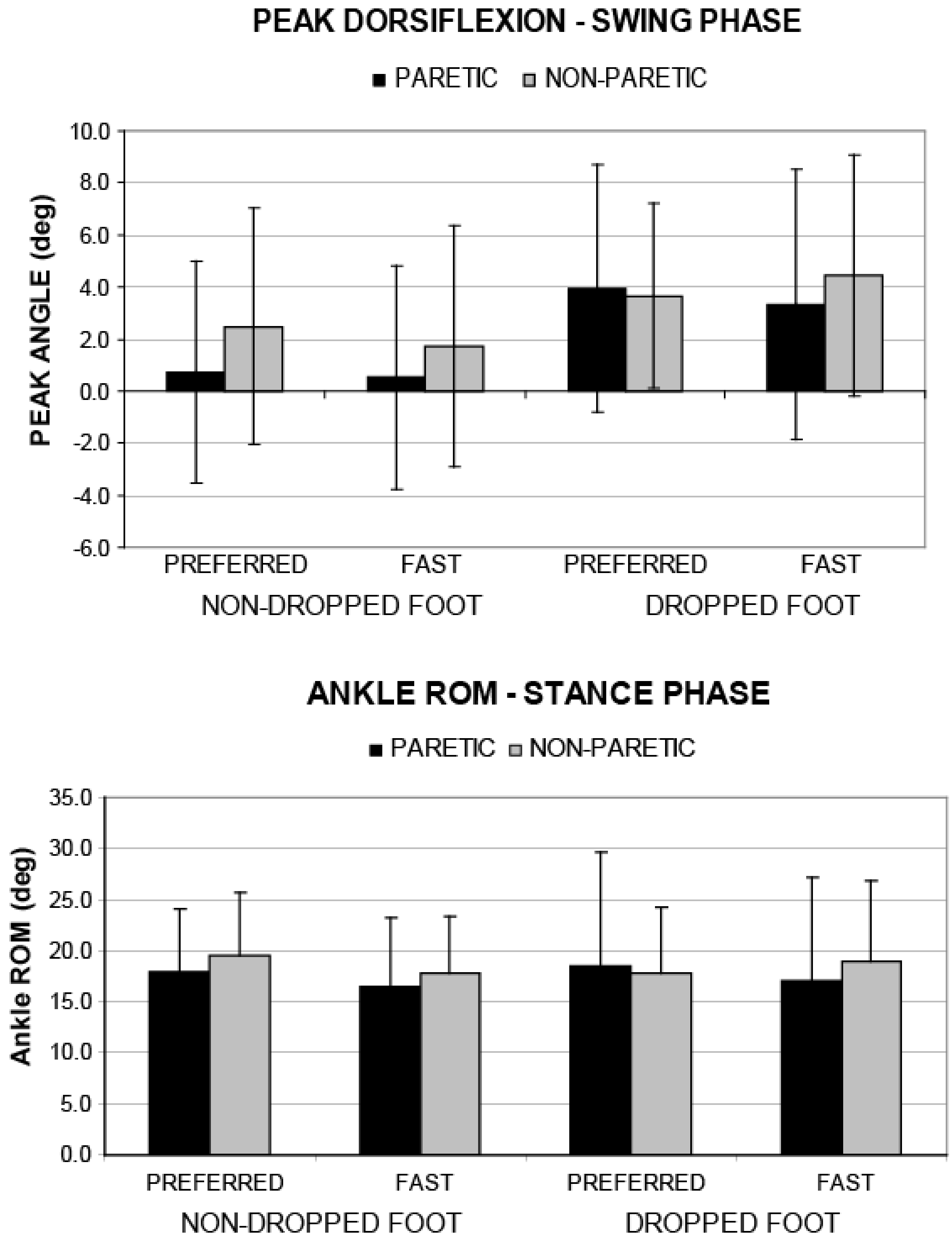


Figure 2.4: Comparison of peak ankle dorsiflexion (minimum value) during swing and ankle ROM during stance between limbs and conditions for both groups. Values illustrated are means with SD error bars. A significant main effect revealed a greater peak dorsiflexion for the non-dropped foot compared to dropped foot group in the swing phase ($p=0.01$). Stance phase ankle ROM was significantly greater in the fast compared to preferred condition ($p=0.015$). Note: negative values represent dorsiflexion and positive values are plantarflexion for peak angle.

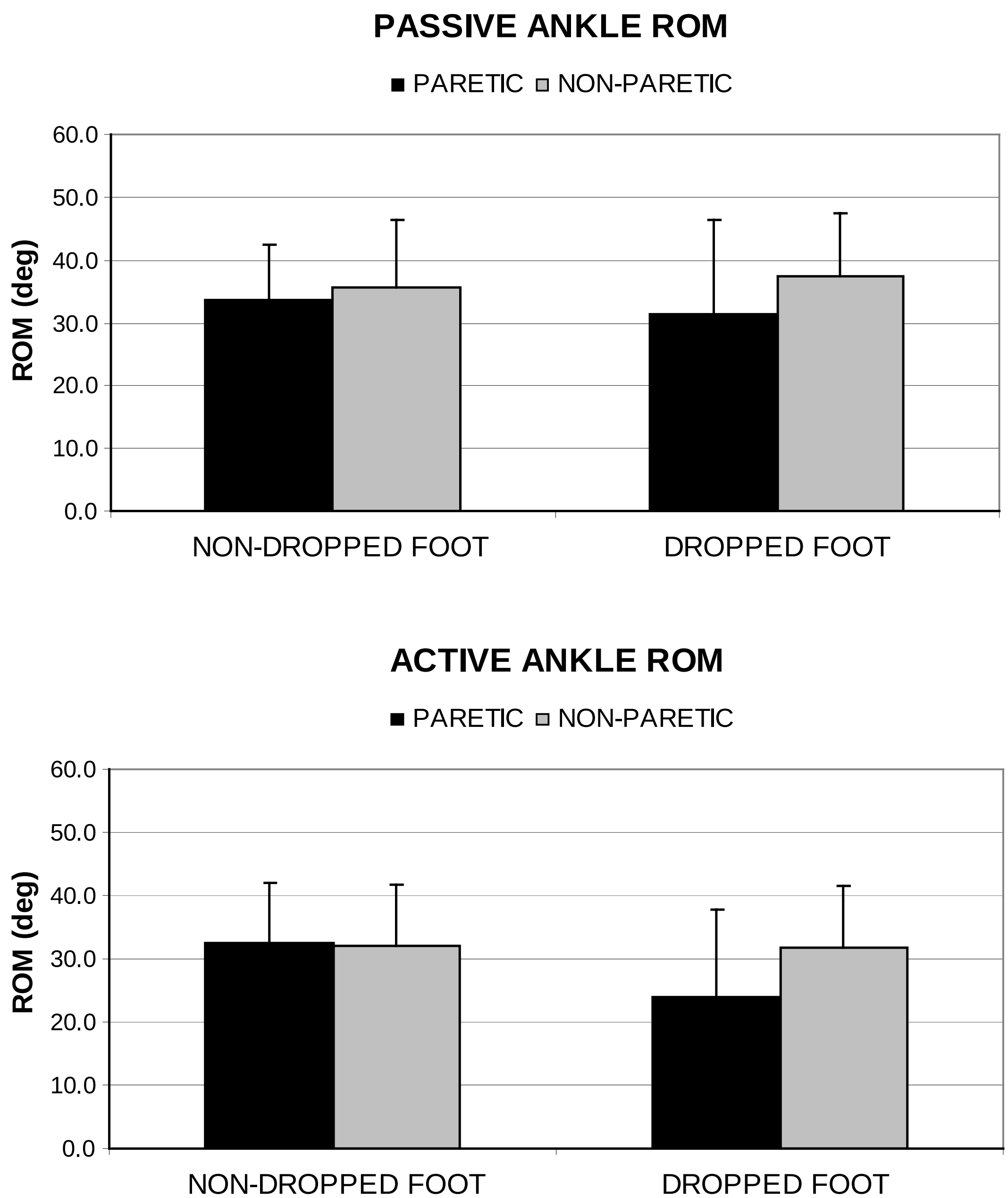


Figure 2.5: Comparison of passive and active ankle ROM between groups. Values illustrated are means with SD error bars. Independent t-tests revealed no difference in passive ROM between groups (p=0.543), while active ROM was significantly lower for the dropped foot compared to non-dropped foot group (p=0.017).

2.4 Discussion

This study found that standardized non-parametric clinical measures quantifying sensorimotor recovery, spasticity and dorsiflexor muscle strength at the ankle-foot complex were not related to sagittal ankle kinematics during gait indicative of dropped foot among stroke survivors at discharge from inpatient rehabilitation. However, individuals with better ankle plantarflexor strength were likely to display a greater peak dorsiflexion during swing. Parametric measures such as ankle isometric dorsiflexors force and passive/active ROM demonstrated significant correlations to peak swing phase dorsiflexion and stance ROM, respectively. Participants who presented with dropped foot had a slower preferred gait velocity and scored lower on all standardized outcome measures, except for the BBS (Table 2.1). As well, these participants displayed a reduced active ankle ROM at rest and isometric plantarflexors force. It is important to note that participants with dropped foot had a longer inpatient length of stay compared to those without dropped foot. Exploring how outcome measures used in a clinical setting relate to dropped foot deviations is important to identify individuals who may benefit from an AFO or FES device before discharge from rehabilitation.

In contrast to our hypothesis, lower limb sensorimotor impairment was not related to peak ankle dorsiflexion during swing and stance ROM. Individuals with stroke who have a lower CMSA score at the foot demonstrate difficulty with active movements in all directions, resistance to passive dorsiflexion, and impaired coordination between dorsiflexion-plantarflexion (Gowland et al., 1995). The absence of association is likely linked to differences in task demands and use of compensatory movements in the swing phase of gait. Lower stages of the CMSA scale require participants to perform isolated dorsi/plantarflexion and inversion/eversion movements in a lying or sitting position (Gowland et al., 1995). Whereas gait performance involves maintenance upright stability, forward propulsion, coupling and synchronizing movements of the lower limbs (Perry, 1992). Although stroke survivors may be able to perform isolate ankle movements, issues such as impaired lower limb coordination and/or reduced proprioception may produce delayed initiation of the swing phase due to difficulty with ankle dorsiflexion. A previous study has reported mild to severe temporal asymmetry between stance and swing phases of the gait cycle among stroke survivors with CMSA foot scores ranging 2-7 (Patterson et al., 2008). One mechanism thought to contribute to temporal gait asymmetry is impaired swing phase initiation, possibly due to problems with generating a lower limb flexion synergy (Patterson et al., 2008). Individuals who have difficulty with ankle dorsiflexion may compensate by increasing knee flexion, hip external rotation and/or pelvic tilt to achieve toe clearance. We observed some compensatory movements with our participants at the knee and hip to improve toe clearance and facilitate forward progression.

Most non-parametric indices of ankle-foot impairments (i.e. MAS and MRC) were not correlated to dropped foot gait deviations. This finding was not surprising as standardized measures used routinely in clinical setting may have components with overlap constructs, such as the CMSA and MAS reflecting components of spasticity and passive ROM. The lack of a relationship may be due to different sensorimotor control programs and large variation across participants for the dropped foot outcome measures. All participants demonstrating no resistance to passive dorsiflexion on the MAS test did not display dropped foot during gait, while individuals with a score of 2 or higher were in the dropped foot group. The significant difference found between groups reflects the contribution of spasticity to impaired ankle-foot function during gait. The MAS is often criticized for lack of specificity between levels and poor reliability between tests (Blackburn et al., 2002; Lamontagne et al., 2001). Previous research has found that the expression of spasticity at rest differs from that observed under a dynamic task, such as walking (Fung & Barbeau, 1994; Knutsson & Richards, 1979; Lamontagne et al., 2001). A few studies have examined methods for measuring spasticity during gait, however the complex method may not be feasible for a clinical setting and interpretation of the outcome requires careful examination of abnormal lower limb movement patterns (Crenna, 1998; Lamontagne et al., 2001). Similarly, the MRC scale demonstrated a difference between groups, possibly due to the inability to perform isolated ankle movements among a few people with dropped foot (n=4). We found a group difference in isometric ankle plantarflexion force, while dorsiflexion force was equivalent. The lack of difference may represent general dorsiflexor muscle weakness among all participants. However, individuals with greater dorsiflexors force were likely to demonstrated higher peak ankle dorsiflexion during the swing phase of gait. Force values from the MVC tests are lower than previous reports (Pohl et al., 2000, Ng & Hui-Chan 2012), likely due to differences in equipment, contraction type and the testing position. Our assessment was easy to administer with low cost equipment, and revealed a difference between stroke survivors with and without dropped foot. Therefore, MVC testing may provide a more discriminatory measure to evaluate muscle strength in relation to ability to perform movements for gait function.

While twelve participants were identified with dropped foot issues during gait, only 4 participants were prescribed an AFO at discharge. The participants with an AFO demonstrated poor lower limb sensorimotor recovery (CMSA foot = 2 and leg = 3 to 4), difficulty with active ankle dorsiflexion against gravity and mild-moderate plantarflexors spasticity (MAS = 1 to 3). These individuals had a slow preferred gait velocity (18.2 – 43.3 cm/s) along with limited stance ROM (n=2), and reduced peak dorsiflexion during swing (n=2) beyond normative values (D. A. Winter, 1991). Previous research has found that stroke survivors discharged with an AFO consistently scored lower on indices of sensorimotor recovery and balance at admission and discharge from rehabilitation (Teasell et al., 2001). For our participants with dropped foot, there was no difference among the standardized clinical measures between individuals with and without an AFO prescription at discharge, expect for the CMSA foot score. It is important to note that other factors that may influence an AFO prescription including participation in other therapy, poor upper limb function to don/doff a device, cognitive and perceptual impairments, and financial cost (Tyson & Thornton, 2001).

This study involved a convenience sample of stroke survivors who were able to ambulate 5m independently without physical assistance. Additionally, individuals with dropped foot were excluded if unable to walk safely across the pressure mat without using an AFO. Sagittal ankle kinematics was our gold standard measure for determining dropped foot impairment during gait; however we did not assess knee flexion angles and toe clearance. Stroke survivors with poor motor recovery of lower limb flexion synergy may demonstrate reduced knee flexion during swing resulting in decreased toe clearance (C. L. Chen et al., 2003). This movement pattern may produce relatively good ankle dorsiflexion during swing with a lower total ROM throughout the gait cycle. Therefore, a combination of sagittal kinematic deviations was utilized as criteria for determining dropped foot and all profiles were examined independently.

To conclude, this study demonstrated that non-parametric clinical measures quantifying sensorimotor recovery, spasticity and muscle strength at the ankle joint were not associated with sagittal ankle kinematics reflecting dropped foot gait deviations. Individuals with dropped foot scored lower on each of the above constructs compared to those without dropped foot during gait. Clinical tests that evaluate ankle-foot function post stroke are important to understand mechanisms of abnormal movement patterns and determine appropriate treatments strategies. We recommend using the CMSA-foot score, MVC dorsiflexors force and active ankle ROM in a sitting position to screen individuals for a detailed gait assessment of dropped foot for consideration of an AFO or FES prescription.

CHAPTER 3

Lower limb kinematics and muscle activation patterns of dropped foot gait impairment post stroke

3.1 Introduction

After stroke, dropped foot impairment can affect important functional tasks, such as walking, sit-to-stand transfers and stair climbing. In walking, the ankle plantarflexor muscles contribute to forward progression by concentrically activating to generate propulsive work starting late in the stance phase, while the dorsiflexors muscles concentrically activate to lift the foot for toe clearance in the swing phase and eccentrically control lowering the foot at initial contact. The inability to generate normal levels of muscle force due to weakness in voluntary movements (Knutsson & Richards, 1979), excessive or insufficient co-activation (Lamontagne et al., 2000) and presence of hyperactive stretch reflexes has been reported among stroke survivors (Olney & Richards, 1996). Previous research has highlighted that muscle activation patterns are varied and complex among individuals with spastic dropped foot impairment (Burridge, Wood, Taylor, & McLellan, 2001). Understanding the relationship between ankle kinematic deviations and muscle activity may provide useful information for development of a clinical tool to identify mechanisms of dropped foot and guide treatments plans.

Dropped foot impairment post stroke is characterized by reduced ankle dorsiflexion during the swing phase, lack of heel strike at initial contact, and decreased ankle range of motion (ROM) during stance (Olney & Richards, 1996; Kinsella & Moran, 2008). Individuals with medial-lateral ankle instability demonstrate greater inversion throughout the gait cycle (C. L. Chen, Yeung, Wang, Chu, & Yeh, 1999). Compensatory movements may be observed at the knee and hip to facilitate toe clearance, including increased knee flexion, and increased hip abduction and external rotation during the swing phase (C. L. Chen et al., 2003; Olney & Richards, 1996). Previous studies have reported kinematic deviations indicative of muscle weakness and poor motor control in the upper leg; less hip extension at toe-off, reduced knee flexion during pre-swing, and knee hyperextension at mid-stance (C. L. Chen et al., 2003; Kim & Eng, 2004; Olney & Richards, 1996). Individuals with dropped foot may engage different strategies to achieve forward progression and stability during stance; therefore the link to muscle activation patterns will provide an understanding of different mechanisms.

Temporal components of muscle activity during walking are often disrupted among stroke survivors due to impaired central control and development of compensatory strategies (Knutsson & Richards, 1979; Shiavi, Bugle, Limbird, 1987a; Shiavi, Bugle, Limbird, 1987b). Common abnormalities in timing observed during hemiparetic gait include absent or reduced amplitudes, prolonged activation, and pre-mature activation (Den Otter et al., 2007). Delayed activation of the tibialis anterior (TA) during the transition from swing to stance has been observed among individuals with spastic dropped foot (Burridge et al., 2001). As well, the ankle plantarflexors displayed reduced activity at push-off and/or inappropriate early activation prior to the stance phase compared to healthy controls (Burridge et al., 2001). Den Otter et al 2007 found that individuals with hemiparetic gait had a longer duration of medial gastrocnemius (MG) activity on the paretic side during weight acceptance compared to healthy controls (Den Otter et al., 2007). Another study found that reduced activation of the MG on the paretic side contributed to low push-off power and slower gait velocity (Lamontagne et al., 2002). Although evaluating the activity of individual muscles have its merits, understanding coordination between the ankle dorsiflexors and plantarflexors may reveal strategies that impair muscle force output during gait.

Coactivation of opposing muscles is an important component of motor control in healthy individuals to regulate joint stiffness and assist with postural stability (Falconer & Winter, 1985). The gait cycle consists of periods with higher levels of coactivation, such as the transitions between swing and stance phases, and lower levels at mid stance (Falconer & Winter, 1985). Excessive or insufficient coactivation of the ankle dorsiflexors and plantarflexors among stroke survivors contributes to poor gait function and reduced postural stability. Abnormal coactivation patterns have also been observed on the non-paretic side, possibly due neurological injury and/or biomechanical compensations (Den Otter et al., 2007). Lamontagne et al 2000 reported less coactivation on the paretic side in the single support phase and greater coactivation on the non-paretic side during both double support phases relative to healthy controls (Lamontagne et al., 2000). Stroke survivors with greater coactivation on the non-paretic side were likely to demonstrate a slower gait velocity, reduced single support time, and poor ankle plantarflexor force (Lamontagne et al., 2000). It is likely that impaired coactivation timing during gait is linked to altered magnitudes of muscle activation. Applying a cross-correlation technique can be useful to investigate spatial and temporal relationships to provide further insight on coordination of muscle activation patterns. Outcome measures with this technique include information on temporal shift or phase delay between muscles and the maximum value can be related to a mechanical event during the gait cycle (Nelson-Wong, Howarth, Winter, & Callaghan, 2009). de Niet et al 2011 determined that the phase shift between the lengthening velocity and electromyographic (EMG) activity of the MG muscle did not correspond with stretch reflex latency among individuals with spastic gait (de Niet, Latour, Hendricks, Geurts, & Weerdesteyn, 2011). Patterns of muscle coactivation among stroke survivors with dropped foot have yet to be investigated and may provide evidence on mechanisms leadings to specific kinematic deviations during gait performance.

This study aims to 1) compare TA-MG activations patterns and sagittal ankle kinematics during gait performance among stroke survivors with and without dropped foot, and 2) evaluate the relationship between TA-MG coactivation and sagittal ankle kinematics. We hypothesize that individuals with dropped foot will demonstrate a delayed non-paretic peak ankle dorsiflexion during stance, reduced paretic peak dorsiflexion during swing, delayed paretic TA onset during stance, and less paretic TA-MG coactivation time compared to individuals without dropped foot. Also, reduced swing phase peak ankle dorsiflexion and stance phase ROM will be related to lower TA-MG coactivation time on the paretic side.

3.2 Methods

Participants

Fifty-five individuals (18 females; 37 males) who had sustained a stroke (48 ischemic; 7 hemorrhagic) participated in this study (Table 3.1). Twenty-eight individuals were affected on the left side, 21 on the right side and 6 bilateral. The paretic side was determined as the more affected side based on the Chedoke McMaster Stroke Assessment (CMSA) scores for those with bilateral impairments. Inclusion criteria were admitted to inpatient stroke rehabilitation, the ability to walk 5 m independently with or without a gait aid, and able to understand instructions. Exclusion criteria were any other neurological or musculoskeletal disorders limiting their walking function. This study was approved by the local university and hospital research ethics boards. All participants provided written informed consent.

Participants completed a standardized clinical assessment with a trained physiotherapist. Data obtained from their medical charts included CMSA leg and foot score, Berg Balance Scale (BBS) and Clinical Outcome Variables Scale (COVS). High inter- and intra-rater reliability scores have been previously reported in the stroke population for the above clinical measures. (Berg et al., 1995; Gowland et al., 1993). Modified Ashworth Scale (MAS) was used to measures ankle plantarflexor spasticity with the knee extended at rest on a 5-point scale (Blackburn et al., 2002). The movement was performed two or three times obtain a score.

Table 3.1: Participant demographic, stroke information and clinical data

	All Participants	Non-Dropped Foot	Dropped Foot
n	55	43	12
Age (yrs)	68.9 ± 12.3	69.5 ± 12.2	66.8 ± 13.2
Height (m)	1.69 ± 0.13	1.68 ± 0.13	1.71 ± 0.08
Weight (kg)	76.9 ± 14.6	77.3 ± 15.2	75.4 ± 12.8
Time post-stroke (days)	40.0 ± 17.8	35.0 ± 11.5	57.9 ± 24.7**
CMSA leg score	4.9 ± 1.0	5.2 ± 1.1	4.3 ± 1.1*
CMSA foot score	4.6 ± 1.2	5.0 ± 1.0	3.4 ± 1.2**
BBS	45.0 ± 8.6	45.7 ± 8.8	41.5 ± 8.3
COVS	76.7 ± 8.5	78.6 ± 7.7	69.7 ± 6.8**
MAS	0.7 ± 1.1	0.2 ± 0.5	2.4 ± 1.1**

Abbreviations: BBS, Berg Balance Scale; CMSA, Chedoke McMaster Stroke Assessment Scale; COVS, Clinical Outcomes Variable Scale; MAS, Modified Ashworth Scale.

Note: Independent t-test between groups *$p<0.05$, **$p<0.001$

Task Protocol

The participants were instructed to walk at their preferred speed and at the fastest speed at which they felt safe over a pressure sensitive mat (GaitRite system). Three trials were performed per condition. Participants were asked to stand upright with equal weight between limbs starting 1 m before the mat and continued to walk 1 m after the mat. Participants wore their regular footwear. Twenty-eight individuals used a gait aid; 18 wheeled walkers and 10 canes. Electrical goniometers were placed bilaterally inferior to the lateral malleoli to record sagittal ankle kinematics. EMG electrodes were placed bilaterally over the TA and MG muscles.

Measurement Tools and Procedures

Spatial-temporal gait parameters recorded using a pressure sensitive mat (GaitRite system, Appendix 2), which is 5.25 m in length, 0.88 m in width and contains a grid pattern of 48 by 288 sensors arranged 1.27 cm on center. Data were sampled at a frequency of 120 Hz. The GaitRite mat signal was synced to the Noraxon system to calculate foot contact times relative to the electrical goniometer and EMG signals.

Custom made electrical goniometers were designed using a 10 kΩ potentiometer, 9V battery and 5 mm thermoplastic to measure sagittal ankle movements. The signal was sampled at 1000 Hz and stored for offline analysis (Noraxon System, Appendix 2). The device was calibrated with a mechanical goniometer. EMG activity was recorded using 30 mm silver-silver chloride surface electrodes (Medi-Trace® Mini, Appendix 2). Manual muscle tests were performed to determine EMG locations. Skin was rubbed with nu-prep and alcohol to reduce the impedance. Pairs of electrodes were placed longitudinally 1 cm apart on center over the proximal aspect of the TA and MG muscles. Data was sampled at 1000 Hz. EMG signals were pre-amplified (2000x), filtered online (bandwidth 10-500 Hz) and stored for offline analysis (Noraxon Telemyo System, Appendix 2).

Data and Statistical Analysis

A custom software program developed in Microsoft Visual Basic (version 6.0) was utilized to merge data from each measurement tool to calculate sagittal ankle kinematics and muscle activation patterns normalized to the gait cycle. Gait cycle duration was defined as the time interval between two consecutive ipsilateral foot contacts on the GaitRite mat. Data analysis software included with the GaitRite system automatically outputs gait velocity, spatial-temporal parameters and foot contact times for each trial. Initial and last foot contact times from the GaitRite data were applied to determine stance and swing phases of the gait cycle. The number of foot contacts analyzed ranges from 4-10 per trial. A second-order low-pass Butterworth filter was applied to electrical goniometer signal with a cutoff frequency of 3 Hz (D. A. Winter, 2005). The participant's ankle position while standing before the walking trial was considered to be neutral (90°). Relative ankle dorsiflexion and plantarflexion motions were calculated from neutral by applying the slope (approx. 17.2 uV/degree) calculated from the manual calibration. Outcome measures include magnitude and time of peak ankle dorsiflexion and plantarflexion during the stance and swing phases. Times of peak joint motions are expressed as percent of gait cycle time. EMG signals were baseline corrected, full-wave rectified and to minimize the effect of movement artifacts a second-order low-pass Butterworth filter was applied at a frequency of 20 Hz (Lamontagne et al., 2000). EMG activity recorded during 10 seconds of quiet standing prior to the walking trial was used to determine baseline amplitude. EMG burst detection onset and offset times were determined as greater than and less than the mean + 4*standard deviations of the baseline amplitude and at least 50 ms in duration, respectively. Onset, offset and activation times are expressed as a percent of gait cycle time. Coactivation duration was calculated as the amount of gait cycle time both the TA and MG activations were above threshold level. Cross-correlation coefficients were calculated between the TA and MG activations during each gait cycle as follows in Matlab (The Math Works Inc, Natick, MA):

$$R_{xy}(\tau)=\frac{\frac{1}{N}\sum_{i=1}^{N}(x_i-\overline{x})(y_{i+\tau f_s}-\overline{y})}{\frac{1}{N}\sqrt{\sum_{i=1}^{N}(x_i-\overline{x})^2\sum_{i=1}^{N}(y_i-\overline{y})^2}}$$

N is the number of data points in the input signal records, τ is the discrete temporal phase shift, and fs is the frequency at which the original signals are sampled (Nelson-Wong et al., 2009). R_{xy} ranges between -1 and +1. A positive correlation indicates the TA and MG signals are increasing and decreasing together, while a negative value indicated an inverse relationship. Outcome measures include the correlation coefficient and temporal phase shift.

Participants were divided into two groups based on the presence of dropped foot during gait. Criteria for dropped foot included 1) an AFO prescription at discharge or 2) peak ankle dorsiflexion during the swing phase and/or stance ROM less than normative values from healthy adults walking at a slow cadence (mean ± 1SD; D. A. Winter, 1991), along with ankle plantarflexion position at initial contact. Toe drag along the mat in the swing phase of gait was noted during the testing session. One participant was unable to complete the fast walking trials due to low tolerance.

A three-way repeated measures analysis of variance (ANOVA), one between factor (group) and two within factors (limb and condition), was utilized to compare sagittal ankle kinematics and muscle activation patterns. There were two levels for each factor; 1) group – dropped foot vs. non-dropped foot, 2) limb – paretic vs. non-paretic and 3) condition – preferred vs. fast walking. Post hoc Tukey analysis was conducted on pair-wise comparisons. Outliers were identified as three standard deviations (SD) from the mean and removed after justification from visual inspection of the data (9 foot contacts removed due to partial foot pressure data on mat). Shapiro-Wilk test was utilized to check for a normal distribution; scores <0.90 were rank-transformed prior to statistical analysis including time of peak ankle dorsiflexion during stance, TA onset time and MG onset time. Pearson's correlations were performed to relate peak swing phase ankle dorsiflexion and stance phase ankle range of motion (ROM) to TA-MG coactivation duration per limb. Reported values are mean ± SD when appropriate. Statistical significance was set at alpha 0.05.

3.3 Results

Twelve participants met our criteria for dropped foot impairment during gait. Gait velocity was significantly slower for individuals with dropped foot compared to those without under both walking conditions (preferred: 0.43 m/s vs. 0.68 m/s, fast: 0.7 m/s vs. 1.06 m/s, respectively, $p=0.013$). All participants were able to increase their gait velocity in the fast condition. The dropped foot group had a significantly shorter step length and longer stance phase percentage of gait cycle time (GC%) compared to the non-dropped foot group (0.33 m vs. 0.46 m, $p=0.003$ and 73.7% vs. 69.5%, $p=0.014$). A significant main effect was found between limbs indicating a shorter stance phase GC% on the paretic compared to non-paretic side (69.1% vs. 72.2%, $p<0.001$). The interaction between group and limb indicates a significantly greater difference in stance phase GC% between limbs for the dropped foot group compared to non-dropped foot group (non-paretic - paretic: 7.2% vs. 1.7%, p=0.003). All participants reduced their stance phase GC% during the fast condition (p<0.001).

Sagittal ankle kinematics

Data for sagittal ankle kinematic parameters are summarized in Table 3.2. The dropped foot group had a significantly reduced swing phase peak dorsiflexion compared to the non-dropped foot group (p=0.01). Total ankle ROM during swing was slightly lower for the dropped foot group (p=0.053). No significant difference was found between groups for stance phase ankle ROM, stance phase peak dorsiflexion, and peak plantarflexion in both phases of the gait cycle (p>0.05). Participants demonstrated a greater stance phase peak dorsiflexion in the preferred compared to fast condition (p=0.022) and for the non-paretic compared to paretic limb (p=0.023). Stance phase ankle ROM significantly decreased in the fast compared to preferred walking condition (p=0.015) and no difference was found between limbs (p=0.173). The main effects of condition and limb was not significant for stance phase peak plantarflexion, swing phase peak dorsiflexion and plantarflexion, and swing ankle ROM (p>0.05).

The dropped foot group demonstrated a delayed peak stance phase ankle dorsiflexion compared to the non-dropped foot group (p=0.05). Time of peak ankle plantarflexion during stance was not significantly different between groups and revealed a high variability within most participants (p>0.05). No difference was found between groups for time of peak dorsiflexion and plantarflexion during the swing phase (p>0.05). In the fast condition, stance and swing phase peak dorsiflexion occurred earlier compared to the preferred condition (p<0.001 and p=0.002, respectively). A significant interaction effect was found between group and condition for swing phase peak dorsiflexion time; participants with dropped foot had a greater difference between conditions indicating an earlier peak dorsiflexion in the swing phase during fast walking (p=0.006). Paretic limb time of peak dorsiflexion in stance and swing occurred earlier in the gait cycle than the non-paretic limb (p<0.001 and p=0.007, respectively). The interaction effect between group and limb was significant for time of peak dorsiflexion in stance and swing, indicating a greater difference between limbs for the dropped foot group (p=0.002 and p=0.041, respectively). No significant difference was found between conditions and limbs for time of peak plantarflexion during stance and swing phases (p>0.05).

Muscle activation patterns

Data for TA and MG activation parameters are summarized in Table 3.3. Participants with dropped foot demonstrated a significantly delayed TA onset time compared to the non-dropped foot group, while MG onset time was similar between groups ($p<0.001$ and $p=0.231$; Figure 3.1). The non-dropped foot group had longer TA activation time in the stance and swing phases than the dropped foot group ($p=0.005$, $p<0.001$; Figure 3.2). No difference was found between groups for stance phase MG activation time, while the swing phase time was slightly greater for the non-dropped foot group ($p=0.175$, $p=0.047$; Figure 3.3). In the fast condition, TA and MG onset times occurred earlier and activation times were greater than the preferred condition ($p<0.001$; Figure 3.1, 3.2 and 3.3). No significant difference was found between limbs for TA and MG onset times, and activation times during stance and swing ($p>0.05$). A significant interaction effect was found between group and limb for stance phase TA activation time; the dropped foot group had a greater activation time for the non-paretic compared to paretic limb, while the non-dropped foot group demonstrated a greater paretic compared to non-paretic limb activation time ($p=0.016$). Two participants in the dropped foot group did not activate the paretic MG above threshold throughout the gait cycle in approximately 50% of the recorded foot contacts.

Stance and swing phases coactivation time between the TA and MG muscles was significantly greater for participants without dropped foot compared to those with dropped foot ($p=0.01$, $p=0.013$; Figure 3.4). Participants significantly increased TA-MG coactivation time during the fast walking condition for both phases of the gait cycle ($p<0.001$). The main effect for limb was not significant for TA-MG coactivation time ($p>0.05$). The non-dropped foot group increased swing phase TA-MG coactivation time greater than the dropped foot group in the fast condition ($p=0.02$). On average, the cross-correlation coefficient between the TA and MG was positive and displayed a low magnitude throughout the gait cycle. There was no significant difference between groups, limbs and conditions for the TA-MG cross-correlation coefficient ($p=0.6$). The max TA-MG displayed a trend towards a greater correlation for the non-dropped foot compared to the dropped foot group ($r=0.41$ vs. $r=0.37$, $p=0.068$). The fast condition had a significantly greater max TA-MG correlation compared to the preferred condition ($r=0.43$ vs. $r=0.37$, $p<0.001$) and no difference was found between limbs ($p=0.38$). The temporal phase shift of max TA-MG was not significantly different between groups, limbs, and conditions ($p>0.05$).

Relationship between sagittal ankle kinematics and TA-MG coactivation

Swing phase TA-MG coactivation time was not significantly related to swing phase peak dorsiflexion on the paretic and non-paretic side (df=54, r=0.153, p=0.284 and r= -0.169, p=0.236, respectively). The non-paretic side revealed a trend towards a negative correlation between stance phase ROM and TA-MG coactivation time (df=54, r= -0.274, p=0.049), while the paretic side showed no relationship (df=54, r= -0.02, p=0.889).

Table 3.2: Summary of sagittal ankle kinematic parameters for stance and swing phases of the gait cycle.

	NON-DROPPED FOOT				DROPPED FOOT			
	PREFERRED		FAST		PREFERRED		FAST	
	PARETIC	NON-PARETIC	PARETIC	NON-PARETIC	PARETIC	NON-PARETIC	PARETIC	NON-PARETIC
STANCE PHASE								
PEAK DF [B,C]	-8.9 ± 3.4	-9.3 ± 4.0	-7.5 ± 3.6	-8.3 ± 3.8	-7.7 ± 5.0	-9.3 ± 4.1	-7.6 ± 6.0	-9.0 ± 5.3
DF TIME %GC [A,B,C,E,G]	46.7 ± 10.7	46.7 ± 10.6	43.3 ± 13.4	44.3 ± 12.5	41.9 ± 10.9	58.1 ± 10.4	40.9 ± 10.9	52.1 ± 9.2
PEAK PF [G]	9.1 ± 4.9	10.1 ± 4.1	9.0 ± 5.9	9.6 ± 3.9	10.9 ± 7.2	8.5 ± 4.5	9.2 ± 6.3	9.9 ± 4.1
PF TIME %GC	25.5 ± 31.6	30.3 ± 35.0	25.4 ± 29.7	30.0 ± 32.7	27.6 ± 33.0	45.0 ± 39.1	24.9 ± 31.5	34.2 ± 38.0
ROM [B]	18.0 ± 6.0	19.5 ± 6.2	16.5 ± 6.7	17.9 ± 5.5	18.6 ± 11.0	17.8 ± 6.4	17.0 ± 10.1	18.9 ± 7.9
SWING PHASE								
PEAK DF [A]	0.7 ± 4.2	2.5 ± 4.5	0.5 ± 4.3	1.7 ± 4.6	3.9 ± 4.8	3.7 ± 3.5	3.3 ± 5.2	4.4 ± 4.6
DF TIME %GC [B,C,D,E,F]	82.1 ± 8.8	82.5 ± 9.8	80.4 ± 8.8	83.3 ± 7.7	82.7 ± 12.8	90.7 ± 8.6	77.4 ± 12.8	86.5 ± 11.1
PEAK PF [G]	8.7 ± 4.6	10.4 ± 4.1	8.0 ± 4.9	9.7 ± 4.2	11.2 ± 7.8	8.4 ± 4.1	9.8 ± 6.9	10.1 ± 3.8
PF TIME %GC	87.9 ± 13.5	87.1 ± 13.0	86.4 ± 15.2	85.4 ± 14.9	90.8 ± 11.0	88.4 ± 9.3	89.2 ± 11.8	87.3 ± 10.9
ROM	7.9 ± 4.2	8.0 ± 4.5	7.3 ± 4.0	7.9 ± 4.0	7.0 ± 6.2	4.8 ± 3.3	6.7 ± 5.6	5.7 ± 3.6

Abbreviations: DF, dorsiflexion; PF, plantarflexion; GC, gait cycle.

Note: Values are mean ± SD. Positive values indicate a plantarflexion position and negative values a dorsiflexion position. ANOVA results were statistically significant at $p<0.05$ for the following effects: [A]Group, [B]Condition, [C]Limb, [D]Group*Condition, [E]Group*Limb, [F]Condition*Limb and [G]Group*Condition*Limb.

Table 3.3: Summary of TA and MG activation during the gait cycle

	NON-DROPPED FOOT				DROPPED FOOT			
	PREFERRED		FAST		PREFERRED		FAST	
	PARETIC	NON-PARETIC	PARETIC	NON-PARETIC	PARETIC	NON-PARETIC	PARETIC	NON-PARETIC
STANCE PHASE								
TA ONSET [A,B]	1.8 ± 9.1	3.8 ± 15.5	0.3 ± 1.5	3.8 ± 15.9	17.1 ± 25.0	7.2 ± 15.2	8.5 ± 18.1	3.6 ± 6.0
TA ACTIVATION [A,B,E]	51.1 ± 18.5	47.8 ± 19.1	59.5 ± 10.9	53.0 ± 18.6	29.0 ± 19.9	43.8 ± 19.3	38.6 ± 17.4	50.4 ± 15.2
MG ONSET [B]	8.8 ± 13.6	8.8 ± 13.3	5.0 ± 11.1	4.2 ± 9.6	7.7 ± 13.0	23.8 ± 26.3	9.6 ± 21.9	16.4 ± 19.0
MG ACTIVATION [B]	42.7 ± 21.6	42.7 ± 20.7	51.7 ± 18.5	55.1 ± 16.2	27.7 ± 23.6	32.1 ± 21.3	41.2 ± 25.1	42.4 ± 21.1
TA-MG COACTIVATION [A,B]	32.9 ± 22.3	30.8 ± 21.1	47.3 ± 19.4	44.8 ± 20.8	15.0 ± 19.5	17.5 ± 17.8	26.4 ± 22.4	28.6 ± 21.5
SWING PHASE								
TA ACTIVATION [A,B]	23.7 ± 10.3	21.1 ± 9.9	31.3 ± 6.9	27.3 ± 9.4	12.1 ± 12.2	13.6 ± 9.1	21.3 ± 11.1	16.7 ± 11.4
MG ACTIVATION [A,B,G]	15.9 ± 12.1	16.2 ± 12.3	23.9 ± 13.0	26.2 ± 11.3	9.7 ± 12.2	7.6 ± 8.9	20.0 ± 13.7	12.0 ± 12.6
TA-MG COACTIVATION [A,B,D]	13.2 ± 11.6	12.6 ± 11.5	23.2 ± 12.9	22.7 ± 11.8	6.1 ± 10.1	5.2 ± 7.4	14.6 ± 12.4	9.5 ± 11.0

Abbreviations: TA, tibialis anterior; MG, medial gastrocnemius.

Note: Values are mean ± SD. ANOVA results were statistically significant at $p<0.05$ for the following effects: [A]Group, [B]Condition, [C]Limb, [D]Group*Condition, [E]Group*Limb, [F]Condition*Limb and [G]Group*Condition*Limb.

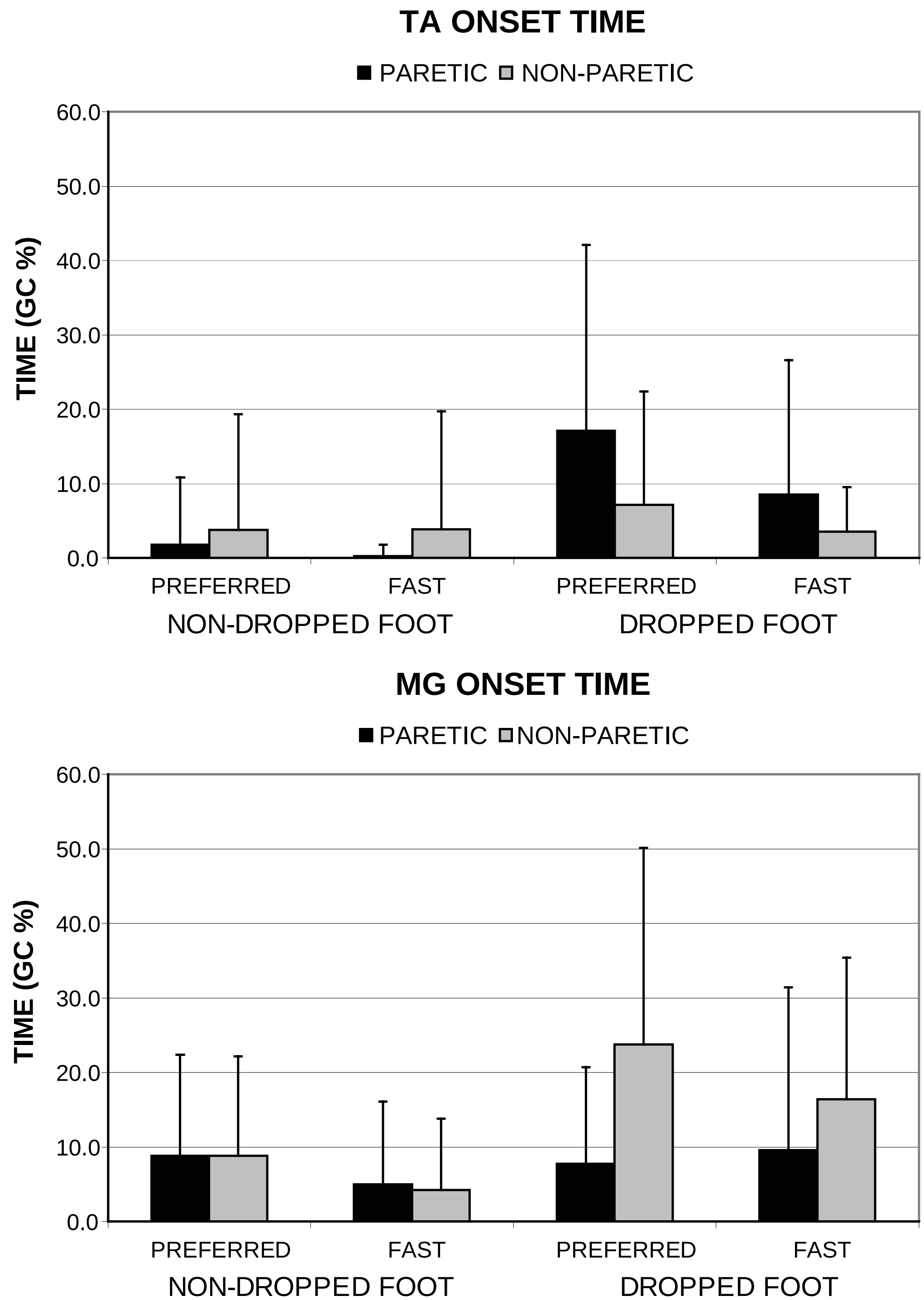
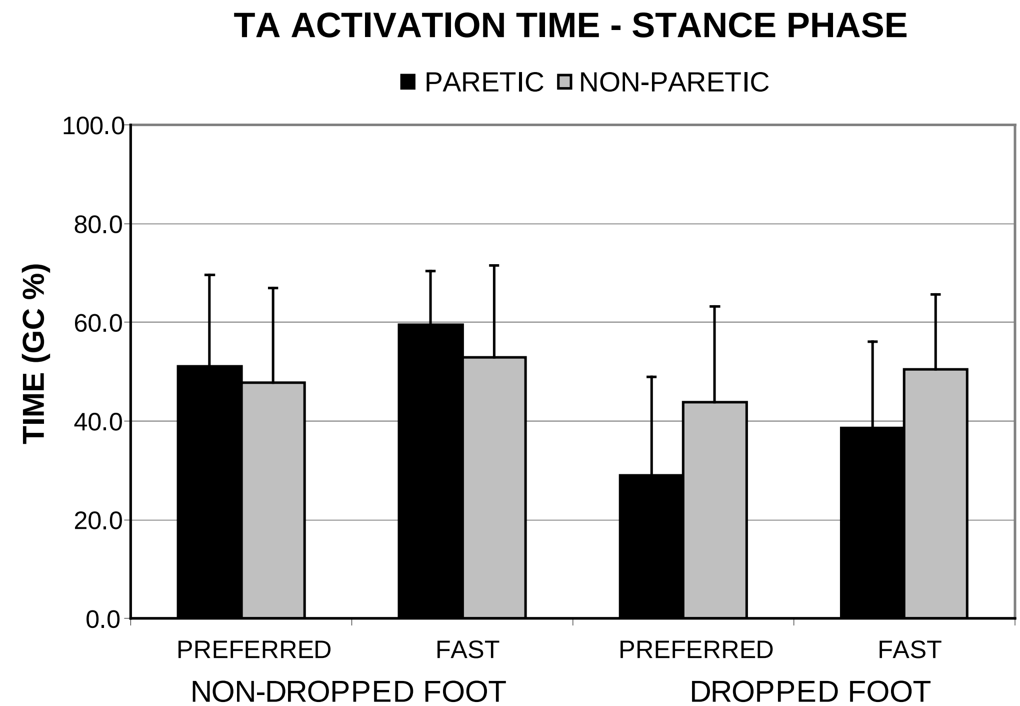


Figure 3.1: Comparison of TA and MG onset times between limbs and walking conditions for both groups. Values illustrated are means with SD error bars. TA onset time was significantly delayed for the dropped foot compared to non-dropped foot group and occurred earlier in the fast compared to preferred walking condition ($p<0.001$). MG onset time was significantly later in the GC during the fast compared to preferred walking condition ($p<0.001$).



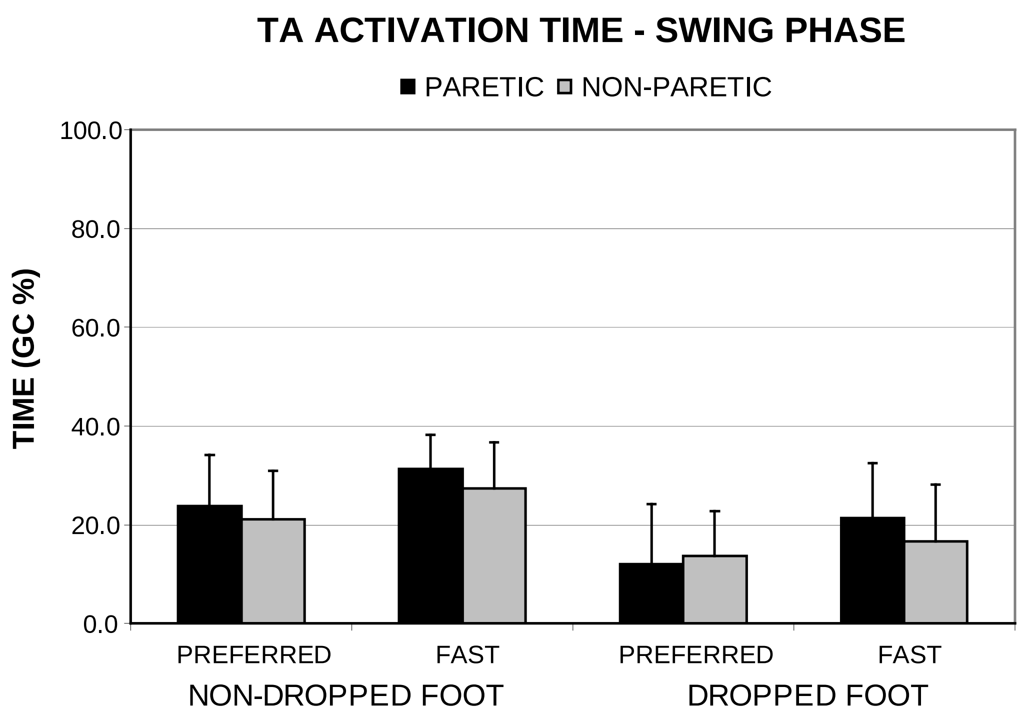
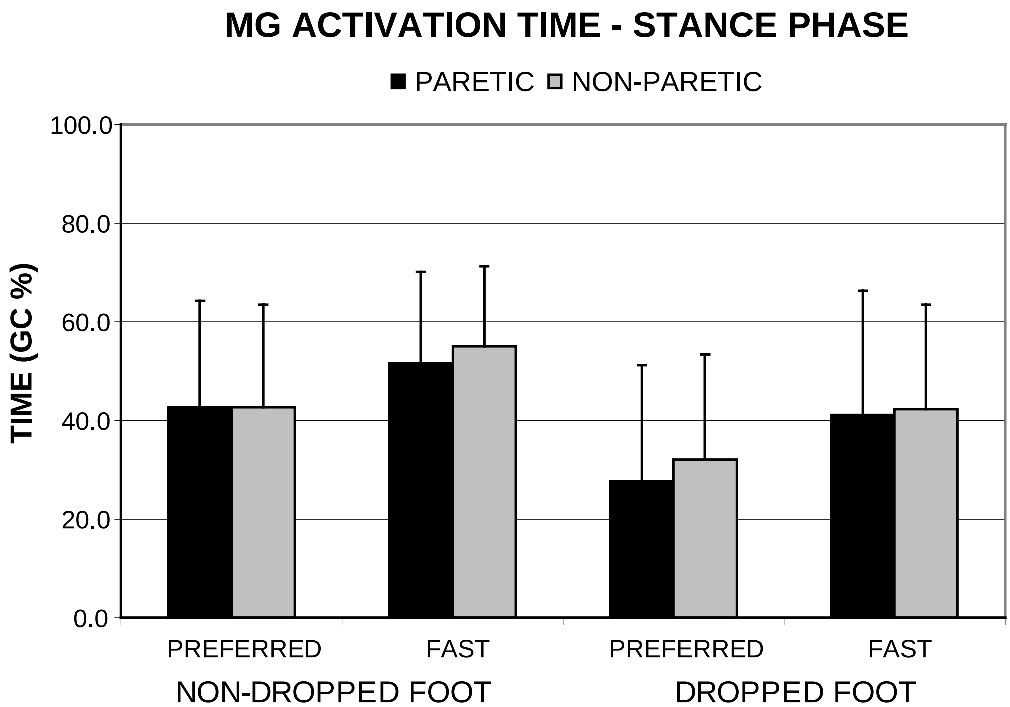


Figure 3.2: Comparison of TA activation time in the stance and swing phases between limbs and walking conditions for both groups. Values illustrated are means with SD error bars. TA activation time during stance had significant main effects between groups ($p=0.005$) and conditions ($p<0.001$), and a group*limb interaction effect ($p=0.016$). In the swing phase, TA activation time was significantly greater for the non-dropped foot compared to dropped foot group and in the fast compared to preferred condition ($p<0.001$).



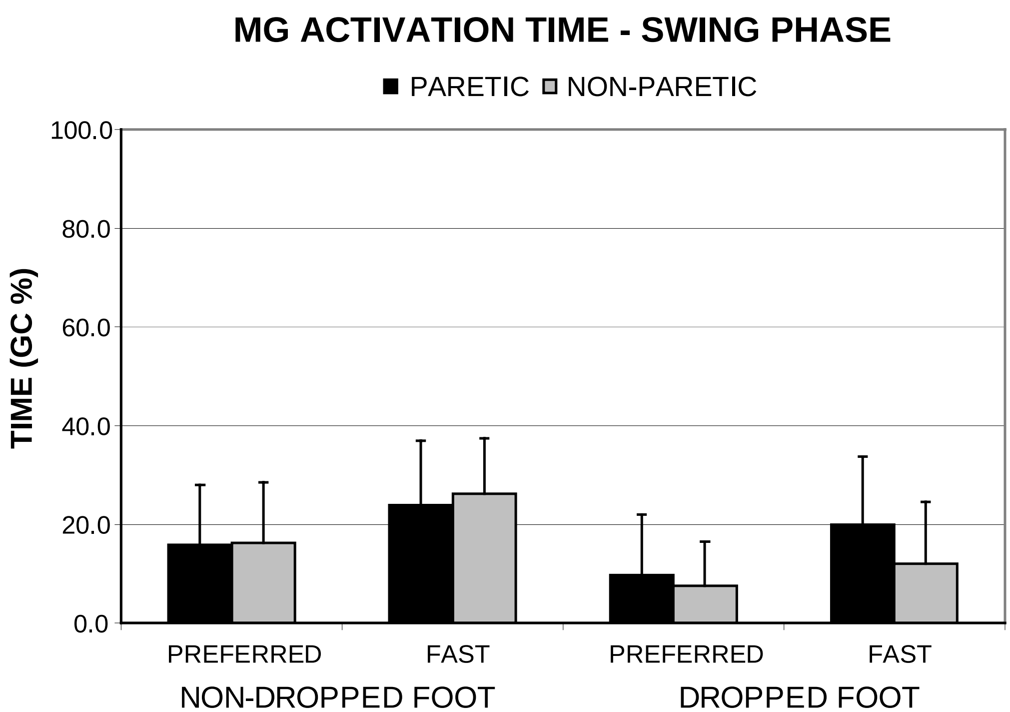


Figure 3.3: Comparison of stance and swing MG activation duration time between limbs and walking conditions for both groups. Values illustrated are means with SD error bars. MG activation time during stance was significantly greater in the fast compared to preferred condition ($p<0.001$). In the swing phase, main effects revealed a greater MG activation time for the non-dropped foot compared to dropped foot group ($p=0.047$) and in the fast compared to preferred condition ($p<0.001$). A significant group*limb*condition interaction effect was found in the swing phase ($p=0.007$).

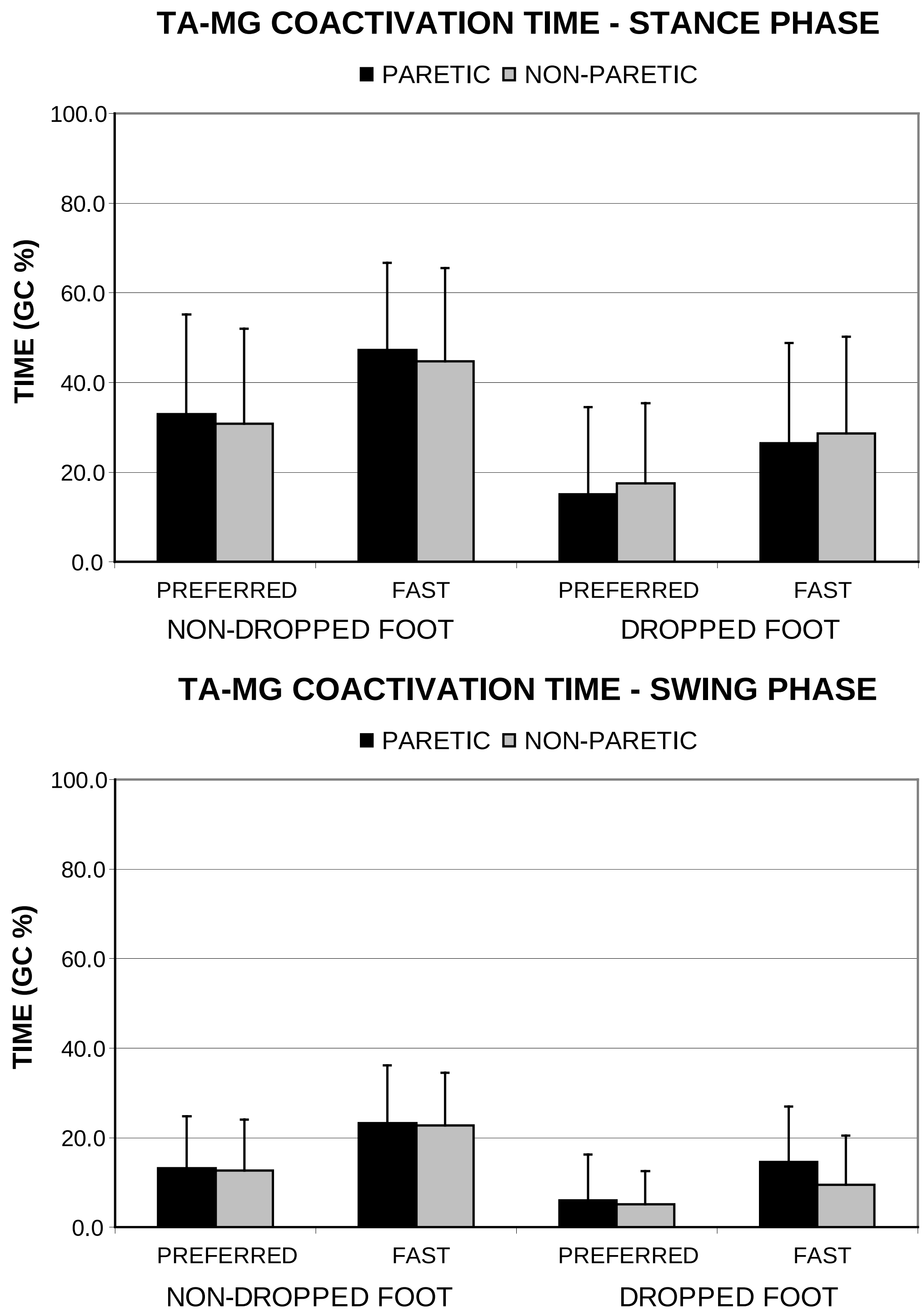


Figure 3.4: Comparison of TA-MG coactivation time during the stance and swing phases between limbs and walking conditions for both groups. Values illustrated are means with SD error bars. TA-MG coactivation time during stance was significantly greater for the non-dropped foot compared to dropped foot group ($p=0.01$) and in the fast compared to preferred condition ($p<.0001$). In the swing phase, main effects were significant for group ($p=0.013$) and condition ($p<0.001$). A significant group*condition interaction effect was found in the swing phase ($p=0.026$).

3.4 Discussion

The results from this study indicate that stroke survivors with dropped foot impairment during gait demonstrated reduced peak dorsiflexion in the swing phase, delayed TA onset time in stance, decreased TA activation time, and reduced TA-MG coactivation time on the paretic side compared to those without dropped foot. Altered timing of lower limb muscle activity may indicate reduced ability to voluntarily generate force and compensatory neuromuscular strategies to facilitate gait function. Reduced and delayed paretic limb TA activation time may represent impaired ability to control the foot position at initial contact and maintain postural stability during stance. Individuals with dropped foot decreased stance time on the paretic limb relative to the non-paretic limb, thus demonstrating a temporally asymmetric gait pattern. Excessive or insufficient TA-MG coactivation is postulated as a mechanism of impaired ankle-foot function during gait (Lamontagne et al., 2000). We found no difference between limbs for coactivation time during the stance and swing phase, and the cross-correlation co-efficient amplitude. In contrast to our hypothesis, TA-MG coactivation time was not related to swing phase peak dorsiflexion and ankle ROM during stance on the paretic side. These findings suggest bilateral muscle weakness and delayed activation on the paretic side in regards to mechanisms of dropped foot impairment.

Approximately 29% and 40% of participants had reduced peak dorsiflexion in the swing phase on the paretic and non-paretic side relative to healthy controls at a slow cadence (D. A. Winter, 1991). Our participants also had a reduced swing phase ankle ROM and decreased peak ankle plantarflexion in the stance phase compared to normative values at a slow cadence (D. A. Winter, 1991). Previous studies have reported increased ankle plantarflexion during swing and decreased ankle plantarflexion at toe-off among stroke survivors, with the deviation magnitude related to walking speed (Olney, Griffin, & McBride, 1994; Olney & Richards, 1996). Possible reasons for altered stance-swing phase kinematics may be found in the positive associations between MG activation, peak plantarflexor moment and walking speed (Lamontagne et al., 2002). Some participants with dropped foot failed to activate the paretic MG above threshold throughout the gait cycle and had a slower gait velocity. Therefore, limiting force generation for push-off and resulting in reduced joint excursions. The slower gait velocity among the dropped foot participants may partially explain group differences in peak joint excursions during the stance phase. On the non-paretic side, participants with dropped foot demonstrated a delayed stance and swing phase peak dorsiflexion. This finding may reflect increased time spent in the stance phase and possibly compensatory strategy to delay forward progression due to difficulty advancing the paretic limb. Five participants in the dropped foot group had paretic limb peak dorsiflexion during swing within the normal range; however 4 demonstrated a reduce stance phase ankle ROM and 3 had an AFO prescription at discharge. Reduced ROM throughout the gait cycle may represent increased passive joint stiffness and/or prolonged coactivation of upper leg muscles during mid-stance to enhance postural stability due to insufficient support from the ankle plantarflexors (Higginson, Zajac, Neptune, Kautz, & Delp, 2006; Lamontagne et al., 2000; Lamontagne et al., 2002). Nine participants in the non-dropped foot group had a peak dorsiflexion value meeting the criteria for too much plantarflexion during swing. They were not considered dropped foot because the stance phase ROM was within normal limits, no toe drag was noted in the testing session and an AFO was not prescribed at discharge. These participants were able to increase knee flexion during the swing phase for toe clearance and limit ankle plantarflexion at initial contact.

Delayed TA onset at initial contact and reduced activation duration in stance and swing appear to be mechanisms of dropped foot impairment during gait. This may be due to the inability to rapidly activate the TA due to reduced central drive for muscle recruitment and/or poor sensory feedback to initiate a response (Den Otter et al., 2007). While no difference was found between groups, early MG onset during the stance phase may indicate an adaptive strategy to control the loading response with a plantarflexed position at initial contact. Another study has reported longer MG activity in the first double support phase among stroke survivors relative to healthy controls (Den Otter et al., 2007). Reduced duration of TA and MG muscle activity in the swing phase may indicate insufficient force output required for toe clearance. Although muscle activation times increased during the fast walking condition, ankle dorsiflexion did not improve in the swing phase. However, TA-MG coactivation among the dropped foot group only demonstrated a slight increase during the fast condition compared to those without dropped foot. This further indicates poor levels of force output as a factor limiting ankle dorsiflexion during swing.

Given our previous findings, it is not surprising that individuals with dropped foot also displayed lower TA-MG coactivation times throughout the gait cycle. As well, there was a trend towards lower peak cross-correlation index suggesting a poor coactivation magnitude (Nelson-Wong et al., 2009). Functionally a deficit in coactivation levels during stance may impact postural stability due to its role in regulating joint stiffness (Levin & Dimov, 1997). Since muscle activation times were reduced, TA-MG coactivation is not likely the factor limiting ankle dorsiflexion in the swing phase. This is supported by the non-significant correlation found between these parameters for both limbs. The present results revealed that increased TA-MG coactivation time was likely to limit stance phase ROM on the non-paretic side. Likewise in the fast walking condition, coactivation time increased with gait velocity and stance phase ROM slightly decreased. Increased coactivation time during stance may represent a mechanism to enhance postural stability during gait on the non-paretic side (Lamontagne et al., 2000). Similar to Den Otter et al 2007, we found no difference between limbs for TA-MG coactivation time in stance and swing (Den Otter et al., 2007). Previously, Lamontagne et al 2000 reported increased of TA-MG coactivation during the double support phases on the non-paretic side and low levels during the single support phase on the paretic side (Lamontagne et al., 2000). A possible explanation for the difference in results may be the criteria used to determine EMG threshold cut-off points.

In the present study, the assessment of motor impairments contributing to poor ankle function during gait did not include the influence at the knee and hip joints. It is likely that proximal motor impairments will affect toe clearance during the swing phase and dynamic stability during stance. Our focus was to identify and quantify impairments most likely to be involved in poor ankle function during gait among stroke survivors. Individuals with dropped foot walked at a significantly slower gait velocity, which may partially explain some of the group differences in peak joint excursions for the stance phase of gait. The effect of gait velocity on joint kinematics can be examined by comparing changes between conditions (i.e. preferred and fast) and limbs (i.e. paretic and non-paretic). Participants with dropped foot had a significantly longer inpatient rehabilitation stay, which may indicate more severe impairments at admission. On average, the assessment was conducted within 8 ± 10 (SD) days from discharge. This time point was selected because patients are more likely to be ambulatory with mild-moderate sensorimotor impairments and physiotherapists may recommend an ankle-foot orthotics for those with dropped foot before discharge. It is possible that mechanisms of poor sensorimotor control related to gait deviations may change over time.

In conclusion, individuals with dropped foot demonstrated reduced swing phase dorsiflexion, and delayed stance phase peak dorsiflexion on the non-paretic side compared to stroke survivors without dropped foot. Delayed TA onset combined with reduced TA and MG muscle activation times contributes to dropped foot gait deviations. Our findings indicate bilateral weakness of the MG may impair gait performance. Continued study needs to examine changes in specific mechanisms of dropped foot impairment over time. Overall this work highlights altered temporal patterns of lower limb muscle activity contributing to dropped foot impairment during gait among stroke survivors.

CHAPTER 4

A case series comparing the immediate effects of ankle-foot orthotic and functional electrical stimulation devices on gait deviations post stroke

4.1 Introduction

Recovery of walking function is a goal most often stated by individuals after stroke and represents primary focus of rehabilitation programs (Bohannon, Andrews, & Smith, 1988). Gait deficits greatly contribute to poor functional mobility, which is associated with a higher incidence of falls (Forster & Young, 1995; Hyndman, Ashburn, & Stack, 2002) and reduced activity participation (Muren, Hutler, & Hooper, 2008). An ankle-foot orthotic (AFO) is often recommended to minimize gait deviations caused by dropped foot impairment. In particular, AFOs manipulate the foot's position to provide medial-lateral stability at the ankle during the stance phase, limit ankle plantarflexion at initial contact and assist with toe clearance in the swing phase. Typical clinical indications for an AFO prescription include ankle dorsiflexor weakness, ankle plantarflexor spasticity and knee instability, wherein significant gait deviations compromise walking safety (Condie et al., 2004). Advantages of using an AFO include easy application, consistent support during stance and useful for many physical activities. Findings from a systematic review suggest AFOs immediately improve gait velocity and ankle dorsiflexion in the swing phase, while the effect on lower limb muscle activity remains inconclusive due to large individual differences (Leung & Moseley, 2003). Hesse et al reported reduced tibialis anterior (TA), increased vastus laterialis and no change in medial gastrocnemius (MG) activity amplitudes with a rigid non-articulating AFO compared to barefoot walking (Hesse et al., 1999). A recent study observed reduced TA amplitudes from pre-swing to loading response only with a more flexible hinged AFO compared to shoed walking, while the rigid non-articulating and dorsi-assist/dorsi-stop AFOs demonstrated no changes in muscle activation among individuals with stroke (Mulroy et al., 2010). These findings support the concern for learned muscle disuse with AFO devices; however variations in mechanical components of the AFO design and sensorimotor impairments among stroke survivors may explain conflicting results. Despite the fact that approximately 22% of stroke survivors required an AFO at discharge from rehabilitation (Teasell et al., 2001), current literature highlights the need for more specific investigation into clinical characteristics of stroke survivors that may benefit from a prescription.

An alternative option is application of a functional electrical stimulation (FES) device, which activates the common peroneal nerve to elicit ankle dorsiflexion with some eversion in the swing and early stance phases of gait. Individuals with dropped foot have reported to perceive improved stability, greater confidence, and less fatigue when walking with an FES device compared to an AFO and no device (Tyson & Thornton, 2001). Application of FES devices with stroke survivors has demonstrated immediate improvement in gait velocity, temporal symmetry and reduced energy cost (Burridge et al., 1997; Burridge et al., 2007; Hausdorff & Ring, 2008; Stein et al., 2010; Taylor et al., 1999). Kesar et al reported increased ankle dorsiflexion and reduced knee flexion during the swing phase, and reduced ankle plantarflexion at toe-off with an FES device compared to no device (Kesar et al., 2009). FES applied to both the ankle dorsiflexors and plantarflexors immediately resulted in greater swing phase knee flexion, greater ankle plantarflexion at toe-off and increased forward propulsion compared to stimulation of only the dorsiflexors (Kesar et al., 2009). This finding is functionally important because the ability to generate propulsive forces at the transition from stance to swing represents a critical indictor of gait dysfunction post stroke due to its association with sensorimotor impairment, preferred gait velocity and temporal symmetry (Bowden et al., 2006; Nadeau, Gravel, Arsenault, & Bourbonnais, 1999b). Kottink et al found increased MG maximum voluntary contractions over a 6 month period of walking with FES; however no therapeutic effect on TA activation magnitude during the swing phase stroke survivors (Kottink et al., 2008). Although FES offers potential long term benefits for improved gait velocity and reduced energy cost (Stein et al., 2010), a current review suggests the immediate therapeutic effect on muscle activity is poorly understood and may depend on specific underlying sensorimotor impairments (Robbins et al., 2006).

To date, the few studies that have compared AFO and FES devices in the stroke population observed similar abilities to perform functional walking tests (Sheffler, Hennessey, Naples, & Chae, 2006), no difference in walking speed after 26 weeks of use (Kottink et al., 2008), and improved obstacle avoidance rates on a treadmill with FES (van Swigchem, van Duijnhoven, den Boer, Geurts, & Weerdesteyn, 2012). The potential advantage of FES over an AFO has not been demonstrated by quantitative measures of ankle kinematics and temporal coordination of TA and MG muscle activation during gait.

Since dropped foot is caused different underlying impairments (i.e. muscle weakness and spasticity), various kinematic and muscle activation patterns during gait may be observed. As well, previous research has highlighted bilateral ankle weakness as a contributing factor to gait deviations among stroke survivors (Lamontagne et al., 2002), therefore it is important to examine the impact of AFO and FES devices on bilateral lower limb neuromuscular control. The purpose of this case series is to explore the immediate effects of AFO and FES devices on sagittal ankle kinematics and TA-MG muscle activation patterns during gait among stroke survivors with dropped foot impairment.

4.2 Patient History and Systems Review

Participant 1 (P1) was admitted to the stroke in-patient rehabilitation program and P2-P4 were receiving out-patient stroke rehabilitation services at the Toronto Rehabilitation Institute. Details on participant demographic, stroke information and clinical measures of stroke-related impairments are outlined in Table 4.1. All participants had ischemic strokes affecting their left hemisphere resulting in right side paresis. The specific locations for each participant are pontine/brainstem, basal ganglia, middle cerebral artery, and posterior cerebral artery/thalamus/occipital lobe, respectively. Stroke mechanisms included small vessel thrombosis, thromboembolic and embolism. Medical history indicated no previous strokes; P1 and P2 had type II diabetes and hypertension. All participants were regularly using an AFO for walking as prescribed by their rehabilitation team on transition to out-patient services; pre-fabricated (P1-P2) and custom (P3-P4). Participants were able to ambulate independently with a gait aid indoors, and P2-P4 were ability to walk short distances in their community.

Participants displayed moderate to severe lower limb sensorimotor control impairment as measured by the Chedoke McMaster Stroke Assessment (CMSA) scores (Table 4.1). These scores indicate resistance to passive ankle dorsiflexion-plantarflexion, some voluntary activation for ankle dorsiflexion and in/eversion, and possibly some coordination of ankle dorsiflexion-plantarflexion while sitting. Participants revealed significant right side dorsiflexor and plantarflexor muscle weakness relative to the left side. In particular, P3 and P4 had difficulty with active voluntary movements against gravity (Table 4.1). P2-P4 displayed hip and knee flexor muscle weakness. For the Modified Ashworth Scale (MAS), P1 and P3 demonstrated increased ankle plantarflexor spasticity while sitting with the knee extended. P2-P4 had impaired sensation to light touch and joint position. P3 and P4 complained of right side numbness in the lower limb.

Visual observation of the participants gait pattern revealed typical characteristics of hemiplegic gait, such as slowed velocity and spatial-temporal asymmetry. When walking without using their AFO, participants displayed significant toe drag during the swing phase and poor stability during stance on the paretic side. In addition, P3 and P4 used compensatory movements, such as hip circumduction, to facilitate toe clearance and forward progression of the paretic limb. P4 had an obvious forefoot initial contact and uncontrolled loading response at the ankle. A more detailed assessment of their gait impairments is presented below from the examination.

Table 4.1: Participant demographic, stroke information and clinical data

	P1		P2		P3		P4	
Age (yrs)	60		63		36		35	
Gender	F		M		F		M	
Height (m)	1.58		1.73		1.63		1.83	
Weight (kg)	68		79.5		79.5		81.8	
Time post-stroke (months)	1.3		6.5		12.1		15.3	
Type of stroke	I		I		I		I	
Hemisphere affected	L		L		L		L	
Gait aid	4 PC		4 PC		SPC		SPC	
CMSA leg score	4		3		5		4	
CMSA foot score	3		4		3		3	
BBS	46		51		52		49	
MAS	1		0		2		0	
MRC DF	4		4		3		1	
MRC PF	4		4		2		1	
6MWT (m)	266		160		313		188	
	P	**NP**	**P**	**NP**	**P**	**NP**	**P**	**NP**
PROM Peak DF (deg)	-9.0	-16.5	-13.1	-9.3	-21.5	-13.5	-20.8	-12.7
PROM Total (deg)	36.6	37.4	28.9	46.3	37.1	36.3	41.3	46.4
AROM Peak DF (deg)	-2.0	-5.7	-9.0	-11.8	-1.8	-14.3	-0.6	-16.3
AROM Total (deg)	18.0	31.9	24.1	27.9	10.6	37.4	16.0	34.9
MVC DF (N)	-4.0	-7.4	-8.3	-6.2	-4.3	-5.2	-4.1	-9.3
MVC PF (N)	5.5	5.6	9.0	7.5	2.1	3.2	4.4	5.6

Abbreviations: 4PC, quad point cane; 6WMT, Six Minute Walk Test; AROM, activate range of motion; BBS, Berg Balance Scale; CMSA, Chedoke McMaster Stroke Assessment; DF, dorsiflexion; MAS, Modified Ashworth Scale; MRC, Medical Research Council – manual muscle test score; MVC, maximum voluntary contraction; NP, non-paretic; P, paretic; PF, plantarflexion; PROM, passive range of motion; SPC, single point cane.

Note: Negative values indicate dorsiflexion for PROM and AROM peak DF relative to neutral. PROM and AROM total values indicate the difference between the maximum joint excursion in the dorsiflexion and plantarflexion directions.

4.3 Examination

Participants completed two gait assessments on separate days in The Balance Mobility and Falls Clinic at the Toronto Rehabilitation Institute. In the gait assessment, participants were asked to walk over a pressure sensitive mat with bilateral recordings of ankle kinematics and lower limb muscle activity. Three trials were performed per condition. Participants completed the following walking conditions in order; baseline (no device) preferred speed, and AFO/FES preferred speed. The 6MWT was administered as standardized practice for walking with the AFO/FES device. After practice (post), participants walked at their preferred speed with no device over the mat. Participants used their AFO on the first day followed by the Odstock Dropped Foot Stimulator (ODFS, Appendix 2) for the second gait assessment (2-7 days later).

Additional quantitative standardized tests were performed to evaluate ankle joint range of motion (ROM) and muscle strength. Passive and active ankle ROM was assessed in a seated position with the lower leg supported and knee extended (Clarkson, 2005). Each movement was performed twice to obtain an average score. Maximal voluntary contractions (MVC) were used to determine isometric muscle strength of the ankle dorsiflexors and plantarflexors muscles for both limbs. Participants were in a seated position with their knee at 90° and foot strapped to a load cell. Instructions were to push/pull on the load cell and hold for 2 seconds. This procedure was repeated twice for each muscle for both limbs after 1 minute of rest (Bohannon, 2007).

The GaitRite pressure sensitive mat was used to record spatial-temporal gait parameters (Appendix 2). The mat is 5.25 m in length, 0.88 m in width and contains a grid pattern of 48 by 288 sensors arranged 1.27 cm on center. Data were sampled at a frequency of 120 Hz. Custom made electrical goniometers were placed bilaterally over the lateral malleoli to record sagittal ankle kinematics. The signal was sampled at 1000 Hz and stored for offline analysis (Noraxon Telemyo System, Appendix 2). The device was calibrated with a mechanical goniometer at approximately 17.2 uV/degree. Surface electromyography (EMG) 30 mm silver-silver chloride electrodes were placed bilaterally over the TA and MG muscles (Medi-Trace® Mini, Appendix 2). Manual muscle tests were performed to determine EMG locations. Skin was rubbed with nu-prep and alcohol to reduce the impedance. Electrodes pairs were placed longitudinally 1 cm apart on center over the proximal aspect of the TA and MG muscles. Data was sampled at 1000 Hz. EMG signals were pre-amplified (2000x), filtered online (bandwidth 10-500 Hz) and stored for offline analysis. Goniometer and EMG data was synced with the pressure mat signal to calculate relative foot contact times.

Outcome measures for sagittal ankle kinematics and muscle activation patterns were calculated with a custom software program (Microsoft Visual Basic version 6.0). All data was normalized to the gait cycle (GC) duration; defined as the time interval between two consecutive ipsilateral foot contacts. The GaitRite data analysis system calculated gait velocity, spatial-temporal parameters, and foot contact times to determine stance and swing phases of the GC. Paretic step length refers to the distance from the non-paretic toe to the paretic heel of the next foot contact, respectively. The number of foot contacts analyzed ranges from 4-10 per trial. A second-order low-pass Butterworth filter was applied to the goniometer signal with a cutoff frequency of 3 Hz (D. A. Winter, 2005). Ankle position when standing was measured before the gait assessment. Ankle dorsiflexion and plantarflexion motions were calculated by applying the slope from the manual calibration. Outcome measures include magnitude and time of peak ankle dorsiflexion and plantarflexion during the stance and swing phases. Time to peak dorsiflexion is expressed as percent of GC time. EMG signals were baseline corrected, full-wave rectified and to minimize the effect of movement artifacts a second-order low-pass Butterworth filter was applied at a frequency of 20 Hz (Lamontagne et al., 2000). EMG activity recorded during 10 seconds of quiet standing prior to the walking trial was used to determine baseline amplitude. EMG burst detection onset and offset times were determined as greater than and less than the mean + 4*standard deviations of the baseline amplitude and at least 50 ms in duration, respectively. Onset, offset and activation times are expressed as a percent of GC time. Coactivation was calculated as the amount of time when both the TA and MG activity was above threshold level and expressed as a percent of the GC time. Outcome measures include TA and MG activation time during stance and swing.

Descriptive statistics were calculated for passive and active ROM at rest, dorsiflexor and plantarflexor muscle force, sagittal ankle kinematics, and TA-MG muscle activation patterns. Positive values indicate ankle plantarflexion and negative values for ankle dorsiflexion. A repeated measures analysis of variance (ANOVA) with two factors, condition (baseline, AFO/FES and post) and limb (paretic and non-paretic), was used to determine the main effect (AFO/FES compared to baseline) and carry-over effect (post compared to baseline) for sagittal ankle kinematics and TA-MG muscle activation patterns within each participant and assessment day (Fisch, 2001). Contrasts were performed to compare the AFO/FES and post walking trials to the baseline condition. Values are reported as mean ± SD. Statistical significance was set at alpha 0.01.

4.4 Intervention

A description of the participants AFO device is summarized in Table 4.2. The ODFS was used to deliver surface stimulation to the paretic ankle dorsiflexor muscles during the swing phase of gait. Stimulation was activated when the foot switch placed under the heel was off and deactivated when the heel contacted the ground. The stimulation intensity was initially set at the minimum threshold for eliciting a dorsiflexion response when sitting and adjustments were made if the response was not observed during gait. The settings for each parameter are outlined in Table 4.2. P1 had one ODFS training session with a physiotherapist prior to our intervention, while P2-P4 had never used a dropped foot FES device.

Table 4.2: Summary of AFO designs and FES parameters

		P1	P2	P3	P4
AFO					
	Type	PLS - NA	PLS - NA	Solid - NA	Hinged - AT
	Material	Thermoplastic	Thermoplastic	Thermoplastic	Thermoplastic
	Angle	DF 0°	DF 0°	DF 0°	DF 5°
	ROM	No PF / mod DF	No PF / mod DF	No PF / min DF	No PF / free DF
	Length	Full	Full	Full	Full
	Trim	Posterior	Posterior	Anterior	Anterior
FES					
	Freq (Hz)	35	40	40	40
	Time (s)	3	3	3	3
	CRT (mA)	80	60	60	60-80
	Level	5	7	3-4	6-8
	Ramp (s)	0.5	0.5	0.5	0.5

Abbreviations: AT, articulating; CRT, current amplitude; DF, dorsiflexion; min, minimal; mod, moderate; NA, non-articulating; PF, plantarflexion; PLS, Posterior spring leaf; ROM, range of motion.

Notes: Angle refers to the alignment of the lower limb relative to the foot when standing. ROM refers to the amount of movement permitted by the AFO. Full indicates a foot plate length to the metatarsal heads. Trim refers to the AFO trim lines relative to the ankle malleoli. Level refers to a percent magnitude of the current amplitude on a scale of 10. Ramp refers to the stimulation time to ramp up and down from the heel switch off and on signal.

4.5 Clinical Impression

The participant's sensorimotor and balance impairments contributed significantly to gait deviations that impede their ability to walk safely. From the clinical examination, paretic ankle active ROM was reduced and bilateral force output was low for dorsiflexion-plantarflexion (Table 4.1). The baseline gait assessment confirmed our visual observations; slow gait velocity below the level required for community ambulation (Holden, Gill, & Magliozzi, 1986; Perry, Garrett, Gronley, & Mulroy, 1995) and temporal asymmetry in favour of the non-paretic limb (Patterson et al., 2008). Tables 4.3 and 4.4 summarize ankle kinematic and EMG outcome measures, respectively.

P1 walked at 0.47 m/s with a greater non-paretic step length (0.39 m vs. 0.33 m) and longer non-paretic stance phase (75.2% vs. 65.5%) compared to the paretic side. She had an ankle plantarflexed position at initial contact and low peak dorsiflexion during swing for the paretic limb, along with reduced stance phase ROM compared to the non-paretic side. Greater TA-MG coactivation time was observed during the stance phase on the non-paretic compared to the paretic side.

P2 walked at 0.26 m/s with a short non-paretic step length (0.21 m vs. 0.29 m) and greater stance phase time (85.3% vs. 65.4%) compared to the paretic side. He had a slightly plantarflexed position at initial contact, low peak plantarflexion at toe-off, and low peak dorsiflexion during swing for the paretic limb. A delayed TA onset time (41.7%), low MG duration and absent TA-MG coactivation was observed during the paretic stance phase.

P3 walked at a speed of 0.47 m/s with a shorter non-paretic step length (0.3 m vs. 0.49 m) and longer stance phase time (81.4% vs. 65.8%) compared to the paretic side. She had an ankle plantarflexion position at initial contact, and inadequate peak dorsiflexion in swing for toe clearance on the paretic side. Time to peak dorsiflexion during non-paretic stance was delayed at 59.2% compared to the paretic side at 32.5%. Paretic limb TA duration was low during the swing phase along with little TA-MG coactivation time in stance.

P4 walked at a speed of 0.31 m/s with a shorter non-paretic limb step length (0.15 m vs. 0.34 m) and greater stance phase time (88.2% vs. 72.8%) compared to the paretic side. The paretic ankle was plantarflexed at initial contact, and low peak dorsiflexion during swing impeded toe clearance. A low MG duration and TA-MG coactivation time is observed during the stance phase, while the TA was activated for most of the swing phase on the paretic side.

4.6 Outcomes

See Appendix 3 for kinematic and EMG time series graphs comparing the AFO/FES and post-intervention conditions to the baseline condition for each participant.

P1 – After application of the AFO, gait speed increased to 0.50 m/s. Peak dorsiflexion during the stance phase increased on the paretic side ($p=0.026$) and decreased on the non-paretic side ($p<0.001$), while stance phase ROM did not change for both limbs ($p>0.05$). Paretic limb peak dorsiflexion during swing improved ($p=0.015$), and peak plantarflexion at initial contact decreased ($p=0.021$). Time to peak dorsiflexion occurred earlier during the non-paretic stance phase ($p<0.001$). TA-MG coactivation during stance increased on the paretic side ($p=0.036$).

Gait speed increased to 0.56 m/s with the FES device. Stance phase peak dorsiflexion increased with an earlier time to peak on the paretic side ($p<0.001$). Swing phase peak dorsiflexion increased ($p<0.001$), while plantarflexion at initial contact and toe-off decreased on the paretic side ($p<0.001$). Non-paretic time of peak dorsiflexion occurred earlier in stance phase ($p<0.001$). TA duration and TA-MG coactivation increased in the non-paretic stance and swing phase ($p<0.005$ and $p<0.001$). Post intervention walking trials were not completed.

P2 – Gait speed increased to 0.32 m/s with the AFO. On the paretic side, peak dorsiflexion decreased during stance ($p=0.017$) and increased during swing ($p<0.001$), along with reduced plantarflexion at initial contact and toe-off ($p=0.001$). On the non-paretic side, peak dorsiflexion and ROM decreased during stance ($p<0.01$). TA onset time did not change ($p>0.05$), while TA duration decreased ($p=0.004$) and MG duration increased ($p=0.012$) in the paretic stance phase. Paretic limb TA duration decreased and MG duration increased during swing ($p<0.001$). TA duration and TA-MG coactivation time increased for the non-paretic stance and swing phase ($p<0.001$). When the AFO was removed after practice compared to baseline, paretic peak dorsiflexion in swing decreased and plantarflexion at initial contact increased ($p<0.001$). Stance phase peak dorsiflexion and ROM remained lower on the non-paretic side ($p<0.01$). Paretic limb TA onset time occurred later with shorter durations in stance and swing, while MG duration remained higher in the swing phase ($p<0.001$). Non-paretic limb MG onset shifted later in stance with a longer duration ($p<0.01$).

Comparing walking with FES device to baseline, gait speed decreased to 0.28 m/s. Paretic limb peak dorsiflexion increased during stance ($p=0.008$) with reduced plantarflexion at initial contact ($p<0.001$) and greater plantarflexion at toe-off ($p<0.001$). Time to peak dorsiflexion increased during the paretic stance phase ($p=0.004$). On the non-paretic side, plantarflexion increased at initial contact and toe-off, along with stance phase ROM ($p<0.01$). MG onset occurred later in stance and TA duration reduced in stance on the non-paretic side ($p<0.01$). Stance and swing phase TA-MG coactivation decreased on the non-paretic side ($p<0.01$). When the FES was turned off after practice, all kinematic outcome measures returned to baseline values except for ankle plantarflexion at toe-off remained higher on the non-paretic side ($p=0.001$). Paretic limb TA and MG durations increased in the stance phase resulting in greater coactivation ($p<0.001$).

P3 – When the AFO was applied, gait speed increased to 0.74 m/s. The AFO restricted paretic ankle ROM in stance by reducing peak dorsiflexion, decreasing plantarflexion at initial contact and at toe-off ($p<0.001$). Paretic swing phase peak dorsiflexion increased ($p<0.001$). Non-paretic peak dorsiflexion was slightly lower and occurred earlier in stance ($p=0.018$, $p<0.001$). Plantarflexion was reduced at initial contact and toe-off on the non-paretic side ($p<0.01$). TA and MG durations in the swing phase increased with greater coactivation time for both limbs ($p<0.001$). When the AFO was removed after practice, peak dorsiflexion in stance was greater than baseline on the non-paretic side ($p=0.004$) and slightly lower than baseline on the paretic side ($p=0.02$). Plantarflexion at initial contact was lower for both limbs ($p<0.001$). Time to peak dorsiflexion remained earlier than baseline on the non-paretic side ($p<0.001$). Stance phase TA and MG durations increased for the paretic limb ($p<0.001$). Swing phase TA duration remained greater than baseline on the paretic side ($p<0.001$), and MG duration was higher than baseline on the paretic ($p<0.001$) and non-paretic sides ($p=0.013$).

When walking with the FES device, gait speed increased to 0.63 m/s. Paretic ankle ROM in stance decreased ($p=0.008$), with no significant changes in peak dorsiflexion and plantarflexion at toe-off ($p>0.05$). Non-paretic peak dorsiflexion was reduced in stance with a lower ROM ($p<0.001$). Plantarflexion at initial contact slightly decreased for the paretic ankle ($p=0.04$) and increased for the non-paretic ankle ($p=0.03$). Stance phase TA duration reduced and swing phase MG duration increased on the non-paretic side ($p<0.001$), along with greater TA-MG coactivation time ($p<0.001$). When the FES device was turned off, time to peak dorsiflexion in stance reduced for paretic ($p<0.001$) and non-paretic ($p=0.003$) limbs compared to baseline. Plantarflexion at initial contact decreased and at toe-off increased on the paretic side ($p<0.001$). MG duration increased in stance and swing on the non-paretic side with more TA-MG coactivation time ($p<0.001$).

P4 – Gait speed increased to 0.35 m/s with a hinged AFO. Time to peak dorsiflexion increased during the paretic stance phase ($p<0.001$) and ankle plantarflexion was reduced at initial contact ($p<0.001$) and toe-off ($p=0.007$). Peak dorsiflexion during swing increased on the paretic side ($p<0.001$). No significant changes in ankle kinematics were observed on the non-paretic side. Stance phase MG duration significantly increased on the paretic side ($p<0.001$) with greater TA-MG coactivation ($p=0.008$). Paretic limb TA duration decreased ($p=0.014$) and MG duration increased ($p<0.001$) in the swing phase with more TA-MG coactivation ($p=0.001$). When the AFO was removed, peak dorsiflexion in non-paretic stance increased ($p<0.001$) with greater total ROM compared to baseline. Time to peak dorsiflexion occurred earlier in stance ($p=0.02$) and peak dorsiflexion in swing was reduced on the paretic side ($p>0.05$). Stance and swing phase paretic TA duration increased ($p<0.001$, $p=0.027$, respectively). Non-paretic limb MG duration increased in stance and swing with greater TA-MG coactivation ($p<0.001$).

When walking with the FES device, gait speed slightly increased to 0.33 m/s. Time to peak dorsiflexion slightly reduced on the paretic side ($p=0.033$). Plantarflexion at toe-off decreased on the paretic side ($p=0.005$) and increased on the non-paretic side ($p<0.001$). Paretic limb peak dorsiflexion in swing did not change ($p>0.05$). MG duration increased during non-paretic stance ($p<0.001$). Greater TA-MG coactivation time was observed in stance and swing for the non-paretic limb ($p<0.001$). When the FES device was turned off, paretic limb ROM in stance reduced below baseline ($p=0.016$). Peak dorsiflexion in swing slightly decreased ($p>0.05$) with a longer TA duration on the paretic side ($p<0.001$). Paretic and non-paretic limb MG duration in stance and swing was greater compared to baseline ($p<0.001$). Greater TA-MG coactivation time was observed in paretic limb stance and swing, and non-paretic limb swing compared to baseline ($p<0.001$).

Table 4.3 – Summary of sagittal ankle kinematic outcome measures for each participant

		BASELINE		AFO		POST		BASELINE		FES		POST	
		P	NP	P	NP	P	NP	P	NP	P	NP	P	NP
P1	**Stance Phase**												
	Peak DF	-6.6	-11.6	-8.0	**-8.8**			-7.0	-10.5	**-15.3**	-10.8		
	DF time %GC	43.3	54.9	**47.5**	**47.4**			45.5	56.4	**35.6**	**49.5**		
	PF at IC	9.3	9.8	7.5	10.2			11.6	12.5	**1.2**	**7.9**		
	PF at TO	6.7	9.6	5.5	12.0			8.6	10.2	**2.0**	10.2		
	Swing Phase												
	Peak DF	6.6	6.5	4.9	7.3			8.5	6.8	**-2.2**	4.1		
P2	**Stance Phase**												
	Peak DF	-8.7	-6.9	-6.0	**-3.5**	-8.7	**-3.9**	-7.8	-6.2	**-10.2**	**-9.8**	-6.8	-4.9
	DF time %GC	39.0	57.7	38.2	58.5	33.7	62.2	39.6	58.9	**43.6**	57.8	36.3	57.5
	PF at IC	4.2	3.6	1.5	3.0	**7.9**	4.0	8.1	5.1	**5.7**	**8.1**	6.9	4.8
	PF at TO	7.7	5.4	4.6	5.7	9.3	5.1	5.6	2.1	**9.5**	**5.4**	5.3	**6.6**
	Swing Phase												
	Peak DF	3.1	0.9	**-1.1**	1.3	**7.1**	1.9	5.2	-0.7	5.7	0.5	4.6	-0.8
P3	**Stance Phase**												
	Peak DF	-10.7	-10.1	**-2.4**	-7.6	-8.3	**-13.0**	-7.0	-15.3	-7.2	**-8.8**	-6.9	-11.3
	DF time %GC	32.5	59.2	35.0	**38.8**	36.1	**43.1**	39.5	58.8	27.7	56.3	**18.8**	**39.8**
	PF at IC	20.8	11.0	**1.2**	**1.3**	**4.8**	**2.3**	13.8	6.5	8.3	12.4	**2.0**	3.8
	PF at TO	12.2	19.8	**1.5**	**16.0**	12.3	17.7	10.6	13.3	6.0	12.4	**18.6**	14.8
	Swing Phase												
	Peak DF	12.1	8.0	**-0.1**	2.6	**5.4**	3.7	9.5	5.9	5.7	7.9	**-4.0**	2.0
P4	**Stance Phase**												
	Peak DF	-6.2	-7.6	-6.2	-9.2	-6.9	**-12.7**	-7.1	-13.8	-8.3	-13.8	-6.5	-12.7
	DF time %GC	50.8	64.0	**61.6**	62.9	42.2	64.7	51.8	66.4	46.0	62.7	47.9	63.6
	PF at IC	16.7	9.1	**-1.1**	7.7	14.1	7.4	16.1	5.0	18.1	4.6	13.6	8.1
	PF at TO	2.2	8.5	**-5.3**	7.8	1.4	11.1	5.8	11.4	**1.5**	**15.8**	3.5	13.3
	Swing Phase												
	Peak DF	2.3	3.9	**-5.5**	3.8	1.4	5.9	1.1	4.0	1.5	3.4	2.6	6.7

Abbreviations: AFO, ankle-foot orthotics; DF, dorsiflexion; FES, functional electrical stimulation; GC, gait cycle; NP, non-paretic; P, paretic; PF, plantarflexion.

Note: Mean values are reported. Angles are measure in degrees. Bold values are significant at $p<0.01$ compared to the respective baseline.

Table 4.4: Summary of temporal TA and MG activation times for each participant

		BASELINE		AFO		POST		BASELINE		FES		POST	
		P	NP	P	NP	P	NP	P	NP	P	NP	P	NP
P1	**Stance Phase**												
	TA Duration	45.6	75.2	51.0	73.1			37.2	47.1	48.2	**61.7**		
	MG Duration	57.2	64.6	62.8	67.9			34.3	62.3	**61.1**	61.7		
	TA-MG Time	38.3	64.6	48.6	67.1			19.7	38.6	**47.0**	**57.0**		
	Swing Phase												
	TA Duration	32.9	22.6	30.9	25.4			24.8	12.7	**36.6**	**25.4**		
	MG Duration	29.3	18.8	32.5	22.1			5.5	5.9	**36.6**	**14.5**		
	TA-MG Time	27.7	16.9	30.7	22.1			5.0	4.3	**36.6**	**11.0**		
P2	**Stance Phase**												
	TA Duration	12.9	47.9	**1.8**	**75.6**	3.9	55.2	21.2	81.5	16.7	**69.3**	**39.9**	81.2
	MG Duration	5.0	43.6	14.9	36.6	12.6	**32.9**	40.7	70.0	**56.0**	64.7	**62.4**	73.0
	TA-MG Time	0.0	20.0	0.7	**32.7**	0.1	21.7	12.5	67.3	14.5	**56.8**	**38.9**	71.1
	Swing Phase												
	TA Duration	19.6	9.7	**4.7**	**15.8**	**5.9**	8.5	0.4	14.7	**12.2**	14.3	0.6	16.3
	MG Duration	2.7	0.0	**19.5**	**9.8**	**10.1**	**8.3**	23.3	15.2	25.3	**10.9**	**29.4**	16.3
	TA-MG Time	2.7	0.0	3.1	**9.2**	1.8	**4.9**	0.4	14.7	**12.2**	**8.8**	0.6	16.3
P3	**Stance Phase**												
	TA Duration	27.2	50.4	33.7	51.3	**46.5**	64.0	63.4	43.7	62.2	**23.7**	54.5	38.0
	MG Duration	49.1	51.2	56.8	57.4	**61.5**	47.8	57.2	34.3	62.5	37.1	57.7	**55.4**
	TA-MG Time	19.3	40.5	31.2	47.0	**45.7**	42.4	56.8	23.3	61.3	16.9	52.0	**34.6**
	Swing Phase												
	TA Duration	3.1	18.6	**18.8**	24.8	**21.8**	23.1	30.5	19.7	**36.7**	21.3	30.5	22.9
	MG Duration	16.3	15.4	**36.4**	**24.8**	**34.6**	22.1	30.4	4.6	**36.7**	**15.6**	32.7	**23.8**
	TA-MG Time	0.5	15.4	**18.8**	**24.8**	**20.9**	21.1	27.2	4.5	**36.7**	**15.3**	26.3	**22.7**
P4	**Stance Phase**												
	TA Duration	25.9	81.6	19.1	**65.6**	**48.2**	78.3	57.5	71.8	64.7	67.9	61.6	**57.3**
	MG Duration	7.6	26.1	**48.5**	26.3	4.0	**40.0**	17.0	23.6	**45.5**	**37.1**	**37.2**	**39.5**
	TA-MG Time	4.0	24.2	**15.3**	19.3	3.8	**36.3**	12.6	18.4	**45.2**	**28.8**	**33.6**	22.1
	Swing Phase												
	TA Duration	19.6	10.1	14.0	12.6	24.5	13.9	23.3	12.4	**29.9**	13.6	**30.1**	**15.7**
	MG Duration	3.5	3.7	**17.3**	4.6	1.3	**11.5**	2.0	0.6	**29.9**	**4.8**	**20.1**	**6.9**
	TA-MG Time	3.0	3.0	**8.9**	4.2	0.9	**11.2**	2.0	0.6	**29.9**	**4.8**	**19.9**	**6.9**

Table 4.4
Abbreviations: AFO, ankle-foot orthotics; FES, functional electrical stimulation; NP, non-paretic; P, paretic; POST, post-intervention.

Notes: Mean values are reported. Muscle activation times are expressed as a percentage of gait cycle time. Bold values are significant at $p<0.01$ compared to the respective baseline.

4.7 Discussion

This case series demonstrates the immediate changes to gait kinematics and muscle activation patterns with application of an AFO and FES device among four stroke survivors with dropped foot impairment. Although the participants displayed toe drag and difficulty swinging the paretic limb forward when walking without an AFO or FES device, underlying sensorimotor impairments contributing to their poor gait mechanics varied from dorsiflexor weakness to increased plantarflexor muscle spasticity and an equinus-varus ankle position. Despite functional differences in gait mechanics, three key characteristics were observed when walking without a dropped foot device; 1) reduced peak dorsiflexion during the paretic swing phase, 2) delayed time to peak dorsiflexion in stance on the non-paretic side, and 3) low magnitude and duration of plantarflexor activation during paretic stance. As well, P2 had a delayed paretic TA activation onset at initial contact and P3 displayed a low paretic TA duration in the swing phase. The present results highlight the need to study a larger sample of stroke survivors to determine the response to AFO and FES devices among individuals with different underlying mechanisms of impairment.

Effects of the AFOs

All participants displayed a greater peak ankle dorsiflexion during swing and at initial contact on the paretic side with their AFO compared to walking without a device. As well, the AFOs reduced ankle joint ROM during stance phase by restricting plantarflexion and limiting dorsiflexion to varying degrees. Different structural characteristics of the AFO designs will affect the ROM permitted at the ankle joint, as determined by trim-line position relative to the malleoli, material stiffness and joint type (Fatone, 2009). The posterior spring-leaf AFO used by P1 and P2 offered the most flexibility and produced only a few changes in ankle kinematics; improved paretic limb swing phase peak dorsiflexion and slightly reduced plantarflexion at initial contact. In contrast, P2 demonstrated reduced TA activation time during the stance and swing phases, while P1 displayed no change in muscle activation time. The solid AFO design used by P3 had little flexibility, and limited ankle plantarflexion throughout the GC and increased TA-MG coactivation time in the swing phase. Hesse and colleagues reported a lower mean TA amplitude from 60%-110% of the GC among stroke survivors walking with a non-articulating AFO (Hesse et al., 1999). It is possible that P3 had a longer TA duration with lower amplitude due to the restricted ROM. The hinged AFO design used by P4 offered free dorsiflexion to maintain forward rotation over the foot during stance, which may have facilitated increased paretic MG duration and TA-MG coactivation time in the stance phase. In contrast, a previous study reported no change in MG amplitude during stance with a non-articulating AFO (Hesse et al., 1999). Our findings show that application of AFO devices post stroke may not impede muscle activation patterns during gait in the short term; however specific design features may be better suited for different underlying mechanism of dropped foot.

Effects of the FES device

Immediate joint kinematic changes observed with application of the FES device occurred with P1 and P2, including improved ankle position at initial contact and increased peak dorsiflexion in stance on the paretic side. P3 had the lowest stimulation level and revealed a trend towards improved ankle dorsiflexion during swing and at initial contact. Most participants had a reduced plantarflexion at toe-off, possibly because the stimulation onset started to ramp up at heel rise and limited their ability to push-off. These findings support a previous report showing a shift towards greater ankle dorsiflexion throughout the GC with FES among stroke survivors (Kesar et al., 2010). However, P4 demonstrated no change in sagittal ankle kinematics with the FES device, despite a higher stimulation amplitude. This may reflect increased TA and MG durations in the swing phase, which were maintained when the stimulation was turned off. Increased coactivation time during paretic stance with the FES observed in P1 and P4 may reflect improved joint stiffness and postural stability (Lamontagne et al., 2000). A carry-over effect was observed with P3 in the post walking trials, specifically improved peak dorsiflexion during swing leading to less plantarflexion at initial contact and greater plantarflexion at toe-off. Increased muscle activation amplitudes may have contributed to the carry-over effect, as the duration of muscle activity returned to baseline levels (Kottink et al., 2008). These findings suggest that FES may enhance postural stability during stance and improve voluntary muscle activation immediately after training.

Changes on the non-paretic side

The non-paretic limb demonstrated a few adaptations in response to the AFO and FES device. Specifically, stance phase peak dorsiflexion was reduced when the PLS AFO was used by P1 and P2. The time to peak dorsiflexion in stance was earlier for P1 and P3 when walking with their AFO compared to baseline, possibly reflecting improved stance/swing time symmetry. P2 displayed a greater ROM, reduced TA duration and less TA-MG coactivation in the stance phase with the FES device. In contrast, P1 and P4 increased coactivation time during the non-paretic stance phase, possibly as mechanism to enhance joint stability as the paretic limb advances forward (Lamontagne et al., 2000). Previous studies have reported excessive coactivation on the non-paretic side as a compensatory mechanism when postural stability is challenged (Den Otter et al., 2007; Lamontagne et al., 2000). Up regulating coactivation levels may be the initial response to increased postural stability demands when walking with a dropped foot device.

Limitations

Although this case series provides important information regarding how AFO and FES devices change gait biomechanics, there are several limitations to consider. Most participants regularly used an AFO for walking within the community; therefore they have adapted to and feel confident when wearing the device. P2-P4 were using the AFO for 2-5 hours daily for ambulating within their community to attend appointments and for general physical activity. The FES device appeared to have a lower impact on gait kinematics compared to the AFO possibly due to the novelty of the device. If participants had more time to practice with the FES device, a training effect may have been observed. A common issue with FES devices is rapid muscle fatigue with extended periods of use (Popovic et al., 2001). It is possible that participants may have experience muscle fatigue by the post intervention trials; however rest breaks were provided between each condition. Participants were instructed that the stimulation would turn-off when they put weight on the affected side; therefore encouraging them to weight-bear on the paretic side between trials. While our assessment focused on the effect at the ankle joint, it is possible that changes in joint excursions and muscle activation patterns occurred at the knee and hip. Few studies to date have investigated the impact of AFO and FES devices at the knee joint and the findings remain inconclusive (Buckon et al., 2001; Chisholm & Perry, 2012; Fatone et al., 2009; Kesar et al., 2009). This protocol was easy to administer and provided valuable information that may guide treatments plans in regards to a dropped foot device; however a full lower limb analysis may be preferred if a patient displays significant proximal deviations.

Subjective feedback

Previous research has revealed positive and negative perceptions by stroke survivors using an AFO (Tyson & Thornton, 2001). In our study, P1 preferred using the FES device compared to an AFO because it's ease and comfortable to fit with shoes. While P3 and P4 preferred their AFO due to the increased support provided when loading weight onto their affected side and ability to "lift the ankle up" to step forward. P2 was indifferent; the AFO was difficult to don/doff with shoes and the FES device was complex with "too many settings". Stroke survivors have previously indicated greater self-confidence and improved ability to perform functional tasks when wearing an AFO compared to no device (de Wit et al., 2004), along with negative aspects such as unpleasantly heavy and reservations with its appearance (Hesse et al., 1996; Tyson & Thornton, 2001). The patient's cost/benefit analysis will likely influence whether the device is used for daily activities. Further study is required to determine challenges in performing complex tasks in their environment with each device.

Conclusion

In conclusion, this case series describes immediate changes in gait biomechanics with application of AFO and FES devices to correct dropped foot impairment post stroke. We observed improved ankle dorsiflexion and increased MG activation during swing with the AFOs, while the response to FES was less consistently across participants. Our findings highlight different underlying impairments as contributing factors to dropped foot, and varying responses bilaterally to walking with AFO and FES device. The implications of these findings warrant further investigation to determine long-term impact on gait function and develop prescription guidelines with specific clinical characteristics.

CHAPTER 5

General Discussion and Future Directions

5.1 General Discussion

This dissertation has presented evidence of the relationship between impaired sensorimotor function and dropped foot gait deviations among stroke survivors. Individuals with greater deviations in ankle joint motion during gait were likely to demonstrate dorsiflexor muscle weakness, and reduced passive and active ankle ROM at rest. In addition, clinical measures of lower limb sensorimotor control and ankle plantarflexor spasticity indicated greater impairment among stroke survivors with dropped foot compared to those within the normal range of healthy adults. Our gait assessment revealed delayed and inadequate TA activation time along with reduced TA-MG coactivation time as possible mechanisms of dropped foot gait deviations. Altered timing of lower limb muscle activity may indicate reduced capacity to generate appropriate levels of voluntary force and compensatory neuromuscular strategies to facilitate gait function. As well, the results of this work have provided insight on the immediate effects of AFO and FES devices on sagittal ankle kinematics and muscle activation patterns during gait performance. Although different AFO designs were utilized, we observed improved peak ankle dorsiflexion in the swing phase and limited ankle ROM in stance. Our participant who had a previous training session with the FES device demonstrated positive effects on gait function, such as improved peak dorsiflexion during swing and increased MG activation time during stance. The FES device had little impact on gait function among the other participants; suggesting a more extensive training program may be required to gain immediate beneficial effects. Overall, these findings highlighted individual differences in response to interventions aimed at improving dropped foot gait deviations. The importance of exploring contributions of different sensorimotor control impairment types to dropped foot deviations aligns with an evidence-based approach to rehabilitation to understand the impact of variations in gait dysfunction and plan interventions to achieve specific outcomes.

The significant relationships between objective measures of ankle-foot impairment and dropped foot gait deviations add to previous evidence emphasizing the important role ankle function in gait performance (Bowden et al., 2006; Hsu et al., 2003; Lin et al., 2006; Parvataneni, Olney, & Brouwer, 2007; Perry, 1992). As opposed to non-parametric indices typically used in a patient evaluation, quantitative measures of dorsiflexor muscle strength and ankle range of motion were significantly associated with sagittal kinematic deviations indicative of dropped foot. These measures would be quick and easy to administer in a clinical setting, while providing valuable information to the clinician. Previous research has observed relationships between standardized clinical tests and temporal measures of gait performance (i.e. velocity and symmetry) (Hsu et al., 2003; Lamontagne et al., 2001; Lin et al., 2006). While most stroke survivors display a slow gait velocity and temporal asymmetry between limbs (Patterson et al., 2008), these measures are not specific enough for a diagnosis of dropped foot gait. Based on our findings, poor ankle function measured at rest may indicate the need for a gait analysis with specific measures of dropped foot. If a clinician suspects an intervention may be required to improve gait function, a readily available tool to provide insight on the mechanisms of dysfunction may help determine if an intervention is required. By improving patient evaluation to include specific and accurate measures that are reliable to guide the clinician, the process of selecting the appropriate intervention may reduce negative aspects of the trial and error approach. Also, the use of gait analysis in planning an intervention may result in a more favourable ratio of positive/negative outcomes.

Mechanisms leading to reduced ankle dorsiflexion during the swing phase, excessive ankle plantarflexion at initial contact and poor stability during the stance phase may result from different underlying motor control impairments. Our results provided evidence of multiple factors contributing dropped foot gait deviations, specifically delayed and reduced TA activation time, and decreased TA-MG coactivation time throughout the gait cycle. Delayed TA onset may be explained by an ankle plantarflexed position at initial contact with poor coactivation to control the loading response. Although no difference was found between stroke survivors with and without dropped foot, MG onset time usually occurred early in the stance phase. A previous study observed longer MG activation times during the first double support phase compared to healthy adults, possibly indicating an adaptive response to forward progression (Den Otter et al., 2007). Individuals with dropped foot deviations demonstrated reduced TA activation during swing, thus limiting the potential to generate higher levels of force. Reduced TA-MG coactivation time on the paretic side is consistent with previous reports and may lead to poor stability during stance because of reduced joint stiffness (Lamontagne et al., 2000). It is important to identify features of ankle kinematics and muscle activation patterns that correlate with successful gait performance. Our findings suggest TA onset time in stance, TA duration in swing and TA-MG coactivation time in stance may represent key features of dropped foot impairment. Thresholds for these features can be set to identify dropped foot and possibly categorize based on deviations in different phases of the gait cycle. After this is accomplished, guidelines for treatment could be developed by using results from gait analysis with different interventions.

Our hypothesis that dropped foot impairment may result from different underlying mechanisms (i.e. weakness, spasticity and flaccidity) was confirmed with findings from the series of case studies. Two participants (P1 and P2) primarily demonstrated moderate ankle dorsiflexor weakness at rest, resulting in reduced peak dorsiflexion during swing and limited ROM during stance. Application of a posterior spring-leaf (PLS) AFO with relatively little restrictions slightly increased ankle dorsiflexion throughout the gait cycle. Stroke survivors with mild to moderate muscle weakness may benefit from training with FES, as our participants improved ankle dorsiflexion during swing and at initial contact via increased TA activation time with repeated bouts of walking. Our participant (P4) with a flaccid ankle due to severe TA and MG muscle weakness displayed a significant ankle plantarflexion in the swing phase and at initial contact. While the hinged AFO allows relative dorsiflexion over the foot in stance, a compensatory strategy was utilized to limited forward progression likely due to difficulty controlling the movement. This design may not be appropriate for individuals who have difficulty voluntarily activating the dorsiflexor and plantarflexor muscles against gravity (Condie et al., 2004). In contrast, FES enhanced the duration of lower limb muscle activation in the flaccid ankle, however not enough to generate forces to improve joint motion. Another proposed mechanism of dropped foot is excessive ankle plantarflexor spasticity, which limits ankle dorsiflexion in swing due to a stretch reflex response at toe-off (Knutsson & Richards, 1979; Lamontagne et al., 2002). Application of a rigid non-articulating AFO successfully limited ankle plantarflexion and controlled medial-lateral instability throughout the gait cycle, while FES did not immediately change joint motion. The observed carry-over effect on ankle joint motion requires further investigation to confirm whether practice enhances muscle fiber recruitment during gait. Previous research has reported a greater magnitude of TA activation at rest after FES training (Kottink et al., 2008). Given the complexity of dropped foot impairment, developing general categories could focus a gait analysis on outcome measures related to the deficit and target the intervention at improving those outcomes. Information from specific outcome measures may be particularly useful for the clinician when a relationship is determined between the interventions effect at the neuromuscular level.

Our instrumented and quantitative assessment of dropped foot impairment provided relevant clinical information for identifying underlying neuromuscular mechanisms contributing to significant kinematic gait deviations. In a clinical setting, physiotherapists (or other rehabilitation professionals) may utilize this assessment with patients to inform diagnosis and guide a treatment plan that facilitates sensorimotor recovery and improves gait biomechanics. The most commonly used method of gait assessment in a clinical setting is visual observation because as it is easy to administer and inexpensive (Toro, Nester, & Farren, 2003). However, it does not provide the appropriate level of detail to determine the potential cause and make a decision on which intervention to recommend for dropped foot. Our standard gait assessment without intervention was approximately 30-40 minutes in length depending on the patient's functional mobility. This time included setup, patient instruction, administration of clinical tests (i.e. ROM, strength and spasticity) and multiple walking trials over the pressure mat. Our participants were able to walk at least 5m independently without physical assistance and completed assessment with ease. Most participants felt the assessment was not invasive and/ or difficult to walk with the equipment. We recruited participants prior to discharge from inpatient rehabilitation, as this is a critical point for the rehabilitation team to determine their recommendations for outpatient therapy based on standardized outcome measures. Individuals who require an intervention for dropped foot may be provided a temporary AFO during inpatient rehabilitation to use as an interim device before a custom prescription is determined at a later time or sensorimotor recovery is achieved to the level where a device is no longer needed. Implementing a gait assessment with specific quantitative outcome measures for dropped foot prior to discharge may advance the prescription process, facilitate walking safety at home and maximize the benefit to the patient as they transition to outpatient therapy.

Limitations

This assessment does have a few limitations in regards to clinical feasibility and comprehensive analysis of gait dysfunction. The cost of technology used in this assessment is an obvious feature that limits its uptake in clinical practice. For gait laboratories associated with teaching hospitals or specialty clinics providing AFO/FES interventions, the cost may not be a deterrent if the analysis is found to improve patient care and enhance gait outcomes. As well, this information is not particularly useful until a relationship is established between the specific gait dysfunction and effect of an intervention. Further work is required to determine specific indices of dropped foot impairment during gait that may be improved by AFO/FES intervention with a larger sample size. As new information is gained, knowledge translation is required to update gait analysis training for clinicians. Time to complete post data collection analysis is a limiting factor, however advanced computer programming to create a streamlined process may assist implementation. The focus of our gait assessment was on measuring joint motion and muscle activity at the ankle as it relates to dropped foot impairment. However, gait performance can be affected by numerous stroke related impairments included more proximal sensorimotor dysfunction, poor balance, reduced sensation, perceptual and cognitive deficits (Bowen et al., 2001; Geurts, de Haart, van Nes, & Duysens, 2005; Lamontagne et al., 2005; Niam et al., 1999; Olney & Richards, 1996; Parvataneni et al., 2007). Therefore, an assessment may need to be modified to understand all factors contributing to the individual's gait deviations and to properly develop an intervention plan.

5.2 Future Directions

The overall goal of research in this area is to 1) understand mechanisms of impaired neuromuscular control during gait performance, and 2) develop effective interventions to facilitate sensorimotor recovery and improve functional mobility. This work has provided recommendations for a clinical gait assessment to identify gait deviations for each mechanism of dropped foot impairment (i.e. weakness, spasticity and flaccidity), and determined the effects of AFO and FES interventions on gait function among stroke survivors. Many questions arise that require further investigation to develop a clinical assessment tool and evidence-based practice guidelines for clinicians, as well as advance current technology to target specific characteristics of dropped foot. Considering the complexity and heterogeneity of stroke-related impairments, it is important that future research incorporate a multi-disciplinary approach to develop a comprehensive clinical assessment and intervention strategies to improve gait dysfunction.

To further knowledge about the impact of dropped foot impairment on gait function, we need to identify specific gait characteristics that can be successfully improved with AFO or FES intervention. Individuals with dropped foot may demonstrate various kinematic and muscle activation patterns due to different mechanisms of impairment and utilization of compensatory strategies. Increased accuracy and detailed gait analysis will yield better information which can guide diagnosis and treatment of dropped foot impairment. There has been considerable advancement of objective and instrumented gait analysis techniques in the research field. As these techniques become more clinically feasible, interventions can be applied to determine if specific gait characteristics improve with therapy. A clinical assessment should include quantitative and objective measures of ankle-foot function to determine whether an advanced gait analysis is required. In addition, other health domains need to be considered, such as upper limb function and cognition that may affect an interventions outcome. Future research needs to determine the influence of AFO and FES devices on more complex activities and functional mobility within the community.

While the immediate effect of AFO and FES technology has demonstrated a few positive outcomes on gait function, the long term impact requires a higher level of evidence from well designed studies with a representative sample size and appropriate outcome measures. As well future work may include a more comprehensive comparison of AFO and FES devices at producing a carry-over effect on muscle activation patterns. Improved voluntary muscle activation is a potential advantage of using an FES system, further work needs to determine the duration of the carry-over effect and the positive outcomes on gait function. Conversely, whether application of an AFO device delays sensorimotor recovery and promotes learned disuse remains unknown. A dose-response relationship has not been established between the duration and frequency of use, and improved gait function. New developments in AFO and FES designs aimed to improve specific features of dropped foot impairment will contribute to guidelines for a prescription.

In conclusion, future research that can be generalized to other neuromuscular conditions with impaired gait function due to dropped foot will help advance diagnostic and intervention strategies. Identifying common mechanisms of dropped foot across different populations may further knowledge of the relationship between an interventions effect at the neuromuscular level. Implementing an instrumented and objective gait analysis that both combines laboratory and standardized clinical measures will lead to the development of more effective assessment and intervention approaches to address the problem of dropped foot impairment.

Appendix 1

Clinical indications for AFO prescription from ISPO consensus conference (Condie et al., 2004).

Type	Clinical Indication
Non-articulated AFO	• Poor balance, instability in stance • Inability to transfer weight onto affected leg in stance • Moderate to severe foot abnormality; equinus, valgus or varus, or a combination • Moderate to severe hypertonicity • As above, but with mild recurvatum or instability of the knee • To improve walking speed and cadence
Articulated AFO	• Dorsiflexor weakness only • Where passive or active range of dorsiflexion is present • Where dorsiflexion is needed for sit-to-stand or stair climbing • To control knee flexion instability only, articulated AFO with dorsiflexion stop • To control recurvatum only, articulated AFO with plantarflexion stop • To improve walking speed and cadence
Posterior Spring Leaf	• Isolated dorsiflexors weakness • No significant problem with tone • No significant medio-lateral instability • No need for orthotic influence on the knee or hip

Appendix 2

Detailed information on equipment used for data collection and analysis.

Equipment	System	Model #	Company
Load cell	Force Transducer	SSM-AJ-500N	Interface Inc Scottsdale, AZ
Pressure mat	GaitRite Electronic Walkway	Platinum Software 4.0	CIR Systems Inc Peekskill, NY
Electrodes	Medi-Trace® Mini	MT130	King Medical Ontario, Canada
Data acquisition system	Noraxon Telemyo System	900	Noraxon Scottsdale, AZ
Functional Electrical Stimulator	Odstock Dropped Foot Stimulator	ODFS II	Odstock Medical Limited, Salisbury, UK

Appendix 3
Kinematic and EMG time series data comparing the effect of the AFO/FES and post-intervention conditions to the baseline condition for each participant.

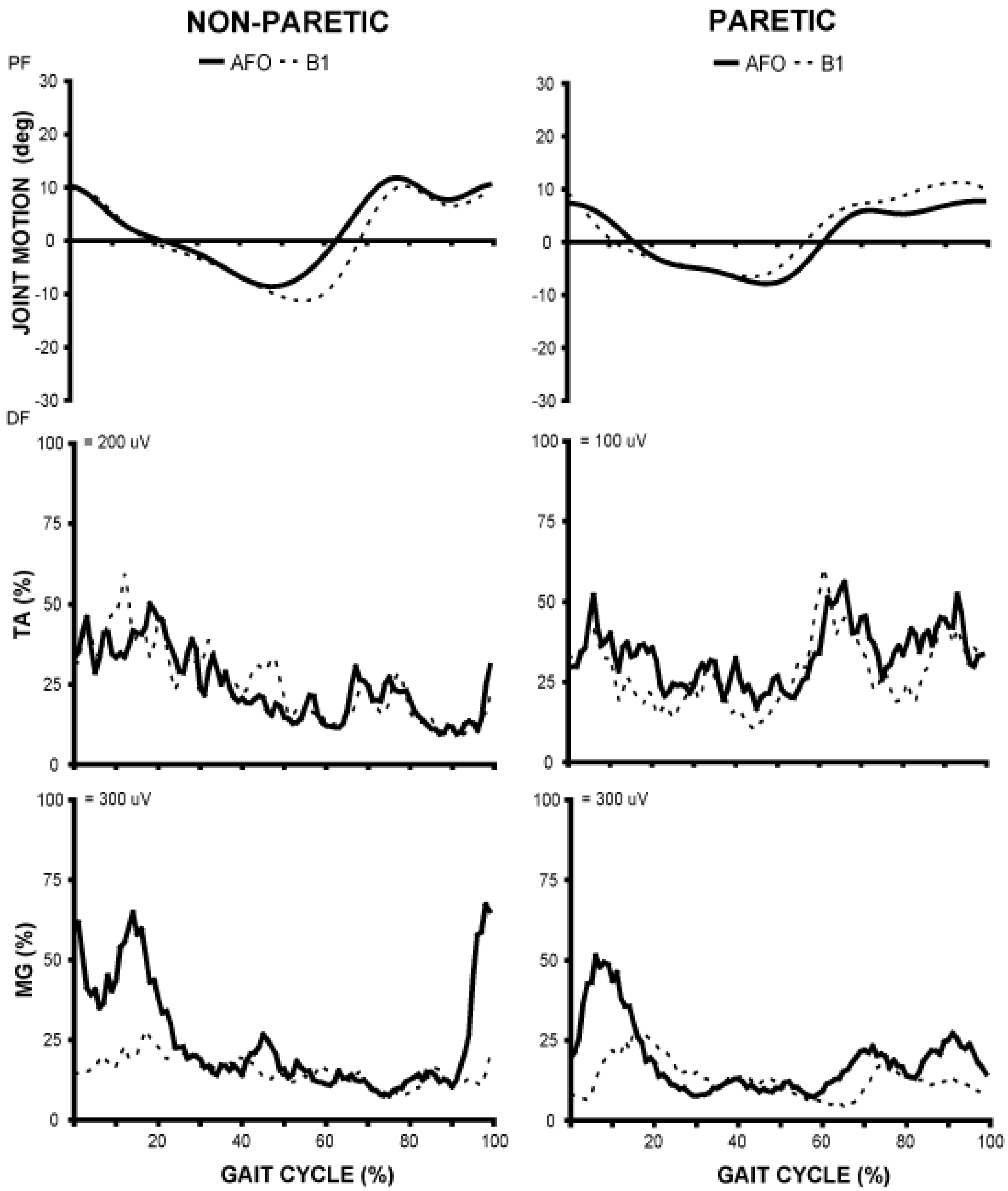


Figure 4.1a: Participant 1 comparing the effect of the AFO device to baseline (B1). Joint motion data are positive for plantarflexion and negative for dorsiflexion.

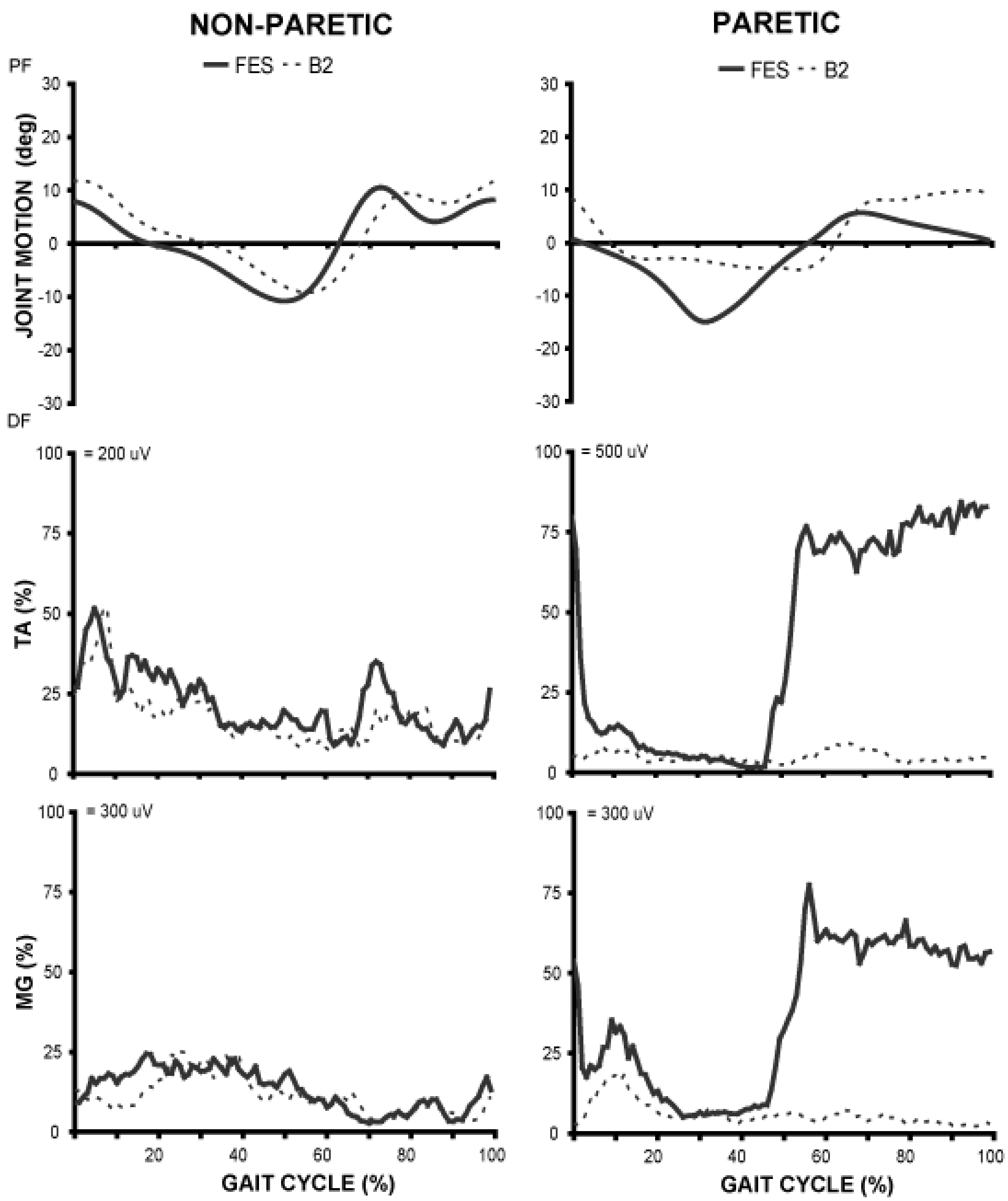


Figure 4.1b: Participant 1 comparing the effect of the FES device to baseline (B2). Joint motion data are positive for plantarflexion and negative for dorsiflexion.

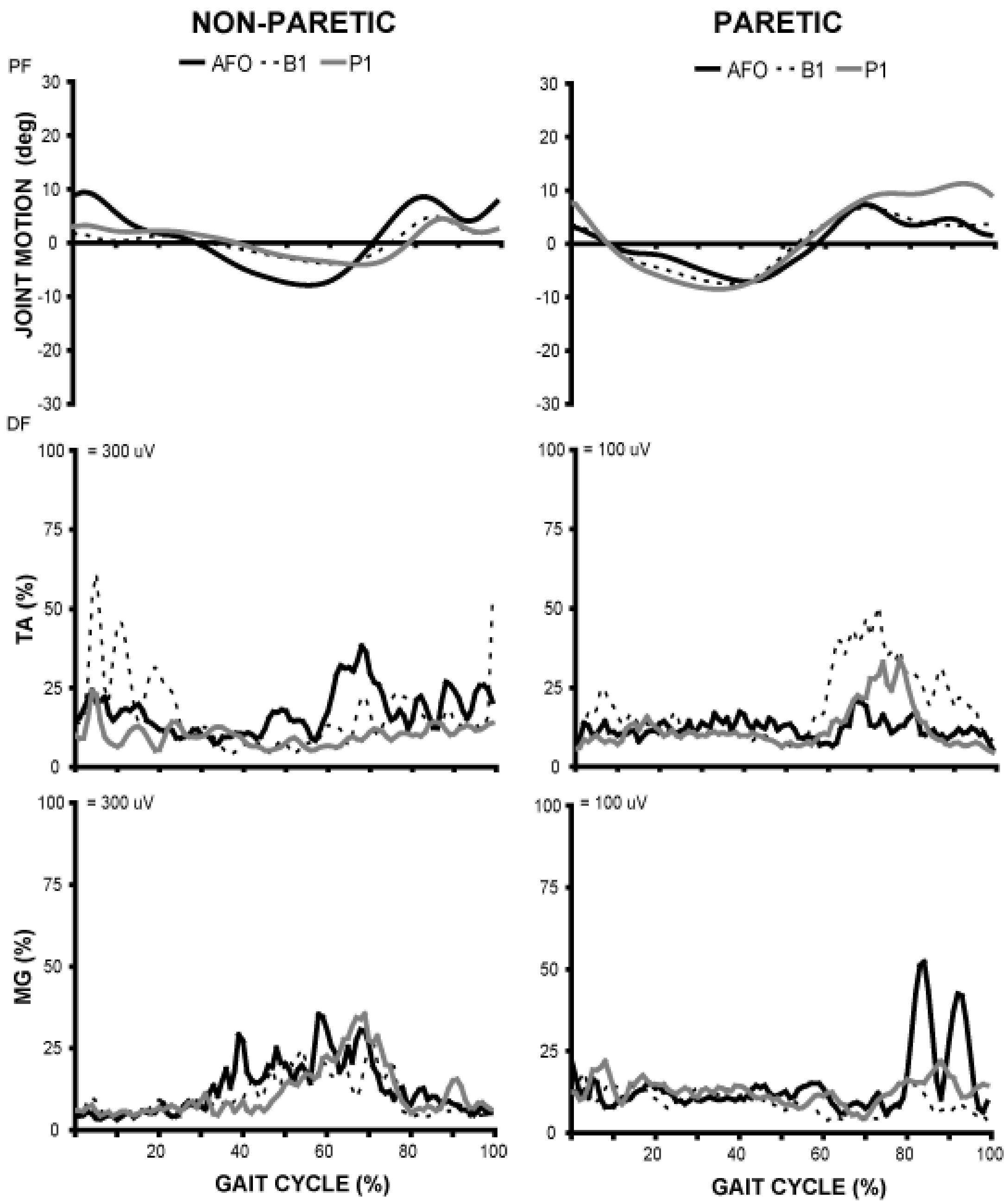


Figure 4.2a: Participant 2 comparing the effect of the AFO device and post-intervention (P1) to baseline (B1). Joint motion data are positive for plantarflexion and negative for dorsiflexion.

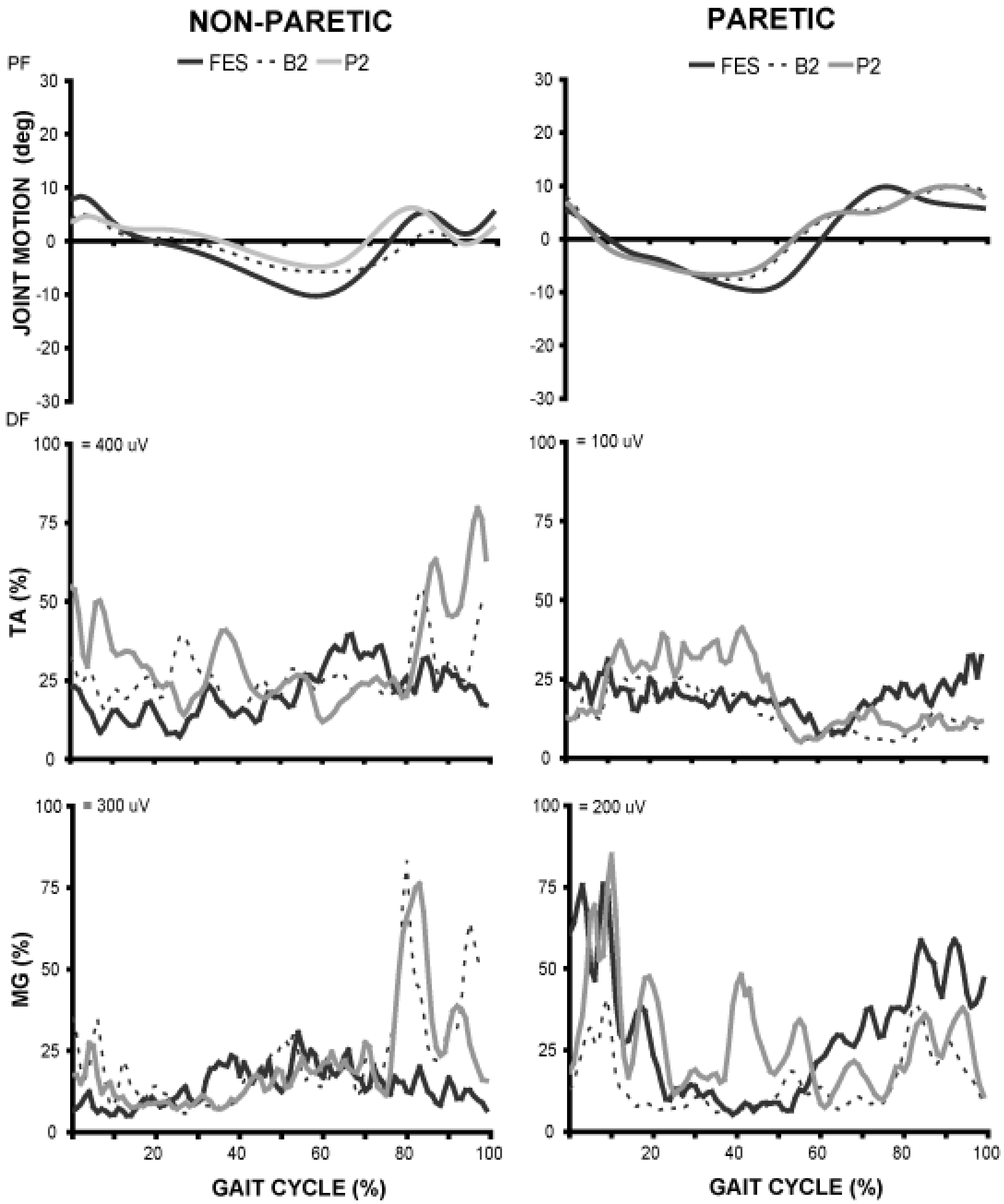


Figure 4.2b: Participant 2 comparing the effect of the FES device and post-intervention (P2) to baseline (B2). Joint motion data are positive for plantarflexion and negative for dorsiflexion.

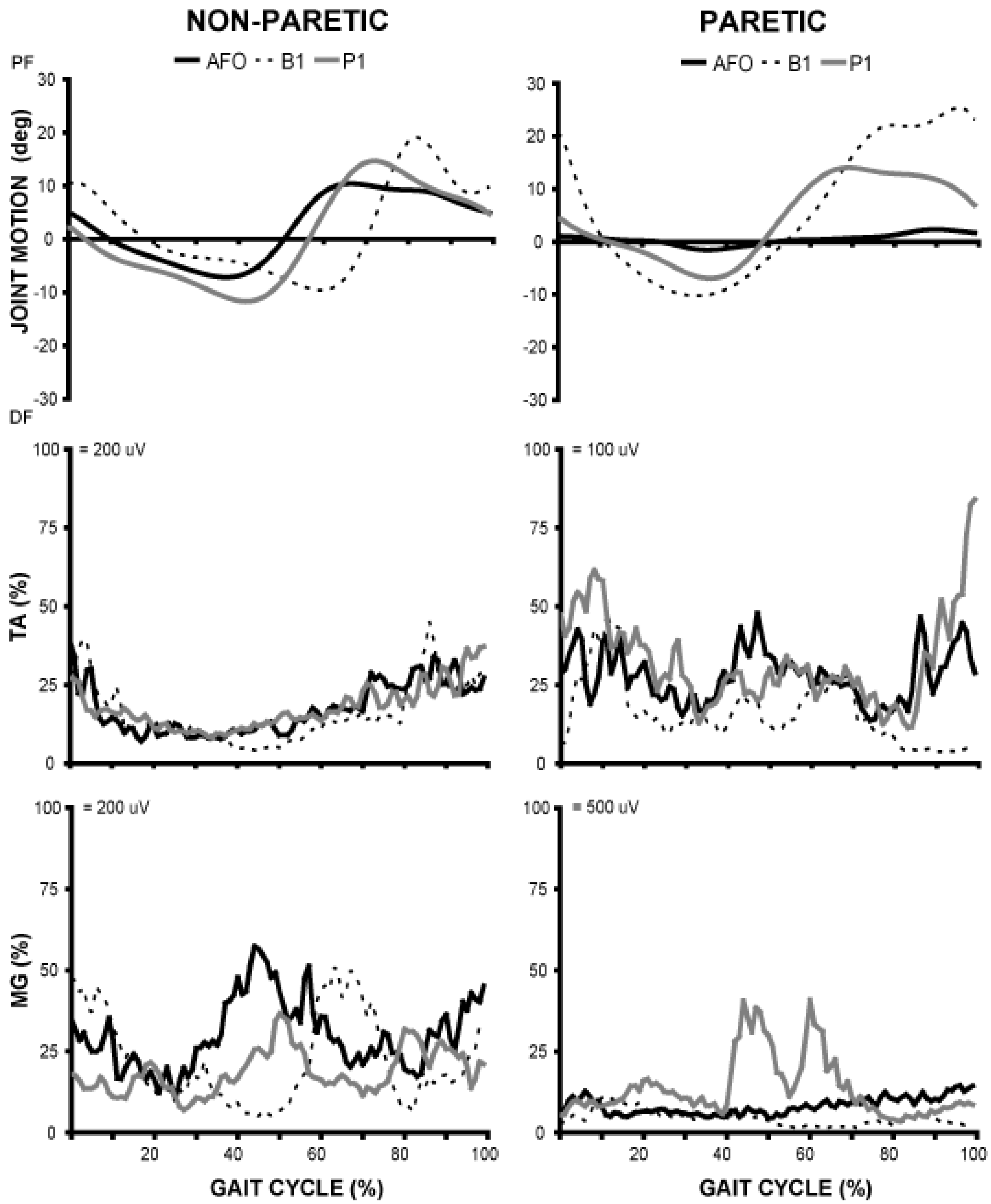


Figure 4.3a: Participant 3 comparing the effect of the AFO device and post-intervention (P1) to baseline (B1). Joint motion data are positive for plantarflexion and negative for dorsiflexion.

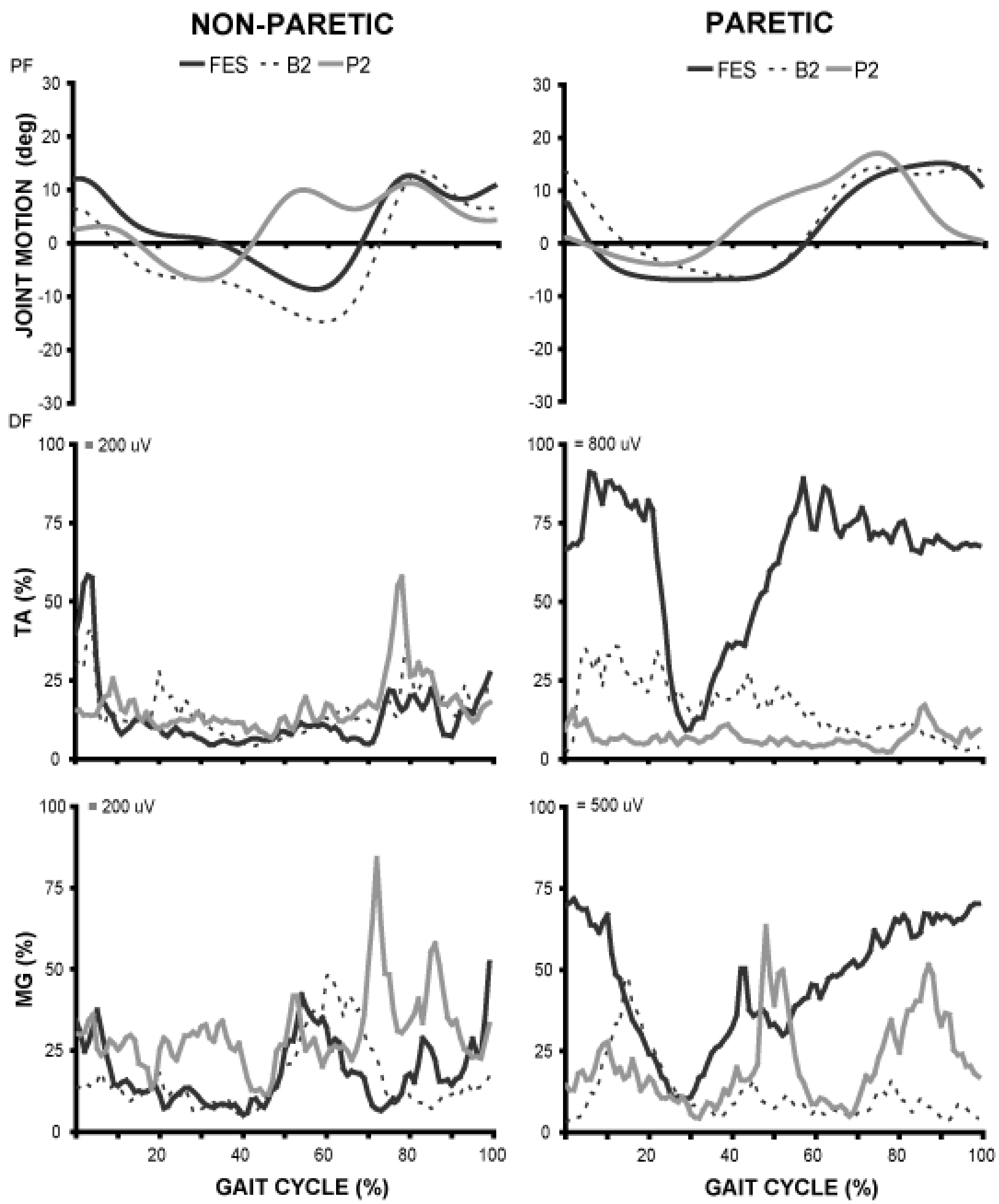


Figure 4.3b: Participant 3 comparing the effect of the FES device and post-intervention (P2) to baseline (B2). Joint motion data are positive for plantarflexion and negative for dorsiflexion.

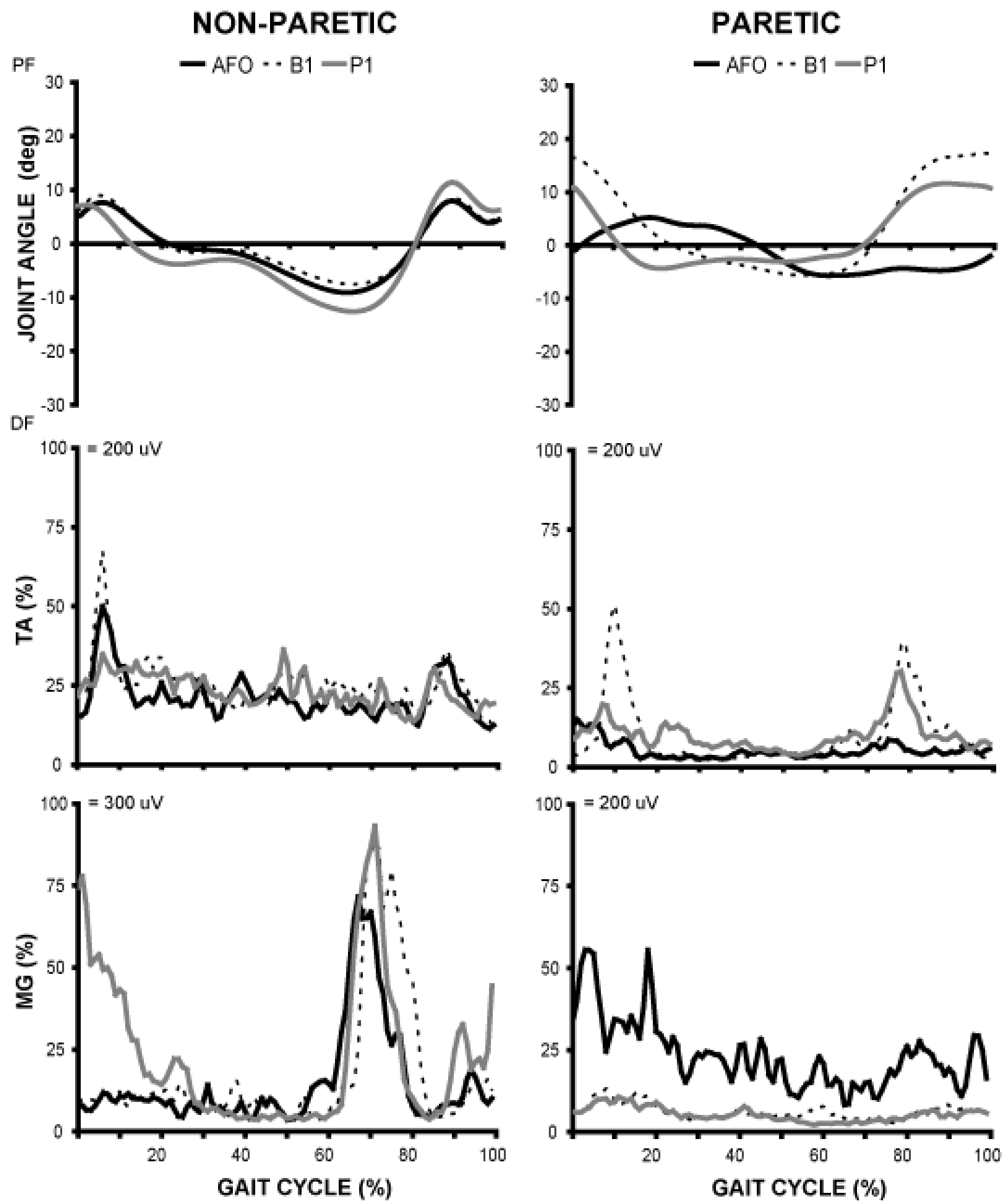


Figure 4.4a: Participant 4 comparing the effect of the AFO device and post-intervention (P1) to baseline (B1). Joint motion data are positive for plantarflexion and negative for dorsiflexion.

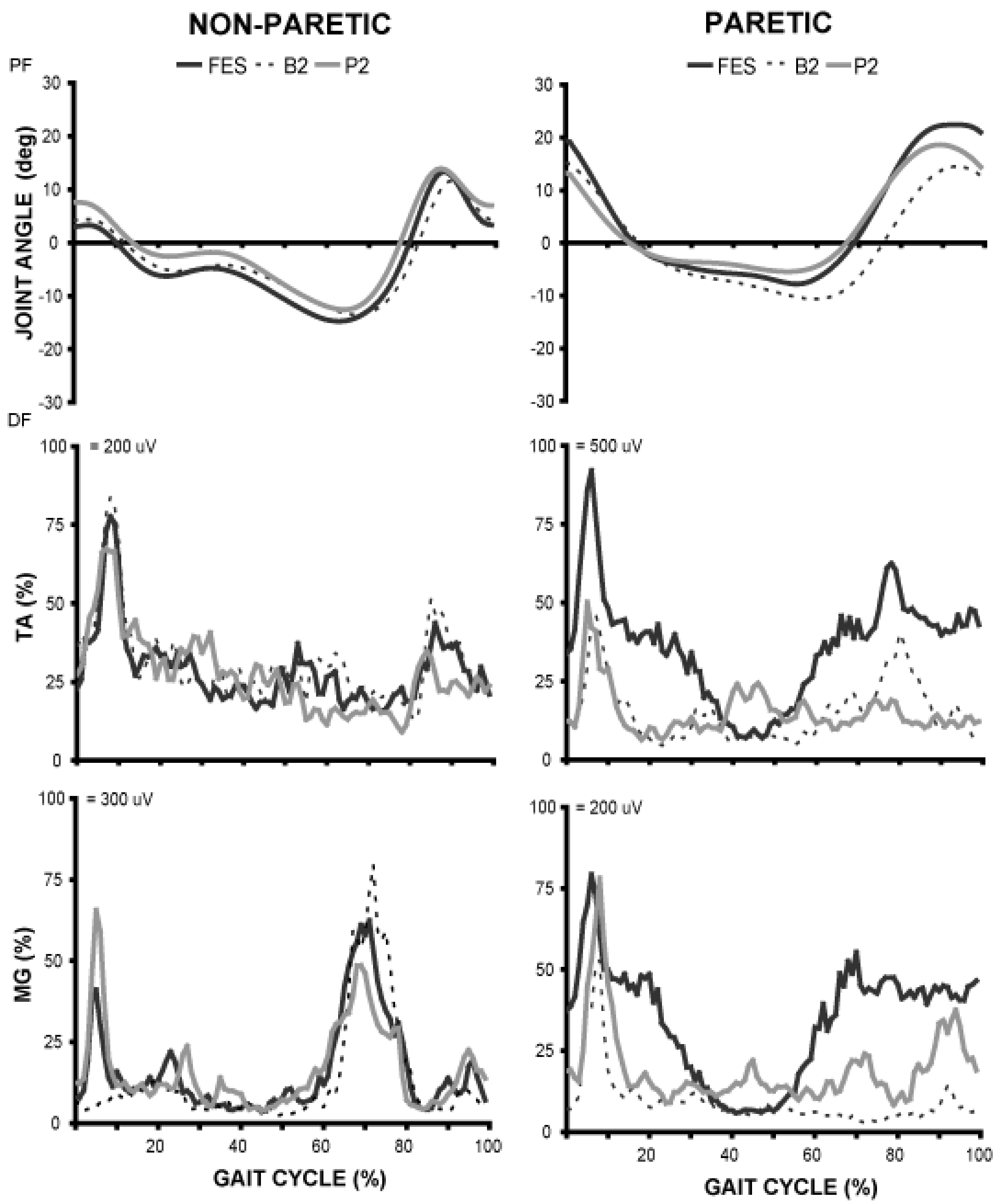


Figure 4.4b: Participant 4 comparing the effect of the FES device and post-intervention (P2) to baseline (B2). Joint motion data are positive for plantarflexion and negative for dorsiflexion.

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