EVALUATION AND TREATMENT OF PATIENTS WITH STROKE: AN EMG AND H-REFLEX STUDY

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DEDICATION

I dedicate this Dissertation to my wife Monica and to our lovely daughters Paula, Daniela and Francisca, who have patiently supported me through these years of graduate study.

...And to our loved and always remembered families in the faraway land of Chile.

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ABSTRACT

Evaluation and Treatment of Patients with Stroke: An EMG and H-Reflex Study

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This study consisted of four experiments that investigated the soleus H-reflex and soleus/tibialis anterior muscle pair activation in healthy subjects and patients with cerebrovascular accident (CVA) or stroke. In Experiment 1, the purpose was to establish test-retest reliability of the soleus H-reflex for both groups during gait-simulated postures between two days. Experiment 2 investigated soleus H-reflex amplitudes and soleus/tibialis anterior EMG activation during gait-simulated postures in healthy adults. Experiment 3 compared the two groups average soleus H-reflex amplitudes and related soleus/tibialis anterior EMG recorded in gait-simulated postures. Experiment 4 assessed (using soleus H-reflex and tibialis anterior EMG) the effects of an exercise program intended to improve the patients' motor control at the ankle joint determined by improvements in gait speed. Ten healthy subjects and 9 patients participated in Experiment 1; Experiment 2 included 21 healthy subjects; Experiment 3 included 21 healthy subjects and 9 patients; and 9 patients participated in Experiment 4. Soleus H-reflex amplitudes and soleus /tibialis anterior EMG activity were recorded for both groups in each of five gait-simulating postures (N: freestanding, PO: push-off, HC: heel-contact, SW: swing and MS: mid-stance) for experiments 1-3. Patients participated in a

4-week exercise (3 sessions/week) that consisted of repetitive ankle dorsiflexion of the paretic limb that simulated the swing phase (open chain) of the gait cycle. Pre- and post-training gait speed measures for self-paced and fast-paced gait speed, soleus H-reflex, and soleus/tibialis anterior EMG were recorded. Results of Experiment 1 showed test-retest of the soleus H-reflex was high for each posture in both groups. Experiment 2 showed a relationship between normal gait and gait-simulated postures for soleus H-reflex/EMG activation. The results of Experiment 3 found the patient group exhibited a lack of inhibition of the soleus H-reflex amplitude in postures HC and SW compared to the healthy subjects. In Experiment 4 results, the patient group had a significant improvement for gait speed in the fast-paced gait condition only. Individual comparisons of pre- and post-training results showed that the majority of patients improved in their control of soleus inhibition and tibialis anterior activation.

Key Words: H-REFLEX, STROKE, CVA, SIMULATED GAIT, REHABILITATION

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CHAPTER I

INTRODUCTION

Although the incidence of cerebrovascular accident (CVA) or stroke appears to be decreasing, the cost of rehabilitation is likely to increase because of improved survival rates and the increased number in the elderly population (Desai, Zhang, & Hennesy, 1999; Dobkin, 1995; Duncan, 1994). Rehabilitation efforts to help sufferers of upper motor neurone (UMN) syndrome, especially CVA, are multifaceted. The treatment includes management from physical and occupational therapy, orthotics, drug treatments, intrathecal techniques and surgery (Barnes, 2001) among other modalities. Since UMN syndromes also involve dysfunction in other non-motor domains such as cognition, perception and memory, its presentation is different for each patient. This requires individualized treatment programs to address individual rehabilitation needs. Since loss of walking function is a major problem after CVA (Friedman, 1990; Wade & Hewer, 1987), recovery of walking is an important goal for most patients (Bohannon, Horton, & Wikholm, 1991; Mumma, 1986).

Physical Treatment Interventions Directed to Improve Gait Function in Patients with Hemiplegia

The residual muscle weakness and loss of motor control seen in the affected limb

of stroke sufferers (e.g., drop-foot) has been a focus for physical therapists and other rehabilitation specialists. Traditional rehabilitative strategies designed to improve gait performance have proposed early and intensive gait-specific physical therapy (Richards, Malouin, Wood-Dauphinee, Williams, Bouchard & Brunet, 1993); strength and coordination development of the involved limb, especially during the pre-swing phase (De Quervain, Simon, Leurgans, Pease, & McAllister, 1996); and specific strengthening of the ankle extensors to improve performance and gait speed (Nadeau, Arsenault, Gravel, & Bourbonnais, 1999). Others have also recommended functional electrical stimulation (Bogataj, Gros, Malezic, Kelih, Kljajic, & Acimovic, 1989) and electromyographic (EMG) biofeedback (Cozean, Pease, & Hubbell, 1988). The efficacy of the latter, however, is still controversial (Glanz, Klawansky, Stason, Berkey, Shah, Phan et al., 1995; Intiso, Santilli, Grasso, Rossi, & Caruso, 1994; Moreland, Thomson, & Fuoco, 1998; Schleenbaker & Manious, 1993). Other therapeutic techniques emphasize neuromuscular reeducation, such as the approaches set forth by Bobath (1990), Brunnstrom (1970), and proprioceptive neuromuscular facilitation (Knott & Voss, 1968).

Another, more recent rehabilitation strategy to improve gait performance in hemiplegia is treadmill walking with partial body-weight support. The method provides an integrative, task-oriented paradigm that, by decreasing the burden of the patient's weight bearing on the affected limb, allows for better performance of repetitive stepping and re-creation of gait pattern activity. It has been shown to improve overall gait ability as assessed by the Functional Ambulation Category assessment (Hesse, Bertelt,

Schaffrin, Malezic, & Mauritz, 1994), gait velocity (Hesse, Bertelt, Jhanke, Schaffrin, Baake, Malezic et al., 1995; Hesse, Werner, Uhlenbrock, von Frackenberg, Bardeleben, & Brandl-Hesse, 2001), and gait speed and endurance (Visintin, Barbeau, Korner-Bitensky, & Mayo, 1998). Also, it has allowed a more regular activation pattern of the shank muscles (compared with floor walking), with less co-contraction between the gastrocnemius and the tibialis anterior (Hesse, Konrad, & Uhlenbrock, 1999). Nevertheless, treadmill walking may not generate similar muscle and reflex activation patterns as in ground walking (Murray, Spurr, Sepic, Gardner, & Mollinger, 1985).

Common elements in the above-mentioned treatment approaches are the repetitive mode of treating and the use of an upright posture. This reiteration of stepping movement implies alternating postural changes for the involved limb: elevated, off-ground during swing phase (open chain) and in contact with the ground during heel-contact and stance phases (closed chain). The reflex and volitional interplay between these two states, through repetition, may contribute to the recovery of motor control. Okuma and Lee (1996) and Morita, Crone, Christenhuis, Petersen and Nielsen (2001) suggested intensive, reiterative dorsiflexion as a means of improving reciprocal inhibition (during open chain or swing) from ankle flexors to ankle extensors (Ia inhibition). This assumption, however, has not been tested experimentally.

Functional Outcome Measures

Functional outcome measures are necessary to assess therapy effectiveness. Natural and fast gait speed has been shown to correlate well with the stage of locomotor recovery and strength in the patient with hemiplegia (Brandstater, de Bruin, Gowland, & Clark, 1983; Friedman, 1990; Goldie, Matyas, & Evans, 1996; Nadeau et al., 1999; Richards, Malouin, Dumas, & Tardif, 1995; Wade, Wood, Heller, Maggs, & Langton Hewer, 1987) with good test-retest reliability (Hill, Goldie, Baker, & Greenwood, 1994). Good correlation has also been reported between gait parameters and scores from clinical tests of function, such as the Fugl-Meyer, Barthel and Berg tests (Brandstater et al., 1983; Goldie et al., 1996; Richards, Malouin, Dumas et al., 1995; Wade & Hewer, 1987). These studies suggested that gait speed is a good measure that reflects physiological and functional changes taking place during locomotor recovery. However, evidence linking functional recovery with restoration of the disrupted neuronal mechanisms underlying motor control is still lacking.

Reflex Pathways in the Spinal Cord

Relevant neural pathways that will be frequently mentioned and referred to throughout this study are described in this section. The following reflex pathways have been briefly defined based on Rothwell's (1994) view.

Classification of Nerve Fibers

Muscle afferent fibers are generally classified on the basis of fiber diameter into four groups (I - IV). Group I has the largest fibers (12-21 μm) and are further divided as Ia, arising from primary spindle endings; and Ib, arising from Golgi tendon organs. Group II fibers (6-12 μm) arise from muscle spindle secondaries, joint receptors, Pacinian corpuscles and other receptors. Group III and Group IV fibers are unmyelinated (1-6 μm) arising from nociceptors, thermoreceptors, mechanoreceptots (gross touch) and nociceptors, chemoreceptors respectively.

Reflex Pathways from Ia Pathways

Monosynaptic Excitation

The monosynaptic reflex from Ia afferents to motoneurones is probably the best known of all spinal reflexes. Ia fibers from one muscle provide homonymous excitation to motor neurones innervating the parent muscle, and heteronymous excitation to those supplying other muscles. Heteronymous Ia monosynaptic input can span more than one joint: in man there is a connection between the quadriceps muscles in the thigh and the soleus muscle in the calf.

Disynaptic Inhibition

Inhibition through two synapses is another major reflex action of Ia afferents on antagonist motoneurones. This is known as reciprocal Ia inhibition, because the inhibition to antagonist muscles is reciprocated by a similar inhibition from the antagonist Ia afferent fibers. The spinal interneurone which mediates disynaptic Ia inhibition has been

termed Ia inhibitory interneurone. Because of this common pattern of connections, α-motoneurones and linked Ia inhibitory interneurones (and γ-motoneurones) are said to form a functional unit in the spinal cord. Inputs to this unit produce α-γ coactivation and reciprocal inhibition of antagonist muscles.

Reflex Pathways from Ib Afferents

The Ib actions may be summarized as: 1) disynaptic inhibition of motoneurones projecting to synergists; and 2) disynaptic or trisynaptic excitation of motoneurones to antagonists. Ib fibers project into the motor nuclei of the ventral horn via Ib interneurones. Ib interneurones, as was the case with the Ia inhibitory interneurones, receive input from a wide range of sources. In particular, they are facilitated at a short latency by low threshold cutaneous and joint afferents, implying that this reflex pathway overlaps with that of servo-control of movement mediated by the stretch reflex.

Flexor Reflex Afferent (FRA) Pathways

Group II and III muscle afferent pathways participate in a much larger system of reflex pathways termed flexor reflex afferent (FRA) system. FRA afferents refer to a multisensorial and interneuronal reflex system that is associated with the generation of locomotion. Although the FRA system seems to refer to flexor activity only, extensor responses are also mediated by this system. Thus, the quality and location of the stimulus mostly determine FRAs flexor or extensor response. The FRA system has a powerful interaction between the two legs. Stimulation of FRAs exerts a weaker, opposite effect on the contralateral limb, with inhibition of flexors and excitation of extensors resulting in

limb extension (the crossed extensor reflex). One purpose of such a reflex would be to withdraw the limb from the stimulus (flexion) while supporting the body on the other extended limb. These ipsilateral flexion/contralateral extension actions have made some to suggest that FRAs may be involved with spinal stepping generators.

The Renshaw Cell (Recurrent Ia Inhibition)

In the 1940s, Renshaw demonstrated that antidromic impulses in motor axons could reduce the excitability of α-motoneurones projecting to the same or synergistic muscles. This phenomenon was called recurrent inhibition and has been shown to be due to activation of a group of interneurones by motor axon collaterals. They receive monosynaptic excitation from α-motoneurones and have a monosynaptic inhibitory connection back onto the homonymous and synergistic motoneurones.

Renshaw inhibition is distributed not only to motoneurones but also to homonymous and synergistic α-motoneurones and Ia inhibitory interneurones, as well as other Renshaw cells. Although still much in debate, the function of Renshaw cells seems to be of an inhibitory effect that would tend to reduce the average resting potential of motoneurones. This would: 1) reduce the frequency of α-motoneurones discharge below that expected in a system without recurrent inhibition; and 2) reduce the sensitivity of motoneurones to excitatory inputs. This is believed for some to be a function that would be analogous to the lateral inhibitory mechanisms of sensory systems.

Presynaptic Inhibition

Presynaptic inhibition is a means of changing reflex transmission, which can affect a variety of neural pathways from monosynaptic to multisynaptic. This inhibition is caused by activity in axo-axonic synapses near the terminals of the afferent nerve fibers. Group Ib fibers have a slightly different organization of presynaptic inhibition than group Ia. They are inhibited by other Ib fibers and also by the descending systems (e.g., corticospinal, rubrospinal, reticulospinal pathways). Cutaneous input suppresses the presynaptic inhibition of some Ib fibers and facilitates inhibition of others. Presynaptic inhibition in group II fibers is less clear. They seem to receive inhibition from Group I and other Group II fibers as well as from the corticospinal tract.

Clinical Electrophysiological Techniques

Clinical electrophysiology has proved invaluable to the understanding of motor control in the normal state. It likely has a similar role to play in understanding the disordered motor control in UMN syndromes.

The H-reflex

The Hoffmann reflex (now known as the H-reflex in his honor) is an indirect method of assessing α-motoneurone excitability (Magladery & McDougal, 1950; Schieppati, 1987; Zehr & Stein, 1999). The H-reflex arc includes input from large, fast-conducting Ia fibers. A characteristic of H reflexes is the delayed indirect response larger than the associated submaximal direct muscle action potential (short latency wave denominated M-wave). Soleus H-reflex testing is known as a sensitive test for S1

radiculopathies (Braddom & Johnson, 1974; Han, Kim, & Paik, 1997). It has been also used in investigating subjects with upper motor neurone lesions in whom disruption of supraspinal inhibitory control reveals disorganized motor control, especially the loss of reciprocal inhibition (Katz & Pierrot-Deseilligny, 1982; Leonard, Diedrich, Matsumoto, Moritani, & McMillan, 1998). The H-reflex has been used to investigate different reflex pathways such as the reciprocal (disynaptic) Ia inhibition (Tanaka, 1974), Ib inhibition (Pierrot-Deseilligny, Morin, Bergego, & Tankov, 1981), Renshaw (concurrent) inhibition (Katz & Pierrot-Deseilligny, 1982, 1984), and presynaptic inhibition (Edamura, Yang, & Stein, 1991; Hultborn, Meunier, Pierrot-Deseilligny, & Shindo, 1987; Schieppati, 1987; Stein & Capaday, 1988). Thus, H-reflex testing is not only a useful tool in routine clinical neurophysiology, but it is also a powerful resource in the study of spinal reflex pathways (Ferris, Aagaard, Simonsen, Farley, & Dyrhe-Poulsen, 2001; Sheean, 2001). Milanov (1992) provided an opposing view regarding the utilization of the H-reflex to assess the excitability of lower motoneurones. He compared three methods: H-reflex, F-wave and reflex from Achilles tendon percussion. These methods were used to test four groups of patients (30 each) with hemiparesis before and after anti-spasticity treatments (3 groups with medication and 1 group with electro-acupuncture). Muscle tone was clinically assessed with the Ashworth Scale. The results showed that patients in all four groups improved tone clinically and that only the F-wave detected the changes. He argued that this was due to the fact that H-reflex and Achilles tendon percussion-reflex depend not only on the excitability of α-motoneurones, but also on the activity of γ-motoneurones

and presynaptic inhibition. He added that F-wave parameters are easy to observe in both upper and lower limbs with no special recording conditions, and it depends solely on the excitability of the α-motoneurones, and thus, it is best indicated to assess the effects of treatment for spasticity. It should be noted, however, that lowering muscle tone (with medication) is not the same as lowering spasticity, since as Milanov also acknowledges, elevated excitability of the α-motoneurones is only one of many mechanisms of spasticity (Lance, 1980). A more detailed analysis of the physiology and clinical applications of H-reflex and F waves can be found in the work of Fisher (1992).

Electromyography

The electromyogram (EMG) is the electrical manifestation of the contracting muscle that can be used to evaluate the activity of muscle fibers or muscle groups during static and dynamic conditions (Basmajian & De Luca, 1985). Surface EMG has been used in the distal lower limb to assess muscle activity during treadmill walking in healthy controls and patients with hemiplegia due to stroke (Burridge, Wood, Taylor, & McLellan, 2001; Yang, Fung, Edamura, Blunt, Stein, & Barbeau, 1991). Surface EMG analysis, either alone or in conjunction with H-reflex testing, provides an uncommon opportunity to interface and synthesize neural and biomechanical analyses of normal and pathological gait (Leonard, 1995). Since the relevance of movement occurring in the distal lower limb is essential to the overall walking function, this investigation focused on investigating the volitional and reflex motor control of the ankle antagonists utilizing the

soleus H-reflex amplitude (reflex component) and surface EMG of the soleus and tibialis anterior muscles (volitional component).

Patterns of Normal Neuromotor Control of the Ankle Antagonists Measured by Soleus H-Reflex and EMG Recordings

Hoffmann, in 1918, found that low intensity stimulation of the tibial nerve in the popliteal fossa could produce a reflex contraction of the triceps surae muscles without direct activation of the muscle by the α-motoneurones. Hoffmann described the changes in amplitude of the soleus muscle H-wave in response to movement at the ankle joint, facilitation with soleus contraction (H-reflex gain) and inhibition with contraction of its antagonist (reciprocal inhibition from tibialis anterior muscle). Several studies investigated motor control of the ankle antagonists during gait in healthy subjects (Capaday & Stein, 1986, 1987; Crenna & Frigo, 1987). It is well known that the soleus H-reflex amplitude as well as soleus and tibialis anterior EMG activity varies greatly throughout the gait cycle. In this study, and as set forth by Capaday and Stein (1986, 1987) and Crenna and Frigo (1987), modulation of the H-reflex is defined as the presence of change (either facilitatory or inhibitory) in the recorded soleus H-reflex amplitude throughout the course of the gait cycle. Normally, soleus H-reflex amplitude during the stance phase and push-off is facilitated (amplitude widens), and during the swing phase and heel-contact it is greatly inhibited or totally absent. Although the inhibition of the soleus H-reflex during heel-contact is primarily the result of reciprocal inhibition from

the highly activated tibialis anterior muscle, its deep inhibition during the swing phase is unlikely to be the result of a similar mechanism, given the subtle activity of the tibialis anterior at the initiation of swing. The depression of the soleus H-reflex during the swing phase of walking is only partially related to tibialis anterior activity, indicating that a different and powerful source of inhibition (probably presynaptic inhibition) is the responsible (Yang & Whelan, 1993). Presynaptic inhibition to the soleus motoneurone pool, suggested to be from central origin, was reported to modulate during the gait cycle (Andersen & Sinkjaer, 1999; Faist, Dietz, & Pierrot-Deseilligny, 1996; Meunier & Pierrot-Deseilligny 1998). The presynaptic inhibition increased to a maximum at mid-stance and decreased to near zero by the end of stance phase of gait. The soleus H-reflex amplitude varied similarly. It increased at mid-stance and decreased by the end of the stance phase (Faist et al., 1996). Increments of presynaptic inhibition to soleus motoneurone pools also was demonstrated as body position is changed from supine to standing (Mynark, Koceja, & Lewis, 1997), reflecting its relevance in the more functional upright posture. Also, presynaptic inhibition to the soleus was reported to increment linearly with age which suggested an adaptive phenomenon in the aging neuromuscular system or, alternatively, a deteriorating process with decreasing flexibility in supraspinal adaptability (Morita, Shindo, Yanagawa, Yoshida, Momoi, & Yanagisawa, 1995). Thus, presynaptic inhibition to the soleus motoneurone pool during late stance and early swing apparently may be a key mechanism to help counteract the stretch reflex triggered in soleus muscle at the end of stance. This would allow the tibialis anterior muscle to

contract unchallenged by its antagonist during the swing phase. Aging, however, may affect negatively this critical reflex interplay, resulting in an increased risk of tripping due to less than optimal dorsiflexion and foot clearance during the gait cycle.

The soleus H-reflex amplitude was highly correlated with soleus muscle activity and inversely correlated with tibialis anterior muscle activity (Capaday & Stein, 1986, 1987; Kameyama, Ogawa, Okamoto, & Kumamoto, 1990; Peat, Dubo, Winter, Quanbury, Steinke, & Grahame, 1976). In general, EMG of the soleus showed the highest activity during the stance phase with its peak at the push-off. It remained silent during the swing phase and activated again just before the heel-contact. Tibialis anterior EMG generally presented two main activation peaks during the gait cycle (Kameyama et al., 1990; Knutsson & Richards, 1979). First, a larger burst originated just before heel-contact (to help absorb body-weight transfer on the landing foot after heel-contact); and second, a smaller burst originated just before swing (toe-off, to clear foot from ground). These modulations are crucial for the successful coordination of the gait cycle (Kameyama et al., 1990; Peat et al., 1976).

Patterns of Neuromotor Control of the Ankle Antagonists in Patients with Stroke Measured by Soleus H-Reflex and EMG Recordings

Drop-foot or the inability to dorsiflex the ankle effectively during the swing phase of walking was reported as a common problem for patients with CVA. Manca, Cavazzini, Cavazza, Salvadori, DeGrandis, and Basaglia (1998) and Leonard et al. (1998)

demonstrated that in subjects with cortical damage, soleus H-reflex inhibition did not occur either during voluntary tibialis anterior contractions or during unexpected anteroposterior postural perturbations in the standing position. A limited tibialis anterior EMG activity was observed when subjects voluntarily dorsiflexed the ankle while in the standing position (closed chain, similar to heel-contact). However, tibialis anterior EMG activity, although insufficient to cause reciprocal inhibition to the soleus, was observed during sudden, unexpected postural perturbations in standing. This suggested that patients in a position with their involved foot in a closed chain state similar to heel-contact, cannot produce enough volitional activity in the tibialis anterior during heel-contact, resulting in a flat foot reception after the swing (Knutsson & Richards, 1979). In patients, tibialis anterior EMG activation during the swing phase (open chain) was shown to be similar to that of healthy subjects (Burridge et al., 2001). Soleus H-reflex amplitude, however, remained elevated (not inhibited) which suggested that a central inhibitory (possibly presynaptic) effect on soleus α-motoneurones was lacking.

This lack of coordinated volitional and reflex modulation in the patients appeared to be caused by several factors. Evidence has shown a pathological increase in stretch reflex activity of the soleus during stance (Morita et al., 2001), impaired presynaptic inhibition (Morin, Katz, Mazieres, & Pierrot-Deseilligny, 1982; Yang & Whelan, 1993), dysfunctional reciprocal inhibition (Katz & Pierrot-Deseilligny, 1982; Okuma & Lee, 1996; Yang, Fung, et al., 1991), and altered cutaneous (sensory) afferents of the hemiparetic ankle (Brooke, Cheng, Misisszek, & Lafferty, 1995; Yang, Stein, & James,

1991). As a result, and in contrast to healthy subjects, the gait of patients with hemiparesis is highly variable. This, and the variability in the location and extent of the central damage, makes it difficult to establish consistent patterns of motor control dysfunction that could be used to allow comparisons between patients.

STATEMENT OF THE PROBLEMS

In this study, the problems were organized into two areas. First, the methodological difficulties that are found in assessing how the soleus H-reflex amplitude and soleus/tibialis anterior EMG vary under dynamic conditions. And second, the lack of electrophysiologic evidence linking functional gait improvements with recovery of underlying neuronal modulation of disrupted neuromotor control of the ankle antagonists.

Methodological Difficulties in Testing the Soleus H-Reflex and Soleus/Tibialis Anterior EMG under Dynamic Conditions

Analysis of soleus H reflexes during normal gait, although already revealing the changes in the patterns of neuromotor control (facilitation during the stance phase and inhibition during the swing phase), still faces significant methodological threats that may affect the repeatability and stability of recordings. One of these problems is the displacement that occurs between the skin where electrodes are placed and the underlying, targeted nerve or muscle. Recordings may differ throughout the movement range reflecting relative displacements of the stimulating and recording sites. Of concern

also is that a large number of stimuli and EMG recordings are needed to sample and profile the modulation of gait cycle under dynamic conditions. The smoothness and similarity of gait movements among healthy subjects minimize these difficulties; however, this was not seen when gait was studied in patients with CVA or UMN syndromes. Patients showed a variety of abnormal responses of the stretch reflex to movement, such as clonus (Yang, Fung, et al., 1991). Also, patients show wide inter-individual variability of gait speed, muscle recruitment and movement patterns during gait and differences in their strength and fatigue levels (Katz & Pierrot-Deseilligny, 1982). Another concern is that in the majority of the reviewed studies, gait was investigated with treadmill equipment. This method may generate patterns of EMG and reflex data that are not based in actual ground walking, introducing the potential for inaccurate extrapolations. In addition, since walking speed of patients is in general lower than that of matched healthy subjects (Goldie et al., 1996; Turnbull, Charteris, & Wall, 1995), studies comparing the two populations under similar gait speeds would require healthy subjects to walk at abnormally slow speeds, which could introduce some distortion in the normal patterns. These elements certainly limit the effectiveness and accuracy of testing electrophysiologic parameters during gait. The use of static postures that mimic gait phases may alleviate some of these difficulties.

Linking Functional Gait Improvements with Recovery of the Underlying Disrupted Neuronal Mechanisms of the Ankle Antagonists

It is well established that rehabilitative intervention has a positive impact in the recovery of gait in stroke survivors. Usually, physical therapy aggressively seeks reinforcement of gait and gait-like activities in these patients. A large portion of these therapeutic activities and exercises are reiterative, involving repeated stepping and reinforcement of ankle dorsiflexion. This reinforcement often occurs by using alternating open and closed chain activity of the involved lower leg and foot (swing and heel-contact/stance phase respectively). Then, as patients recover, they progress to more complex gait activities.

Evidence has shown that during ankle open/closed chain activity several electrophysiologic changes occur in the interplay of the ankle antagonists. In healthy subjects, open chain (swing phase) is associated with lowered or absent soleus H-reflex amplitudes, which are in parallel with a depressed soleus EMG activity and a greater activity in the tibialis anterior muscle (Capaday & Stein, 1986, 1987; Crone & Frigo, 1987; Morin et al., 1982; Stein & Capaday, 1988). These responses are suggested to reflect a direct reciprocal inhibition to the soleus from the tibialis anterior (postsynaptic) and a greater presynaptic inhibition of central (cortical) and peripheral (i.e., cutaneous, muscle and joint receptors) origin. Open chain (swing) in patients is characterized by an abnormally elevated (not inhibited) soleus H-reflex, possibly reflecting the lack of a presynaptic inhibitory volley. And, a natural tibialis anterior EMG activity comparable to

that of healthy subjects, which suggested the existence of a neural processing center that locates sub-cortically (unaffected by the cortical insult). This neural center located probably at the spinal level may involve central pattern generators (CPG) and/or flexor reflex afferents (FRA). These latter reflex mechanisms are known to participate in the generation and control of alternating limb movement (Grillner, 1985; Leonard, 1995; McCrea, 1986; Rothwell, 1994).

Normally, during closed chain (heel-contact and stance phase) soleus H-reflex amplitudes are larger than during open chain (swing phase). This is in parallel with the greater soleus muscle EMG activity seen towards the end of the stance phase or push-off (Capaday & Stein, 1986). Tibialis anterior EMG activity is progressively lowered as the foot makes full contact with the ground (Knutsson & Richards, 1979; Yang, Fung, et al., 1991). In patients with stroke, the soleus muscle reveals abnormal EMG activity bursts while being stretched by the shank as it travels forward over the fixed foot. These bursts are associated with the presence of spasticity (abnormal, velocity dependent stretch reflex hyperexcitability) (Yang, Fung, et al. 1991). Preservation and improvement of volitional tibialis anterior activity correlated positively with good recovery in gait function in the presence of mild spasticity, which seems enhanced by early upright stepping and gait training (Morita et al., 2001; Okuma & Lee, 1996).

One common outcome measure used to document gait function improvements is gait speed. Patients who walk faster are believed to have better motor control (Brandstater et al., 1983; Goldie et al., 1996; Witte & Carlsson, 1997). Repetitive ankle

dorsiflexion (open chain) may have a positive influence on improving motor control of the ankle antagonists (as reflected by improved gait speed), possibly due to recovery of a reciprocal inhibition mechanism (Okuma & Lee, 1996). However, evidence showing recovery of reciprocal inhibition as related to recovery of gait function and the restoration of soleus H-reflex modulation is lacking.

JUSTIFICATION FOR THE STUDY

Pattern of Soleus H-Reflex and Soleus/Tibialis Anterior EMG Activity Using Gait-Simulating Postures

Evaluative procedures that disclose abnormal patterns of motor control are essential to defining baselines for treatment planning and developing of testing protocols that could serve to measure change over time or the effect of therapeutic intervention. Evidence favors the use of the soleus H-reflex and associated soleus/tibialis anterior EMG as a means to gain insight into the disordered motor control of the ankle antagonists after stroke.

Some preliminary evidence has shown that in healthy subjects, soleus H reflexes and soleus/tibialis anterior EMG recorded in sustained (static) postures that simulated phases of gait vary in a manner comparable with those seen in dynamic studies (Crenna & Frigo, 1987). In addition, static H-reflex measurements were reported to have improved intra-individual reliability (Handcock, Williams, & Sullivan, 2001). Similarly, static conditions were recommended for the accuracy in surface EMG recording (De

Luca, 1997). In this study, five standing gait-simulating postures (N: free standing, PO: push-off, HC: heel-contact, SW: swing, and MS: mid-stance) were used to reproduce the changes in soleus H-reflex amplitude and soleus/tibialis anterior EMG reported during gait.

A testing paradigm based on gait-simulating postures that reflects the changes (modulation) of the soleus H-reflex amplitude and soleus/tibialis anterior EMG activity during gait may be a useful and practical tool for the clinician to objectively assess motor control of the ankle antagonists. It would also provide assessment uniformity (for patients and healthy subjects) regardless of a patient's functional status, thus allowing for intra- and inter-individual comparisons.

Soleus H-reflex and Soleus/Tibialis Anterior EMG in Patients with Stroke

At present, there is a need for studies that investigate the effects of exercise on the recovery of the volitional and reflex modulation of the ankle antagonists in patients with CVA. Repetitive dorsiflexion exercise may positively influence the volitional and reflex interplay of the ankle antagonists impaired by CVA. Neurophysiologically, a possible mechanism may involve Ia inhibitory influence from the tibialis anterior, which may influence the recovery (normalization) of soleus H-reflex play. This reflex mechanism may be enhanced by gains in the volitional control of the tibialis anterior associated with improvement in spasticity levels. The extent of this influence on different patients and its

relation to gait function may help to identify those patients more likely to benefit from therapy.

PURPOSES OF THE STUDY

The main purposes of this study were first, to investigate in healthy subjects the volitional and reflex interplay of the ankle antagonists in static gait-simulating postures and to determine its similarity with that seen in normal gait studies. The second purpose was to investigate these same changes in patients with CVA and compare them with those seen in healthy subjects. The third purpose was to assess the individual and group responses to a rehabilitative intervention directed to improve ankle motor control as measured by gait speed. Stability and repeatability of the soleus H-reflex amplitude was assessed through a test-retest reliability study for both healthy subjects and patients.

HYPOTHESES

1. In healthy subjects the pattern of changes in soleus H-reflex amplitude recorded in gait-simulating postures will reproduce those profound changes seen in previously published normal gait studies. Throughout the gait cycle, the soleus H-reflex amplitude will be closely paralleled by the soleus EMG activity and it will be reciprocal to the tibialis anterior EMG activity.

2. In patients with CVA, the soleus H-reflex amplitude will not vary (i.e., modulate) throughout the gait cycle as seen on the healthy subjects. Hence, the soleus H-

reflex amplitude will not parallel the soleus EMG activity, and it will not show reciprocity with tibialis anterior EMG activity.

3. The changes in the soleus H-reflex amplitude throughout the gait cycle in patients with CVA will be enhanced (i.e., recovered) by an exercise program directed to restore disynaptic inhibition from tibialis anterior. Gains in the neuromotor control of the ankle antagonists will be reflected by improvements (volitional) in tibialis anterior EMG and in gait speed.

CHAPTER II

LITERATURE REVIEW

Electromyography

When a muscle contracts a series of action potentials are generated. These are called motor unit action potentials (MUAP). These changes in potentials can be detected by myoelectric electrodes, amplified and recorded (Basmajian & De Luca, 1985).

Electromyography (EMG) has been used extensively to assess muscle activity, either from individual or groups of muscles in healthy and neurologically impaired individuals (Turker, 1993). Other examples of clinical EMG use include assessing muscle function during or as a result of exercise and therapeutic procedures (Callaghan, Gunning, & McGill, 1998), providing visual and auditory feedback to patients (Cozean et al., 1988; Draper, 1990), and assessing gait function (Burridge et al., 2001; Knutsson & Richards, 1979). Elements to consider in using EMG instrumentation are the myoelectric electrodes (transducers), amplification, filtering and signal processing (Soderberg & Cook, 1984).

Electrodes

The electrode converts the bioelectric current of the EMG signal into electron current (Sorderberg & Cook, 1984). There are basically three types of EMG electrodes: surface; needle and fine-wire electrodes. In this study, surface EMG electrodes were

used. Surface electrodes are useful for deriving general information from superficial muscles, whereas indwelling electrodes (needle or fine-wire) are designed to discriminate superficially located motor units, or to ensure sampling from muscles located deep in the body (Soderberg & Knutson, 2000). Good reliability of EMG recordings has been reported with use of surface electrodes in isometric conditions (Komi & Buskirk, 1970), dynamic and isometric conditions (Giroux & Lamontagne, 1990), and during gait activity (Kadaba, Wootten, Gainey, & Cochran, 1985). Since a more global view of muscle activity is obtained by using surface electrodes, their use is commonly favored by physical therapists (Sorderberg & Knutson, 2000).

Electrode Configuration

The bipolar configuration is currently the most commonly used surface electrode arrangement. This configuration is readily available, easily applied and causes minimal discomfort to the user (Sorderberg & Knutson, 2000; Turker, 1993). Jonas, Bischoff, and Conrad (1999) recommended that surface electrodes should have adjustable distances between the recording pairs, to allow for reproducibility of measuring conditions between different laboratories and different times.

Amplifier

The purpose of the amplification of the myoelectric signal is to convert the electrical current to voltage and to enhance the signal with minimal distortion and noise (Sorderberg & Cook, 1984). According to Sorderberg and Cook (1984) relevant characteristics of the amplifier are: 1) the input impedance (ratio of the applied voltage to

the current drawn from the signal source); 2) the differential amplifier (used to amplify the difference between two electrical signals only); 3) the voltage gain (ratio between input and output signal that ranges between multipliers of 100 and 10,000); 4) the frequency bandwidth of the amplifier (considered a window of the frequency domain, which usually ranges between 10 to 1,000 Hz for surface EMG and 10 to 10,000 Hz for indwelling EMG); and 5) the common mode rejection ratio or CMRR (ratio between common mode signals at input and those at output). For a differential amplifier its optimal level is at about 1000 to 1 (60 decibels [dB] or higher).

Quantification of EMG Recordings

Once the EMG signals are detected, filtered and amplified, raw or analog data is obtained (Sorderberg & Cook, 1984). The analog data then can be converted to digital data using an analog-to-digital converter. Digital data can be storage for later display and analysis. During data analysis, further rectification and filtering may take place (Turker, 1993). De Luca and Van Dyk (1975), Lawrence and De Luca (1983), and Basmajian and De Luca (1985) have recommended the use of integration and the root mean square (RMS) to quantify the EMG signal. These researchers stated that the RMS more completely represents motor unit behavior during muscle contraction compared to other parameters of the myoelectric signal (e.g., the mean rectified value) used to describe the output and the state of the contracting muscle.

Normalizing Procedures

The decision to normalize or not is based on the type of descriptions or comparisons to be made. For example, if comparisons are made between subjects, days, muscles, or studies, the process is required (Winter, 1991). Conversely, if subjects serve as their own control and contrasts are made within a day and on the same muscle, with the electrode not being removed, normalization is generally not necessary (Sorderberg & Knutson, 2000). However, if normalization is necessary, its standardization has not been established (Sorderberg & Knutson, 2000). Some researchers have suggested, however, some procedures to normalize EMG recordings. Yang and Winter (1983) divided the EMG data of interest by that recorded during a maximum or submaximum voluntary isometric contraction and expressing the resultant as a percentage of the isometric contraction. Alternatively, Winter and Yack (1987) used the peak EMG value obtained during a dynamic activity to normalize the data.

The H-Reflex

A characteristic of H reflexes is the delayed indirect response (wave) which is larger than the associated submaximal direct muscle action potential (short latency wave denominated M-wave). This difference between the two waves occurs only if there is central amplification of the motor response that is due to reflex activation of α-motoneurones; that is, reflex contraction of the triceps surae muscles without direct activation of the muscle by the α-motoneurones. The reflex occurs because the group Ia

fibers are larger than, and have a lower threshold to electrical stimulation, than the α-motoneurones. Therefore, at low stimulus intensities, Ia afferents may be the first fibers to be activated. The relatively pure and synchronous Ia afferent volley then produces reflex activation of triceps surae muscles. The H reflexes are largest at submaximal nerve stimulation and are inhibited by greater stimulation. With increased stimulus strength, the axons of α-motoneurones are stimulated and a direct muscle response (M-wave) is elicited. This is due to two factors: 1) antidromic firing of motor fibers rendering the motoneurones refractory to the reflex input; and 2) the antidromic motor volleys collideing with the orthodromic reflex volley set up by the Ia input (Rothwell, 1994). With a stepwise incrementation of the stimulus intensity to the posterior tibial nerve, and once the threshold level of the Ia fibers have been reached, the pulse invades the Ia axons orthodromically. It passes through the posterior horn to synapse on α-motoneurone cells in the anterior horn of the spinal cord (Sabbahi, 1976). In newborns, H reflexes may be widely distributed in their location, but beyond the age of 2 years, these are found only in calf muscles, primarily the soleus, vastus medialis and homologous forearm flexors. This restricted distribution reflects refinement of motoneurone pool activation with central nervous system (CNS) maturation (Fisher, 1992).

The H-reflex does not include muscle spindle activation, but the reflex arc is similar to that of the spindle-dependent phasic myotatic (deep tendon) reflex produced by dynamic muscle lengthening. Generally, the H-reflex arc has been considered to be monosynaptic (Fisher, 1992; Schieppati, 1987); however, recent reports have shown that

H reflexes involve both oligo- and polysynaptic pathways (and thus interneurones) that are influenced by supraspinal and other segmental influences (Rothwell, 1994; Sheean, 2001).

Soleus H-Reflex Reliability and Stability

High intersession and intrasession reliabilities (ICC [3,1]) of the soleus H-reflex measured in a one-leg standing position (0.803 and 0.853 respectively) were reported by Hopkins, Ingersoll, Cordova, and Edwards (2000). High intra-individual reliabilities (ICC [3,1]) for soleus H-reflex and M response (0.96 and 0.87 respectively) were also provided by Handcock et al. (2001) for measurements taken with subjects in standing, weight bearing postures. Also, stability of the amplitude and latency of soleus H-reflex was better preserved if measured in the upright posture compared with such less-functional postures as prone lying (Ali & Sabbahi, 2000). These studies demonstrated that the soleus H-reflex in weight bearing standing is stable and resistant to potentially confounding postural influences, or other sources of biological variation.

Studies Investigating Normal Soleus H-Reflex and Soleus/Tibialis Anterior EMG

Studies during Steady Standing

Capaday and Stein (1986) recorded soleus H-reflex amplitude and soleus and tibialis anterior EMG in six healthy subjects during steady standing and compared it to recordings obtained during treadmill walking. Their results found that soleus H-reflex

was higher in amplitude during quiet standing than during walking, and had less soleus EMG activity during standing than walking.

Hayashi, Tako, Tokuda, and Yanagisawa (1992) showed in seven healthy subjects that soleus H-reflex gain was less during standing with and without support compared to that obtained in sitting. They suggested that when postural stability was affected, the presynaptic inhibition of Ia fibers to the soleus motoneurones would increase. This increment in presynaptic inhibition would lower the stretch reflex gain to stabilize posture.

Koceja, Markus, and Trimble (1995) studied the changes in soleus H-reflex amplitude in 10 young and 15 older persons during static standing and prone. On average, the young group showed a downward shift of the H-reflex amplitude from prone to standing. The old group did not show such change in amplitude. The authors pointed out that the increased H-reflex amplitude during standing would easily be accounted for by the greater background activity of the soleus muscle when the standing posture was assumed. The EMG activity of the soleus muscle during both standing and prone was not significantly different between the two groups, but it increased for both groups from prone to standing. Therefore, despite increased muscle activity when standing, the young subjects exhibited depressed H-reflex gain, suggesting the involvement of mechanisms other than background muscle activation levels.

Studies during Passive Standing and Active Stepping

Crenna and Frigo (1987) described soleus H-reflex excitability during stepping without progressing to walk. The stepping reproduced the step cycle including stance, swing, and the stance-swing transition. Soleus H-reflex amplitude was elevated (compared to a standing weight bearing posture) toward the end of the stance phase and started to decrease just before the beginning of the swing phase, and it remained inhibited throughout swing. Soleus EMG activity showed two main bursts: one at the initiation of the stance phase; and a second toward the end of the stance phase. Tibialis anterior EMG activity was mostly silent during the stance phase, but EMG activity increased during the swing phase. Based on these observations the authors believed that different mechanisms (pre- and postsynaptic influences) could be proposed to explain the H-wave inhibition during the end of the stance phase and throughout the swing phase.

Brooke et al. (1995) investigated active and passive stepping in a total of 21 healthy subjects. The postures were natural bipedal and unipedal stepping in a fixed point on the ground, passive stepping (body was tilted 20 degrees from vertical to silence postural EMG) with external manipulation of the limb, and during passive stepping with concomitant tonic contraction of the soleus muscle. It was found that soleus H-reflex inhibition occurred in all postures regardless of whether it was an active or passive stepping motion. It was concluded that the profound inhibition of soleus H-reflex appeared to have a presynaptic origin along with a sensory component (mechanoreceptors of quadriceps muscle sensitive to changes in muscle length) arising

from the movement itself. Later, work from Garrett, Kerr, and Caufield (1999) and Schneider, Lavoie, and Capaday (2000) showed evidence against the findings suggested by Brooke et al. (1995). Garrett et al. (1999) studied 15 healthy subjects to test the effect of restricted knee motion on soleus H-reflex during active walking. In their results, soleus H-reflex was attenuated during the swing phase even though the subject's knees were locked with a bivalved cast in full extension. The authors concluded that sensory input was unlikely to have an effect in attenuating soleus H-reflex, at least those originated from motion at the knee (stretch-evoked afferent activity from mechanoreceptors of the quadriceps), supporting the notion of central control of locomotion. The authors acknowledged that bracing the knee could trigger sensory signals from spindles and Golgi tendon organs. However, the authors reported that care was taken to assure that the pressure exerted by the cast on the skin surface was evenly distributed. Schneider et al. (2000) studied 24 healthy subjects in a similar experiment adding support to the findings provided by Garrett et al. (1999).

Kasai, Kawanishi, and Yahagi (1998) demonstrated in eight healthy subjects that the effect of reciprocal inhibition during initiation of ankle dorsiflexion is posture-dependent. Ankle dorsiflexion was investigated in sitting and standing postures. They found that H-reflex depression was significantly larger in the standing than in the sitting posture in spite of the same dorsiflexion movement. Furthermore, the depression upon initiation of dorsiflexion movement appeared earlier in the standing than in sitting. The authors concluded that these changes in reciprocal inhibition (from tibialis anterior to

soleus motoneurone pool) during standing was appropriate to maintain a stable standing posture prior to and during forthcoming voluntary movement.

Studies during Normal Walking

Ground or Treadmill Walking

In 1984, Garrett, Ireland, and Luckwill reported on H reflexes measured in the gastrocnemius muscle of 15 healthy subjects during walking. Compared to H-reflex amplitudes obtained in a standing non-weight bearing posture, H-reflex amplitude was reduced by 60% at mid-swing and it augmented by 85% at mid-stance. Further, during swing phase the H-reflex amplitude (excitability) remained constant. In contrast, during stance, excitability increased from 50% of the control value at the beginning (heel strike) to peak mid-stance at 185% of the control value and then fell sharply to 17% of the control value at the end of stance (toe-off). They concluded that the changes in excitability (amplitude) would appear to facilitate ankle extensor activity required during the stance phase of walking and reduce the likelihood of unwanted plantarflexion during swing.

Capaday and Stein (1986, 1987) demonstrated how the soleus H-reflex amplitude varied greatly during treadmill walking in humans. The authors analyzed H reflexes and soleus/tibialis anterior EMG activity from six healthy subjects during level walking on a treadmill at a comfortable speed. The authors correlated the changes in the soleus H-reflex amplitude during the gait cycle with the soleus and tibialis anterior EMG recordings. The EMG activity of the soleus usually began before the heel made ground

contact. It then increased during most of the stance phase, and terminated just before the toes were lifted off the ground. The tibialis anterior EMG activity was mostly reciprocal to the soleus EMG and consisted of two prominent bursts. The first was associated with the ankle dorsiflexion late in the swing phase. The second, usually larger burst began at about the time of heel contact and continued until about the time the foot was at mid-stance. Since the soleus muscle was also active during heel contact, a considerable co-contraction with the tibialis anterior was observed. The amplitude of the soleus H-reflex during a step cycle varied greatly (facilitated during stance and inhibited during swing) in all six subjects studied. Of the six subjects, three showed a soleus EMG activity that paralleled closely the soleus H-reflex amplitude changes throughout the step cycle. In all subjects the soleus H-reflex amplitude was reciprocal to the tibialis anterior EMG activity.

Crenna and Frigo (1987) also investigated the changes in the soleus H-reflex amplitude and soleus/tibialis anterior EMG recordings in eight healthy subjects during treadmill walking. They showed that soleus EMG was largest in the second half of the stance phase (push-off), and decreased sharply just before the toe-off time (stance-swing transition). It remained mostly absent throughout the swing phase, and began to rise slightly just before heel contact. For the tibialis anterior EMG activity, they reported two main activity peaks. One, at the beginning of the swing phase, and another of greater magnitude around the time of heel contact (more likely to control the speed of the landing foot into the upcoming stance phase). The authors also observed that soleus H-reflex

amplitude varied highly during the course of the step cycle. The largest reflex gain was seen at the time of push-off and the lowest at the beginning of the swing phase (toe-off).

Yang, Fung, et al. (1991) and Petersen, Morita, and Nielsen (1999) reported similar results showing large changes in the soleus H-reflex amplitude during the gait of healthy subjects walking on a treadmill. Soleus H-reflex inhibition was large in the swing phase and absent in the stance phase. Also, the soleus EMG was reciprocal with the tibialis anterior EMG activity and it paralleled closely the changes in amplitude of the soleus H-reflex during the stance and swing phases.

Kameyama et al. (1990) investigated EMG patterns in the ankle muscles during natural gait in 147 healthy subjects (112 men and 35 women, age range 19 - 35 years). At the ankle two muscles were studied, tibialis anterior and lateral gastrocnemius. Their results showed four types of activation patterns for the tibialis anterior. However, a common discharge pattern observed in all subjects was the double-burst pattern, where the first burst appeared before heel-contact and the second burst appeared before toe-off. The activation pattern for the lateral gastrocnemius showed three discharge patterns. However, a common discharge pattern observed in all subjects was a burst at mid-stance phase with no activation in the mid-swing phase. The authors concluded that these EMG discharges were the essential EMG patterns of the ankle muscles during normal gait. These results were similar to those reported earlier by Knutsson and Richards (1979). The authors stated that even though all subjects followed general rules of EMG discharges, it is wise to acknowledge that individual traits do exist. Reservation then is suggested in

interpreting pooled EMG data, because the individual peculiarities are often averaged out, especially with respect to the soleus discharge patterns.

Beam Walking

Soleus H reflexes were investigated by Llewellyn, Yang, and Prochazka (1990) in five healthy subjects during beam walking and walking on a treadmill. Background soleus EMG activity was matched for the two walking conditions by adjusting the walking speed on the treadmill for that of beam walking. The authors found that the normal reciprocal activation pattern of tibialis anterior and soleus muscles in treadmill walking was replaced by a pattern dominated by co-contraction on the beam. The H reflexes varied highly in both tasks, the amplitude being greater in the stance phase and lower in the swing phase. Overall, the averaged soleus H-reflex amplitude during the stance phase of beam walking was 40% lower than those obtained during treadmill walking.

Graded Walking

Simonsen, Dyrhe-Poulsen, and Voigt (1995) studied the soleus H-reflex in five healthy subjects during level walking, as well as during uphill and downhill walking at an 8% grade (on a treadmill). Their findings showed that soleus H-reflex amplitude varied highly in all three walking conditions. It was elevated during the stance phase and low or absent during the swing phase. This absence of the H-reflex during the swing phase was considered by the authors to be a decisive factor for normal human locomotion. Shortly after toe-off the ankle is dorsiflexed rapidly by the tibialis anterior muscle and if this

dorsiflexion were to elicit a stretch reflex in the soleus muscle the movement pattern would be severely disturbed as seen in spastic patients. During level walking, a fast rise in reflex excitability was observed just after heel strike with low or absent soleus EMG. The mean soleus EMG was lower during downhill than during uphill or level walking, but the mean H-reflex amplitude was similar in all three conditions. The authors concluded that regulation of ankle joint stiffness was the more likely functional implication regarding the differences found in the three walking conditions.

Studies during Running

Capaday and Stein (1987) studied the soleus H-reflex in eight healthy subjects during walking and running on a treadmill. There were two main findings reported by the authors. First, they found that the H-reflex varied in amplitude during running and also in walking. Second, during running the soleus H-reflex amplitude was on average less than during walking, despite the fact that the soleus EMG activity during running was on average 2-4 times greater than during walking. The authors argued that these modulations were appropriate to the requirements of running since elevated reflex activity is observed during the stance phase where the soleus muscle undergoes a lengthening contraction. At the same time, spindle afferents presumably are also firing at high rates during stance, and the stretch reflex would contribute to the muscle tension required to decelerate the downward and forward motions of the body and to lift the body off the ground. The reflex is absent during the swing phase, since a high reflex sensitivity at this time would activate the soleus muscle and oppose the active dorsiflexion of the ankle. The authors

used the term "automatic gain compensation" to explain the lowered reflex gain during running as compared to walking (and from previous work, walking compared to standing). This lowered gain during running, they stated, may be the appropriate response to reduce saturation of motor output and potential instability of the stretch reflex feedback loop. It is important to note that the investigators acknowledged the technical problems of using surface electrodes during the highly dynamic tasks of walking and running, which could cause the inevitable displacements of the tibial nerve and muscle fibers. Therefore, the effective stimulus strength (current density) is not the same throughout the locomotor cycle. The authors argued that these stimulating and recording inconveniences were largely controlled by repeating the experiment at several stimulus intensities and using the standing Mmax wave to scale direct M-wave during locomotion. Then, H reflexes occurring at various phases of the step cycle could be compared at essentially the same stimulus intensity.

Simonsen and Dyrhe-Poulsen (1999) arrived at somewhat different results in their measures of soleus H-reflex amplitude done in seven healthy subjects during similar walking and running activities. H-reflex amplitudes became larger as subjects transitioned to running from walking, and they further augmented with increments in running speed. A suggested cause for this difference in H-reflex behavior was their finding that the size of the maximal M-wave varied during the gait cycle and this variation was consistent for each subject although different among subjects. Since this variation of the M-wave would more likely influence the recorded levels of H-reflex

excitability, they used an alternative method (to that of Capaday and Stein, 1987) which was to compensate for the variations in Mmax due to changes in muscle and nerve geometry. The alternative method added an additional submaximal stimulus to generate a second M-wave just after the H-reflex wave was evoked. A computer program was then developed to allow the adjustment of stimulus strength based on the fluctuations in Mmax during the gait cycle and avoid subject fatigue by reducing total experiment time. This method and the one by Capaday and Stein were compared on measures taken from the subjects in standing. During the stance phase the reflex was facilitated and during the swing phase it was suppressed. And also, the soleus EMG activity increased up to 3 times during running compared to walking.

Walking and Running under Reduced Gravity

The study by Ferris et al. (2001) investigated soleus H-reflex gain during walking and running with different levels of muscle activity. This was achieved by studying eight healthy subjects while walking and running on a treadmill and while being suspended by a harness in order to simulate reduced gravity (1.0, 0.75, 0.50 and 0.25 G). To calculate H-reflex gain (slope line from H-reflex and EMG interaction) the authors used a previously reported method that adjusted continuously the Mmax (Simonsen & Dhyre-Poulsen, 1999). The authors found no difference in the gain slopes between walking and running, but there was a difference in y-intercept. They concluded that the primary difference in the H-reflex between walking and running was a difference in H-reflex threshold and not a difference in H-reflex gain. Walking has a lower reflex threshold than

running. Thus, a given afferent signal from soleus muscle spindles will produce a greater efferent response during walking compared to running, independent of muscle recruitment level. Also, the authors found H-reflex facilitation during the stance phase and H-reflex inhibition during the swing phase on both walking and running in all four levels of gravity.

Summary of Studies Investigating Normal H-Reflex and EMG of the Ankle Antagonists

There is ample agreement in that the amplitude of the soleus H-reflex varies greatly during the course of normal gait and gait like activities such as running and stepping. It is suggested that this variation could be reproduced in static postures simulating phases of the gait cycle.

The greater soleus H-reflex amplitude is seen in the stance phase, and during foot push-off. It is suggested that this elevated soleus H-reflex response involve a stretch-triggered mechanism (lower leg moving forward on a fixed foot).

The lowest soleus H-reflex amplitude (and sometimes its complete absence) is seen in the beginning of the swing phase just before toe-off, and it remains diminished for most of the swing phase.

Soleus EMG activity is greater at the stance phase and mostly absent during swing phase. Soleus EMG activity closely parallels the changes of the soleus H-reflex amplitude during the course of the gait cycle.

Tibialis anterior EMG activity has two main activation peaks, at heel-contact (help to absorb heel strike) and during toe-off (dorsiflexion to clear the ground). Tibialis anterior EMG activity shows a reciprocal relationship with the soleus H-reflex amplitude during the course of the gait cycle.

An important amount of muscle co-contraction between the soleus and the tibialis anterior is seen during heel-contact at the beginning of the stance phase. At this point soleus H-reflex remains low.

Although tibialis anterior reciprocal inhibition to the soleus muscle has been shown to affect soleus H-reflex amplitude, this is likely to be only partially responsible for its variation during the step cycle. Other possible mechanisms affecting the soleus H-reflex amplitude are central presynaptic inhibition and cutaneous afferent influences.

Spasticity

A definition of spasticity widely accepted was given by Lance (1980): "Spasticity is a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motor neurone syndrome". This definition emphasizes the fact that spasticity is only one of the various features found in the UMN syndrome. The UMN syndrome can occur following any lesion affecting some or all of the descending motor pathways.

The clinical features of the UMN syndrome can be divided into two broad groups (Mayer, 1997; Sheean, 2001): 1) negative phenomena (i.e., the absence of or the impairment of an ability or condition): muscle weakness, loss of dexterity, fatigueability; and 2) positive phenomena (i.e., the presence of abnormal or exaggerated responses): increased tendon reflexes, clonus, positive Babinsky sign, spasticity, extensor spasms, flexor spasms, mass reflex, dyssynergic patterns of co-contraction during movement, associated reactions and other dyssynergic and stereotypical spastic dystonias). Among the positive signs, spasticity is the hallmark of the UMN syndrome (Sheean, 2001).

Neurophysiology of Spasticity

It is common for clinicians to picture the whole UMN syndrome and all the mentioned positive features of this syndrome as spasticity, since the positive features of UMN (e.g., tendon hyperreflexia, extensor plantar responses) tend to occur together. For example, increasing flexor spasms is often seen as a sign showing worsening of spasticity (Sheean, 2001).

In spasticity the α-motoneurone pool becomes hyperexcitable at the segmental level, and in cerebral lesions, this augmented excitability has a bias toward involvement of the antigravity muscles (Mayer, 1997; Milanov, 1994). A characteristic feature of spasticity (hypertonia) is that it is dependent upon the velocity of the muscle stretch; therefore, greater resistance is felt with faster stretches (Lance, 1980). Common consequences from hypertonia are that the muscle has a tendency to remain in a

shortened position. Thus, if maintained for prolonged periods of time it may result in soft tissue changes and eventually lead to the development of contractures (O'Dwyer, Ada, & Neilson, 1996; Thillman, Fellows, & Garms, 1991). In addition, triggered resistance to lengthening in the antagonists restricts attempted agonistic movements. Thus, hypertonia often has both a neural component (secondary to the spasticity) and a biomechanical component (secondary to the soft tissue changes). The relative contribution of each of these two components needs to be taken into account when planning a treatment intervention (Mayer, 1997). Most of these positive phenomena of the UMN syndrome can occur at rest. Some other problems can be seen during movement, such as inappropriate co-contraction of antagonistic muscle groups and disrupted limb movements (Knutsson, 1981; Knutsson & Richards, 1979).

Studies Investigating Soleus H-Reflex and Soleus/Tibialis Anterior EMG in Hemiplegia

Knutsson and Richards (1979) investigated 26 patients with gait disorders due to spastic hemiparesis (mean age 50 years, age range 19 - 71 years). Patients were asked to walk five meters at their own pace and EMG activity from muscle groups of the involved limb, including tibialis anterior and triceps surae, were recorded. In their results, the authors acknowledged the large variability of the EMG patterns during walking. In spite of this, three different types of abnormal activation patterns were defined.

Type I. This pattern was characterized by premature activation of the triceps surae during the stance phase of gait cycle, usually combined with a relatively lower than

normal degree of activity in both the tibialis anterior and triceps surae (9 patients). Tibialis anterior EMG at the initiation of swing phase, however, was not clearly different from the averaged normal. The abnormal activation (in some cases with more than one abnormal activity burst) of the triceps surae occurred in periods where the muscles were lengthening. Thus, this may be related to the stretch imposed to the triceps surae as the shank moves forward over a fixed foot, caused by hyperexcitable stretch reflex responses (spasticity). The authors also observed this premature triceps surae activation in patients with normal toe elevation during the swing phase. This suggested that triceps surae stretch, produced by the dorsiflexing foot during the swing phase could also generate abnormal bursts of triceps surae EMG activity. Functionally, this may affect negatively the clearance of the advancing foot in spite of normal or nearly normal tibialis anterior EMG.

Type II. This pattern was characterized by a low or absent activation (possibly due to muscle weakness) in both the triceps surae and tibialis anterior muscles (9 patients). Patients had a flatfoot reception (4 cases), toe reception (2 cases) or an impaired toe elevation (2 cases). Some triceps surae activation bursts were seen as the body-weight incremented in stance. This probably was caused by the sudden stretch of the triceps surae from body-weight loading.

Type III. Abnormal co-activation, but no significant lowering in the degree of EMG activity characterized this pattern (4 patients). In contrast, the level of activity was greater than normal in some cases. Reciprocal muscle activation was disrupted.

A fourth group of patients could not be included in the previous Types I-III. This was characterized by complex activation patterns where different elements from the other types were mixed in each patient (4 patients). The fact that there were a considerable number of cases that could not fit in any of the three defined groups revealed the multidimensionality of the underlying causes of hemiplegia generating complex, mixed forms which are difficult to classify.

Another study investigating interplay of EMG activation patterns of the ankle antagonists in stroke survivors was recently reported by Burridge et al. (2001). The authors analyzed muscle activation patterns in 15 patients (walking aids permitted) and 12 healthy control subjects while walking on a treadmill. Each subject was allowed to walk at a speed that was comfortable, but confined to a range between 1-2 km/h. This apparently implied that patients with slower gait speed needed to force themselves to walk faster and possibly generating patterns that were different than their usual pace. Also, control subjects needed to restrain themselves in order to accommodate the required experimental gait pace, introducing potential changes to their normal activation patterns. The authors argued that treadmill walking would enforce a more rhythmic gait on participants than they may have achieved during free walking. This assumption, however, was not substantiated objectively and they acknowledged that patterns of activation might differ from that of ground walking. The author's objective was to further develop the earlier clinical classification proposed by Knutsson and Richards (1979) by defining indices to describe patterns of triceps surae and tibialis anterior muscle

activation in patients and healthy subjects. The indices were: 1) premature calf activation index (PCAI) obtained in early stance; 2) tibialis anterior activation index (TAAI) obtained during swing; and 3) push-off index (POI) obtained at push-off. The results showed that the PCAI index was significantly higher in the patients than in the controls. It was concluded that premature triceps surae activity during the first 20% of stance phase was more likely due to a stretch response during the passive ankle dorsiflexion occurring when both, heel and toe were on the ground. The TAAI index did not distinguish controls from the patients. Although the TAAI was useful to identify subjects who were unable to activate their tibialis anterior muscles during swing, it cannot be assumed that a normal TAAI was indicative of a normal profile of activity. The POI index was always greater in the controls than in the patients; however, it was not sensitive to distinguish differences in triceps surae activation levels in the patients. In conclusion, statistically significant differences were found between controls and patients in patterns of triceps surae activity. But the indices did not detect a significant difference in tibialis anterior activity despite clearly different profiles (especially the lack of an activity burst during heel-contact). In addition the study highlighted variation between patients, and the observation that in many cases inappropriate triceps surae activity may have contributed to the drop-foot as much as, if not more than, an inability to activate the tibialis anterior muscle.

Yang, Fung, et al. (1991) investigated soleus H-reflex amplitude and its relation with soleus and tibialis anterior EMG activity in 21 patients with hemiplegia and a healthy subject. All patients and the healthy subject were tested while walking on a

treadmill. Their results showed that in the patients the soleus H-reflex pattern was in general less variable than that of the healthy subject. The authors organized patient's results in three groups by their spasticity level and correlated them with the soleus H-reflex and soleus/tibialis anterior EMG. A group of five patients with mild spasticity level showed a reciprocal pattern for soleus and tibialis anterior muscles, although large repetitive bursts of the soleus during early stance was commonly seen. Increasing the walking speed could enhance these stretch related bursts. The early tibialis anterior burst at heel-contact was reduced or absent, as ground contact was made with either the forefoot or with the foot flat. The soleus H-reflex was low in early stance and rose progressively to its peak during the push-off phase. Another group of 10 patients with moderate spasticity showed a soleus and tibialis anterior EMG activity that remained largely reciprocal. Stretch-induced responses were visible in soleus muscle during stance. The soleus H-reflex was elevated throughout the stance phase, with a slight inhibition during the swing phase. A third group of six patients who were severely spastic showed sharp oscillating bursts in soleus muscle throughout the stance phase. The tibialis anterior activity was very low in stance and swing phases. The soleus H-reflex amplitude varied minimally or was absent throughout the gait cycle. Stretch-induced responses (clonus) were common in the stance phase in the majority of patients. These were observed during early stance. The magnitude of the abnormal stretch response appeared to be associated with the severity of spasticity. Reciprocity between EMG activity of the soleus and tibialis anterior muscles apparently was conserved in mild and moderately spastic

patients. The soleus H-reflex amplitude did not vary (lacked inhibition) especially in those patients who presented with a high level of spasticity. However, the lack of inhibition over the soleus H-reflex amplitude did not interfere with walking ability, since all studied patients were ambulatory.

Yanagisawa, Tanaka, and Ito (1976) reported their findings in elucidating the reciprocal Ia inhibitory connection in ankle antagonists in cases of capsular hemiplegia. They observed an unbalanced reciprocal innervation that was presumed to be one of the essential neuronal mechanisms underlying this neuromotor disorder. They studied 11 patients with spastic hemiplegia, age range 36 - 72 years. Illness onset ranged from 3 months to 15 years. Inhibition of ankle flexor (dorsiflexor) motoneurones from afferents arising from ankle extensor (plantarflexor) muscles was large, whereas Ia inhibition in the opposite direction (from dorsiflexor afferents to extensor motoneurones) was minimal. They suggested that excessive Ia inhibition of flexor motoneurones might be one of the factors contributing to loss of function in the ankle flexors. Evidence supporting this was provided by the observation that alcohol injections at the motor points of extensor muscles not only relieved spasticity but also improved strength in the ankle flexor muscles. Knutsson (1970) described the effects of cooling as having a similar mechanism to depress stretch reflex and to increase the power of the antagonists. Yanagisawa and Tanaka (1978) in a follow-up study further investigated the previous work of Yanagisawa et al. (1976) where they reported similar conclusions. In neither study, however, were healthy controls included under the same experimental conditions.

Thus, it is uncertain to what extent the abnormalities described were specific for hemiplegia.

The work of Burridge and McLellan (2000) provided additional support for the hypothesis of reciprocal Ia inhibition to soleus from an activated tibialis anterior muscle. They stimulated the peroneal nerve of 18 patients with drop-foot. As a result, it was reported that patients who had poor control of ankle movement and spasticity demonstrated by stretch reflex and co-activation, were more likely to respond well to stimulation (inducing Ia reciprocal inhibition to extensors). Those with mechanical resistance to passive movement and with normal activation responded less well. This latter point may suggest that in addition to some control over tibialis anterior muscle to induce Ia inhibition to extensors, actual movement at the ankle joint needs to take place to enhance it.

Leonard et al. (1998) reported additional work in soleus H-reflex in four patients with CVA compared to twelve healthy subjects. These authors investigated changes in soleus H-reflex amplitude in response to voluntary ankle dorsiflexion and dorsiflexion induced by perturbation (sudden forward movement of the standing surface) while standing. The results showed that soleus H-reflex was inhibited during voluntary dorsiflexion and that it was independent of afferent feedback from the contracting tibialis anterior muscle (inhibition occurred before tibialis anterior activation) reflecting the supraspinal component of reciprocal inhibition (presynaptic inhibition). During the postural perturbation in the healthy subjects, however, soleus H-reflex suppression did

not occur prior to tibialis anterior activation. Inhibition was observed only after the onset of tibialis anterior EMG activity. Hence, reciprocal inhibition of soleus motoneurones might be more dependent on afferent feedback from the contracting tibialis anterior muscle or other sources. The patients were greatly limited in their ability to activate voluntarily the tibialis anterior while standing as reflected in EMG recordings. But, during the automatic perturbations, patients showed considerable tibialis anterior EMG activity. However, even in the presence of tibialis anterior activity, patients did not exhibit soleus H-reflex inhibition. The authors suggested that in light of the results, intact corticofugal projections appeared to be necessary for control of reciprocal inhibition during automatic postural reactions.

Crone, Nielsen, Petersen, Ballegaard, and Hultborn (1994) also investigated reciprocal inhibition of ankle extensors (plantarflexors). They studied the effect of electrical stimulation of the common peroneal nerve in 74 healthy subjects and 39 patients with spasticity in the lower extremities due to multiple sclerosis. Upon stimulation soleus H-reflex was depressed in the healthy group and as it was reported in an earlier work (Crone, Hultborn, Jespersen, & Nielsen, 1987) the most probable cause was disynaptic (reciprocal) Ia inhibition. Such soleus H-reflex inhibition was only observed in four patients. These four patients did not differ from the other patients regarding the degree of spasticity or any other clinical parameter, except that they used an external peroneal nerve stimulator daily as a walking aid. It was suggested that the lack of reciprocal inhibition reflected a deficient control of the interneurones, which mediate this

inhibitory spinal mechanism between antagonistic muscles in man. This might contribute to the pathophysiology of spasticity and it might be related to the frequent occurrence of co-contraction of functionally antagonistic muscles during gait in spastic patients. Thus, the existence of a pronounced reciprocal inhibition in patients receiving frequent stimulation of the peroneal nerve may suggest that regular activation of peripheral nerves is of importance for the maintenance of the activity in spinal pathways.

In a more recent work Morita et al. (2001) also investigated reciprocal Ia inhibition of ankle extensors in healthy controls and spastic patients due to multiple sclerosis. As it was in the work of Crone et al. (1994) these authors suggested that the abnormal regulation of disynaptic reciprocal Ia inhibition and presynaptic inhibition in patients with spasticity (from multiple sclerosis) was responsible for the abnormal changes in the stretch reflexes in relation to voluntary movement in these patients. Lack of an increase in reciprocal inhibition at the onset of dorsiflexion may be responsible for the tendency to elicitation of unwanted stretch reflex activity and co-contraction of antagonistic muscles in patients with spasticity. The authors further suggested that therapy should be directed at improving reciprocal inhibition as a means to relieve some of the functional disability of patients with spasticity. The authors, however, did not mention how this improvement (of reciprocal inhibition) could be achieved. These suggestions are yet to be investigated in stroke survivors.

Okuma and Lee (1996) investigated reciprocal inhibition in 16 patients with hemiplegia and 26 healthy subjects. Associations were made between recovery of

function and improvement in spasticity level with changes in disynaptic reciprocal Ia inhibition from ankle flexors to extensor motoneurones. The results showed that excitability of Ia inhibitory system undergoes a number of changes during the recovery period following CVA. It was concluded that in patients who had good functional recovery there was increased Ia inhibition from ankle dorsiflexors to extensors (plantarflexor) motoneurones. This might contribute to the recovery by serving as a compensatory mechanism for the reduced descending motor commands to flexor Ia interneurones. In patients with poor recovery and marked spasticity in the soleus this compensatory mechanism would not be effective. In contrast, Ia inhibition from extensors to flexor motoneurones showed a reverse trend with greater Ia inhibition being associated with poor recovery and marked extensor spasticity. This overriding Ia inhibition would suppress the activity of both flexor (tibialis anterior) α-motoneurones and the corresponding Ia interneurones (inhibitory). As a result there would be persisting weakness of the tibialis anterior and reduced reciprocal inhibition of the soleus muscle. Further, the authors suggested that what may account for the increased Ia inhibition from the flexor afferents to the extensor motoneurones in the patients with good recovery is the "intensive physical therapy and repeated attempts to produce ankle dorsiflexion movements" (one of the most difficult tasks for patients). The authors did not mention, however, what types of specific activity were included when they stated intensive physical therapy and repeated attempts to produce dorsiflexion movements. Probably, they were referring to stepping and gait activities, which would involve the alternating

use of open and closed chain activity in the affected limb. This could over a long term, modify synaptic transmission in the Ia inhibitory pathway to the antagonist extensors because Ia inhibition to the extensors becomes active during voluntary ankle dorsiflexion. This concept appears to be related to the improvement in tibialis anterior activation along with reduced soleus stretch reflex responses obtained recently in weight supported treadmill-walking training in hemiparetic subjects (Hesse et al. 1999). The effect of repetitive dorsiflexion in the recovery of Ia inhibition to ankle extensors is, however, yet to be investigated experimentally.

Okuma and Lee (1996) summarized their conclusions as follows: In control subjects Ia inhibition from tibialis anterior afferents to soleus motoneurones was weak, whereas inhibition to tibialis anterior was profound. In the patients who had poor recovery and marked spasticity, the asymmetry of Ia inhibitory effects between ankle dorsiflexors and extensors was comparable to that of healthy subjects. Further, these authors suggested the presence of a release mechanism that would indicate a reduction of descending tonic inhibitory influences, which are present under normal conditions. The powerful Ia inhibition from extensor afferents to flexor systems may cancel a large part of these release effects on the flexors. Thus the tibialis anterior motoneurones cannot become sufficiently excitable to compensate for the severe loss of descending motor drive. As a result, ankle dorsiflexion remained weak.

Summary of Studies Investigating H-Reflex and EMG of the Ankle Antagonists in Patients with Hemiplegia

The coupling or interplay between antagonist muscles of the distal leg is disrupted following UMN lesions, such as CVA. The soleus muscle shows inappropriate activity early in the stance phase and cyclic abnormal bursts are common (clonus). The tibialis anterior normal burst of activity during heel-contact is diminished or absent in the patients.

Inappropriate, unwanted co-contraction is commonly seen in the ankle antagonists early in the stance and swing phases. Although present, EMG activation reciprocity between ankle antagonists is highly variable between patients, and may be correlated to the severity of brain lesions.

UMN syndromes such as those resulting from CVA lesions may produce a release of the spinal reflexes from inhibition by the higher levels of the brain. These cause elevated excitability in flexors and extensors muscle groups. In the lower extremity, this is much more marked in extensor muscle groups and extensor spasticity becomes a dominant sign clinically. Any release effect on the flexor system is largely canceled by the high activity of the reciprocal Ia inhibitory pathway from extensors and only a fragment of it is occasionally revealed in some patients as weak Ia inhibition of the triceps surae.

Normally, the soleus H-reflex amplitude varies considerably during the course of walking. In patients, it varies from being relatively normal (i.e., facilitation during stance

and inhibition during swing) to a complete absence of inhibition during swing in others. This may be associated with the severity of spasticity and may indicate a failure to facilitate transmission in the disynaptic reciprocal inhibitory pathway.

The soleus H reflexes in patients are similar in size to those of normal subjects during tonic plantar flexion. This may help to explain why increased muscle tone and reflex hyperexcitability is not seen during agonist soleus contraction in patients with spasticity. It is therefore only in the resting state and during antagonist contraction (stretch) that there is evidence of reflex hyperexcitability and increased muscle tone.

For patients, tibialis anterior contraction alone, either voluntary or as a result of automatic perturbations, is not sufficient to cause reciprocal inhibition over the soleus muscle, suggesting the lack of influence from other inhibitory sources (e.g., presynaptic).

Patients with UMN syndromes are heterogeneous. This may be due in part to the complexity of the neural elements at the site of brain damage. And, to the fact that this not only involves motor pathways but also non-motor domains such as memory, cognition, and perception.

Although unclear, some suggestions indicate that reinforcement of dorsiflexion, that is, the alternating play between open and closed chain activity of the involved limb may, in the long term, improve the volitional control of dorsiflexion. This in turn would enhance the reciprocal inhibition to the soleus allowing a more normal motor control or interplay.

CHAPTER III

METHODS

In this Investigation four experiments were undertaken. Since the four experiments had some common grounds in their experimental procedures, the Methods were organized into two sections: Parts A and B. Part A describes general aspects of the Methods, and Part B describes the design, subjects, setup and procedures, with data and statistical analyses that were specific for each one of the experiments.

PART A

Participants and Inclusion/Exclusion Criteria - General

Healthy Participants

Participants were excluded if they reported any history of stroke, cancer, diabetes, low back pain, or neurologic or orthopedic pathologies.

Patients

Patients were stroke survivors who were independent ambulators (with/without gait devices) and able to walk at least 10 meters without stopping. Patients were not included if they were not independent ambulators and required physical support or assistance from a helper to walk. Patients were examined by their primary physicians

within six months prior to participating, and had been cleared from having unstable blood pressure, unstable heart disease, diabetes (risk of neuropathy), or any orthopedic pathologies. Participating patients were not taking prescription medications to reduce spasticity. No priority or bias based on sex, national origin, or institutional affiliation was used in selecting appropriate participants.

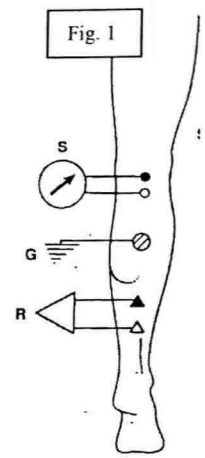
Informed Consent Form - General

All selected participants signed an informed consent form, approved by the Human Subjects Review Committee from Texas Woman's University and Baylor College of Medicine.

Instrumentation - General

H-Reflex Evoking and Recording

The H-reflex of the soleus muscle was evoked and recorded according to the methods of Sabbahi and Khalil (1990) using the Cadwell 5200A[1] electromyograph. Figure 1 shows the location of the electrodes on the lower limb (S = stimulating; G = ground; R = recording).

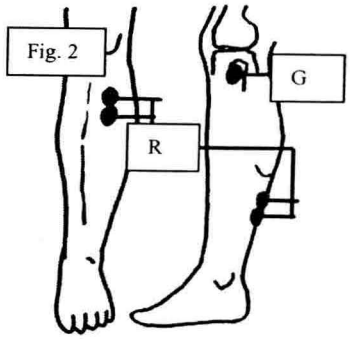


Electromyography

Soleus and tibialis anterior muscles were recorded and analyzed off-line using the Myosystem 1200[2] and its Norquest EMG

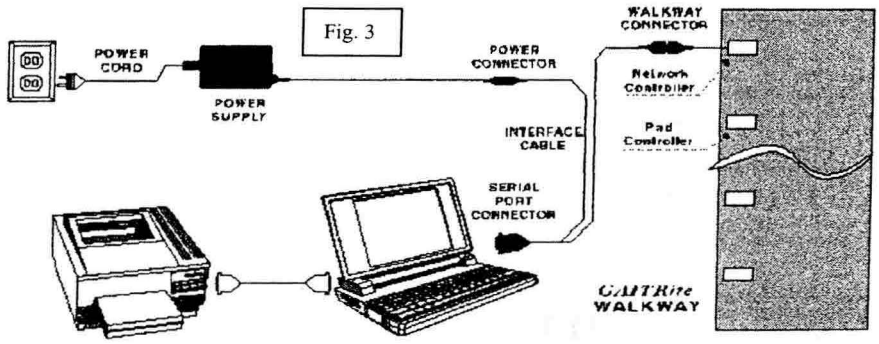
[1] Cadwell, Inc., USA
[2] Noraxon, Inc., USA

evaluation/analysis software. An analog/digital KPCMCIA-12AI-C[3] card was used to link the Myosystem to a Lifebook[4] C340-233 Mhz laptop computer. Figure 2 shows the location of the electrodes on the lower limb (R = recording; G = ground).



Gait Measurements

The GAITRite walkway and its application software were utilized to calculate gait speed (Figure 3). The application software loaded into a computer, controlled the functionality of the walkway, processed the raw data and automatically computed temporal and spatial parameters. In this study a walkway containing eight sensor pads was used. The sensors are encapsulated in a roll-up carpet to produce an active area 61 cm (24 inches) wide and approximately 488 cm (192 inches) long. The walkway is portable, can be laid over any flat surface, requires minimum setup and test time, and does not require the placement of any devices on the subject/patient.



[3] Keithley Instruments, Inc., USA

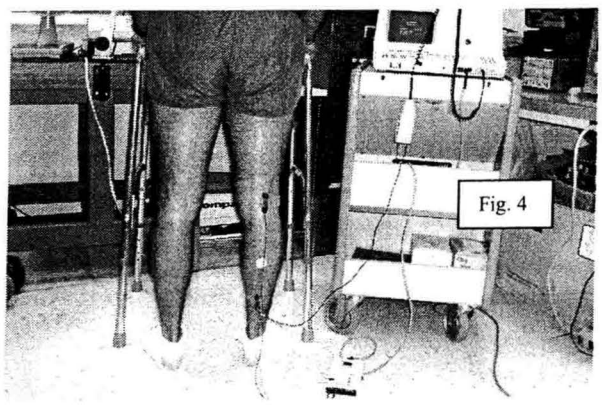
Clinical Indexes and Scales

Barthel Activities of Daily Living (ADL) Index

All patients were interviewed to complete the Barthel ADL Index (Appendix A). In some cases, some help to provide the required information was obtained from family members and caregivers. The Barthel ADL Index (Mahoney & Barthel, 1965) has been reported to provide a valid and reliable estimate of disability of patients with CVA (Collin, Wade, Davies, & Horne, 1988; Wade & Collin, 1988).

Modified Asworth Scale

In order to provide a quantifiable level of spasticity, the modified Ashworth Scale (Appendix B) was used to grade the level of spasticity at the knee and ankle joints in all patients. This scale has been reported to provide reliable measures of spasticity (Bohannon & Smith, 1987).



General Testing Setup

H-Reflex Amplitude

Maximum soleus H reflexes waves were elicited using a CADWELL 5200-A EMG unit. The tibial nerve was stimulated at the popliteal fossa with percutaneous electrical stimulus of 0.5 ms squared-wave pulses, with a stimulating silver-

[4] Fujitsu, Inc. Japan.

silver chloride surface electrode bar, at a frequency of 0.2 Hz to elicit maximum H-reflex amplitudes with minimal M response (see Figure 4). The recording surface bar electrodes (center-to-center distance between the two electrodes was 25 mm., active lead-off surface of electrodes was 1 cm2 each) were placed over the soleus, 3 cm distal the gastrocnemii/Achilles tendon junction. A metal ground electrode 2 cm in diameter was applied on the skin between the stimulating and recording electrodes. Once the maximum H-reflex amplitude and minimal M response were determined with the subjects in the standing position, the minimal M response was kept constant throughout the eliciting/recording procedures for all the gait-simulating postures. The soleus H-reflex signal was amplified (1000-5000X) using a band pass of 10 Hz-10 KHz.

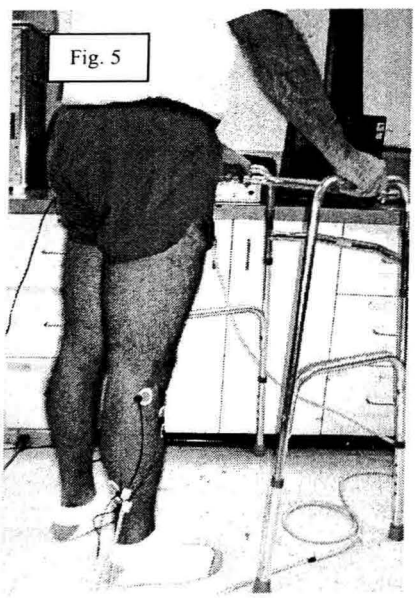


Fig. 5

Electromyography

Soleus and tibialis anterior surface EMG was obtained using the Myosystem 1200. Two round surface electrodes[5] were placed side-by-side, over the tibialis anterior muscle belly, and over the soleus 1 cm below the bifurcation of the gastrocnemii and on line with the Achilles tendon. A single ground electrode was placed on the skin covering the head of the fibula (Figure 5). The raw EMG signal was sampled at 1000 Hz, rectified and smoothed with a 30 ms averaging window for online monitoring and storage. Stored

[5] Blue Sensor (M-00-S), Medicotest, Inc. Denmark.

data was quantified using an automatic root mean square (RMS, expressed in (μV) algorithm with a 50 ms averaging window. Surface electrode EMG recordings have been reported to have good reliability for within session measurements and satisfactory reliability for between sessions (Giroux & Lamontagne, 1990; Komi & Buskirk, 1970).

Gait

Patients were required to walk 10 meters (approximately 30 feet) at their maximal speed (fast-pace) and at their natural pace (self-pace). Each condition was repeated twice. Of the 10 meters, approximately five meters were covered by the instrumented GAITRite walkway. The walkway was placed centrally, so that the effects of acceleration and de-acceleration on gait speed would be minimized. Stand-by assistance from a helper was provided during the walking to assure balance and safety. As the patient ambulated along the walkway, the system captured the pressure of each footfall as a function of time (Figure 6). Data recorded was stored into the linked computer by the application software for later analyses. Gait testing was carried out at the beginning and at the end of a training period. It has been reported that

Fig. 6

the GAITRite System[6] is a valid and reliable tool for measuring selected spatial and temporal parameters of gait (McDonough, Batavia, Chen, Kwon, & Ziai, 2001). Gait speed has been reported as a good indicator of recovery in hemiplegic patients (Brandstater et al., 1983; Friedman, 1990; Goldie et al., 1996; Richards et al., 1995), with good reliability (Hill et al., 1994; Wade et al., 1987).

Testing Procedures - General

Gait-Simulating Postures

H reflexes and EMG were obtained from the involved lower limb in the patients and a from a randomly chosen lower limb in the healthy subjects in the following five gait-simulating postures:

- (N) neutral: freestanding posture (weight evenly distributed).
- (PO) push-off: standing on toes (both legs), mimics push-off during late stance phase of gait.
- (HC) heel-contact: standing on heels (both legs), mimics transition between swing and stance phases of gait (heel-contact).
- (SW) swing: foot just cleared from ground, mimics toe-off and beginning of swing phase.

[6] CIR Systems, Inc. USA.

- (MS) mid-stance: standing on tested leg, while contralateral foot is lifted as in the SW posture. Mimics mid-stance phase of gait

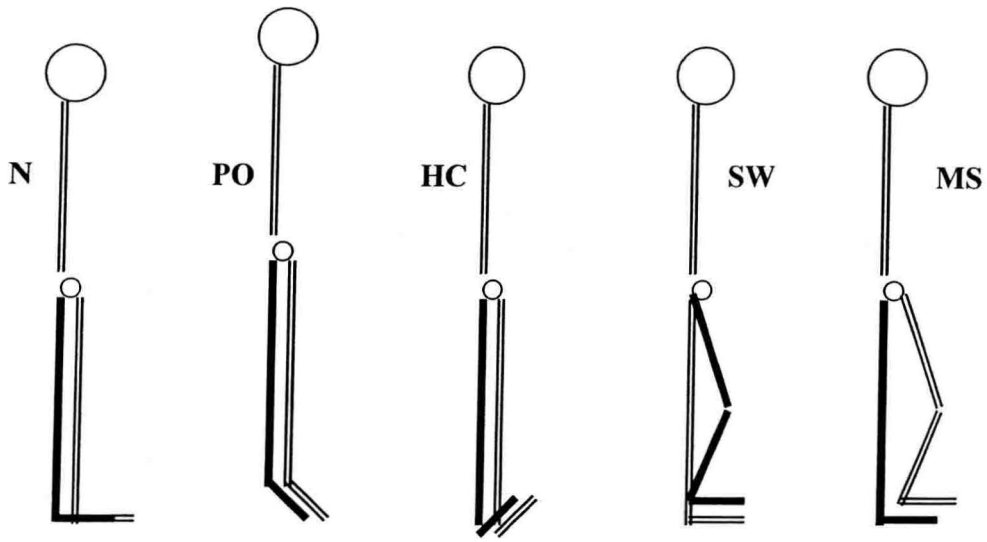


Figure 7. Gait-simulating postures. N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance. The solid black lines represent the tested limb.

The above-defined postures and their schematic drawings (Figure 7) are described in reference to the tested limb, which is represented as solid black lines on the drawings. Subjects adopted all postures standing in front of an adjustable standard walker (healthy subjects) or platform walker (patients) to provide postural stability. All subjects were required to maintain their heads facing straightforward during testing/recording. Practice trials were performed in all postures before actual testing. Data collection was initiated once subjects learned the postures.

Sequence of Testing

Soleus H-reflex and soleus/tibialis anterior EMG were recorded in the following sequence: N, PO, HC, SW, and MS. First, participants were tested for H-reflex, then for EMG.

Skin Preparation and Electrodes Placement

The skin covering the soleus/tibialis anterior and the popliteal fossa were lightly sanded with fine grade sandpaper and cleaned with alcohol. For H-reflex testing, electrodes were moistened with conductive gel and secured to the skin with surgical tape.

PART B

Experiment 1

The purpose of this experiment was to assess the test-retest reliability (intrasession and intersession) of the soleus H-reflex amplitude in five gait-simulating postures (N: freestanding, PO: push-off, HC: heel-contact, SW: swing, and MS: mid-stance) over two separate sessions (days), from two population samples: healthy subjects and patients with CVA.

Participants

Ten healthy subjects (3 women, 7 men), age range 33 to 73 years (40.2 ± 12.6); and, nine patients with CVA (5 women, 4 men), age range 44 to 73 years (60.1 ± 8.3) participated.

Design

Test-retest of the soleus H-reflex amplitude recorded during five gait-simulating postures. All recordings were repeated within one week.

Data and Statistical Analyses

Three consecutive, soleus H-reflex amplitudes were measured and averaged for each posture in each of the two testing occasions. Two H-reflex parameters were recorded: the peak-to-peak amplitude and the onset latency to the first deflection. The H reflexes amplitudes and latencies were measured manually in millimeters from paper printouts and converted to mV equivalents (amplitude) and to ms equivalents (latencies). Data were screened for skewness and outliers by frequencies and histograms.

Multiple Pearson product-moment correlation coefficients (r) were calculated for each posture between the two testing occasions and between postures within each occasion. Alpha was set at .05. All calculations and statistical analyses were completed using Microsoft Excel[7] spreadsheet and the SPSS[8] statistical software package, version 10.0.

Experiment 2

The main purpose of this experiment was to investigate in a group of healthy subjects the soleus H-reflex amplitude and EMG activity of the ankle antagonists during five gait-simulating postures N: freestanding, PO: push-off, HC: heel-contact, SW: swing

[7] Microsoft, Inc., USA

and MS: mid-stance). It was hypothesized that the soleus H-reflex and EMG activity of the ankle antagonists would modulate throughout the gait-simulating postures in a manner similar to that observed during walking.

Participants

Twenty-one healthy subjects (9 women, 12 men), age range 22 to 69 years (36.7 $\pm$ 12.6) participated.

Design

A one-way repeated measures analysis of variance (ANOVA) design (univariate approach) was used to assess differences among postures for soleus H-reflex amplitude. Max M-waves and latencies of the H-reflex were also recorded and measured. Additionally, in order to assess their relationship to the soleus H-reflex amplitude, surface EMG activity was recorded and processed from the soleus and tibialis anterior muscles in the same five gait-simulating postures.

Data and Statistical Analyses

H-Reflex

Three parameters were recorded: Max H-wave peak-to-peak amplitude, Max M-wave peak-to-peak amplitude, and the onset latency to the first deflection. The soleus H-reflex and M amplitudes were measured manually in millimeters from paper printouts and converted to mV equivalents. Soleus H-reflex latencies were expressed in ms. Three consecutive H-reflex (and their latencies) and M-waves amplitudes were measured and

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[8] SPSS, Inc., USA

averaged for each posture. H-reflex and M-wave averaged amplitudes were processed to obtain H/M ratios. To examine the relationship between the H/M ratios and the H-reflex amplitude, a Pearson product-moment correlation coefficient (r) was calculated. Data was screened for skewness and outliers by frequencies and histograms. A one-way repeated measures ANOVA (univariate approach) with Greenhouse-Geisser Epsilon adjustment (correction of the F test) was used to assess the means of soleus H-reflex amplitude across the postures. The univariate approach is recommended for designs with small sample sizes and when the sphericity assumption is not met (Maxwell & Delaney, 1990). Alpha was set at .05, with Bonferroni adjustments and Post-Hoc follow-up where appropriate.

Electromyography

Two 3-second soleus and tibialis anterior EMG signals were recorded and stored for each posture. Later, the signals were rectified and quantified using a root mean square (RMS, expressed in μV) algorithm with a 50 ms window and the results averaged for each posture.

All calculations, graphs and figures were completed using Microsoft Excel spreadsheet and the SPSS statistical software package, version 10.0.

Experiment 3

The main purpose of this experiment was to investigate in a group of patients with CVA the soleus H-reflex amplitude and EMG activity of the ankle antagonists during five gait-simulating postures (N: freestanding, PO: push-off, HC: heel-contact, SW: swing and MS: mid-stance). Results were analyzed and compared to that of healthy subjects who were assessed by the same testing paradigm. It was hypothesized that the patterns of soleus H-reflex amplitude and associated EMG activity of the ankle antagonists recorded from the gait-simulating postures would be different between the two groups.

Participants

Twenty-one healthy subjects (9 women, 12 men), age range 22 to 69 years (36.7 $\pm$ 12.6); and, nine patients with CVA (5 women, 4 men), age range 44 to 73 years (60.1 $\pm$ 8.3) participated.

Design

A split-plot design (univariate approach) with a within-subject factor (posture, with five levels) and a between-subject factor (group, with two levels) was used to assess main effects and the interaction. The independent variable was the soleus H-reflex amplitude. Additionally, to assess its relationship to the soleus H-reflex amplitude, surface EMG activity was recorded and processed from the soleus and tibialis anterior muscles in the same five gait-simulating postures.

Data and Statistical Analyses

Soleus H-reflex parameters

Two parameters were recorded, the H-reflex peak-to-peak amplitude and the onset latency to the first deflection. Three consecutive, H-reflex amplitudes and their latencies were measured and averaged for each posture. The H-reflex amplitudes and latencies were measured manually in millimeters from paper printouts and converted to mV and ms respectively. Data was screened for skewness and outliers by frequencies and histograms. A split-plot design (univariate) with Greenhouse-Geisser Epsilon adjustment (correction of the F test) was used to assess the soleus H-reflex means across postures. The univariate approach is recommended for designs with small sample sizes and when the sphericity assumption is not met (Maxwell & Delaney, 1990). Alpha was set at .05, with Bonferroni adjustments and Post-Hoc follow-up where appropriate.

Electromyography

Two 3-second soleus and tibialis anterior EMG signals were recorded and stored for each posture. Later, the signals were rectified and quantified using a root mean square (RMS - expressed in μV) algorithm with a 50 ms window and the results averaged for each posture.

All calculations, graphs and figures were completed using Microsoft Excel spreadsheet and the SPSS statistical software package, version 10.0.

Experiment 4

The main purpose of this experiment was to observe the individual and group responses to a 4-week exercise intervention directed to improve ankle motor control. It was hypothesized that the soleus H-reflex amplitude would be positively affected (i.e., recovered or normalized) by an exercise program that consisted of repeated ankle dorsiflexion in standing (simulating initiation of swing phase) aided with EMG biofeedback from the tibialis anterior muscle. The recovery of the soleus H-reflex inhibition during swing would be associated with recovery in motor control of the tibialis anterior (improved EMG activity) and reflected by improvements in gait speed.

Participants

Nine patients with CVA (5 women, 4 men) with an age range 44 to 73 years (60.1 $\pm$ 8.3) participated. And, data from 17 healthy subjects (6 women, 11 men), age range 22 to 69 years (37.5 $\pm$ 14.4) from a related study were used for comparisons.

Design

An AB (A: baseline, B: intervention) single-case design with repeated measures format was used to assess patient's individual soleus H-reflex amplitude over time. Soleus H-reflex was recorded before, throughout, and after a 4-week training period. Soleus H-reflex amplitude was recorded from five gait-simulating postures: N: freestanding, PO: push-off, HC: heel-contact, SW: swing and MS: mid-stance.

Tibialis anterior EMG activity was recorded before and after the training period from two postures: heel-contact (HC) and swing (SW). A previous related study showed

that these two postures accounted for most of the tibialis anterior EMG activity during the step cycle. Self- and fast-paced gait speed was also recorded before and after the training period. Pre- and post-training comparisons (individual and group) were made for soleus H-reflex, gait speed and tibialis anterior EMG activity

Exercise Training and EMG Feedback Protocol Setup and Procedures

After placement of surface electrodes over the tibialis anterior muscle, patients were asked to dorsiflex maximally the involved foot while in the swing posture. A self-directed target of tibialis anterior EMG activity was simultaneously displayed on a monitor screen at eye level to provide feedback to the patient. A pre-set EMG-feedback program[9] with 10 contract-up repetitions lasting approximately 5 seconds each was used to exercise the tibialis anterior muscle. Ten repetitions made 1 set. Patients performed 3 sets in each training session for a total of 30 contractions and movements. Patients were also asked to perform two more sets of 10 repetitions for a total of 20 contractions and movements while sitting. Resting time (1-2 min) was provided between sets. Each patient attended training for 3 sessions/week for 4 weeks.

Data and Statistical Analyses

Soleus H-Reflex

Three consecutive, soleus H-reflex peak-to-peak amplitudes were measured and averaged for each posture. The amplitudes were measured manually in millimeters from paper printouts and converted to mV equivalents. Data was screened for skewness and

[9] Myoclinical - Noraxon, Inc., USA

outliers by frequencies and histograms. Individual and group data was graphically displayed for inspection and analysis. Student's paired t tests were used to compare group pre- and post-training soleus H-reflex amplitude means across postures. Alpha was set at .05.

Electromyography

Two 3-second tibialis anterior EMG signals were recorded and stored for the heel-contact and swing postures. Later, the signals were processed using a root mean square (RMS - expressed in μV) algorithm with a 50 ms window and results averaged for each of the two postures. Individual and group data was graphically displayed for inspection and analysis. Student's paired t tests were used to compare group pre- and post-training tibialis anterior EMG. Alpha was set at .05.

Gait

Stored gait data was retrieved by the GAITRite applications software. The software provided (after processing recorded footprints downloaded from the walkway) automatic calculation and display of gait speed values (cm/sec) for each trial. Gait speed results from two trials (2 trials before and 2 trials after the training) were averaged for the self- and fast-paced gait conditions. Individual and group data was graphically displayed for inspection and analysis. Student's paired t tests were used to compare group pre- and post-training gait speed averages. Alpha was set at .05.

CHAPTER IV

RESULTS

The results were reported in the form of manuscripts for each one of the experiments outlined in Chapter III - Methods.

Experiment 1

Test-Retest Reliability of the Soleus H-Reflex in Five Gait-Simulating Postures in Healthy Subjects and in Patients with Stroke

Introduction

The Hoffmann reflex (H-reflex), especially from the soleus muscle, has been used as a diagnostic tool for neurological disorders, developmental deficits and in detection of nerve root lesions (Braddon & Johnson, 1974; Fisher, 1992). It has also been used to study normal soleus motoneurone pool excitability (Capaday & Stein, 1986; Crenna & Frigo, 1987; Morin, Katz, Mazieres, & Pierrot-Deseilligny, 1982). Some researchers have also tested soleus H-reflex in standing and walking to gain insight into the known disrupted motoneurone interplay found in upper motor neurone (UMN) syndromes such as cerebrovascular accident or CVA (Leonard, Diedrich, Matsumoto, Moritani, & McMillan, 1998; Okuma & Lee, 1996; Yanagisawa, Tanaka, & Ito, 1976; Yang, Fung,

Edamura, Blunt, Stein, & Barbeau, 1991). Williams, Sullivan, Seaborne, and Morelli (1992) reported high inter- and intra-individual reliability of soleus H-reflex recordings (with coefficients r above 0.86) with as few as four trials measured in the prone position.

Hopkins, Ingersoll, Cordova, and Edwards (2000) reported high reliability (ICC[3,1] - from 5 trials or recordings) values of soleus H-reflex when recorded in supine and in one-leg standing. In supine, intersession reliability was 0.94 and intrasession was 0.93. In standing, intersession reliability was 0.80 and intrasession was 0.86.

Ali and Sabbahi (2001) reported test-retest (intersession) and within-subject reliability (intrasession) of the soleus H-reflex amplitude (averages from 4 trials or recordings) of both lower extremities evaluated in three different positions: pronelying, freestanding and standing while subjects (8 women and 7 men, 20 -50 years) were holding 20% of their respective body weights. Intersession reliability of soleus H-reflex in the left leg ranged from $r = 0.19$ in prone position to $r = 0.46$ in the loaded standing position. For the right leg it ranged from $r = 0.39$ in prone position to $r = 0.45$ in the loaded standing position. Intrasession reliability for the first session was high between the standing and the loaded standing with $r = 0.94$ and 0.97, for the left and right leg respectively. For the second session, intrasession reliability was high between the standing and the loaded standing position with $r = 0.82$ and 0.96, for the left and right leg respectively. The correlation values decreased between the prone and standing positions with $r = 0.77$, 0.78 for the first session and with $r = 0.67$, 0.67 for the second session, left and right leg respectively. The correlation values between the prone and loaded standing

positions for the first session were, r = 0.67, 0.78 and for the second session were r = 0.56, 0.60, for the left and right leg respectively. For the H-reflex latency, the intersession correlation coefficients were high for all three positions: the correlation values for the left leg were r = 0.94, 0.94, 0.94; and for the right leg were r = 0.92, 0.92, 0.93 for the prone, standing and loaded standing positions respectively. Latency showed high intrasession correlation (from r = 0.96 to 0.99) for both the first and second sessions, between the three positions and for the two legs.

Handcock, Williams, and Sullivan (2001) studied soleus H-reflex and M-wave response reliability in standing healthy subjects (2 women and 18 men, age range 19 - 40 years). Data were collected over two sessions (separated by 2 - 4 days), with each session consisting of 4 blocks of 20 recording trials. Their results showed high intrasession reliability for both parameters (r = 0.97). Intersession reliability was r = 0.87 and r = 0.80 for the H-reflex and M-wave respectively. These high reliability coefficients of the H-reflex and M-wave were maintained even though as few as four recording trials were used in the analysis.

The results of these studies suggested that the H-reflex is a reliable assessment tool for healthy subjects when measured in supine and quiet standing. Of the two, standing probably relates better to a functional activity such as walking. Studies reporting reliability of recording the H-reflex in patients with UMN syndromes such as CVA were not found. The stability of recording soleus H-reflex in standing may be used to measure changes in motoneurone pool excitability over time.

This report attempted to assess the reliability of a testing procedure using the H-reflex amplitude that may be useful to evaluate patients with CVA and other neuromuscular conditions. The testing procedure was also performed in healthy subjects under the same experimental conditions. Therefore, the purpose was to assess the intersession and intrasession reliability of recording the H-reflex amplitude in five gait-simulating postures (N: freestanding; PO: push-off; HC: heel-contact; SW: swing; and MS: mid-stance) over two separate sessions (days), and from two population samples: healthy subjects and patients with CVA.

Material and Methods

Participants

Ten healthy subjects (3 women, 7 men), age range 33 - 73 years (40.2 ± 12.6). And, nine patients with CVA (5 women, 4 men) age range 44 - 73 years (60.1 ± 8.3) participated in this study. The healthy subjects reported having no history of significant low back pain, diabetes, or neurological or orthopedic pathologies. Patients were community dwellers able to walk at least 10 meters without stopping, and reported having no diabetes, low back pain, cancer, or orthopedic pathologies. All patients had visited their physicians within the last six months before participating in the study. None of the patients was taking anti-spasticity medications. Demographic and clinical information of patients are summarized in Table 1.

Instrumentation and H-Reflex Recording

The Cadwell 5200A electromyography unit was used to elicit and record the soleus H-reflex. Initially the skin location of stimulation and recording were slightly abraded with fine sandpaper in order to reduce skin impedance. For electrical stimulation of the tibial nerve silver-silver chloride surface bar electrodes (active lead-off surface of electrodes was 1 cm2 each, center-to-center distance between the 2 electrodes was 25 mm) were placed over the tibial nerve in the popliteal fossa. Stimulation electrodes were positioned with the cathode proximal to the anode to avoid anodal block (Fisher, 1992). Another similar bar recording electrode was placed over the soleus muscle along its mid-line, with the active electrode 3 cm distal to the distal margin of the two heads of the gastrocnemius muscle. A 2 cm diameter metal ground electrode was applied between the stimulating and recording electrodes over the calf muscle group. Electroconductive gel was applied on all electrodes' surfaces for good coupling. Once in place the electrodes were not removed until the whole experiment was completed to ensure exact placement and consistent results. A percutaneous electrical stimulus of 0.5 ms square-wave pulses was delivered at a frequency of 0.2 Hz to the tibial nerve to elicit the maximum H-reflex with minimal M response. Once the maximum H-reflex amplitude and minimal M response were determined with the subjects in the standing position, the minimal M response was kept constant throughout the eliciting/recording procedures for all the gait-simulating postures. Signal was amplified (1000-5000X) using a band pass of 10 Hz-10 KHz.

Experimental Procedure

After signing the consent form, soleus H-reflex amplitude recordings were obtained during each of the following five gait-simulating postures: 1) N: freestanding, 2) PO: push-off (standing on toes, mimics push-off during late stance phase of gait), 3) HC: heel-contact (standing on heels, mimics transition between swing and stance phases of gait), 4) SW: swing (foot just cleared from ground, mimics toe-off and beginning of swing phase), and 5) MS: mid-stance (standing on tested leg, while contralateral foot is lifted as on the SW posture; mimics mid-stance phase of gait). Postures are schematically shown in Figure 8. In healthy subjects, the tested limb was chosen alternating right or left as they arrived to the testing site. Patients were only tested in their affected limb. The sequence of testing was the same as defined above. Subjects adopted all postures standing in front of an adjustable standard walker and were asked to have their hands resting on the walker's handles to ensure balance. For patients, a platform walker was used. All subjects were tested twice (2 separate sessions) within 7-10 days in all five postures.

Data and Statistical Analysis

Three representative H-reflex amplitudes were recorded and averaged for each posture. Two parameters were measured, the H-reflex peak-to-peak amplitude and the onset latency to the first deflection. Data was screened for skewness and outliers by frequencies and histograms. Multiple Pearson product-moment correlation coefficients (r) were calculated between the two sessions (intersession) and between postures

(intrasession). The SPSS 10.0 statistical software was employed for the statistical analyses.

Results

Healthy Participants

Soleus H-Reflex Amplitude (mV)

Three recorded H-reflex amplitudes were used to calculate means and their corresponding standard deviations for each of the five postures. These calculations were done for the two sessions and the results are shown in Table 2.

The intersession Pearson correlation coefficients were high (0.80 or higher) for postures N ($r = 0.88$), PO ($r = 0.82$), SW ($r = 0.95$), MS ($r = 0.82$), and moderate for posture HC ($r = 0.78$) (Table 4).

The intrasession Pearson correlation coefficients between postures in each of the two sessions were: In the first session, the r values ranged from 0.59 to 0.97; and in the second session, the r values ranged from 0.34 to 0.91 (Table 4).

Soleus H-Reflex Latency (mV)

Three recorded H-reflex latencies were used to calculate means and their corresponding standard deviations for each of the five postures. These calculations were done for the two sessions and the results are shown in Table 3.

The intersession Pearson correlation coefficients were high for postures N ($r = 0.97$), PO ($r = 0.97$), HC ($r = 0.86$), SW ($r = 0.97$), and MS ($r = 0.93$) (Table 5).

The intrasession Pearson correlation coefficients between postures in each of the two sessions were: In the first session, r values ranged from 0.84 to 0.97; and in the second session, r values ranged from 0.88 to 0.98 (Table 5).

Patients

Soleus H-Reflex Amplitude (mV)

Three recorded H-reflex amplitudes were used to calculate means and their corresponding standard deviations for each of the five postures. These calculations were done for the two sessions and the results are shown in Table 6.

The intersession Pearson correlation coefficients were high (0.80 or higher) for postures N (r = 0.94), PO (r = 0.92), HC (r = 0.80), MS (r = 0.87), and moderate for posture SW (r = 0.61) (Table 8).

The intrasession Pearson correlation coefficients between postures in each of the two sessions were: In the first session, the r values ranged from 0.78 to 0.98; and in the second session, the r values ranged from 0.47 to 0.97 (Table 8).

Soleus H-Reflex Latency (mV)

Three recorded H-reflex latencies were used to calculate means and their corresponding standard deviations for each of the five postures. These calculations were done for the two sessions and the results are shown in Table 7.

The intersession Pearson correlation coefficients were high for postures N (r = 0.88), PO (r = 0.87), HC (r = 0.96), SW (r = 0.96), and MS (r = 0.93) (Table 9).

The intrasession Pearson correlation coefficients between postures in each of the two sessions were: In the first session, r values ranged from 0.95 to 0.98; and in the second session, r values ranged from 0.88 to 0.99 (Table 9).

Discussion

Soleus H-Reflex Amplitude in Healthy Subjects

The results of this study showed high intersession and intrasession reliability coefficients for the H-reflex amplitude and H-reflex latency in all five postures. The intersession reliability found in this study for the mid-stance posture (MS) in the healthy subjects was similar to that reported by Hopkins et al. (2000), which was from H-reflex recordings obtained in one-leg standing (0.82 and 0.80 respectively). The intersession reliability values found in the present study for the freestanding posture (N) in the healthy subjects was similar to that reported by Handcock et al. (2001), which was from H-reflex recordings obtained in a comparable standing posture (0.88 and 0.87 respectively). Intersession reliability in these gait-simulating postures demonstrated the consistency needed to detect changes in maximum H-reflex over a period of several days. This consistency of the recordings in standing weight-bearing posture, and in agreement with Hopkins et al. (2000) and Handcock et al. (2001), is important to assess changes in motoneurone excitability due to injury or trauma. Ali and Sabbahi (2001) also pointed out that during weight-bearing postures in standing (as opposed to lying supine) the spinal reflexes become more stable, more consistent and with less variability.

In the present study intrasession reliability coefficients were high between some postures and of lower order between others. A possible explanation of this could be that among the postures used to record the H-reflex, some were relatively more relaxed and stable with minimal muscle activation at the ankle such as freestanding (N) and mid-stance (MS), and others were actively sustained such as push-off (PO) and heel-contact (HC). In the latter two postures and in contrast with freestanding and mid-stance, there was a degree of co-contraction of the ankle antagonists since balance was challenged by balancing the body-weight either on heels or toes (small base of support). This possibly involved constant adjustment (increasing variability) in the muscles firing rates, which may have affected the consistency of the soleus H-reflex recordings. Supporting this notion, Funase and Miles (1999) demonstrated that the amplitude of the H-reflex was consistently dependent on the level of background EMG activity. Larger H-reflex amplitudes were exhibited at higher levels of activity and smaller when the tonic activity was low; and that trial-to-trial variability in H-reflex amplitude responses was also dependent on the small changes in the level of activity in the motoneuronal pool that occur from one instant to the next.

Soleus H-Reflex Amplitude in Patients

The results showed high intersession reliability coefficients in four postures for the H-reflex amplitude and in all five postures for the H-reflex latency. The lower order coefficient seen for the swing (SW) posture (0.61) may be explained by the presence of some triggering of stretch-induced discharges in the soleus muscle as a result of the

dorsiflexion movement. With the foot off the ground (open chain) its cutaneous and proprioceptive stimuli have been reduced significantly. Ali and Sabbahi (2000) previously recorded similar results, when comparing lying (open chain) with standing (closed chain) posture. Apparently, such afferent information generated from the skin, joint and muscle proprioceptors during standing helped to stabilize the reflexes inducing less variability. The intrassession reliability coefficients, calculated for each of the two sessions, were similar to those of the healthy subjects, indicating a comparable level of variability.

Soleus H-Reflex Latency in Healthy Subjects and Patients

Reliability of the H-reflex was high in all postures for both, healthy subjects and patients. Reflex latency is a measure of the conduction time of the neural signal along the reflex pathway/arc. This conduction time is not altered by subject position or between different testing sessions. The latency parameter does not change unless axonal conduction is affected as in radiculopathy, demyelination or axonal loss (Braddom & Johnson, 1974). The difference of H-reflex latency between healthy subjects (approximately 31 ms) and patients (approximately 34 ms) although under the normal upper limit of 35 ms, could be explained by the mean age difference (approximately 20 years) between the two groups (Fisher, 1992).

Conclusions

The reliability of testing the H-reflex amplitude in five gait-simulating postures with repeated observations on each of two sessions was investigated in a group of healthy

subjects and in patients with stroke under similar experimental conditions. To our knowledge, this is the first reliability study carried out in patients with CVA that assessed recordings of the soleus H-reflex amplitude elicited in gait-simulating postures. This study demonstrated that, in the studied healthy subjects and patients, the H-reflex can be reliably recorded in weight-bearing gait-simulating postures. The reliability of obtaining repeated measures using this testing paradigm was high, and for clinical practicality, the average of as few as three H-reflex amplitude recordings might be used as an estimate of soleus motoneurone pool excitability.

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Table 1. Demographic and Clinical Data of Participating Stroke Survivors.

Subject	Age (yr)	Sex	Mass (Kg)	Height (m)	TFO (mo)	Paretic side	HD	Speech ability	Sensory	WAid	AFO	ASHs K / A	Clonus	BARi
CC	63	F	81.6	1.65	67	R	R	Cons	Cons	Q-cane	Yes	3 / 4	Yes	19
CS	44	F	63.5	1.68	25	R	R	Imp	Cons	Q-cane	Yes	0 / 0	No	19
HH	62	F	65.8	1.75	20	L	R	Cons	Cons	None	Yes	0 / 1	Yes	20
WB	64	F	78.9	1.63	69	L	R	Imp	Cons	None	Yes	1 / 2	Yes	20
BL	66	M	77.1	1.70	180	R	R	Cons	Cons	Cane	No	3 / 4	No	16
BT	56	M	74.8	1.75	46	R	R	Imp	Imp	None	No	0 / 1	Yes	20
EF	53	M	80.3	1.73	51	L	R	Cons	Imp	None	Yes	0 / 4	Yes	20
GA	60	M	88.5	1.75	15	R	R	Imp	Imp	Q-cane	Yes	2 / 4	Yes	20
FS	73	F	56.7	1.63	41	R	R	Cons	Cons	None	No	0 / 0	No	20

Sex (M=male,F=female) TFO=time from CVA onset (months) R=right Imp=impaired
HD=hand dominance ASHs=modified Ashworth Scale (1-5, K=knee, A=ankle) L=left Cons=conserved

Waid=walking aid AFO=ankle foot orthosis
BARi=Barthel Index (0-20)

Table 1. (Continuation)

Subject	Location of brain lesion(s)
CC	Infarction involving the left middle cerebral artery.
CS	Clot in left internal carotid artery with large infarct involving the left middle cerebral artery.
HH	Focal lesion (infarction) within the right posterior corona radiata adjacent to the right occipital horn.
WB	Middle cerebral artery infarction secondary to clot mechanism.
BL	Left internal carotid artery clot involving the middle cerebral artery.
BT	Occluded left internal carotid artery resulting in left middle cerebral artery infarction.
EF	Trombotic occlusion of right internal carotid artery with large infarct involving the right middle cerebral artery.
GA	Complete occlusion of left internal carotid artery with infarct involving the left middle cerebral artery.
FS	Posterior cerebral artery trombotic occlusion.

Table 2. Means and SD of the H-Reflex Amplitude (mV) for the Five Postures in the Two Sessions - Healthy Subjects.

	Session 1		Session 2	
	Mean	SD	Mean	SD
N	2.61	2.0	2.20	1.5
PO	5.89	2.7	5.60	3.5
HC	.88	1.0	.60	.6
SW	.92	1.1	.93	1.0
MS	2.28	1.8	1.63	1.5

N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance

Table 3. Means and SD of the H-Reflex Latency (mV) for the Five Postures in the Two Sessions - Healthy Subjects.

	Session 1		Session 2	
	Mean	SD	Mean	SD
N	31.0	2.2	30.7	2.0
PO	30.7	2.4	30.4	2.4
HC	31.0	2.1	31.0	1.7
SW	31.4	1.7	31.2	2.0
MS	30.8	2.2	30.9	2.5

N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance

Table 4. Correlation Matrix of the Soleus H-Reflex Amplitude for the Five Postures in Two Sessions - Healthy Subjects.

	N1	PO1	HC1	SW1	MS1	N2	PO2	HC2	SW2	MS2
N1										
PO1	0.77**									
HC1	0.91**	0.67*								
SW1	0.59	0.77**	0.74*							
MS1	0.97**	0.81**	0.89**	0.63*						
N2	0.88**	0.86**	0.74*	0.71*	0.87**					
PO2	0.55	0.82**	0.43	0.63	0.60	0.72*				
HC2	0.64*	0.63*	0.78**	0.86**	0.60	0.65*	0.34			
SW2	0.64*	0.84**	0.71*	0.95**	0.69*	0.75*	0.78*	0.78**		
MS2	0.78**	0.88**	0.69*	0.80**	0.82**	0.91**	0.84**	0.66*	0.91**	

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance

Table 5. Correlation Matrix of the Soleus H-Reflex Latency for the Five Postures in Two Sessions - Healthy Subjects.

	N1	PO1	HC1	SW1	MS1	N2	PO2	HC2	SW2	MS2
N1										
PO1	0.92**									
HC1	0.97**	0.92**								
SW1	0.97**	0.93**	0.94**							
MS1	0.96**	0.84**	0.95**	0.91**						
N2	0.97**	0.90**	0.97**	0.97**	0.95**					
PO2	0.94**	0.97**	0.91**	0.96**	0.87**	0.92**				
HC2	0.91**	0.90**	0.86**	0.88**	0.88**	0.91**	0.94**			
SW2	0.99**	0.88**	0.94**	0.97**	0.95**	0.97**	0.92**	0.89**		
MS2	0.95**	0.89**	0.94**	0.96**	0.93**	0.98**	0.90**	0.88**	0.95**	

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance

Table 6. Means and SD of the H-Reflex Amplitude (mV) for the Five Postures in the Two Sessions - Patients.

	Session 1		Session 2	
	Mean	SD	Mean	SD
N	3.37	2.6	2.90	1.8
PO	4.60	3.1	4.46	2.2
HC	3.70	2.9	3.13	2.0
SW	3.77	2.6	2.98	1.8
MS	3.19	2.7	3.09	1.5

N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance

Table 7. Means and SD of the H-Reflex Latency (mV) for the Five Postures in the Two Sessions - Patients.

	Session 1		Session 2	
	Mean	SD	Mean	SD
N	33.9	2.1	34.0	2.1
PO	34.0	2.2	34.1	2.2
HC	34.2	2.3	34.3	1.6
SW	34.2	1.9	34.2	1.8
MS	34.0	2.1	34.3	1.7

N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance

Table 8. Correlation Matrix of the Soleus H-Reflex Amplitude for the Five Postures in Two Sessions - Patients.

	N1	PO1	HC1	SW1	MS1	N2	PO2	HC2	SW2	MS2
N1										
PO1	0.89**									
HC1	0.96**	0.78*								
SW1	0.87**	0.84**	0.91**							
MS1	0.98**	0.86**	0.96**	0.88**						
N2	0.94**	0.81**	0.88**	0.72*	0.90**					
PO2	0.81**	0.92**	0.73*	0.78*	0.78*	0.77*				
HC2	0.78*	0.50	0.80**	0.54	0.77*	0.89**	0.47			
SW2	0.65	0.52	0.71*	0.61	0.70*	0.72*	0.52	0.80**		
MS2	0.91**	0.71*	0.89**	0.69*	0.87**	0.97**	0.73*	0.91**	0.68*	

** Correlation is significant at the 0.01 level (2-tailed). N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance

* Correlation is significant at the 0.05 level (2-tailed).

Table 9. Correlation Matrix of the Soleus H-Reflex Latency for the Five Postures in Two Sessions - Patients.

	N1	PO1	HC1	SW1	MS1	N2	PO2	HC2	SW2	MS2
N1										
PO1	0.96**									
HC1	0.98**	0.97**								
SW1	0.95**	0.96**	0.96**							
MS1	0.97**	0.95**	0.97**	0.95**						
N2	0.88**	0.87*	0.86**	0.86**	0.92**					
PO2	0.88**	0.87*	0.87**	0.83**	0.89**	0.98**				
HC2	0.95**	0.93**	0.96**	0.93**	0.97**	0.95**	0.95**			
SW2	0.91**	0.94**	0.93**	0.96**	0.94**	0.90**	0.88**	0.97**		
MS2	0.94**	0.89*	0.94**	0.93**	0.97**	0.93**	0.91**	0.99**	0.96**	

** Correlation is significant at the 0.01 level (2-tailed). N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance

* Correlation is significant at the 0.05 level (2-tailed).

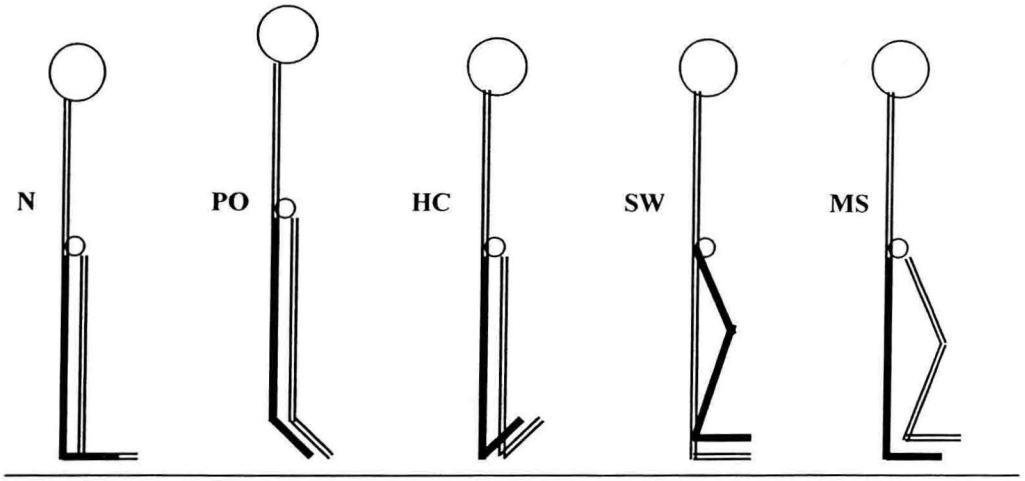

Figure 8. Gait-Simulating Postures. N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance. The solid black lines represent the tested limb.

Experiment 2

Volitional and Reflex Activity of the Ankle Antagonists of Healthy Subjects during Gait-Simulating Postures: An EMG and H-Reflex Study

Introduction

Motor control at the ankle joint is important for the integrity of walking. Inability to control the ankle joint during the gait cycle, is a common impairment that affects patients with cerebrovascular accidents or CVA. Understanding of the normal pattern of neuromotor interplay (reflex and volitional) at the ankle joint during the gait cycle is important to rehabilitative strategies.

The Hoffmann reflex has been used as a means of assessing spinal reflex excitability (Magladery & McDougal, 1950; Schiepatti, 1987). Several studies have reported how the soleus H-reflex amplitude varies in healthy subjects during gait or gait-like activities. Some of these studies have tested soleus H-reflex in: quiet standing (Capaday & Stein, 1986; Morin, Katz, Mazieres, & Pierrot-Deseilligny, 1982); stepping movements (Crenna & Frigo, 1987; Kasai, Kawanishi, & Yahagi, 1998); comparing active and passive stepping movements (Brooke, Cheng, Misiaszek, & Lafferty, 1995); during walking (Capaday & Stein, 1986; Crenna and Frigo, 1987; Morin et al., 1982; Petersen, Morita, & Nielsen, 1999); beam walking and normal treadmill walking (Llewelyn, Yang, & Prochazka, 1990); graded walking (Simonsen, Dhyre-Poulsen, & Voigt, 1995); walking and running (Capaday & Stein, 1987; Simonsen & Dyrhe-Poulsen,

1999); and, walking and running under simulated reduced gravity (Ferris, Aagaard, Simonsen, Farley, & Dyrhe-Poulsen, 2001).

In general, the majority of these studies are in agreement in that the soleus H-reflex amplitude varies greatly during the course of the gait cycle (i.e., the amplitude enhanced during stance and almost completely depressed or absent during swing). Although known to contribute to the soleus H-reflex depression during swing, reciprocal inhibition to soleus from the tibialis anterior muscle has been demonstrated to be only a partially responsible source for this depression, suggesting that central mechanisms may be involved (Morin et al., 1982; Yang & Whelan, 1993).

Evoking the H-reflex and recording electromyographic (EMG) responses during gait movement does however, present a number of methodological challenges (Handcock, Williams, & Sullivan, 2001). First, with movement and changes in posture, alterations in the distance and position of the stimulating electrode relative to the target nerve are highly probable. The common practice to ensure a uniform stimulus to the nerve during movement is to adjust its intensity in order to provoke an M response that is of consistent amplitude (Capaday & Stein, 1986; Crenna & Frigo, 1987). Second, to adequately describe the changes in the H-reflex amplitude, repeated stimuli throughout the phases of the gait cycle (potentially adding more discomfort) are required to complete a profile of H-reflex amplitude for the entire cycle. These methodological concerns show that such investigations necessitate the gathering of a considerable number of measurements from each subject. These needs are likely to be translated into long testing

sessions, adding additional confounding factors such as fatigue, distraction, and the potential for incrementing intra-individual variance.

It was suggested that tonic gait-simulating postures may show soleus H-reflex amplitude changes comparable to those seen in dynamic studies (Crenna & Frigo, 1987). Testing soleus H-reflex in gait-simulating postures may potentially serve as a more practical alternative as a testing paradigm. Soleus H-reflex measured in the upright posture was reported to be more consistent (i.e., stable) compared to less functional postures such as prone lying (Ali & Sabbahi, 2000). Hopkins, Ingersoll, Cordova, and Edwards (2000) reported high inter and intrasession reliabilities (ICC [3,1]) of the soleus H-reflex amplitude measured in one-leg standing position (0.803 and 0.853 respectively). High intrasession reliabilities (ICC [3,1]) for soleus H-reflex and M response (0.97 and 0.87 respectively) were also provided by Handcock et al. (2001). These studies demonstrated that the soleus H-reflex in weight bearing standing is stable and resistant to potentially confounding postural influences, or other sources of biological variation.

By using static postures, variability created at the stimulating and recording sites would likely contribute less to the variability of the measures. Additionally, a lesser number of recordings would be required, making testing sessions shorter. For clinical practicality (evaluation and treatment monitoring), it would be useful to have a pattern of soleus H-reflex and soleus/tibialis anterior EMG activity correlating with different phases of the gait cycle, that could be readily obtained by adopting key static postures. This would be even more relevant in testing patients with upper motor neurone (UMN), who

are known to have wide inter-individual variability in their movement patterns, especially during gait.

The main purposes of this study were to investigate the volitional and reflex activity of the ankle antagonists during five gait-simulating postures. It was hypothesized that the patterns of neuromotor control would be comparable to those observed during regular walking.

Material and Methods

Participants

Twenty-one healthy subjects (9 women, 12 men), age range 22 to 69 years (36.7 $\pm$ 12.6) were recruited for this study. To participate subjects needed to have no previous history of CVA, cancer, diabetes, low back pain, or neurologic or orthopedic pathologies.

Instrumentation

H-Reflex Recording

The Cadwell 5200A electromyography unit was used to elicit and record the soleus H-reflex. Initially the skin location of stimulation and recording were slightly abraded with fine sandpaper in order to reduce skin impedance. For electrical stimulation of the tibial nerve silver-silver chloride surface bar electrodes (active lead-off surface of electrodes was 1 cm2 each, center-to-center distance between the 2 electrodes was 25 mm) were placed over the tibial nerve in the popliteal fossa. Stimulation electrodes were positioned with the cathode proximal to the anode to avoid anodal block (Fisher, 1992).

Another similar bar recording electrode was placed longitudinally at mid-line over the soleus muscle, with the active electrode 3 cm distal to the distal margin of the two heads of the gastrocnemius muscle. A 2 cm diameter metal ground electrode was applied between the stimulating and recording electrodes over the calf muscle group. Electroconductive cream was applied on all electrodes' surfaces for good coupling. Once in place the electrodes were not removed until the whole experiment was completed to ensure exact placement and consistent results. A percutaneous electrical stimulus of 0.5 ms square-wave pulses was delivered at a frequency of 0.2 Hz to the tibial nerve to elicit the maximum H-reflex with minimal M response. Once the maximum H-reflex amplitude and minimal M response were determined with the subjects in the standing position, the minimal M response was kept constant throughout the eliciting/recording procedures for all the gait-simulating postures. The signal was amplified (1000-5000X) using a band pass of 10 Hz-10 KHz.

Electromyography

EMG from soleus and tibialis anterior muscles were recorded and analyzed off-line using the Myosystem 1200 and its Norquest EMG evaluation/analysis software. An analog/digital KPCMCIA-12AI-C card was used to link the Myosystem to a Lifebook C340-233 MHz laptop computer. A pair of surface electrodes was placed side-by-side, along the belly of the tibialis anterior muscle and over the soleus, 1 cm below the bifurcation of the gastrocnemii and on line with the Achilles tendon. A single ground electrode was placed on the skin covering the head of the fibula.

Experimental Procedure

After the subjects signed the consent form, soleus H-reflex and EMG recordings of the soleus and tibialis anterior muscles were obtained in the following five gait-simulating postures: 1) N: freestanding (standing posture), 2) PO: push-off (standing on toes, mimics push-off during late stance phase of gait), 3) HC: heel-contact (standing on heels, mimics transition between swing and stance phases of gait), 4) SW: swing (foot just cleared from ground, mimics toe-off and beginning of swing phase), and 5) MS: mid-stance (standing on tested leg, while contralateral foot is lifted as on the SW posture (mimics mid-stance phase of gait). Postures are schematically shown in Figure 9. The limb to be tested was chosen by alternating right or left as subjects arrived to the testing site. The sequence of testing was the same as defined above. Subjects adopted all postures standing in front of an adjustable standard walker and were asked to have their hands resting on the walker's handles to ensure balance.

Data Analysis

H-Reflex

Three representative H-reflex amplitudes were recorded and averaged for each posture. Three parameters were measured: the H-reflex peak-to-peak amplitude, the maximum peak-to-peak M-wave, and the onset latency to the first deflection. H/M ratios were subsequently calculated. Data was screened for skewness and outliers by frequencies and histograms.

Electromyography

Two 3-sec rectified soleus and tibialis anterior EMG signals were recorded, processed and averaged for each posture. Raw EMG signal was sampled at 1000 Hz rectified and smoothed with 30 ms averaging window for online monitoring and storing. Stored data was quantified using an automatic root mean square (RMS) algorithm with a 50 ms window. The soleus and tibialis anterior EMG data was correlated with the soleus H-reflex.

Statistical Analysis

A univariate one-way repeated measures analysis of variance (ANOVA) was used to assess soleus H-reflex amplitude (dependent variable) differences between postures (independent variable). The SPSS 10.0 statistical software was employed for the statistical analyses.

Results

Soleus H-Reflex amplitude (mV)

Soleus H-reflex amplitude in each posture was: N = 3.46 $\pm$ 2.3, PO = 6.11 $\pm$ 2.6, HC = 0.75 $\pm$ 1.2, SW = 1.21 $\pm$ 1.6, and MS = 3.40 $\pm$ 2.4 (Table 10 and Figure 17). The values for individual soleus H-reflex amplitudes across postures for all subjects are shown in Figure 11. Soleus H-reflex variability (SD values) was higher during freestanding (N), push-off (PO) and mid-stance (MS) and lower during heel-contact (HC) and swing (SW).

There was a statistically significant difference in soleus H-reflex amplitude between postures. The adjusted F (Greenhouse-Geisser Epsilon method) was: $F(2.61, 33.95) = 41.69$, $p < .0005$. Follow-up comparisons using Bonferroni's procedures (.05/10 comparisons) showed that with an alpha level of .005: 1) freestanding (N) was significantly smaller ($p < .005$) than push-off (PO), heel-contact (HC), and swing (SW). Freestanding (N) was not significantly different ($p > .005$) from mid-stance (MS). 2) push-off (PO) was significantly higher ($p < .005$) than freestanding (N), heel-contact (HC), swing (SW), and mid-stance (MS). 3) Heel-contact (HC) was significantly smaller ($p < .005$) than freestanding (N), push-off (PO), and mid-stance (MS). Heel-contact (HC) was not significantly different from swing (SW). 4) Swing (SW) was significantly smaller ($p < .005$) than freestanding (N), push-off (PO), and mid-stance (MS). Swing (SW) was not significantly different from heel-contact (HC). And, 5) mid-stance (MS) was significantly smaller ($p < .005$) than push-off (PO), and significantly higher than heel-contact (HC) and swing (SW). Mid-stance (MS) was not significantly different ($p > .005$) from freestanding (N).

The peak-to-peak amplitude of the soleus H-reflex was significantly higher during push-off (PO) when compared to all the other postures. The peak-to-peak amplitude of the soleus H-reflex showed a significant reduction during heel-contact (HC), swing (SW) and mid-stance (MS) as compared to push-off (PO). The range of change of soleus H-reflex between push-off (PO) and swing (SW) was about 5 mV (80% change) (Figure 10).

Soleus H-Reflex Latency (mV)

Soleus H-reflex latency in each posture was: N = 29.7 + 2.3, PO = 29.4 + 1.9, HC = 32.4 ± 1.7, SW= 31.1 ± 2.9, and MS = 30.0 ± 2.0. Pooled H-reflex latency was 30.5 ± 2.2 (Table 11).

H/M Ratio

The soleus mean H/M ratio for each posture was: N = 0.4, PO = 0.6, HC = 0.1, SW = 0.2, and MS = 0.4. Soleus H/M ratio showed a high correlation (r = 0.96) with soleus H-reflex amplitude. The percentage of activated soleus motoneurone pool (H/M), compared to N (100%) posture was 150% for PO, 25% for HC, 50% for SW, and 100% for MS.

Soleus EMG (RMS - μV)

Individual recordings of soleus EMG activity across postures for all subjects are shown in Figure 13. Although some variation was seen in individual recordings, the averaged soleus EMG activity (Figure 12) showed a pattern that paralleled that of the soleus H-reflex (Figure 10). As it was with the soleus H, the highest soleus EMG activity was in push-off (PO) and the lowest in swing (SW).

Tibialis Anterior EMG (RMS - μV)

Although some variation was seen in individual recordings (Figure 13), the averaged tibialis anterior EMG activity (Figure 14) showed a pattern that was reciprocal to that of the soleus H-reflex (i.e., higher tibialis anterior EMG - lower H-reflex amplitude) (Figure 17). The highest tibialis anterior EMG activity was in heel-contact

(HC), followed by swing (SW) and push-off (PO); the lowest was in freestanding (N) and mid-stance (MS).

Discussion

Soleus H-Reflex Amplitude

Soleus H-reflex amplitude was obtained during selected static postures that were simulating specific phases and transition points of the gait cycle. Our results showed that the soleus H-reflex amplitude varied greatly across the postures, thus confirming our initial hypothesis. The results of this study confirmed the early observations made by Crenna and Frigo (1987) suggesting that comparable soleus H-reflex variation (compared to that from gait studies) could be seen by adopting static gait-simulating postures. As it was expected, the large changes in H-reflex amplitude seen in these static postures paralleled those reported for the stance and swing phases of gait from previous studies (Capaday & Stein, 1986, 1987; Crenna & Frigo, 1987; Ferris et al., 2001; Llewellyn et al., 1990; Simonsen & Dhyre-Poulsen, 1999; Simonsen et al., 1995).

Of all five postures, push-off (PO) showed the largest H-reflex amplitude and paralleled that of the stance phase of walking, where a large number of soleus α-motoneurones are activated. Swing (SW) and heel-contact (HC) showed the smallest H-reflex amplitude (no statistical difference between these two) and paralleled that of the heel-contact and swing phases of walking (Figures 10-11 and 12-13). The soleus H-reflex amplitude was, as previously reported by Capaday and Stein (1986), similarly inhibited

during gait at the two points where tibialis anterior was more active (heel-contact and swing). During swing (SW), however, tibialis anterior EMG activity was considerably lower than in heel-contact (HC). This finding adds support to the theory suggesting that changes in the soleus H-reflex amplitude during the course of the gait cycle depends, among others, on central mechanisms (e.g., pre-synaptic inhibition, central pattern generator) beyond the excitation level of α-motoneurones (Morin et al., 1982; Yang & Whelan, 1993).

Mid-stance (MS) posture was not different from the freestanding (N) meaning that H-reflex amplitude during static unipedal standing is comparable to that found in static bipedal standing. This stability or invariability between these two postures maybe explained due to the fact that subjects were required to rest their hands onto a walker for balance support during testing. Soleus H/M ratios found in this study in all postures were below 0.7. Delwaide (1984) reported this ratio as the normal upper limit activation in healthy subjects. The H/M ratios correlated highly ($r = 0.96$) with soleus H-reflex amplitude, fact that may alleviate the need to record Mmax (requiring an often uncomfortable stimulus) in the assessment of the soleus H-reflex.

As expected, because subjects were healthy individuals, the soleus H-reflex latency was not affected across postures. The average latency found in this study (30.53 ± 1.80, Table 11), was similar to that reported by Braddom and Johnson (1974) for the calf H reflexes (29.8 ± 2.74). The present findings also matched closely the 30 ms suggested by Han, Kim, and Paik (1997) as cut-off criteria for normality.

Soleus and Tibialis Anterior EMG Activity

As expected, soleus EMG activity (Figure 12) also varied greatly across postures and paralleled that of the soleus H-reflex (Figure 10). The greater soleus EMG activation (amplitude) occurred during the push-off (PO) corresponding to stance and push-off of walking. The lesser soleus activation occurred in the swing (SW) posture where the foot was lifted off ground (to initiate swing phase in gait), indicating the almost complete absence of activation of the soleus α-motoneurone pool. This is critical in normal gait, to allow an undisturbed ankle dorsiflexion for the advancing foot to clear the ground. Soleus activity during mid-stance (MS) was comparable to that seen in freestanding (N), possibly indicating that support provided by the walker in the one-leg posture was enough to maintain ankle stability. That is, at higher levels of soleus activation, higher soleus H-reflex amplitude was observed (i.e., reflecting the large activation of the soleus motoneurone pool), and the soleus EMG activity varied the most in the push-off (PO) posture and the least in the swing (SW) posture (Table 11). This variability in soleus activity between-subjects was noted by Arsenault, Winter, and Marteniuk (1986). These authors found, in a small sample of eight subjects, significant differences in the profiles of EMG activity, especially for the rectus femoris and soleus muscles. Although intra-subject variability was very low, he warned against the use of normal EMG profiles, since biological differences could be averaged out in the pooled between-subject data. The low variability seen for the soleus EMG in swing (SW) could possibly been

originated in the low activation levels of the muscle during this phase, which was similarly displayed by all the subjects (Figure 13).

As it was with the soleus EMG, the tibialis anterior EMG activity varied considerably across the postures (Figure 12). Previous gait studies have shown bursts of tibialis anterior activation mostly during the heel-contact (co-contraction with soleus) and early swing. Similarly, our results showed that the greater tibialis anterior activation was in the heel-contact (HC) posture, followed by swing (SW). Heel-contact (HC) was also the posture with the greater variability (Table 10). This variability may be explained by the large amounts of tibialis anterior activation (in co-contraction with the soleus) that was required from the heel-contact (HC) posture to maintain standing on heels within a reduced base of support. During push-off (PO) posture, tibialis anterior showed some activation that possibly was originated from co-contraction (with soleus) to increase ankle stiffness. This activation was relatively small, since it was similar to that seen in the swing (SW) posture. Tibialis anterior activation in the mid-stance (MS) and freestanding (N) posture was low, indicating possibly the more stable conditions of the postures with reduced demand of muscle activation to off-set balance fluctuations (Capaday & Stein, 1986; Stein & Capaday, 1988).

The tibialis anterior showed an EMG activity that was reciprocal to the soleus H-reflex amplitude (Figures 14 and 10). That is, where soleus H-reflex was high (e.g., push-off) tibialis anterior EMG was low, and vs. This suggested that in healthy subjects, and as previously reported by Stein and Capaday (1988) and others (Crenna & Frigo, 1987;

Petersen et al., 1999), a close functional link exist between the volitional and reflex interplay of the ankle antagonists that are closely matched to the requirements of locomotion. Since the role of the soleus muscle (and other ankle extensors) is to control the rate and extent of ankle flexion from heel-contact (HC) to push-off (PO) and to propel the body forward and upward, a large reflex would be most desirable to assist with this propulsive period. During early swing (SW), the toe comes off the ground and the ankle starts to dorsiflex to clear the ground, the soleus muscle is again stretched. But, a high reflex with a contracting soleus muscle would be inappropriate, because it would counteract the tibialis anterior muscle contraction.

As in the swing (SW) phase of gait, maximum inhibition of soleus H-reflex can be achieved by simply lifting the foot off the ground (SW). This phenomenon may prove to be an important element for further investigation in subjects who have suffered a CVA. It may be a potential mechanism of inhibition for abnormal soleus α-motoneurone activity, which has been suggested to contribute to the impairment of dorsiflexion ability in patients with CVA.

Conclusions

In this study several gait-simulating postures were investigated to assess their capability to reproduce the volitional and reflex activity of the ankle antagonists as reported in gait studies. The results of this study demonstrated that the variation in the soleus H-reflex amplitude and the associated EMG activity (from soleus and tibialis anterior muscles) can be comparably reproduced by adopting the proposed gait-

simulating postures. As opposed to the complexity and methodological difficulties of testing ankle motor control during gait, these proposed postures offer a practical method to assess volitional and reflex activity of the ankle antagonists. This would be a useful testing paradigm for healthy subjects as well as patients with impaired ankle motor control of peripheral or central origin.

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Table 10. Soleus H-Reflex Amplitude (mV) in Five Upright Postures.

Posture	Mean ± SD	*SEM*	p value
N	3.46 ± 2.3	0.62	0.005*
PO	6.11 ± 2.6	0.70	----
HC	0.75 ± 1.2	0.31	0.005*
SW	1.21 ± 1.6	0.41	0.005*
MS	3.40 ± 2.4	0.63	0.005*

*PO significantly different from N, HC, SW, and MS ($p < .005$)
HC not significantly different from SW and N not different from MS ($p > .005$)

Table 11. Soleus H-Reflex Latency (mV) in Five Upright Postures.

Posture	Mean ± SD	*SEM*
N	29.7 ± 2.3	0.6
PO	29.4 ± 1.9	0.5
HC	32.4 ± 1.7	0.5
SW	31.1 ± 2.9	0.8
MS	30.0 ± 2.0	0.5
Total	30.5 ± 2.2	

No difference among postures ($p > .05$)

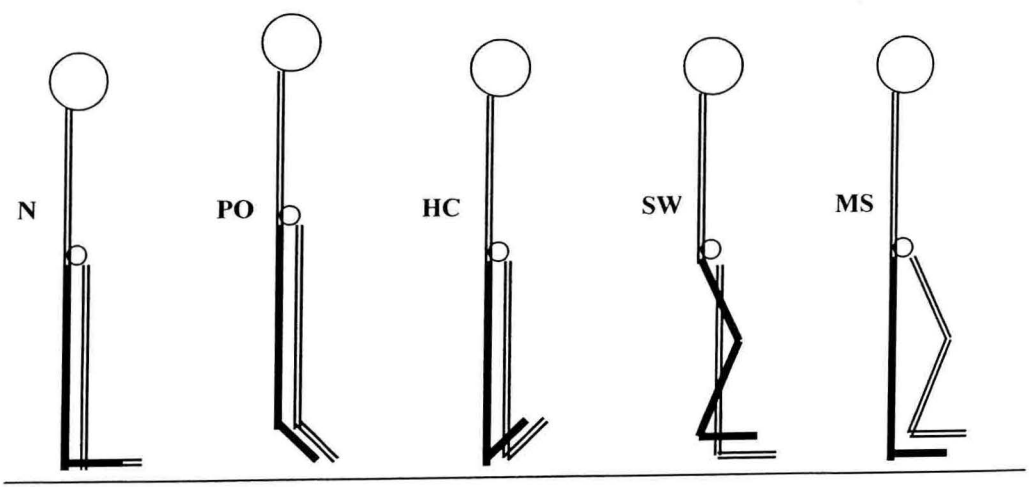

Figure 9. Gait-Simulating Postures. N= free standing, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance. The solid black lines represent the tested limb.

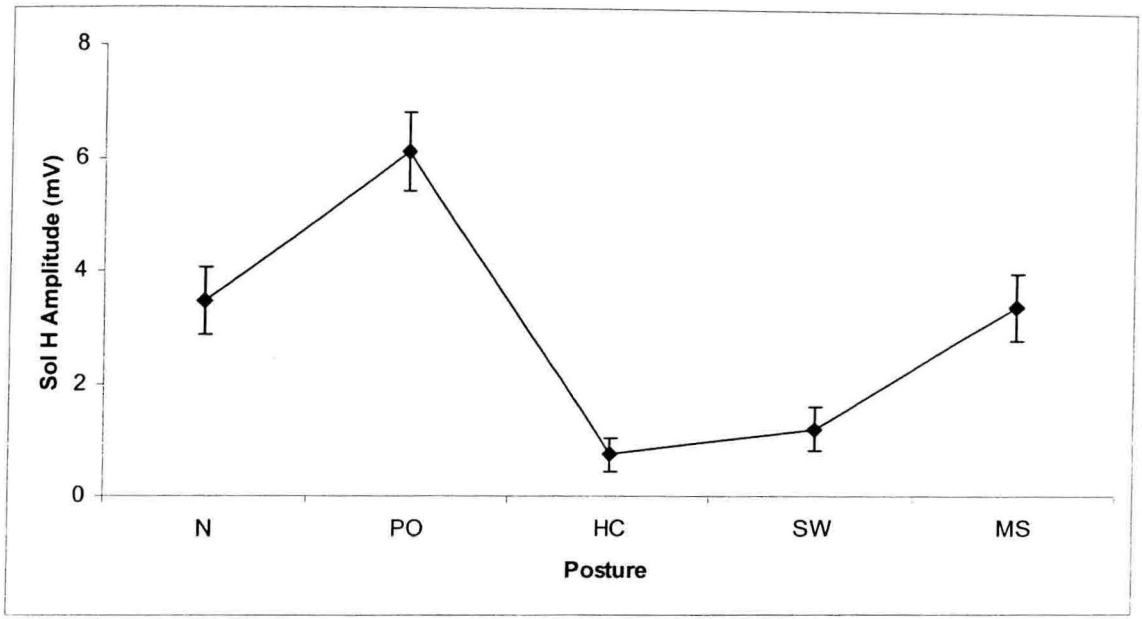

Figure 10. Soleus H-Reflex Amplitude (Mean and *SEM*).

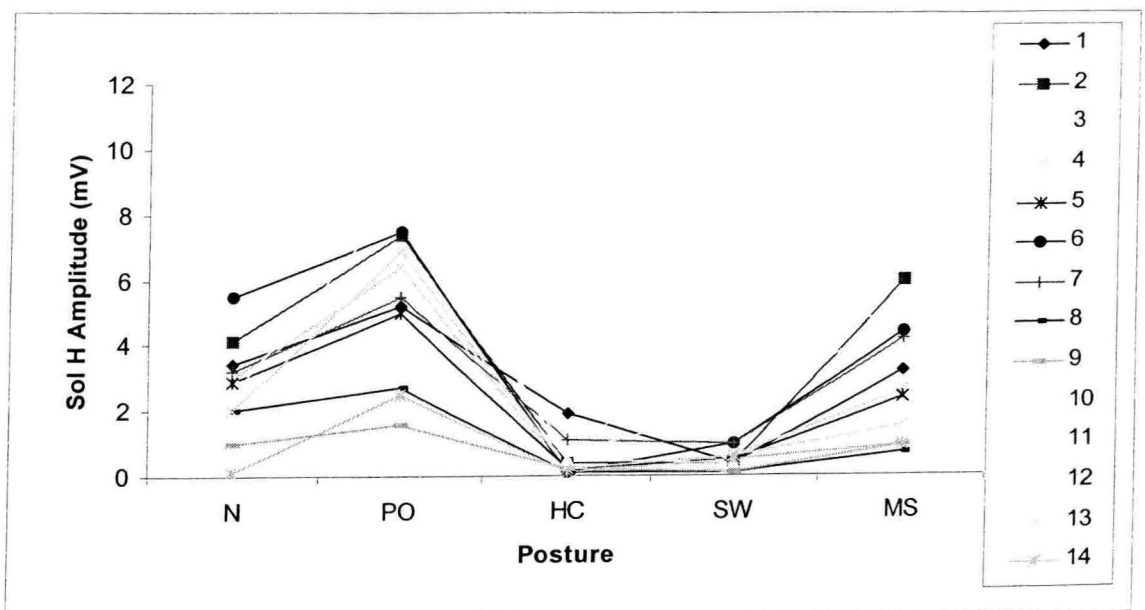

Figure 11. Soleus H-Reflex Amplitude (Individual Traces).

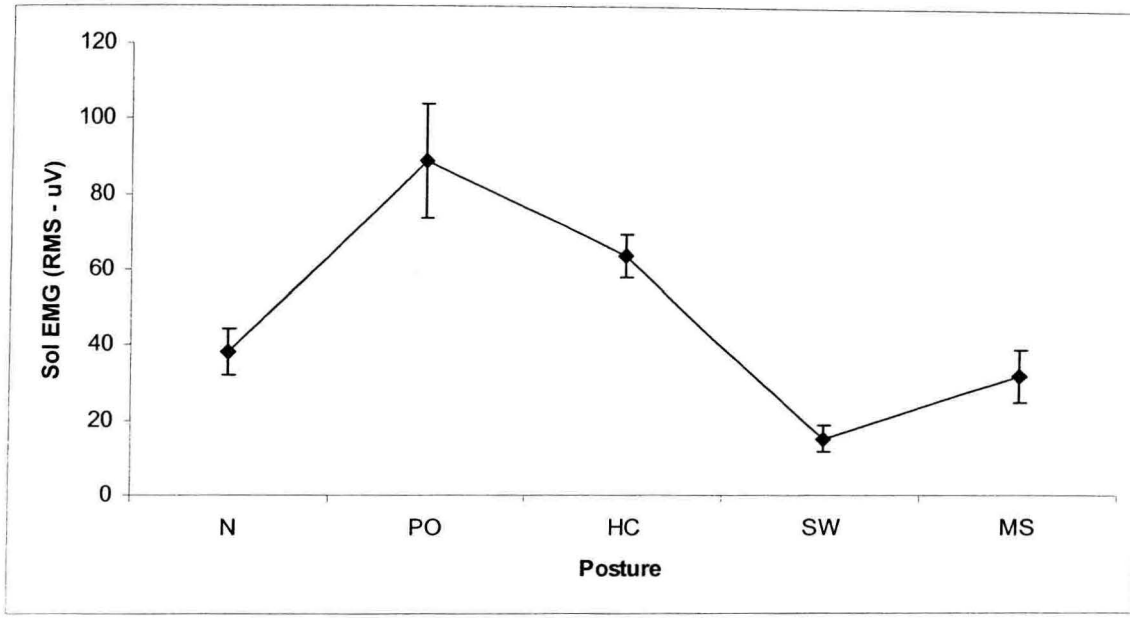

Figure 12. Soleus EMG Activity (Mean and *SEM*).

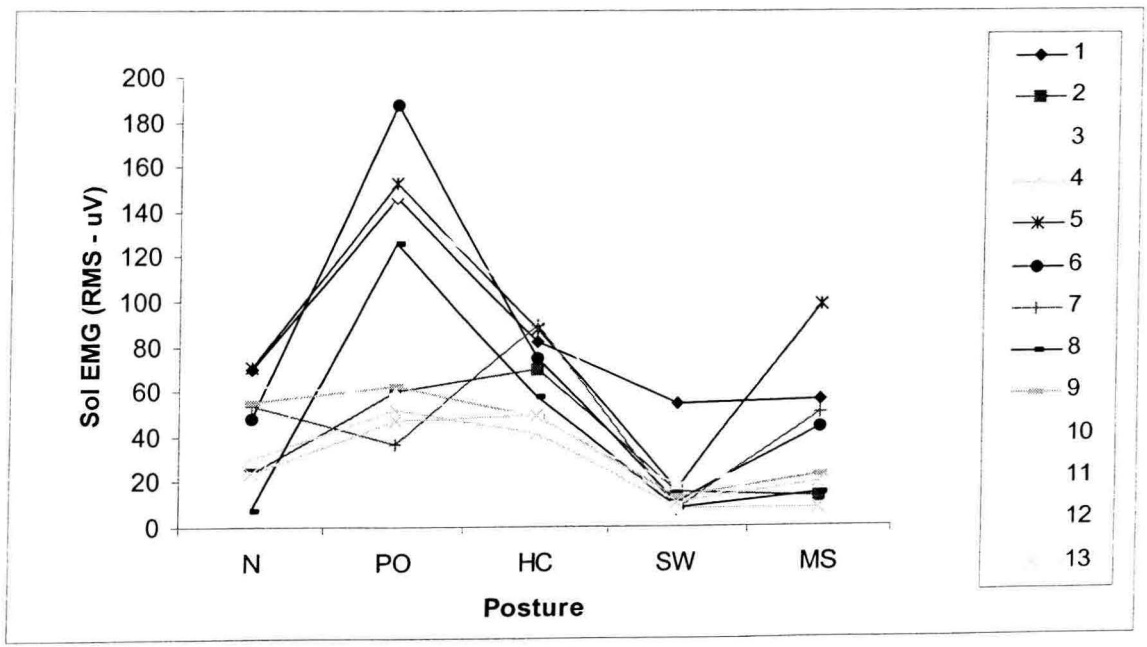

Figure 13. Soleus EMG Activity (Individual Traces).

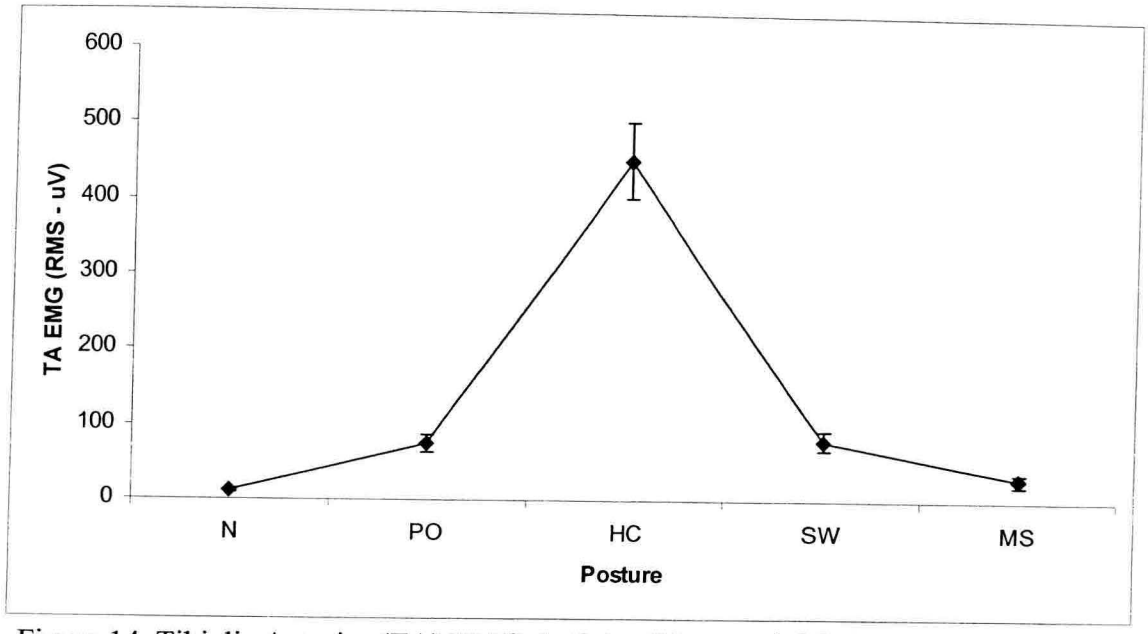

Figure 14. Tibialis Anterior (TA) EMG Activity (Mean and *SEM*).

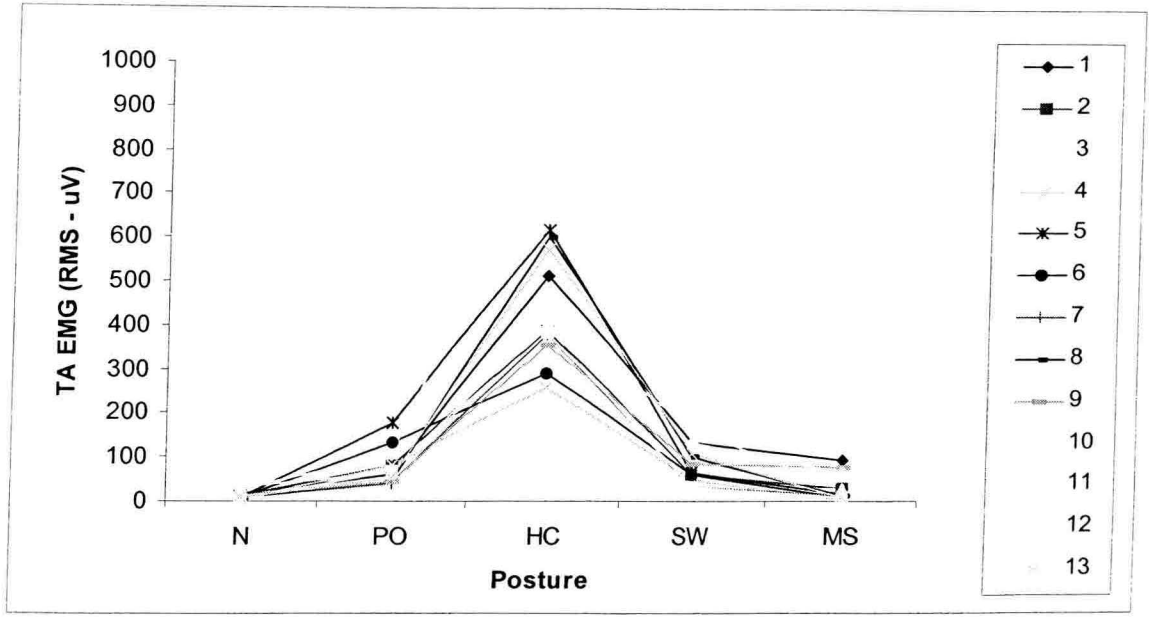

Figure 15. Tibialis Anterior (TA) EMG (Individual Traces).

Experiment 3

Volitional and Reflex Activity of the Ankle Antagonists of Patients with Stroke during Gait-Simulating Postures: An EMG and H-Reflex Study

Introduction

Motor control at the ankle joint is important for the integrity of walking. Inability to control the ankle joint during the gait cycle is a common impairment that affects patients with cerebrovascular accidents (CVA) or stroke. The Hoffmann reflex (H-reflex) has long been used as a means of assessing spinal reflex excitability (Magladery & McDougal, 1950; Schiepatti, 1987). Several studies have reported the variation of the soleus H-reflex amplitude in healthy subjects during gait or gait-like activities (Capaday & Stein, 1986; Crenna & Frigo, 1987; Kasai, Kawanishi, & Yahagi, 1998; Morin, Katz, Mazieres, & Pierrot-Deseilligny, 1982). In general, these studies are in agreement that the soleus H-reflex amplitude varies greatly during the course of the gait cycle (i.e., amplitude enhanced during stance and almost completely depressed or absent during the swing).

Few studies have reported the changes in the soleus H-reflex amplitude during gait in patients with CVA. Yang, Fung, Edamura, Blunt, Stein, and Barbeau (1991) investigated H reflexes of soleus muscles from patients during treadmill walking. Their main finding was that H-reflex amplitude varied from being relatively normal (i.e., facilitated during stance and inhibited during swing) to a complete absence of inhibition

during swing. Further, these authors pointed out that some difficulties are found in recording soleus H-reflex in patients. Among them, they mentioned the presence of clonus, which contaminated the recordings, and the diversity of gait patterns, making difficult the comparisons between patients. Okuma and Lee (1996) reported increased disynaptic Ia inhibition from peroneal to soleus motoneurones in patients who showed good recovery of function with mild spasticity. However, it was not changed, or even sometimes diminished in patients who made a poor recovery and had more marked spasticity. In another study with patients with CVA, Leonard, Diedrich, Matsumoto, Moritani, and McMillan (1998) demonstrated lack of soleus H-reflex inhibition during dorsiflexor activity, either voluntary or during automatic postural perturbations.

These studies showed that disruption of soleus H-reflex amplitude (i.e., lack of inhibition during swing) varied largely among patients, as well as their walking patterns. In addition, these dis-similarities are worsened by the various levels of spasticity and extent of cortical and central nervous system (CNS) damage, making patients to differ greatly in the manifestation of their impairments and functional abilities.

Evoking the H-reflex and recording electromyographic (EMG) responses during gait movement does present a number of methodological difficulties (Handcock, Williams, & Sullivan, 2001). First, with movement and changes in posture, alterations in the distance and position of the stimulating electrode relative to the target nerve are highly probable. The common practice to ensure a uniform stimulus to the nerve during movement is to adjust its intensity in order to provoke an M response that is of consistent

amplitude (Capaday & Stein, 1986; Crenna & Frigo, 1987). This method has been utilized satisfactorily in the testing of healthy subjects but it may not be as useful in patients whose movements may be unsteady and jerky due to the presence of abnormal reflexes. Second, to adequately describe the changes in the soleus H-reflex amplitude throughout the phases of the gait cycle, repeated stimuli are required to complete a profile of H-reflex amplitude for the entire cycle. These methodological concerns show that such investigations necessitated the gathering of a considerable number of measurements from each subject. These needs were likely to be translated into long testing sessions, adding additional confounding factors such as fatigue, distraction, and the potential for incrementing intra-individual variance.

By using static postures, variability created at the stimulating and recording sites would likely to contribute less to the variability of measures. Also, a lesser number of recordings would be required, making testing sessions shorter. For clinical practicality (evaluation and treatment monitoring), it would be useful to have a normal pattern that shows the variation of the soleus H-reflex amplitude and soleus/tibialis anterior EMG, occurring in the course of the gait cycle, that could be readily obtained by adopting key static postures. In addition, by assessing the soleus H-reflex in predetermined postures would allow comparisons to be made among subjects since the same experimental paradigm would be applied to them. This would be even more relevant in testing patients with upper motor neurone (UMN), who are known to have wide inter-individual variability in their movement patterns, especially during gait.

Understanding of the pattern of neuromotor interplay (or the absence of it) at the ankle joint during the gait cycle is important so that rehabilitative strategies can be improved. The main purposes of this study were to investigate the volitional and reflex activity of the ankle antagonists of patients with CVA during five gait-simulating postures, and compare them to that of healthy subjects who were assessed by the same testing paradigm. It was hypothesized that the patterns of soleus H-reflex amplitude and associated EMG activity of the ankle antagonists recorded during gait-simulating postures would be different for the two groups.

Material and Methods

Participants

Nine patients with CVA (5 women, 4 men), age range 44 to 73 (60.1 ± 8.3); and, twenty-one healthy subjects (9 women, 12 men) age range 22 to 69 years (36.7 ± 12.6) participated in this study. Patients were community dwellers able to walk at least 10 meters (30 feet) without stopping and reported having no history of diabetes, low back pain, cancer or lower extremity orthopedic pathologies. None of the patients was taking anti-spasticity medications. Demographic and clinical information is summarized in Table 12. The healthy subjects reported having no history of significant low back pain, or neurological or orthopedic pathologies, or diabetes.

Instrumentation

H-Reflex Recording

The Cadwell 5200A electromyography unit was used to elicit and record the soleus H-reflex. Initially the skin location of stimulation and recording were slightly abraded with fine sandpaper in order to reduce skin impedance. For electrical stimulation of the tibial nerve silver-silver chloride surface bar electrodes (active lead-off surface of electrodes was 1 cm2 each, center-to-center distance between the 2 electrodes was 25 mm) were placed over the tibial nerve in the popliteal fossa. Stimulation electrodes were positioned with the cathode proximal to the anode to avoid anodal block (Fisher, 1992). Another similar bar recording electrode was placed on the mid-line over the soleus muscle, with the active electrode 3 cm distal to the distal margin of the two heads of the gastrocnemius muscle. A 2 cm diameter metal ground electrode was applied between the stimulating and recording electrodes over the calf muscle group. Electroconductive cream was applied on all electrodes' surfaces for good coupling. Once in place the electrodes were not removed until the whole experiment was completed to ensure exact placement and consistent results. A percutaneous electrical stimulus of 0.5 ms square-wave pulses was delivered at a frequency of 0.2 Hz to the tibial nerve to elicit the maximum H-reflex with minimal M response. Once the maximum H-reflex amplitude and minimal M response were determined with the subjects in the standing position, the minimal M response was kept constant throughout the eliciting/recording procedures for all the

gait-simulating postures. The signal was amplified (1000-5000X) using a band pass of 10 Hz-10 KHz.

Electromyography

Soleus and tibialis anterior muscles were recorded and analyzed off-line using the Myosystem 1200 and its Norquest EMG evaluation/analysis software. An analog/digital KPCMCIA-12AI-C card was used to link the Myosystem to a Lifebook C340-233 MHz laptop computer. A pair of surface electrodes was placed side-by-side, over the tibialis anterior muscle belly and over the soleus, 1 cm below the bifurcation of the gastrocnemii and on line with the Achilles tendon. A single ground electrode was placed on the skin covering the head of the fibula.

Questionnaires and Indexes

All selected patients were tested using two scales: 1) the Barthel ADL Index (Mahoney & Barthel, 1965) to provide information on overall functional status. And, 2) the Modified Ashworth Scale (Bohannon & Smith, 1987) to score knee and ankle joint spasticity.

Experimental Procedure

After signing the consent form, the participants were assessed for spasticity with the Ashworth Scale and completed the Barthel ADL Index. Soleus H-reflex and EMG recordings were obtained in the following five gait-simulating postures: 1) N: freestanding (standing posture), 2) PO: push-off (standing on toes, mimics push-off during late stance phase of gait), 3) HC: heel-contact (standing on heels, mimics

transition between swing and stance phases of gait), 4) SW: swing (foot just cleared from ground, mimics toe-off and beginning of swing phase), and 5) MS: mid-stance (standing on tested leg, while contralateral foot is lifted as on the SW posture (mimics mid-stance phase of gait). Postures are schematically shown in Figure 16. Patients were tested in their affected limb only. In the healthy subjects, the limb to be tested was chosen by alternating right or left as subjects arrived to the testing site. The sequence of testing was the same as defined above. Healthy subjects were tested while standing in front of an adjustable standard walker and were asked to have their hands resting on the walker's handles to ensure balance. Patients were tested while standing in front of a platform adjustable walker and were asked to have their forearms resting on the walker's platforms to ensure balance.

Data Analysis

H-reflex

Three representative H-reflex amplitudes were recorded, measured and averaged for each posture. Two parameters were measured: the H-reflex peak-to-peak amplitude and the onset latency to the first deflection. Data was screened for skewness and outliers by frequencies and histograms.

Electromyography

Two 3-sec rectified soleus and tibialis anterior EMG signals were recorded, processed and averaged for each posture. Raw EMG signal was sampled at 1000 Hz rectified and smoothed with 30 ms averaging window for online monitoring and storing.

Stored data was quantified using an automatic root mean square (RMS) algorithm with a 50 ms window. Soleus and tibialis anterior EMG data was correlated with the soleus H-reflex amplitude.

Statistical Analysis

A univariate split-plot one-way repeated measures analysis of variance (ANOVA) was used to assess differences between postures (within-subject factor), and between groups (between-subject factor) for soleus H-reflex amplitude. The dependent variable was soleus H-reflex amplitude. The independent variables were 1) group (healthy subjects and patients), and 2) gait-simulating postures (freestanding, push-off, heel contact, swing, and mid-stance).

Results

Soleus H-Reflex Amplitude (mV) - Healthy Subjects

Soleus H-reflex amplitude in each posture was: N = 3.46 $\pm$ 2.3, PO = 6.11 $\pm$ 2.6, HC = 0.75 $\pm$ 1.2, SW = 1.21 $\pm$ 1.6, and MS = 3.40 $\pm$ 2.4 (Figure 16 and Table 13).

Soleus H-reflex Amplitude (mV) - Patients

Soleus H-reflex amplitude in each posture was: N = 3.37 $\pm$ 2.6, PO = 4.60 $\pm$ 3.1, HC = 3.70 $\pm$ 2.9, SW = 3.77 $\pm$ 2.6, and MS = 3.19 $\pm$ 2.7 (Figure 17 and Table 14).

The results of the univariate split-plot analysis revealed a statistically significant interaction between posture and group (Figure 17). The adjusted F (Greenhouse-Geisser method) was: $F(2.62, 55.13) = 15.68$, $p < .0005$. The interpretation of the interaction indicated that the effect of posture in soleus H-reflex amplitude depends on group.

Follow-up of simple effects revealed that with an alpha level of .01 (with Bonferroni's adjustment procedures), healthy controls and patients were significantly different in the heel-contact (HC, $p = .003$) and swing (SW, $p = .007$) postures. Inhibition of the soleus H- reflex did not occur in the heel-contact (HC) and swing (SW) postures in the patients group.

Soleus H-Reflex Latency (mV) - Healthy Subjects and Patients

Healthy Subjects

Soleus H-reflex latency in each posture was: N = 29.7 $\pm$ 2.3, PO = 29.4 $\pm$ 1.9, HC = 32.4 $\pm$ 1.7, SW = 31.1 $\pm$ 2.9, and MS = 30.0 $\pm$ 2.0. Pooled H-reflex latency was 30.5 $\pm$ 2.2 (Table 15).

Patients

Soleus H-reflex latency in each posture was: N = 33.9 $\pm$ 2.1, PO = 33.9 $\pm$ 2.2, HC = 34.2 + 2.3, SW = 34.2 $\pm$ 1.9, and MS = 34 $\pm$ 2.1. Pooled H latency was 34.0 $\pm$ 2.1 (Table 16).

Soleus EMG (RMS - μV) - Healthy Subjects

Soleus EMG activity across postures for all subjects is shown in Figure 18. Although some variation was seen in individual amplitudes, the overall mean of the soleus EMG (Figure 19) showed a pattern that paralleled that of the soleus H-reflex (see Figure 17). The greatest soleus EMG activity was in push-off (PO) and the lowest was in swing (SW).

Soleus EMG (RMS - μV) - Patients

Soleus EMG activity across postures for all patients is shown in Figure 20. Although some variation was seen in individual amplitudes, the overall mean of the soleus EMG showed a pattern that paralleled that of the soleus EMG from healthy subjects (Figure 19). The highest soleus EMG activity was in push-off (PO) and the lowest was in swing (SW).

Tibialis Anterior EMG (RMS - μV) - Healthy Subjects

Tibialis anterior EMG activity across postures for all subjects is shown in Figure 21. Although some variation was seen in individual amplitudes, the overall mean of the tibialis anterior EMG activity (Figure 22) showed a pattern that was reciprocal to that of the normal soleus H-reflex (see Figure 17). The highest tibialis anterior EMG activity was in heel-contact (HC) followed by swing (SW), and the lowest was in freestanding (N) and mid-stance (MS).

Tibialis Anterior EMG (RMS - μV) - Patients

Tibialis anterior EMG activity across postures for all patients is shown in Figure 23. Although some variation was seen in individual amplitudes, the overall mean of the tibialis anterior EMG activity, compared to that from healthy subjects, showed a pattern with a peak shifted to swing (SW) from heel-contact (HC) (Figure 22). The highest tibialis anterior EMG activity was in swing (SW), which showed a level of activation comparable to that of normal subjects. The lowest was in freestanding (N), push-off (PO) and mid-stance (MS).

Raw Soleus/Tibialis Anterior EMG Activity and Soleus H-Reflex Amplitudes Recorded during Each Posture

A display of individual (1 healthy subject and 9 patients) raw EMG activity from soleus and tibialis anterior muscles and soleus M and H-reflex waves for each of the postures are shown in Figures 24 through 33 (TA= tibialis anterior, Sol= soleus; and N= freestanding, PO= push-off, HC= heel-contact, SW= swing, and MS= mid-stance). Note that the EMG activity from the tibialis anterior (TA) was almost absent during heel-contact (HC) for the majority of the patients. However, during swing, it was comparable to that of the healthy subject. Also, note that the patient FS showed a profile that was comparable to that of the healthy subject.

Discussion

Soleus H-reflex Amplitude - Comparison Between Healthy Subjects and Patients

Our results showed, and as previously reported (Yang et al., 1991), that the large changes observed in the soleus H-reflex amplitude in the healthy subjects was profoundly disrupted (i.e., lack of inhibition during swing) in patients with CVA. The pattern of soleus H-reflex amplitude varied considerably among patients, from being almost normal in one patient (i.e., facilitated during stance and inhibited during swing) to a complete absence of inhibition during swing in others. In this study, soleus H-reflex differences were clearer in the heel-contact (HC) and swing (SW) postures. This indicated that in patients and as opposed to the healthy subjects, soleus motoneurone pool excitability

remained high (i.e., was not inhibited) in the heel-contact (HC) and swing (SW) postures. During push-off (PO), patients showed some increase in soleus H-reflex excitability, but not at the level of the healthy subjects.

In healthy subjects, soleus H-reflex inhibition at heel-contact (HC) may possibly be the result of reciprocal inhibition from tibialis anterior, muscle that was strongly contracted in this posture. In swing (SW), the profound soleus H-reflex depression may only be partially due to the result of reciprocal inhibition from tibialis anterior muscle, since here a much lower tibialis anterior contraction (i.e., to overcome the weight of the foot) was taking place. Morin et al. (1982) and Yang and Whelan (1993), suggested that an additional powerful mechanism of soleus inhibition may be the responsible, most likely central presynaptic inhibition of the primary afferents. In any case, the mechanism was profoundly disrupted in the patients.

The average latency found in the patients (Table 16) was slightly higher than that of the healthy subjects (Table 15), but still under the 35 ms suggested by Fisher (1992) as the upper limit of normality. Age is related to increased soleus H-reflex latency (Fisher, 1992), and possibly explains the differences observed between healthy subjects and patients (a 20 year difference in their averaged ages).

Soleus EMG Activity - Comparison between Healthy Subjects and Patients

Unexpectedly, soleus EMG activity in the patients was remarkably similar to that of the healthy subjects (Figure 19). In gait studies, patients are reported to generate abnormal EMG discharges during the stance phase (in this study the push-off [PO]

posture) triggered by the muscle stretch (eccentric contraction of soleus) induced by the forward moving shank over the fixed foot (Yang et al., 1991). In this study push-off (PO) required the soleus to contract concentrically, since while lifting the heel the shank was not simultaneously being advanced, as it is the case during normal gait. This suggested that the paretic soleus behave "normally" during concentric activity. Further, since the soleus EMG activity of the patients was apparently similar to that of the healthy subjects, it was "normally low" at heel-contact (HC) and swing (SW), contrasting with the high excitability of its motoneurone pool (soleus H-reflex, Figure 17). This exposed a disruption of the association between the soleus H-reflex with its observed level of background activity (EMG) that was clearly different from that of the healthy subjects, where the soleus H-reflex was largely correlated to the volitional EMG activation of the soleus muscle.

Tibialis Anterior EMG Activity - Comparison between Healthy Subjects and Patients

Normally, tibialis anterior EMG activation during gait cycle was reported to show two peaks (Burridge, Wood, Taylor, & McLellan, 2001; Knutsson & Richards, 1979; Yang et al., 1991): one at the heel-contact (HC in this study) related to the active landing of the foot on the ground; and, another at the early swing (SW in this study) related to the clearance of the ground by the advancing foot. In the patients of this experiment and as reported in previous gait studies (Burridge et al., 2001; Knutsson & Richards, 1979; Yang et al., 1991) lack of motor control and poor (totally absent in some cases) muscle activity

of the tibialis anterior muscle were clearly exposed in the heel-contact (HC) posture where patients could not lift their toes or dorsiflex their involved foot off-ground.

The tibialis anterior EMG activity of the patients was unexpectedly similar to that of the healthy subjects in the swing (SW) posture (Figure 22), since patients showed difficulty dorsiflexing their ankles and had various levels of drop-foot. The patients in this study, and in agreement with previous work (Burridge et al., 2001; Knutsson & Richards, 1979) could generate tibialis anterior EMG activity, only in doing the act of stepping, which may be the expression of a spinal non-volitional mechanism. This finding opposes the suggestion that low levels of tibialis anterior activity (i.e., strength) would be the likely source of drop-foot in patients (Yang et al., 1991). Here, patients were able to do the task (SW posture, open chain) by "gross" movement of their limb, done effortlessly. On the contrary, during heel-contact (HC posture, closed chain) they needed to volitionally attempt to focus their action at toes and ankle, showing their inability to produce and control the movement, in spite of demonstrated conscious and considerable physical effort.

Remarkably, the majority of patients during swing (SW) did lift their feet off-ground in an action that was not much different from the way healthy subjects moved. Some patients showed some ability to control and generate some response in their tibialis anterior muscle. It appears that for patients with some level of volitional control over the tibialis anterior, regardless of their spasticity level, were able to show a better functioning, although still disrupted, of their walking ability (Okuma & Lee, 1996).

Further studies are needed to bring light to the relevance of tibialis anterior activity, under different levels of volitional control, in affecting the soleus H-reflex during heel-contact (HC) and swing (SW). This, however, will require to examine individual cases since the large variability observed in patients with CVA.

Conclusions

In this study, a testing paradigm with several gait-simulating postures was used to assess and compare patients with healthy subjects for soleus H-reflex and associated EMG activity of the ankle antagonists. Patients were established, fully functional community dwelling CVA survivors. The testing paradigm, investigated in a related study with healthy subjects, was found to reflect the profound volitional and reflex activity of ankle muscle antagonists seen in gait studies. The results revealed that soleus H-reflex was profoundly disrupted in patients during heel-contact and swing postures, where soleus H-reflex inhibition was absent. Unexpectedly, the patient group and the healthy group showed a similar level of soleus EMG activity for each posture. Also unexpected, was the comparable levels of tibialis anterior EMG activity recorded for the two groups during swing (SW). Muscle weakness, however, and lack of control over tibialis anterior was clearly exposed for patients during heel-contact (HC). This lack of volitional control over tibialis anterior in the patients appeared to have a more direct association to the disrupted soleus H-reflex than to the levels of hyperexcitability (spasticity) of the ankle antagonists.

As opposed to the complexity of testing ankle motor control dynamically, these gait-simulating postures offer a practical and repeatable method to assess volitional and reflex activity of the ankle antagonists. This may be a useful testing paradigm for patients with impaired ankle motor control of peripheral or central origin.

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Table 12. Demographic and Clinical Data of Participating Stroke Survivors.

Subject	Age (yr)	Sex	Mass (Kg)	Height (m)	TFO (mo)	Paretic side	HD	Speech ability	Sensory	WAid	AFO	ASHs K / A	Clonus	BARi
CC	63	F	81.6	1.65	67	R	R	Cons	Cons	Q-cane	Yes	3 / 4	Yes	19
CS	44	F	63.5	1.68	25	R	R	Imp	Cons	Q-cane	Yes	0 / 0	No	19
HH	62	F	65.8	1.75	20	L	R	Cons	Cons	None	Yes	0 / 1	Yes	20
WB	64	F	78.9	1.63	69	L	R	Imp	Cons	None	Yes	1 / 2	Yes	20
BL	66	M	77.1	1.70	180	R	R	Cons	Cons	Cane	No	3 / 4	No	16
BT	56	M	74.8	1.75	46	R	R	Imp	Imp	None	No	0 / 1	Yes	20
EF	53	M	80.3	1.73	51	L	R	Cons	Imp	None	Yes	0 / 4	Yes	20
GA	60	M	88.5	1.75	15	R	R	Imp	Imp	Q-cane	Yes	2 / 4	Yes	20
FS	73	F	56.7	1.63	41	R	R	Cons	Cons	None	No	0 / 0	No	20

Sex (M=male,F=female) TFO=time from CVA onset (months) R=right Imp=impaired
HD=hand dominance ASHs=modified Ashworth Scale (1-5, K=knee, A=ankle) L=left Cons=conserved

Waid=walking aid AFO=ankle foot orthosis
BARi=Barthel Index (0-20)

Table 12. (Continuation)

Subject	Location of brain lesion(s)
CC	Infarction involving the left middle cerebral artery.
CS	Clot in left internal carotid artery with large infarct involving the left middle cerebral artery.
HH	Focal lesion (infarction) within the right posterior corona radiata adjacent to the right occipital horn.
WB	Middle cerebral artery infarction secondary to clot mechanism.
BL	Left internal carotid artery clot involving the middle cerebral artery.
BT	Occluded left internal carotid artery resulting in left middle cerebral artery infarction.
EF	Trombotic occlusion of right internal carotid artery with large infarct involving the right middle cerebral artery.
GA	Complete occlusion of left internal carotid artery with infarct involving the left middle cerebral artery.
FS	Posterior cerebral artery trombotic occlusion.

Table 13. Soleus H-Reflex Amplitude (mV) in Five Upright Postures in Healthy Subjects.

Posture	Mean ± SD	*SEM*
N	3.46 ± 2.3	0.6
PO	6.11 ± 2.6	0.7
HC	0.75 ± 1.2	0.3
SW	1.21 ± 1.6	0.4
MS	3.40 ± 2.4	0.6

Table 14. Soleus H-Reflex Amplitude (mV) in Five Upright Postures in Patients.

Posture	Mean ± SD	*SEM*
N	3.37 ± 2.6	0.9
PO	4.60 ± 3.1	1.0
HC	3.70 ± 2.9	1.0
SW	3.77 ± 2.6	0.9
MS	3.19 ± 2.7	0.9

N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance

Table 15. Soleus H-Reflex Latency (mV) in Five Upright Postures in Healthy Subjects.

Posture	Mean ± SD	*SEM*
N	29.7 ± 2.3	0.6
PO	29.4 ± 1.9	0.5
HC	32.4 ± 1.7	0.5
SW	31.1 ± 2.9	0.8
MS	30.0 ± 2.0	0.5
Total	30.5 ± 2.2	

Table 16. Soleus H-Reflex Latency (mV) in Five Upright Postures in Patients.

Posture	Mean ± SD	*SEM*
N	33.9 ± 2.2	0.7
PO	33.9 ± 2.2	0.7
HC	34.2 ± 2.3	0.8
SW	34.2 ± 1.9	0.7
MS	34.0 ± 2.1	0.7
Total	34.0 ± 2.1	

N= freestanding, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance

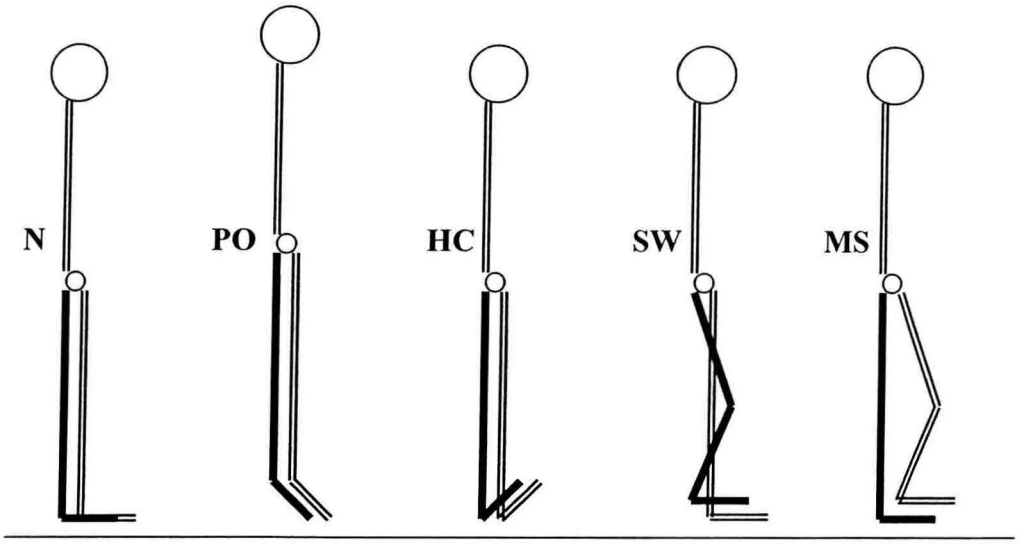


Figure 16. Gait-Simulating postures. N= free standing, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance. The solid black lines represent the tested limb.

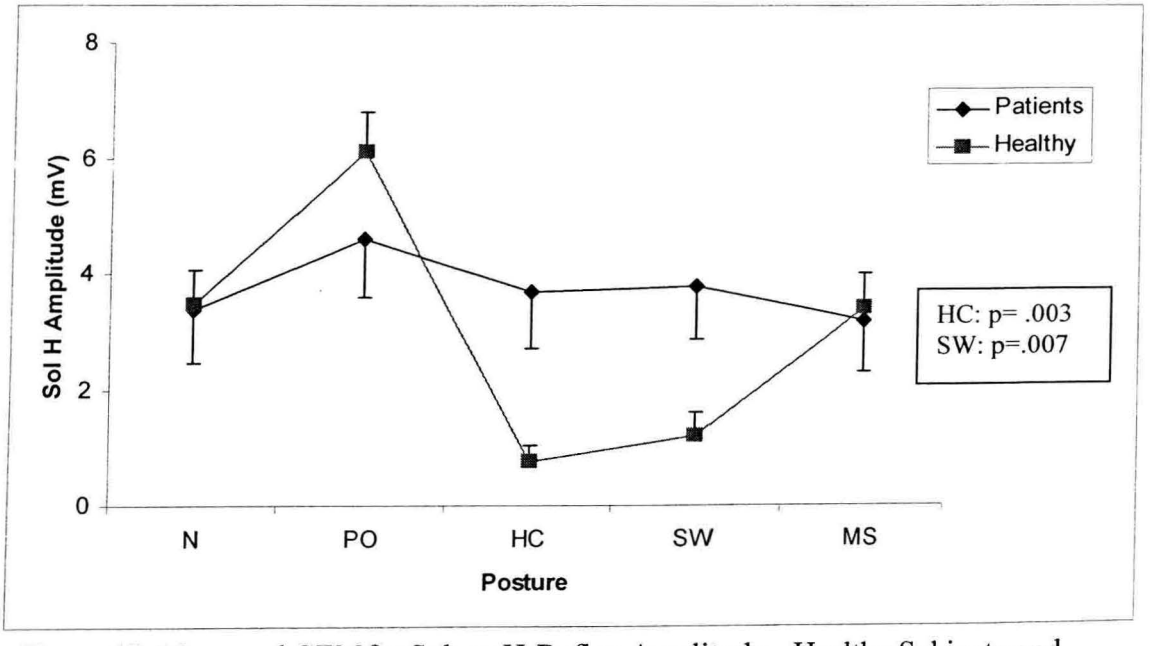


Figure 17. Mean and *SEM* for Soleus H-Reflex Amplitude - Healthy Subjects and Patients.

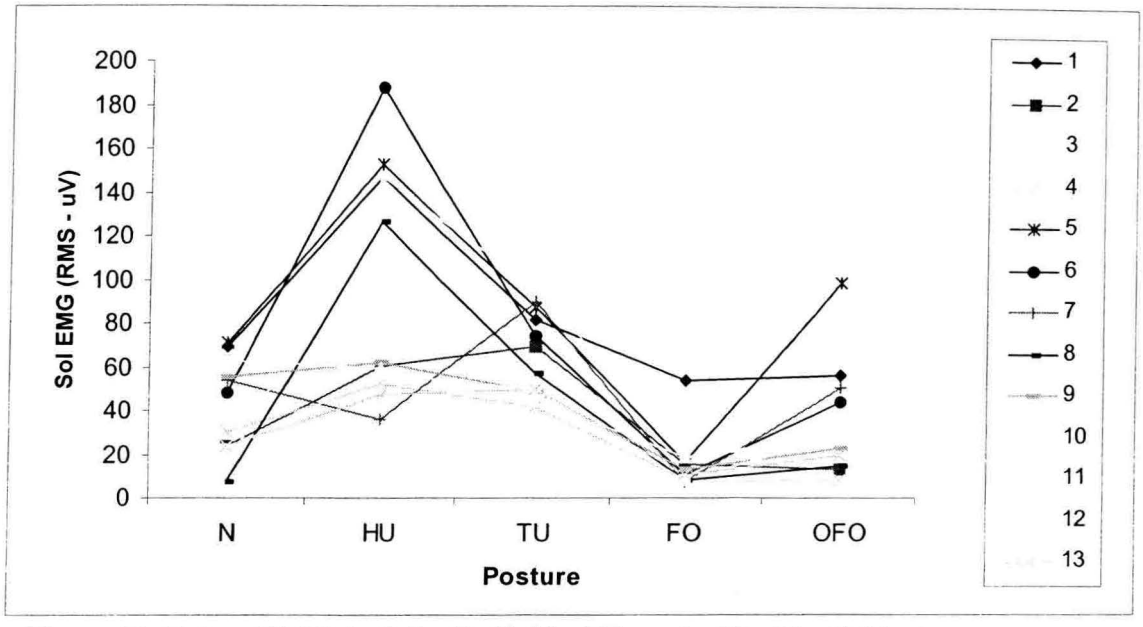


Figure 18. Soleus EMG Activity (Individual Traces) - Healthy Subjects

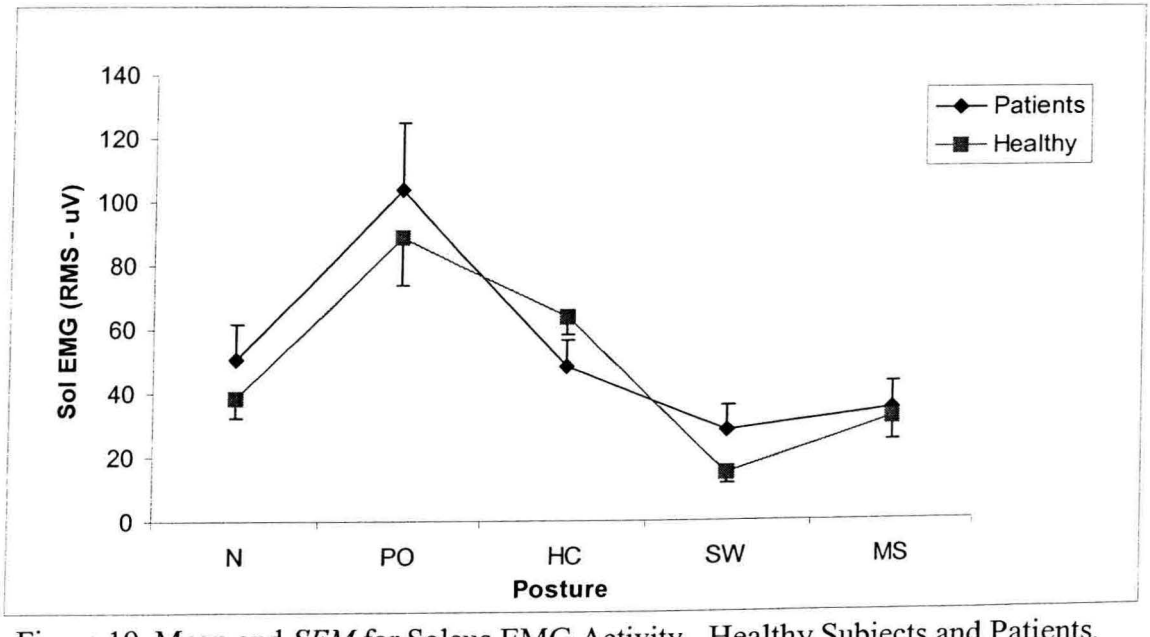


Figure 19. Mean and *SEM* for Soleus EMG Activity - Healthy Subjects and Patients.

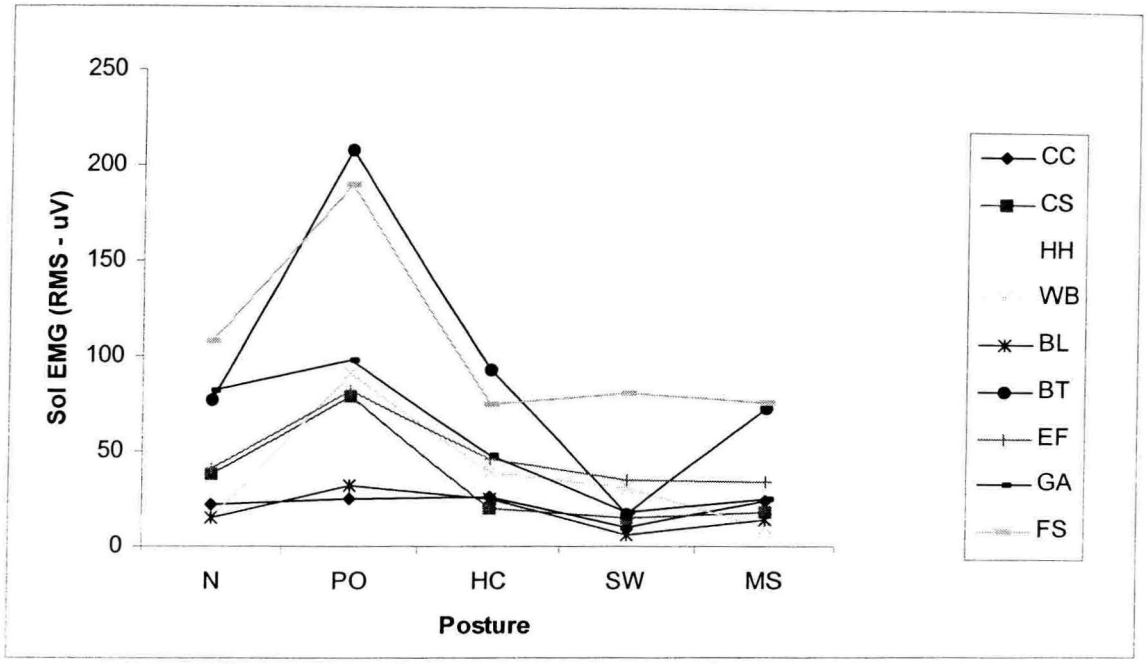


Figure 20. Soleus EMG Activity (Individual Traces) - Patients.

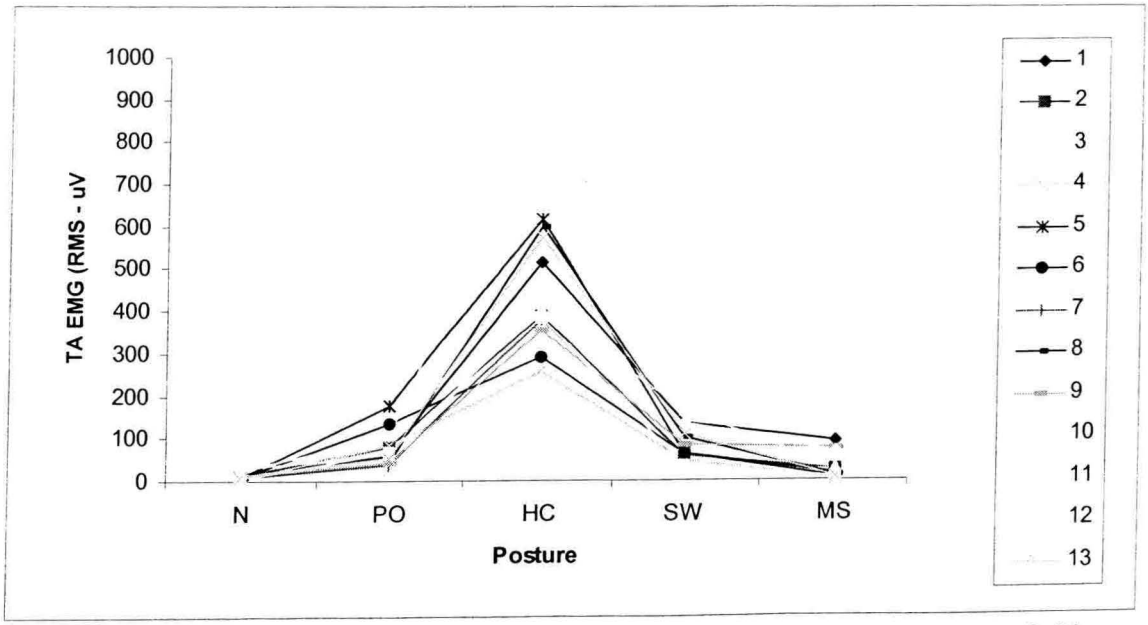


Figure 21. Tibialis Anterior (TA) EMG Activity (Individual Traces) - Healthy Subjects.

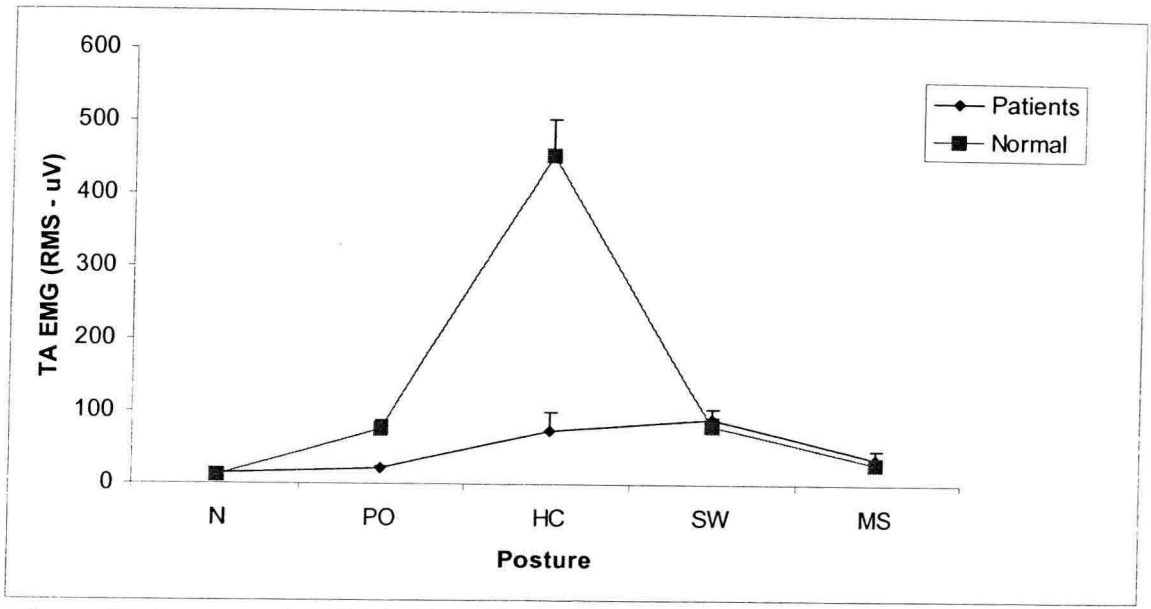


Figure 22. Means and *SEM* for Tibialis Anterior (TA) EMG - Healthy Subjects and Patients.

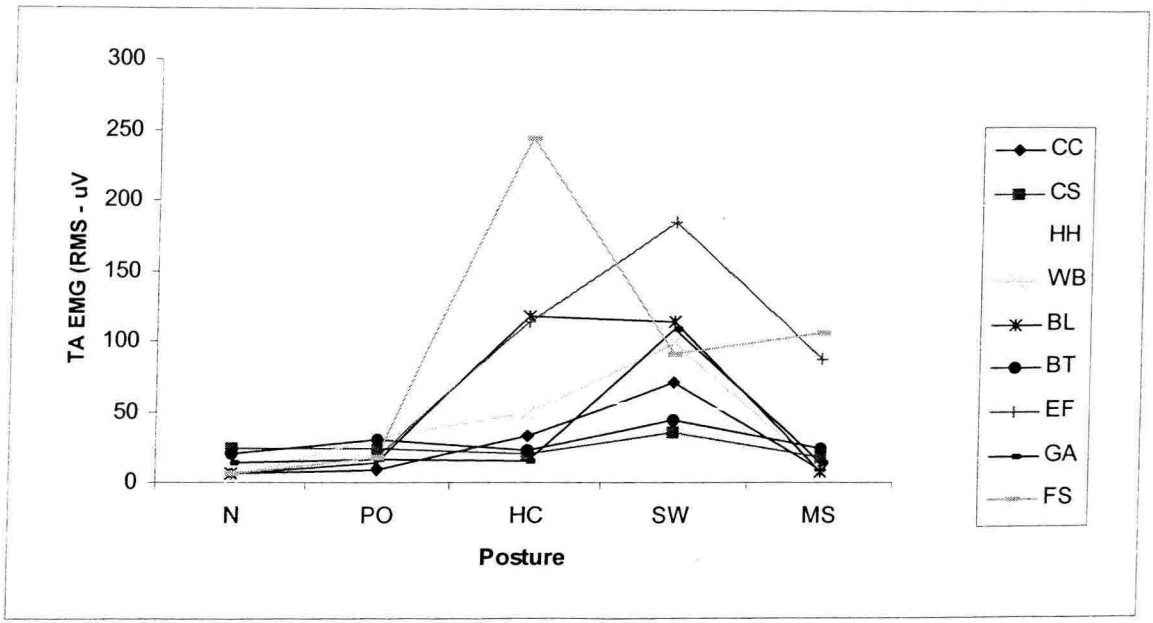


Figure 23. Tibialis Anterior (TA) EMG Activity (Individual Traces) - Patients.

Fig. 24

HEALTHY SUBJECT

Electromyogram (3 sec of raw signal)	Soleus H M-wave H-wave	Posture
TA Sol		
TA Sol		
TA Sol		
TA Sol		
TA Sol		

Fig. 25

PATIENT CC

Electromyogram (3 sec of raw signal)	Soleus H M-wave H-wave	Posture
TA Sol		N
TA Sol		PO
TA Sol		HC
TA Sol		SW
TA Sol		MS

Fig. 26

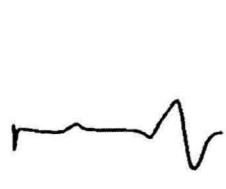
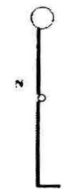
PATIENT CS

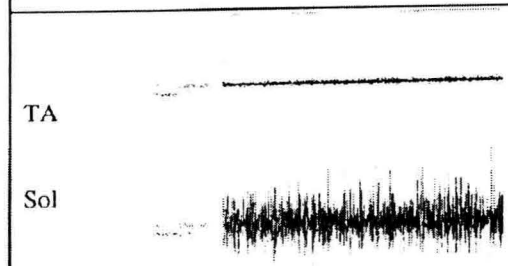
Electromyogram (3 sec of raw signal)	Soleus H M-wave H-wave	Posture
TA Sol		N
TA Sol		PO
TA Sol		HC
TA Sol		SW
TA Sol		MS

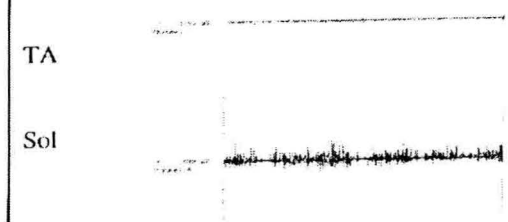
Fig. 27

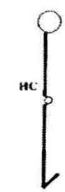
PATIENT WB

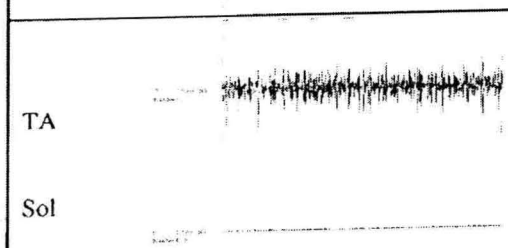
Electromyogram (3 sec of raw signal)	Soleus H M-wave H-wave	Posture
TA Sol		
TA Sol		
TA Sol		
TA Sol		
TA Sol		

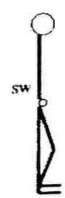














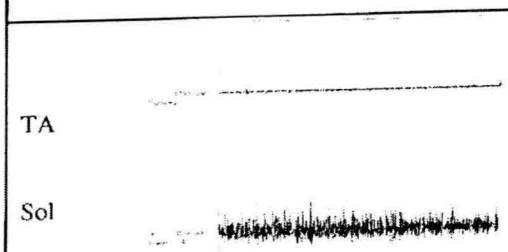


Fig. 28

PATIENT HH

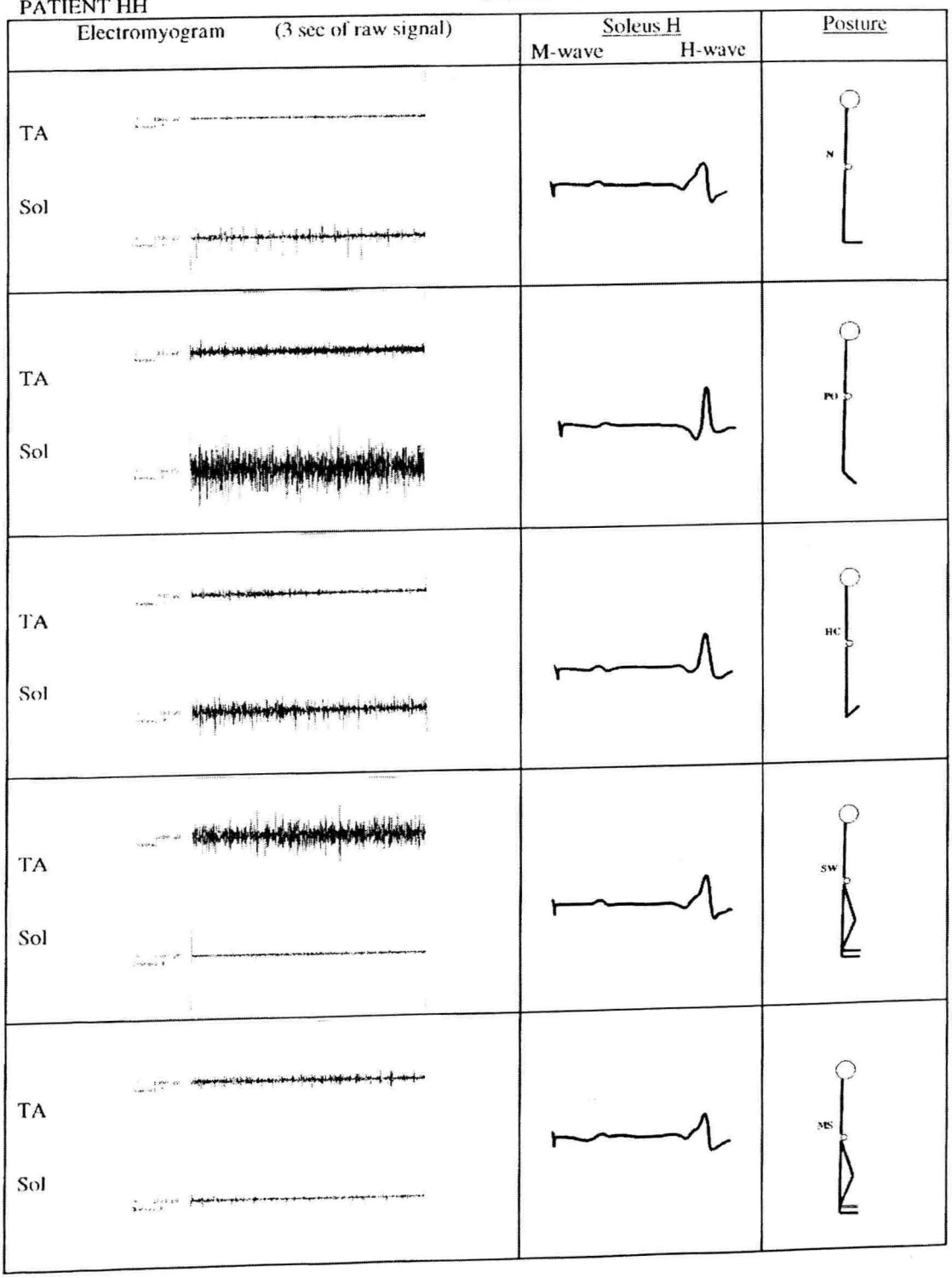


Fig. 29

PATIENT BL

Electromyogram (3 sec of raw signal)	Soleus H M-wave H-wave	Posture
TA Sol		N
TA Sol		PO
TA Sol		HC
TA Sol		SW
TA Sol		MS

Fig. 30

PATIENT BT

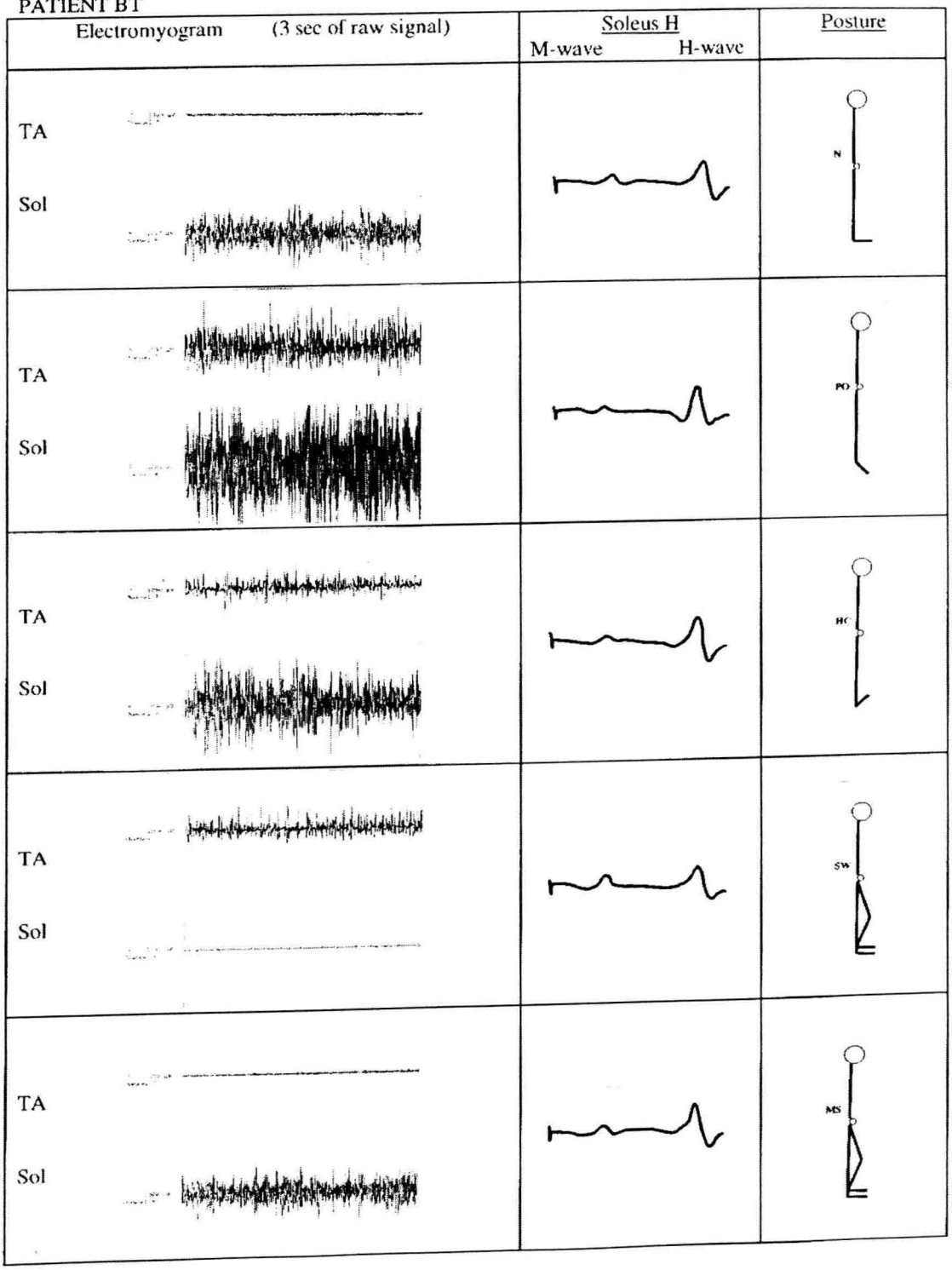


Fig. 31

PATIENT GA

Electromyogram (3 sec of raw signal)	Soleus H M-wave H-wave	Posture
TA Sol		N
TA Sol		PO
TA Sol		HC
TA Sol		SW
TA Sol		MS

Fig. 32

PATIENT EF

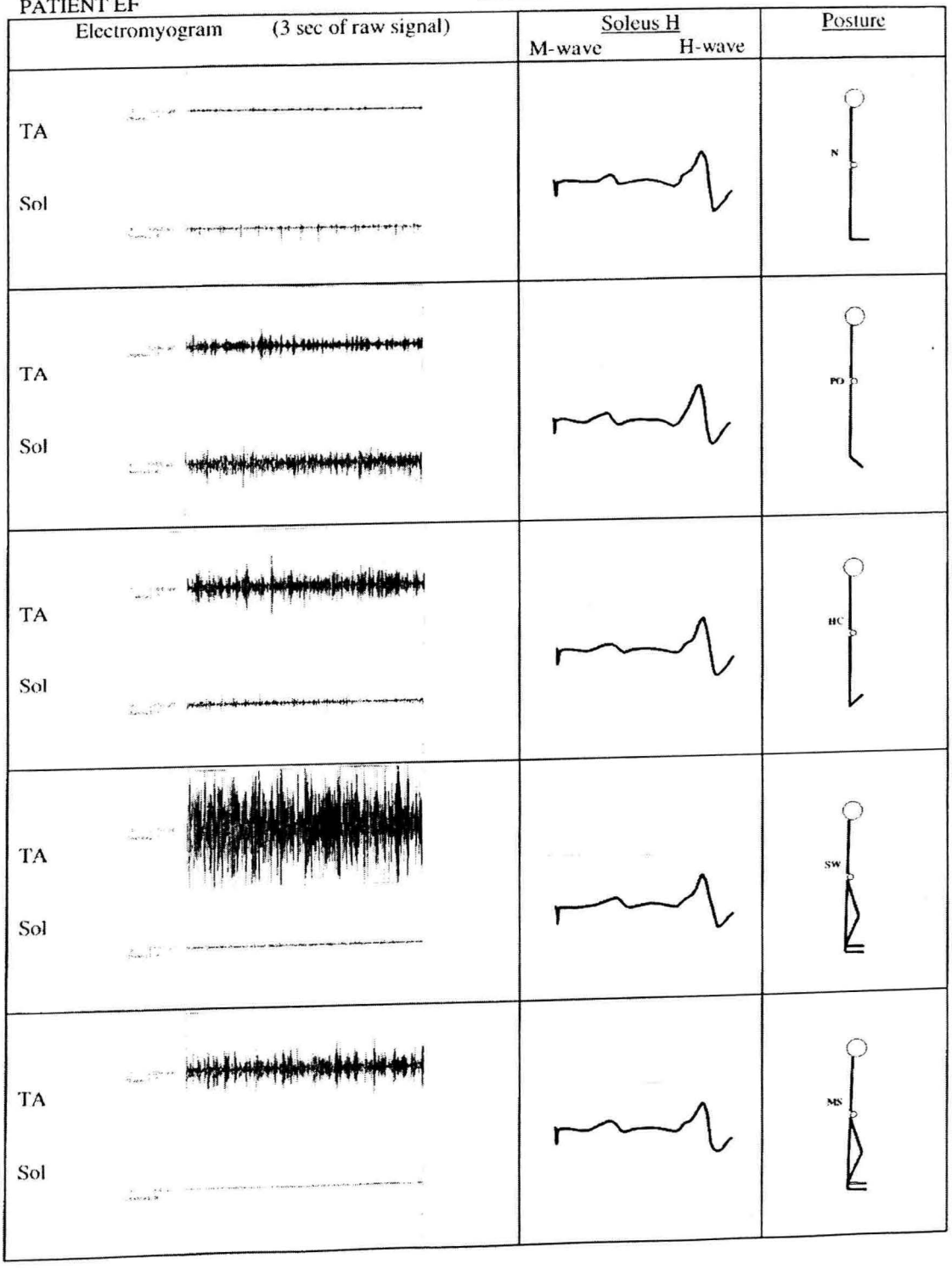
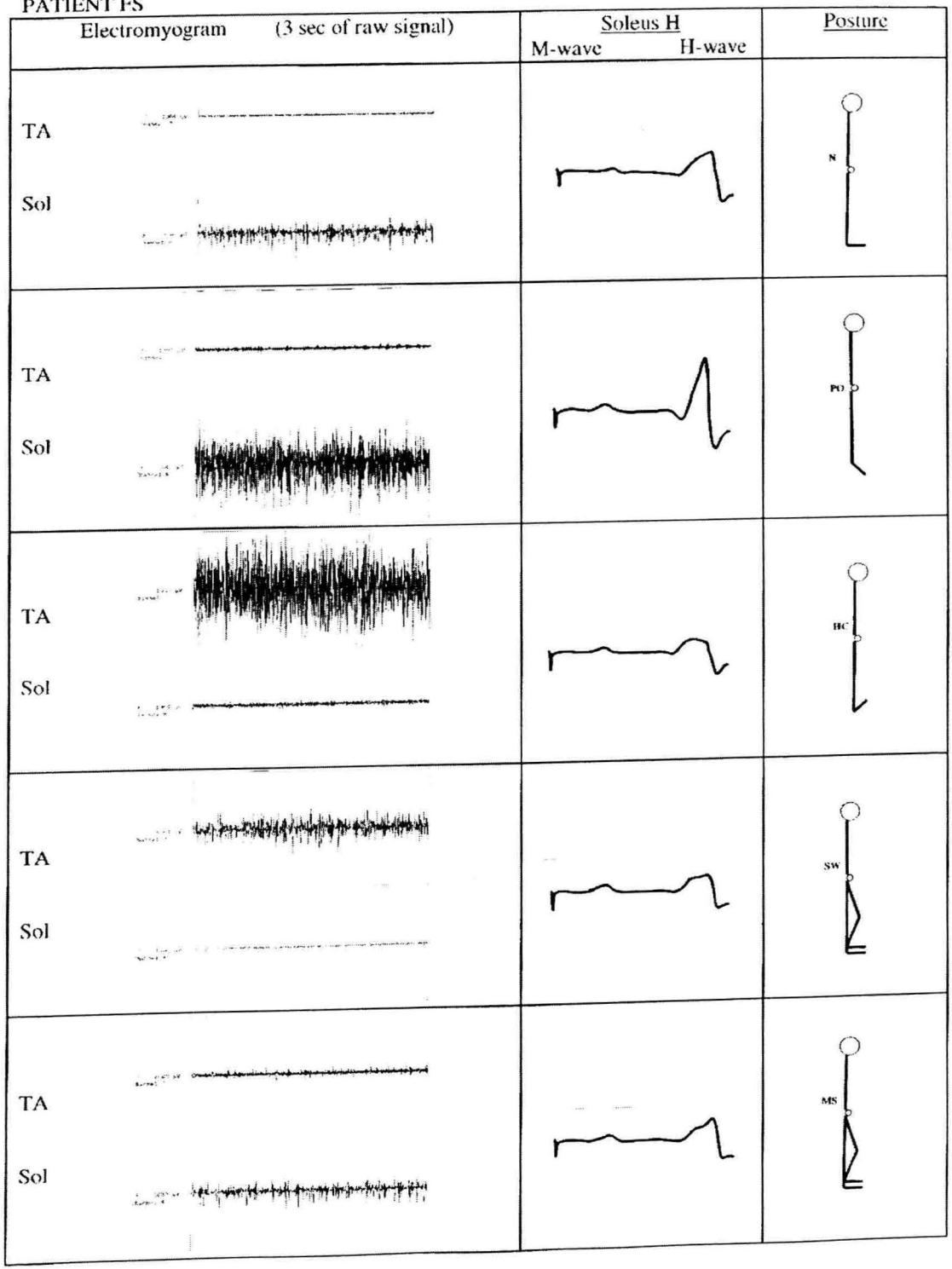


Fig. 33

PATIENT FS



Experiment 4

Effect of Reiterative Dorsiflexion on Gait Speed of Patients with Stroke: Linking the Electrophysiologic Parameters of Ankle Motor Control to Function

Introduction

Motor control at the ankle joint is important for the integrity of walking. Inability to control the ankle joint during the gait cycle, is a common impairment that affects patients with cerebrovascular accidents (CVA) or stroke. This lack of coordinated volitional and reflex interplay appears to be caused by several factors. Evidence has shown a pathological increase in stretch reflex activity of soleus during stance (Okuma & Lee, 1996), impaired presynaptic inhibition (Morin, Katz, Mazieres, & Pierrot-Deseilligny, 1982), dysfunctional reciprocal inhibition (Katz & Pierrot-Deseilligny, 1982; Okuma & Lee 1996; Yang, Fung, Edamura, Blunt, Stein, & Barbeau, 1991), and altered cutaneous (sensory) afferents of the hemiparetic ankle (Brooke, Cheng, Misiaszek, & Lafferty, 1995; Yang, Stein, & James, 1991).

It is well established that rehabilitative intervention has a positive impact in the recovery of gait in patients with CVA (Homberg, 1993). Usually, physical therapy seeks reinforcement of gait and gait-like activities aggressively in these patients. A large portion of these therapeutic activities and exercises are reiterative in nature, involving repeated stepping and reinforcement of ankle dorsiflexion (Okuma & Lee, 1996). Patients then are progressed into more complex gait activities as they recover. One common

outcome measure used to document improvement in gait function (and therefore motor control) is gait speed (Brandstater, de Bruin, Gowland, & Clark, 1983; Richards, Malouin, Dumas, & Tardif, 1995). Thus, patients that walk faster are believed to have better motor control (Friedman, 1990; Goldie, Matyas, & Evans, 1996). Reiterative ankle dorsiflexion may have influence in improving motor control of the ankle antagonists (as reflected in improvement of gait speed), possibly due to recovery of reciprocal inhibition. Okuma and Lee (1996) suggested that intensive physical therapy and the repeated attempts to produce ankle dorsiflexion (one of the most difficult tasks for patients), could over time modify synaptic transmission in the Ia inhibitory pathway to the antagonist extensors (because Ia inhibition to the extensors becomes active during voluntary ankle dorsiflexion). Morita, Crone, Christenhuis, Petersen, and Nielsen (2001) also concluded that failure to increase reciprocal inhibition, and thereby to depress abnormal stretch reflex activity in the antagonist muscle, does reflect a serious impediment for the execution of fast ankle movements in the patients. They proposed that therapy should be directed at strengthening reciprocal inhibition that could be of benefit in relieving some of the functional disability of patients with spasticity. The use of reiterative stepping and gait to benefit the increase in tibialis anterior activation along with reducing soleus stretch reflex responses has been reported from research using weight supported treadmill-walking training in hemiparetic subjects (Hesse, Werner, Uhlenbrock, von Franckenberg, Bardeleben, & Brandl-Hesse, 2001).

In a related study, we investigated patients with CVA who in spite of great conscious and physical effort were unable to activate the tibialis anterior muscle to dorsiflex their ankles while standing (closed chain). But when they were asked to lift the involved foot off the ground, as in swing phase of gait (open chain), tibialis anterior EMG activity was generated to levels observed (under the same experimental conditions) in healthy subjects. The fact that tibialis anterior EMG activity, could be generated while the foot is lifted, may provide a means to influence the recovery of the reflex and volitional interplay (Ia inhibitory pathway) as suggested by Okuma and Lee, (1996) and Morita et al. (2001). However, evidence showing that recovery of reciprocal inhibition to soleus, reflected in the inhibition of the soleus H-reflex amplitude during swing, is in fact related to functional improvement of motor control such as gait, is still lacking.

The Hoffmann reflex (H-reflex) has long been used as a means of assessing spinal reflex excitability (Magladery & McDougal, 1950; Schiepatti, 1987). In this experiment, a testing paradigm that utilized gait-simulated postures, which was used in a related study was employed to assess changes in soleus H-reflex amplitude in patients with CVA.

Therefore, the main purpose of this study was to observe the individual and group responses to a rehabilitative exercise intervention directed to improve ankle motor control as measured by gait speed. It was hypothesized that the soleus H-reflex amplitude would be positively affected (i.e., recovered or normalized) by an exercise program that consisted of repeated ankle dorsiflexion in standing (simulating initiation of swing phase) aided with EMG biofeedback from the tibialis anterior muscle. The recovery of the soleus

H-reflex inhibition during swing would be associated with recovery in motor control of the tibialis anterior (improved EMG activity) and reflected by improvements in gait speed.

Material and Methods

Participants

Nine patients with CVA (5 women, 4 men), age range 44 to 73 years (60.1 ± 8.3) participated in this study. Patients were community dwellers able to walk at least 10 meters (30 feet) without stopping, reported having no history diabetes, low back pain, cancer or orthopedic pathologies, and none was taking anti-spasticity medications.

Demographic and clinical information is summarized in Table 17. Data from seventeen healthy subjects (6 women, 11 men), age range 22 to 69 years (37.5 ± 14.4) from a related study were used for comparisons.

Design

An A-B single-case design (A: baseline, B: intervention) with repeated measures format, was used for each patient to graphically display soleus H-reflex amplitude changes before, throughout, and after the exercise training period.

Pre- and post-training group comparisons were made for gait speed (self- and fast-pace), tibialis anterior EMG (postures HC and SW), and soleus H-reflex amplitude (postures N, PO, HC, SW, and MS).

Instrumentation

H-Reflex Recording

The Cadwell 5200A electromyography unit was used to elicit and record the soleus H-reflex. Initially the skin location of stimulation and recording were slightly abraded with fine sandpaper in order to reduce skin impedance. For electrical stimulation of the tibial nerve silver-silver chloride surface bar electrodes (active lead-off surface of electrodes was 1 cm2 each, center-to-center distance between the 2 electrodes was 25 mm) were placed over the tibial nerve in the popliteal fossa. Stimulation electrodes were positioned with the cathode proximal to the anode to avoid anodal block (Fisher, 1992). Another similar bar recording electrode was placed on the mid-line over the soleus muscle, with the active electrode 3 cm distal to the distal margin of the two heads of the gastrocnemius muscle. A 2 cm diameter metal ground electrode was applied between the stimulating and recording electrodes over the calf muscle group. Electroconductive cream was applied on all electrodes' surfaces for good coupling. Once in place the electrodes were not removed until the whole experiment was completed to ensure exact placement and consistent results. A percutaneous electrical stimulus of 0.5 ms square-wave pulses was delivered at a frequency of 0.2 Hz to the tibial nerve to elicit the maximum H-reflex with minimal M response. Once the maximum H-reflex amplitude and minimal M response were determined with the subjects in the standing position, the minimal M response was kept constant throughout the eliciting/recording procedures for all the gait-

simulating postures. The signal was amplified (1000-5000X) using a band pass of 10 Hz-10 KHz.

Tibialis Anterior Electromyography

Tibialis anterior muscle was recorded and analyzed off-line using the Myosystem 1200 and its Norquest EMG evaluation/analysis software. An analog/digital KPCMCIA-12AI-C card was used to link the Myosystem to a Lifebook C340-233 MHz laptop computer. A pair of surface electrodes was placed side-by-side, over the tibialis anterior muscle belly. A single ground electrode was placed on the skin covering the head of the fibula.

Gait Speed

The GAITRite walkway was utilized in this study to calculate gait speed. The walkway and its application software was utilized to calculate gait speed. The application software, loaded into a Lifebook C340-233 MHz laptop computer, controlled the functionality of the walkway, processed the raw data and computed automatically temporal and spatial parameters. In this study a walkway containing 8 sensor pads was used. The sensors are encapsulated in a roll-up carpet to produce an active area 61 cm (24 inches) wide and approximately 488 cm (192 inches) long. The walkway is portable, can be laid over any flat surface, requires minimum setup and test time, and does not require the placement of any devices on the subject/patient.

Exercise Training and EMG Feedback Protocol

After placement of surface electrodes over the tibialis anterior muscle, patients were asked to dorsiflex maximally the involved foot while in the swing (SW) posture. A self-directed target of tibialis anterior EMG activity displayed on a monitor screen at eye level assisted the patients with the activity. A pre-set EMG-feedback program with 10 contract-up repetitions lasting approximately 5 seconds each was used to exercise the tibialis anterior muscle. Ten repetitions make 1 set. Patients performed 3 sets in each training session. Patients were also asked to perform 2 more sets of 10 repetitions while in sitting. EMG signals were sent to a computer using an analog/digital card for simultaneous display. Resting time (1-2 min) was provided between sets. Each patient attended training for 3 sessions/week for 4 weeks.

Questionnaires and Indexes

All patients were tested using two scales: 1) the Barthel ADL Index (Mahoney & Barthel, 1965) to provide information on overall functional status. And, 2) the Modified Ashworth Scale (Bohannon & Smith, 1987) to score knee and ankle joint spasticity.

Experimental Procedures

After signing the consent form, patients were scored in the Ashworth Scale for spasticity and completed the Barthel ADL Index. Soleus H-reflex and soleus and tibialis anterior EMG recordings were obtained in the following five gait-simulating postures: 1) N = freestanding (standing posture), 2) PO = push-off (standing on toes, mimics push-off during late stance phase of gait), 3) HC = heel-contact (standing on heels, mimics

transition between swing and stance phases of gait), 4) SW = swing (foot just cleared from ground, mimics toe-off and beginning of swing phase), and 5) MS = mid-stance (standing on tested leg, while contralateral foot is lifted as on the SW posture (mimics mid-stance phase of gait). Tibialis anterior EMG activity was recorded in postures HC and SW only. Postures are schematically shown in Figure 34. Patients were tested in their affected limb only. Patients were tested while standing in front of a platform adjustable walker and were asked to have their forearms resting on the walker's platforms to ensure balance. Patients were tested in several occasions throughout the experiment. Patients were required to walk approximately 10 meters (30 feet) at self- and fast-pace two times. Of the 10 meters, approximately 5 meters were covered by the instrumented walkway. The walkway was placed in the middle of the walking distance, so that the effects of acceleration/de-acceleration on gait speed would be minimized. Stand-by assistance from a helper was provided during the walking to assure balance and safety. As the patient ambulated on the walkway, the system captured the pressure of each footfall as a function of time. Data recorded was stored into the linked computer by the application software for later analyses. Gait testing was carried out at the beginning and at the end of the training period.

Data and Statistical Analyses

H-Reflex Amplitude

Three consecutive H-reflex peak-to-peak amplitudes were measured and averaged for each posture. The amplitudes were measured manually in millimeters from paper

printouts and converted to mV equivalents. Data was screened for skewness and outliers by frequencies and histograms. Student's paired t tests were used to compare pre- and post-training soleus H-reflex amplitude means across postures. Alpha was set at .05.

Tibialis Anterior Electromyography

Two 3-sec rectified tibialis anterior EMG signals were recorded, processed and averaged for each of the two postures (HC and SW). Rectified and smoothed EMG signals were quantified using a root mean square (RMS, expressed in μV) algorithm with 50 ms windows. Student's paired t tests were used to compare pre- and post-training tibialis anterior EMG. Alpha was set at .05.

Gait Speed

Stored gait data was retrieved using the GAITRite software that provided automatic display of gait speed values (cm/sec). Averages from two trials were calculated for self- and fast-paced gait. Student's paired t tests were used to assess differences between pre- and post-training. Alpha was set at .05.

Results

Gait Speed (cm/s) - Group Results

Self-paced walking was 39.8 ± 25.0 at pre-training, and increased to 45.3 ± 26.1 at post-training. Fast-paced walking was 71.8 ± 33.4 at pre-training, and increased to 88.5 ± 39.3 at post-training (Figure 35). There were two patients that could not produce a fast-

paced walking (CS and BL) Therefore, analysis of fast-paced walking was based on seven patients only.

There was a statistically significant difference between the pre- and post-training for the fast-paced walking condition (paired, two-tailed t-test, $p = .04$), but not for the self-paced walking condition (paired, two-tailed t-test, $p = .08$).

Gait Speed (cm/s) - Individual Results

Comparisons between pre- and post-training showed that gait speed was enhanced in some patients but not in others (Figure 36).

During self-paced walking, patients GA and HH showed a considerable increment in their gait speed after training. This was not the case for the remaining seven patients. During fast-paced walking, patients HH, WB, BT and GA showed a considerable increment in their gait speed after training. No changes in speed were seen for patients FS, EF and CC. According to the classification of gait speed in patients with CVA provided in the work of Richards et al. (1995), patients CC, CS and BL were very slow (Group I: under 28 cm/s); patients HH, WB, BT and GA were intermediate (group II: 28 to under 55 cm/s); and patient FS was a fast walker (Group III: above 55 cm/s). After the training, patients HH and GA became fast walkers.

Tibialis Anterior EMG (RMS - μV) - Group Results

Pre-training tibialis anterior EMG in each posture was: HC = 50.9 ± 59.4 and SW = $88.0 + 54.7$. At post-training they were: HC = 83.2 ± 89.0 and SW = 71.9 ± 36.9 (Figure 37).

There were no statistically significant differences (paired, two-tailed t-test) between pre- and post-training for HC ($p = .13$) and SW ($p = .40$).

Tibialis Anterior EMG (RMS - μV) - Individual Results

Comparisons between pre- and post-training showed that tibialis anterior EMG activity was enhanced in the heel-contact (HC) posture in relation to swing (SW) in some patients (Figures 38 and 39).

Tibialis anterior EMG activity in HC (ankle dorsiflexion in weight bearing - closed chain) represented approximately 80% of the total activity in the tibialis anterior muscle in the healthy subjects (Figure 38). After training, heel-contact (HC) improved for patients CC, CS, HH, WB, BL, and BT. It was not changed for patients EF and GA. The largest improvement in heel-contact (HC) EMG was in patients CS, HH and WB, followed by patients BT and BL. Patient FS showed a profile similar to that of healthy subjects (before training), which did not change after the training.

Tibialis anterior EMG activity in SW (ankle dorsiflexion in non-weight bearing - open chain) represented approximately 20% of the total tibialis anterior activity in the healthy subjects (Figure 38). Patients WB and BL were the only ones that showed a shift towards a normal percentage after training. Patient FS showed a similar percentage share to that of the healthy subjects and it did not change after training.

Soleus H-Reflex Amplitude (mV) - Group Results

Pre-training soleus H-reflex amplitude in each posture was: $N = 3.6 \pm 2.7$, $PO = 4.7 \pm 3.3$, $HC = 3.8 \pm 3.1$, $SW = 3.6 \pm 2.7$, and $MS = 3.4 \pm 2.9$. At post-training they

were: N = 2.3 ± 1.1, PO = 4.9 ± 2.9, HC = 2.9 ± 1.8, SW = 3.3 ± 2.0, and MS = 2.6 ± 1.0 (Figure 40).

There were no statistically significant differences (paired, two-tailed t-test) between pre- and post-training for soleus H-reflex amplitude in all five postures: N ($p = .12$), PO ($p = .64$), HC ($p = .24$), SW ($p = .33$), and MS ($p = .43$).

Soleus H-Reflex Amplitude (mV) - Individual Results

Comparisons between pre- and post-training, revealed that soleus H-reflex amplitude showed inhibition gains in heel-contact (HC) and swing (SW) postures in some patients but not in others (Figures 41 and 42).

The pattern of soleus H-reflex amplitude showed little or no change at all throughout the postures for patients CC, CS, EF and GA. In patient GA, although did not present changes in its pattern, the overall trace (i.e., the line linking the five postures) was shifted upward, toward the boundaries of that of the healthy subjects (Figure 41, discontinued line). Intermediate changes were observed for patients HH and WB. A most definitive recovery towards the normal pattern of soleus H-reflex amplitude inhibition during heel-contact (HC) and swing (SW) was observed for patients BL and BT. In patient BT, the overall trace was also shifted toward the normal excitability levels (Figure 41, discontinued line). Patient FS showed a pattern that matched the normal modulation, which did not change after the training.

Discussion

In this study patients were investigated before and after a training exercise program directed to improve the motor control, as reflected in gait speed improvements, of the ankle antagonists. The exercise paradigm emphasized reiterative ankle dorsiflexion in standing (open chain mechanism) simulating the early swing movement of gait cycle.

The results of this study showed, and in agreement with the reports of Okuma and Lee (1996) and Morita et al. (2001), that patients differ greatly in the manifestation of their abnormal neurophysiological profiles (e.g., pattern of soleus H-reflex amplitude changes throughout postures, tibialis anterior EMG activity), from being relatively normal (patient FS in this study) to a complete disruption or lack of soleus H-reflex inhibition in HC and SW postures (patients CS and EF). Correlation of the electrophysiological parameters (i.e., soleus H-reflex amplitude and tibialis anterior EMG) and clinical data of the patients allows to suggest: (1) Those patients that showed improvement in the fast-pace mode of gait (HH, WB, BT and GA), all but GA showed an improvement in tibialis anterior EMG activity, mainly in the heel-contact (HC) posture. For soleus H-reflex amplitude, out of four patients, three (HH, WB and GA) showed some recovery of inhibition of the H-reflex in postures HC and SW. Patient BT showed good recovery of inhibition of the soleus H-reflex in postures HC and SW. And of the four, only GA had an Ashworth score of 4 at the ankle, the other three had their spasticity scores at 2 or lower. Thus, change in gait speed (fast-paced) appeared to relate to those who show the most improvements in tibialis anterior EMG activity in the closed-chain

HC posture (possibly improving Ia inhibition pathway to soleus muscle) with low spasticity scores, and modest to good recovery of inhibition of the soleus H-reflex amplitude in HC or SW, or both. (2) Those patients who did not change gait speed (CC and EF, at fast-paced mode and those who could not produce a fast-paced speed, CS and BL) all improved moderately tibialis anterior EMG activity in the HC posture, but patient EF. Patient BL made the largest tibialis anterior EMG gain, although no improvement in gait speed was made. For soleus H-reflex, of the four, patient BL was the only to show a remarkably recovery of amplitude inhibition in the HC and SW postures. And of the four, all but CS had an Ashworth score of 4 at the ankle. Thus, the lack of change in speed (fast-paced) appeared to relate to those who showed low or none tibialis anterior EMG activity gains (specifically, during HC), had higher spasticity scores, and continued to display a lack of soleus H-reflex amplitude inhibition during HC and SW.

Patient BL, although showed the most improved recovery of the soleus H-reflex amplitude inhibition in HC and SW, was also the one with the lowest overall functional status (Barthel score of 16). Patient FS was in all assessed aspects within normal limits, showing a fully working state of motor control interplay at the ankle antagonists.

Changes in gait speed, however, are also dependent in performance at other levels of the lower limb different from the ankle. For example, self-paced walking or natural gait speed was reported in adults with CVA to depend mainly on the strength of the hip flexors ($R^2 = 0.69$); and for fast-paced walking, it depends on the strength of the hip flexors and sensation integrity ($R^2 = 0.85$) (Nadeau, Arsenault, Gravel, & Bourbonnais,

1999). Thus, gait speed may be improved by strength gains made proximally in the affected limb, without intrinsic motor control recovery made distally in the limb (ankle).

Conclusions

The results of this study showed that patients who were established CVA survivors differ greatly in their manifestations of abnormal motor control at the ankle antagonists, from being relatively normal to a complete disruption. It appears that repetitive ankle dorsiflexion (during open chain or swing) exercise in standing, assisted with EMG biofeedback may have contributed to the recovery of the neuromotor interplay at the ankle muscles in the majority of the patients. Recovery of inhibition of soleus H-reflex amplitude during heel-contact (HC) and swing (SW) postures appeared to be more associated with the recovery of EMG activity in the tibialis anterior (during HC or closed chain posture), implying an improved volitional control, than to the level of soleus spasticity. Lack of recovery of inhibition of the soleus H-reflex amplitude during heel-contact (HC) and swing (SW) appeared to relate more to those who showed higher spasticity scores associated with low EMG activity of the tibialis anterior muscle during HC posture, implying a severely impaired volitional control. Gait speed improvements, however, were achieved in some patients although they retained persistent spasticity and poor soleus H-reflex inhibition recovery. In addition, gait speed improvements may also depend on performance at other levels of the lower limb different from the ankle, especially strength of the hip flexors, and on the integrity of sensation.

Due to the complexity of the underlying factors involved in a CVA and the disparity of its clinical and functional manifestations, individual differences in recovery profiles may not be seen if patients are assessed with methods using group analyses. Research using single-case format seemed justified to continue seeking further understanding of the abnormal motor control in stroke.

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Table 17. Demographic and Clinical Data of Participating Stroke Survivors.

Subject	Age (yr)	Sex	Mass (Kg)	Height (m)	TFO (mo)	Paretic side	HD	Speech ability	Sensory	WAid	AFO	ASHs K / A	Clonus	BARi
CC	63	F	81.6	1.65	67	R	R	Cons	Cons	Q-cane	Yes	3 / 4	Yes	19
CS	44	F	63.5	1.68	25	R	R	Imp	Cons	Q-cane	Yes	0 / 0	No	19
HH	62	F	65.8	1.75	20	L	R	Cons	Cons	None	Yes	0 / 1	Yes	20
WB	64	F	78.9	1.63	69	L	R	Imp	Cons	None	Yes	1 / 2	Yes	20
BL	66	M	77.1	1.70	180	R	R	Cons	Cons	Cane	No	3 / 4	No	16
BT	56	M	74.8	1.75	46	R	R	Imp	Imp	None	No	0 / 1	Yes	20
EF	53	M	80.3	1.73	51	L	R	Cons	Imp	None	Yes	0 / 4	Yes	20
GA	60	M	88.5	1.75	15	R	R	Imp	Imp	Q-cane	Yes	2 / 4	Yes	20
FS	73	F	56.7	1.63	41	R	R	Cons	Cons	None	No	0 / 0	No	20

Sex (M=male,F=female) TFO=time from CVA onset (months) R=right Imp=impaired
HD=hand dominance ASHs=modified Ashworth Scale (1-5, K=knee, A=ankle) L=left Cons=conserved

Waid=walking aid AFO=ankle foot orthosis
BARi=Barthel Index (0-20)

Table 17. (Continuation)

Subject	Location of brain lesion(s)
CC	Infarction involving the left middle cerebral artery.
CS	Clot in left internal carotid artery with large infarct involving the left middle cerebral artery.
HH	Focal lesion (infarction) within the right posterior corona radiata adjacent to the right occipital horn.
WB	Middle cerebral artery infarction secondary to clot mechanism.
BL	Left internal carotid artery clot involving the middle cerebral artery.
BT	Occluded left internal carotid artery resulting in left middle cerebral artery infarction.
EF	Trombotic occlusion of right internal carotid artery with large infarct involving the right middle cerebral artery.
GA	Complete occlusion of left internal carotid artery with infarct involving the left middle cerebral artery.
FS	Posterior cerebral artery trombotic occlusion.

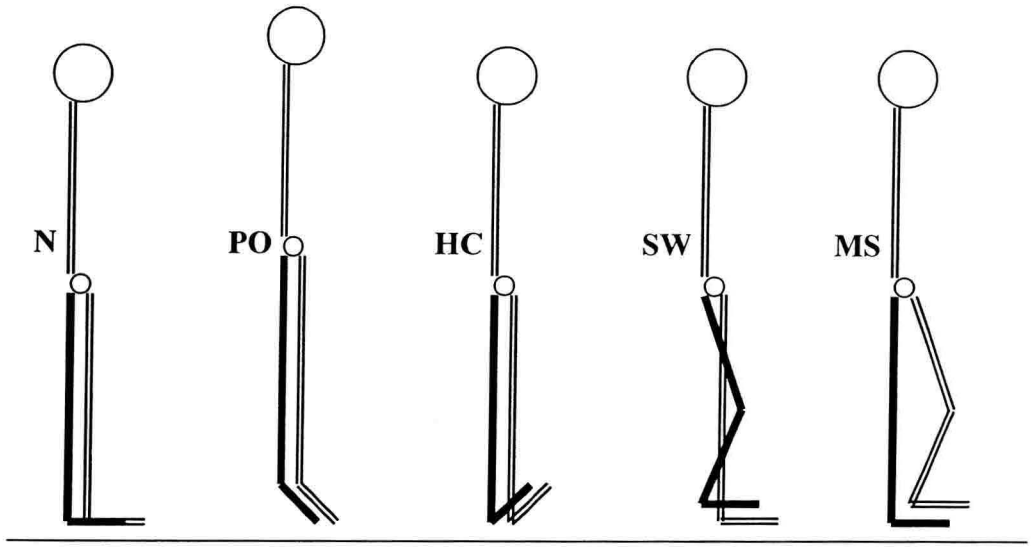

Figure 34. Gait-Simulating postures. N= free standing, PO= push-off, HC= heel-contact, SW= swing, MS= mid-stance. The solid black lines represent the tested limb.

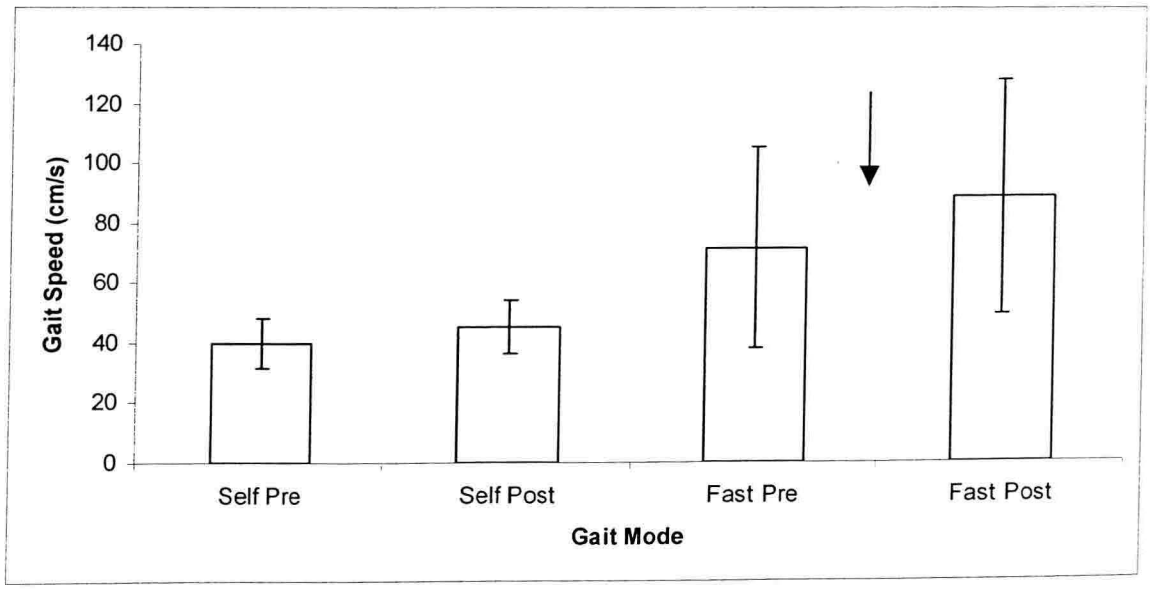

Figure 35. Means and *SEM* for Self-Paced and Fast-Paced Gait Speed. The Arrow Indicates a Statistically Significant Difference between Pre- and Post-Training for the Fast-Speed Condition only.

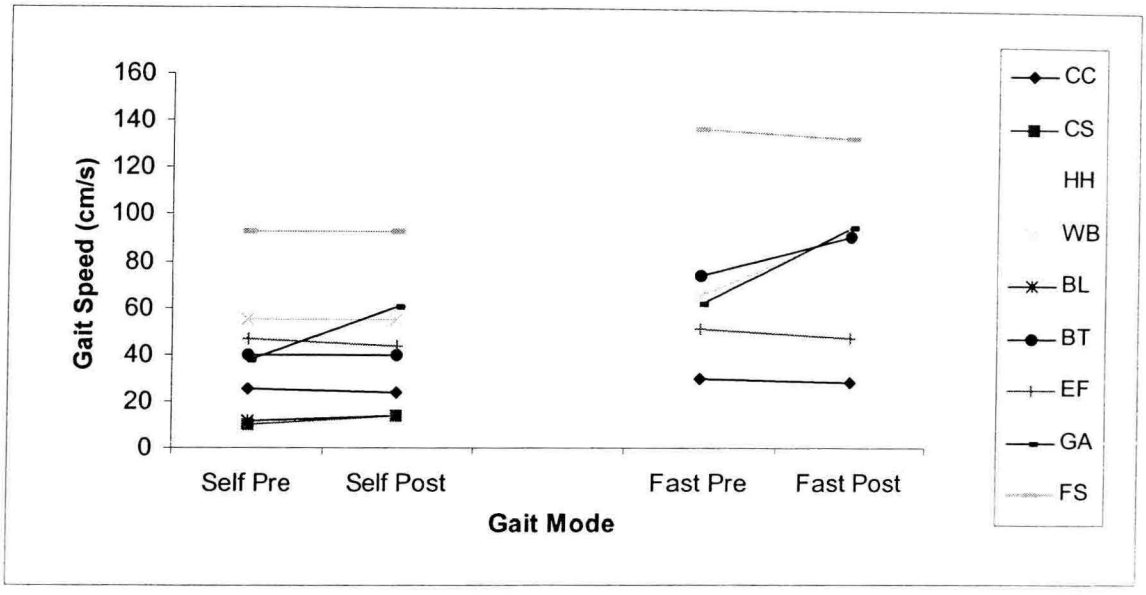

Figure 36. Individual Measures for Self-Paced and Fast-Paced Gait Speed.

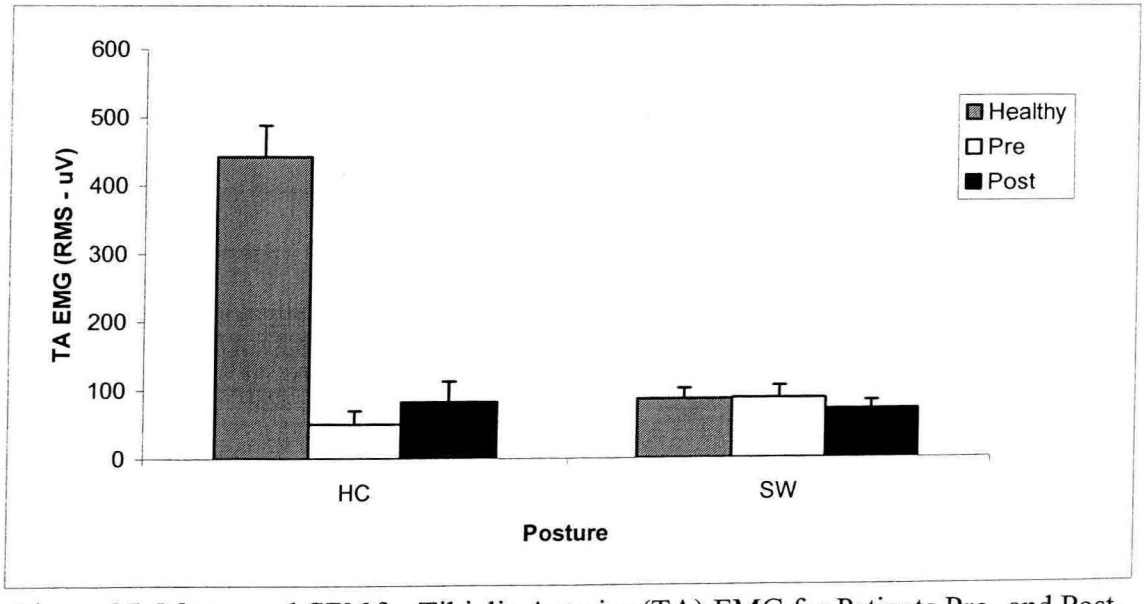

Figure 37. Means and *SEM* for Tibialis Anterior (TA) EMG for Patients Pre- and Post-Training. Mean and *SEM* of Healthy Subjects is also Displayed for Comparison. (HC: Heel-contact, SW: Swing).

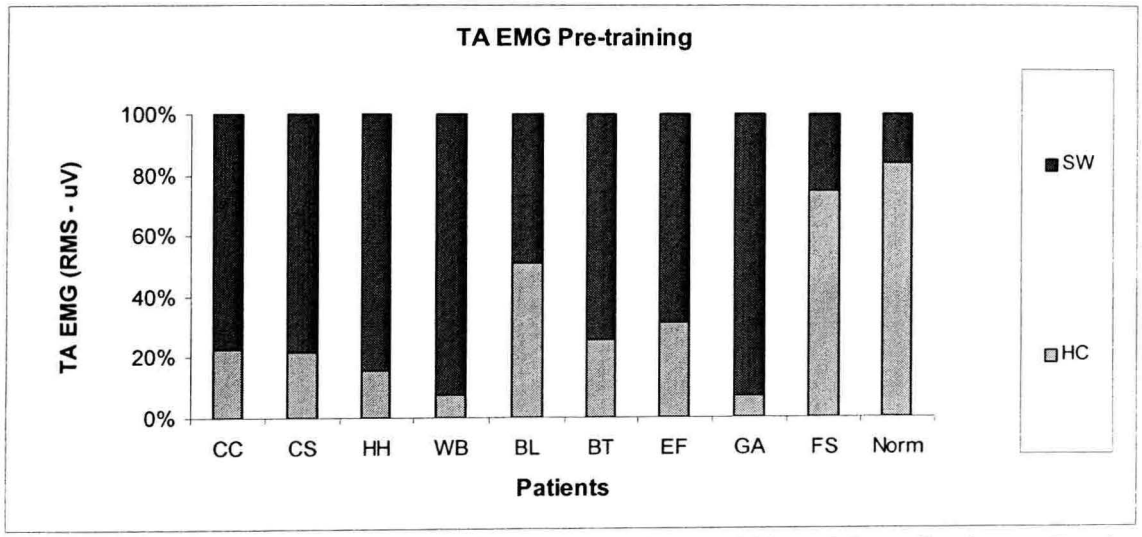

Figure 38. Individual Tibialis Anterior (TA) EMG (% of Shared Contribution to Total TA EMG) during heel-contact (HC) and swing (SW). Norm = Healthy Subjects.

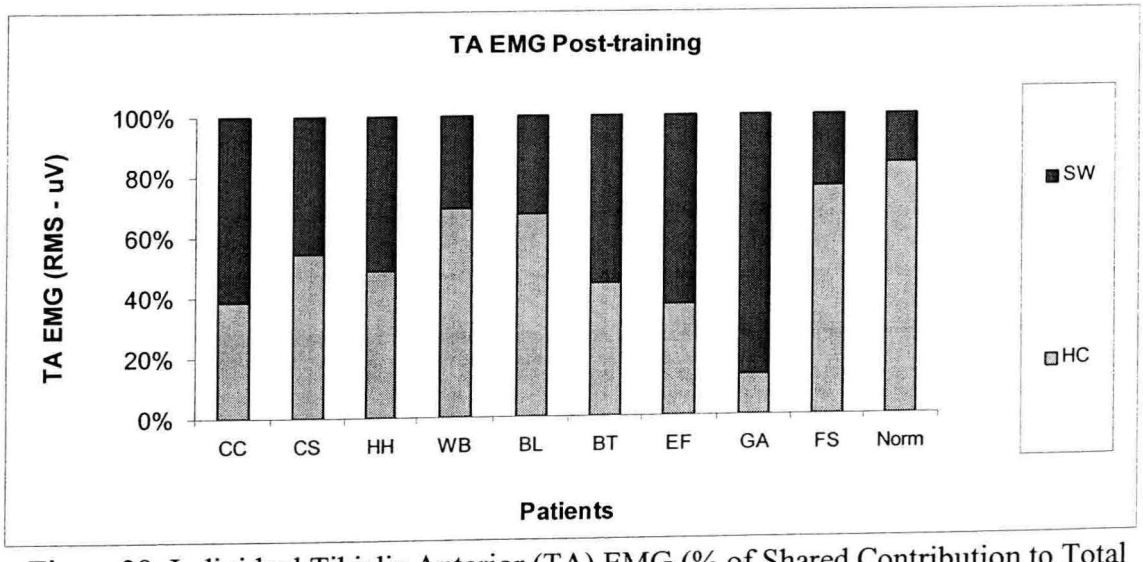

Figure 39. Individual Tibialis Anterior (TA) EMG (% of Shared Contribution to Total TA EMG, as in Figure 38), during HC and SW. Norm = Healthy Subjects

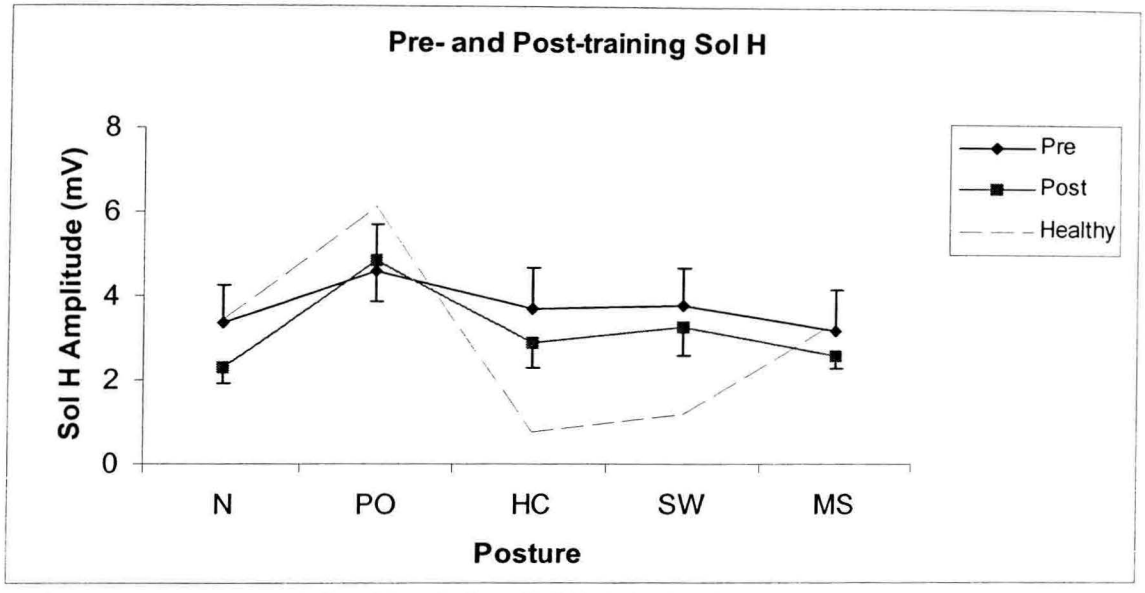

Figure 40. Means and *SEM* for Soleus H Modulation in Patients (solid lines) Pre- and Post-Training. Soleus H-Reflex Modulation from Healthy Subjects is shown for Comparison (discontinued line).

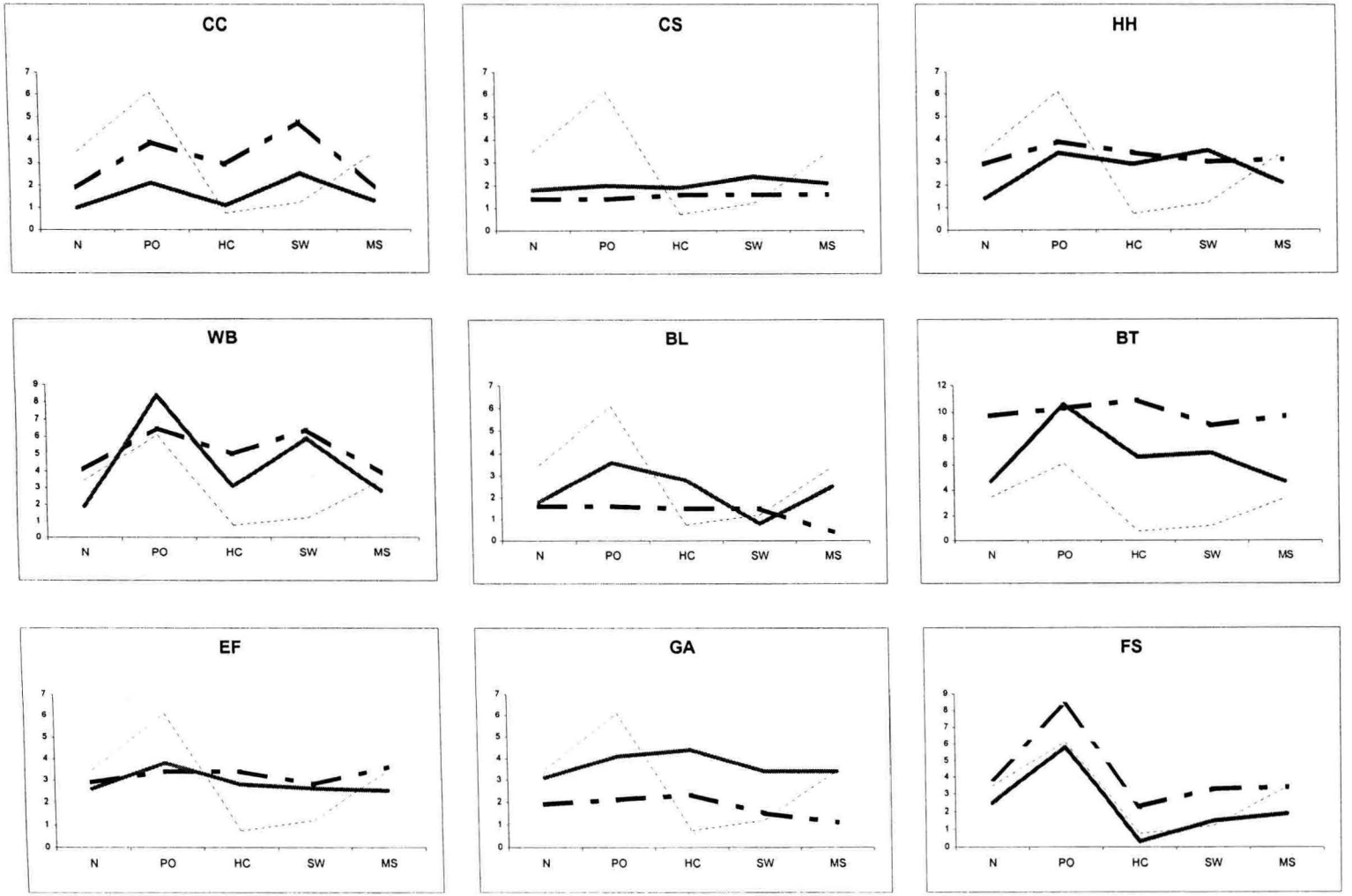

Figure 41. Pre-training (chain-like, gray line) and Post-training (solid, black line) Soleus H Pattern of Amplitude Modulation for each Patient.
Soleus Normal H-Reflex Pattern (thin, discontinued line). Y Axis= H Amplitude; X Axis= Postures.

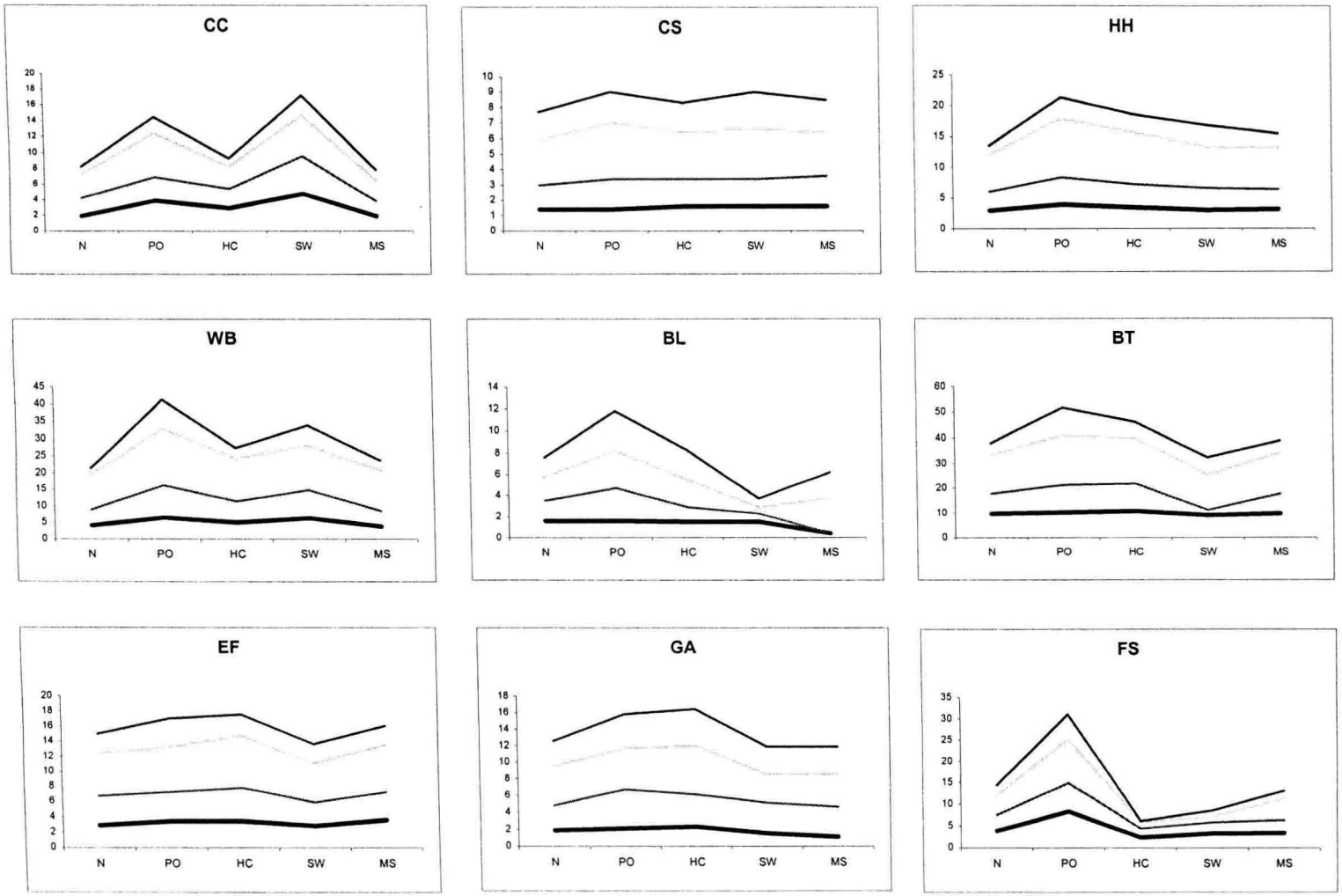

Figure 42. Time SeriesGraphs for each Patient Showing the Relative Weekly Changes in the Soleus H-Reflex Amplitude. Baseline= Solid, Thick Line. Weeks 1-4= Solid, Thin Lines (weeks in ascending order); N= Freestanding, PO= Push-Off, HC= Heel-Contact, SW= Swing, MS= Mid-Stance.

CHAPTER V

GENERAL DISCUSSION AND CONCLUSIONS

In this Chapter, Discussion and Conclusions from each one of the completed experiments were combined to present an integrated view of the outcome of this investigation.

Soleus H-Reflex Amplitude in Healthy Subjects

The results of this study showed high intersession and intrasession reliability coefficients for the H-reflex amplitude and H-reflex latency in all five postures. The intersession reliability found in this study for the mid-stance posture (MS) in the healthy subjects was similar to that reported by Hopkins et al. (2000), which was from H-reflex recordings obtained in one-leg standing (0.82 and 0.80 respectively). The intersession reliability values found in the present study for the freestanding posture (N) in the healthy subjects was similar to that reported by Handcock et al. (2001), which was from H-reflex recordings obtained in a comparable standing posture (0.88 and 0.87 respectively). Intersession reliability in these gait-simulating postures demonstrated the consistency needed to detect changes in maximum H-reflex over a period of several days. This consistency of the recordings in standing weight-bearing posture, and in agreement with Hopkins et al. (2000) and Handcock et al. (2001), is important to assess changes in motoneurone excitability due to injury or trauma. Ali and Sabbahi (2001) also pointed out

that during weight-bearing postures in standing (as opposed to lying supine) the spinal reflexes become more stable, more consistent and with less variability.

In the present study intrasession reliability coefficients were high between some postures and of lower order between others. A possible explanation of this could be that among the postures used to record the H-reflex, some were relatively more relaxed and stable with minimal muscle activation at the ankle such as freestanding (N) and mid-stance (MS), and others were actively sustained such as push-off (PO) and heel-contact (HC). In the latter two postures and in contrast with freestanding and mid-stance, there was a degree of co-contraction of the ankle antagonists since balance was challenged by balancing the body-weight either on heels or toes (small base of support). This possibly involved constant adjustment (increasing variability) in the muscles firing rates, which may have affected the consistency of the soleus H-reflex recordings. Supporting this notion, Funase and Miles (1999) demonstrated that the amplitude of the H-reflex was consistently dependent on the level of background EMG activity. Larger H-reflex amplitudes were exhibited at higher levels of activity and smaller when the tonic activity was low; and that trial-to-trial variability in H-reflex amplitude responses was also dependent on the small changes in the level of activity in the motoneuronal pool that occur from one instant to the next.

Soleus H-Reflex Amplitude in Patients

The results showed high intersession reliability coefficients in four postures for the H-reflex amplitude and in all five postures for the H-reflex latency. The lower order

coefficient seen for the swing (SW) posture (0.61) may be explained by the presence of some triggering of stretch-induced discharges in the soleus muscle as a result of the dorsiflexion movement. With the foot off the ground (open chain) its cutaneous and proprioceptive stimuli have been reduced significantly. Ali and Sabbahi (2000) previously recorded similar results, when comparing lying (open chain) with standing (closed chain) posture. Apparently, such afferent information generated from the skin, joint and muscle proprioceptors during standing helped to stabilize the reflexes inducing less variability. The intrassession reliability coefficients, calculated for each of the two sessions, were similar to those of the healthy subjects, indicating a comparable level of variability.

Soleus H-Reflex Latency in Healthy Subjects and Patients

Reliability of the H-reflex was high in all postures for both, healthy subjects and patients. Reflex latency is a measure of the conduction time of the neural signal along the reflex pathway/arc. This conduction time is not altered by subject position or between different testing sessions. The latency parameter does not change unless axonal conduction is affected as in radiculopathy, demyelination or axonal loss (Braddom & Johnson, 1974). The difference of H-reflex latency between healthy subjects (approximately 31 ms) and patients (approximately 34 ms) although under the normal upper limit of 35 ms, could be explained by the mean age difference (approximately 20 years) between the two groups (Fisher, 1992).

Soleus H-Reflex Amplitude

Soleus H-reflex amplitude was obtained during selected static postures that were simulating specific phases and transition points of the gait cycle. Our results showed that the soleus H-reflex amplitude varied greatly across the postures, thus confirming our initial hypothesis. The results of this study confirmed the early observations made by Crenna and Frigo (1987) suggesting that comparable soleus H-reflex variation (compared to that from gait studies) could be seen by adopting static gait-simulating postures. As it was expected, the large changes in H-reflex amplitude seen in these static postures paralleled those reported for the stance and swing phases of gait from previous studies (Capaday & Stein, 1986, 1987; Crenna & Frigo, 1987; Ferris et al., 2001; Llewellyn et al., 1990; Simonsen & Dhyre-Poulsen, 1999; Simonsen et al., 1995).

Of all five postures, push-off (PO) showed the largest H-reflex amplitude and paralleled that of the stance phase of walking, where a large number of soleus α-motoneurones are activated. Swing (SW) and heel-contact (HC) showed the smallest H-reflex amplitude (no statistical difference between these two) and paralleled that of the heel-contact and swing phases of walking (Figures 10-11 and 12-13). The soleus H-reflex amplitude was, as previously reported by Capaday and Stein (1986), similarly inhibited during gait at the two points where tibialis anterior was more active (heel-contact and swing). During swing (SW), however, tibialis anterior EMG activity was considerably lower than in heel-contact (HC). This finding add support to the theory suggesting that changes in the soleus H-reflex amplitude during the course of the gait cycle depends,

among others, on central mechanisms (e.g., pre-synaptic inhibition, central pattern generator) beyond the excitation level of α-motoneurones (Morin et al., 1982; Yang & Whelan, 1993).

Mid-stance (MS) posture was not different from the freestanding (N) meaning that H-reflex amplitude during static unipedal standing is comparable to that found in static bipedal standing. This stability or invariability between these two postures maybe explained due to the fact that subjects were required to rest their hands onto a walker for balance support during testing. Soleus H/M ratios found in this study in all postures were below 0.7. Delwaide (1984) reported this ratio as the normal upper limit activation in healthy subjects. The H/M ratios correlated highly ($r = 0.96$) with soleus H-reflex amplitude, fact that may alleviate the need to record Mmax (requiring an often uncomfortable stimulus) in the assessment of the soleus H-reflex.

As expected, because subjects were healthy individuals, the soleus H-reflex latency was not affected across postures. The average latency found in this study (30.53 ± 1.80, Table 11), was similar to that reported by Braddom and Johnson (1974) for the calf H reflexes (29.8 ± 2.74). The present findings also matched closely the 30 ms suggested by Han, Kim, and Paik (1997) as cut-off criteria for normality.

Soleus and Tibialis Anterior EMG Activity

As expected, soleus EMG activity (Figure 12) also varied greatly across postures and paralleled that of the soleus H-reflex (Figure 10). The greater soleus EMG activation (amplitude) occurred during the push-off (PO) corresponding to stance and push-off of

walking. The lesser soleus activation occurred in the swing (SW) posture where the foot was lifted off ground (to initiate swing phase in gait), indicating the almost complete absence of activation of the soleus α-motoneurone pool. This is critical in normal gait, to allow an undisturbed ankle dorsiflexion for the advancing foot to clear the ground. Soleus activity during mid-stance (MS) was comparable to that seen in freestanding (N), possibly indicating that support provided by the walker in the one-leg posture was enough to maintain ankle stability. That is, at higher levels of soleus activation, higher soleus H-reflex amplitude was observed (i.e., reflecting the large activation of the soleus motoneurone pool), and the soleus EMG activity varied the most in the push-off (PO) posture and the least in the swing (SW) posture (Table 11). This variability in soleus activity between-subjects was noted by Arsenault, Winter, and Marteniuk (1986). These authors found, in a small sample of eight subjects, significant differences in the profiles of EMG activity, especially for the rectus femoris and soleus muscles. Although intra-subject variability was very low, he warned against the use of normal EMG profiles, since biological differences could be averaged out in the pooled between-subject data. The low variability seen for the soleus EMG in swing (SW) could possibly been originated in the low activation levels of the muscle during this phase, which was similarly displayed by all the subjects (Figure 13).

As it was with the soleus EMG, the tibialis anterior EMG activity varied considerably across the postures (Figure 12). Previous gait studies have shown bursts of tibialis anterior activation mostly during the heel-contact (co-contraction with soleus) and

early swing. Similarly, our results showed that the greater tibialis anterior activation was in the heel-contact (HC) posture, followed by swing (SW). Heel-contact (HC) was also the posture with the greater variability (Table 10). This variability may be explained by the large amounts of tibialis anterior activation (in co-contraction with the soleus) that was required from the heel-contact (HC) posture to maintain standing on heels within a reduced base of support. During push-off (PO) posture, tibialis anterior showed some activation that possibly was originated from co-contraction (with soleus) to increase ankle stiffness. This activation was relatively small, since it was similar to that seen in the swing (SW) posture. Tibialis anterior activation in the mid-stance (MS) and freestanding (N) posture was low, indicating possibly the more stable conditions of the postures with reduced demand of muscle activation to off-set balance fluctuations (Capaday & Stein, 1986; Stein & Capaday, 1988).

The tibialis anterior showed an EMG activity that was reciprocal to the soleus H-reflex amplitude (Figures 14 and 10). That is, where soleus H-reflex was high (e.g., push-off) tibialis anterior EMG was low, and vs.. This suggested that in healthy subjects, and as previously reported by Stein and Capaday (1988) and others (Crenna & Frigo, 1987; Petersen et al., 1999), a close functional link exist between the volitional and reflex interplay of the ankle antagonists that are closely matched to the requirements of locomotion. Since the role of the soleus muscle (and other ankle extensors) is to control the rate and extent of ankle flexion from heel-contact (HC) to push-off (PO) and to propel the body forward and upward, a large reflex would be most desirable to assist with this

propulsive period. During early swing (SW), the toe comes off the ground and the ankle starts to dorsiflex to clear the ground, the soleus muscle is again stretched. But, a high reflex with a contracting soleus muscle would be inappropriate, because it would counteract the tibialis anterior muscle contraction.

As in the swing (SW) phase of gait, maximum inhibition of soleus H-reflex can be achieved by simply lifting the foot off the ground (SW). This phenomenon may prove to be an important element for further investigation in subjects who have suffered a CVA. It may be a potential mechanism of inhibition for abnormal soleus α-motoneurone activity, which has been suggested to contribute to the impairment of dorsiflexion ability in patients with CVA.

Soleus H-reflex Amplitude - Comparison Between Healthy Subjects and Patients

Our results showed, and as previously reported (Yang et al., 1991), that the large changes observed in the soleus H-reflex amplitude in the healthy subjects was profoundly disrupted (i.e., lack of inhibition during swing) in patients with CVA. The pattern of soleus H-reflex amplitude varied considerably among patients, from being almost normal in one patient (i.e., facilitated during stance and inhibited during swing) to a complete absence of inhibition during swing in others. In this study, soleus H-reflex differences were clearer in the heel-contact (HC) and swing (SW) postures. This indicated that in patients and as opposed to the healthy subjects, soleus motoneurone pool excitability remained high (i.e., was not inhibited) in the heel-contact (HC) and swing (SW) postures.

During push-off (PO), patients showed some increase in soleus H-reflex excitability, but not at the level of the healthy subjects.

In healthy subjects, soleus H-reflex inhibition at heel-contact (HC) may possibly be the result of reciprocal inhibition from tibialis anterior, muscle that was strongly contracted in this posture. In swing (SW), the profound soleus H-reflex depression may only be partially due to the result of reciprocal inhibition from tibialis anterior muscle, since here a much lower tibialis anterior contraction (i.e., to overcome the weight of the foot) was taking place. Morin et al. (1982) and Yang and Whelan (1993), suggested that an additional powerful mechanism of soleus inhibition may be the responsible, most likely central presynaptic inhibition of the primary afferents. In any case, the mechanism was profoundly disrupted in the patients.

The average latency found in the patients (Table 16) was slightly higher than that of the healthy subjects (Table 15), but still under the 35 ms suggested by Fisher (1992) as the upper limit of normality. Age is related to increased soleus H-reflex latency (Fisher, 1992), and possibly explains the differences observed between healthy subjects and patients (a 20 year difference in their averaged ages).

Soleus EMG Activity - Comparison between Healthy Subjects and Patients

Unexpectedly, soleus EMG activity in the patients was remarkably similar to that of the healthy subjects (Figure 19). In gait studies, patients are reported to generate abnormal EMG discharges during the stance phase (in this study the push-off [PO] posture) triggered by the muscle stretch (eccentric contraction of soleus) induced by the

forward moving shank over the fixed foot (Yang et al., 1991). In this study push-off (PO) required the soleus to contract concentrically, since while lifting the heel the shank was not simultaneously being advanced, as it is the case during normal gait. This suggested that the paretic soleus behave "normally" during concentric activity. Further, since the soleus EMG activity of the patients was apparently similar to that of the healthy subjects, it was "normally low" at heel-contact (HC) and swing (SW), contrasting with the high excitability of its motoneurone pool (soleus H-reflex, Figure 17). This exposed a disruption of the association between the soleus H-reflex with its observed level of background activity (EMG) that was clearly different from that of the healthy subjects, where the soleus H-reflex was largely correlated to the volitional EMG activation of the soleus muscle.

Tibialis Anterior EMG Activity - Comparison between Healthy Subjects and Patients

Normally, tibialis anterior EMG activation during gait cycle was reported to show two peaks (Burridge, Wood, Taylor, & McLellan, 2001; Knutsson & Richards, 1979; Yang et al., 1991): one at the heel-contact (HC in this study) related to the active landing of the foot on the ground; and, another at the early swing (SW in this study) related to the clearance of the ground by the advancing foot. In the patients of this experiment and as reported in previous gait studies (Burridge et al., 2001; Knutsson & Richards, 1979; Yang et al., 1991) lack of motor control and poor (totally absent in some cases) muscle activity of the tibialis anterior muscle were clearly exposed in the heel-contact (HC) posture where patients could not lift their toes or dorsiflex their involved foot off-ground.

The tibialis anterior EMG activity of the patients was unexpectedly similar to that of the healthy subjects in the swing (SW) posture (Figure 22), since patients showed difficulty dorsiflexing their ankles and had various levels of drop-foot. The patients in this study, and in agreement with previous work (Burridge et al., 2001; Knutsson & Richards, 1979) could generate tibialis anterior EMG activity, only in doing the act of stepping, which may be the expression of a spinal non-volitional mechanism. This finding opposes the suggestion that low levels of tibialis anterior activity (i.e., strength) would be the likely source of drop-foot in patients (Yang et al., 1991). Here, patients were able to do the task (SW posture, open chain) by "gross" movement of their limb, done effortlessly. On the contrary, during heel-contact (HC posture, closed chain) they needed to volitionally attempt to focus their action at toes and ankle, showing their inability to produce and control the movement, in spite of demonstrated conscious and considerable physical effort.

Remarkably, the majority of patients during swing (SW) did lift their feet off-ground in an action that was not much different from the way healthy subjects moved. Some patients showed some ability to control and generate some response in their tibialis anterior muscle. It appears that for patients with some level of volitional control over the tibialis anterior, regardless of their spasticity level, were able to show a better functioning, although still disrupted, of their walking ability (Okuma & Lee, 1996).

Further studies are needed to bring light to the relevance of tibialis anterior activity, under different levels of volitional control, in affecting the soleus H-reflex

during heel-contact (HC) and swing (SW). This, however, will require to examine individual cases since the large variability observed in patients with CVA.

In this study patients were investigated before and after a training exercise program directed to improve the motor control, as reflected in gait speed improvements, of the ankle antagonists. The exercise paradigm emphasized reiterative ankle dorsiflexion in standing (open chain mechanism) simulating the early swing movement of gait cycle.

The results of this study showed, and in agreement with the reports of Okuma and Lee (1996) and Morita et al. (2001), that patients differ greatly in the manifestation of their abnormal neurophysiological profiles (e.g., pattern of soleus H-reflex amplitude changes throughout postures, tibialis anterior EMG activity), from being relatively normal (patient FS in this study) to a complete disruption or lack of soleus H-reflex inhibition in HC and SW postures (patients CS and EF). Correlation of the electrophysiological parameters (i.e., soleus H-reflex amplitude and tibialis anterior EMG) and clinical data of the patients allows to suggest: (1) Those patients that showed improvement in the fast-pace mode of gait (HH, WB, BT and GA), all but GA showed an improvement in tibialis anterior EMG activity, mainly in the heel-contact (HC) posture. For soleus H-reflex amplitude, out of four patients, three (HH, WB and GA) showed some recovery of inhibition of the H-reflex in postures HC and SW. Patient BT showed good recovery of inhibition of the soleus H-reflex in postures HC and SW. And of the four, only GA had an Ashworth score of 4 at the ankle, the other three had their spasticity scores at 2 or lower. Thus, change in gait speed (fast-paced) appeared to relate to those

who show the most improvements in tibialis anterior EMG activity in the closed-chain HC posture (possibly improving Ia inhibition pathway to soleus muscle) with low spasticity scores, and modest to good recovery of inhibition of the soleus H-reflex amplitude in HC or SW, or both. (2) Those patients who did not change gait speed (CC and EF, at fast-paced mode and those who could not produce a fast-paced speed, CS and BL) all improved moderately tibialis anterior EMG activity in the HC posture, but patient EF. Patient BL made the largest tibialis anterior EMG gain, although no improvement in gait speed was made. For soleus H-reflex, of the four, patient BL was the only to show a remarkably recovery of amplitude inhibition in the HC and SW postures. And of the four, all but CS had an Ashworth score of 4 at the ankle. Thus, the lack of change in speed (fast-paced) appeared to relate to those who showed low or none tibialis anterior EMG activity gains (specifically, during HC), had higher spasticity scores, and continued to display a lack of soleus H-reflex amplitude inhibition during HC and SW.

Patient BL, although showed the most improved recovery of the soleus H-reflex amplitude inhibition in HC and SW, was also the one with the lowest overall functional status (Barthel score of 16). Patient FS was in all assessed aspects within normal limits, showing a fully working state of motor control interplay at the ankle antagonists.

Changes in gait speed, however, are also dependent in performance at other levels of the lower limb different from the ankle. For example, self-paced walking or natural gait speed was reported in adults with CVA to depend mainly on the strength of the hip flexors ($R^2 = 0.69$); and for fast-paced walking, it depends on the strength of the hip

flexors and sensation integrity ($R^2 = 0.85$) (Nadeau, Arsenault, Gravel, & Bourbonnais, 1999). Thus, gait speed may be improved by strength gains made proximally in the affected limb, without intrinsic motor control recovery made distally in the limb (ankle).

Conclusions

The reliability of testing the H-reflex amplitude in five gait-simulating postures with repeated observations on each of two sessions was investigated in a group of healthy subjects and in patients with stroke under similar experimental conditions. To our knowledge, this is the first reliability study carried out in patients with CVA that assessed recordings of the soleus H-reflex amplitude elicited in gait-simulating postures. This study demonstrated that, in the studied healthy subjects and patients, the H-reflex can be reliably recorded in weight-bearing gait-simulating postures. The reliability of obtaining repeated measures using this testing paradigm was high, and for clinical practicality, the average of as few as three H-reflex amplitude recordings might be used as an estimate of soleus motoneurone pool excitability.

In this study several gait-simulating postures were investigated to assess their capability to reproduce the volitional and reflex activity of the ankle antagonists as reported in gait studies. The results of this study demonstrated that the variation in the soleus H-reflex amplitude and the associated EMG activity (from soleus and tibialis anterior muscles) can be comparably reproduced by adopting the proposed gait-simulating postures. As opposed to the complexity and methodological difficulties of

testing ankle motor control during gait, these proposed postures offer a practical method to assess volitional and reflex activity of the ankle antagonists. This would be a useful testing paradigm for healthy subjects as well as patients with impaired ankle motor control of peripheral or central origin.

In this study, a testing paradigm with several gait-simulating postures was used to assess and compare patients with healthy subjects for soleus H-reflex and associated EMG activity of the ankle antagonists. Patients were established, fully functional community dwelling CVA survivors. The testing paradigm, investigated in a related study with healthy subjects, was found to reflect the profound volitional and reflex activity of ankle muscle antagonists seen in gait studies. The results revealed that soleus H-reflex was profoundly disrupted in patients during heel-contact and swing postures, where soleus H-reflex inhibition was absent. Unexpectedly, the patient group and the healthy group showed a similar level of soleus EMG activity for each posture. Also unexpected, was the comparable levels of tibialis anterior EMG activity recorded for the two groups during swing (SW). Muscle weakness, however, and lack of control over tibialis anterior was clearly exposed for patients during heel-contact (HC). This lack of volitional control over tibialis anterior in the patients appeared to have a more direct association to the disrupted soleus H-reflex than to the levels of hyperexcitability (spasticity) of the ankle antagonists.

As opposed to the complexity of testing ankle motor control dynamically, these gait-simulating postures offer a practical and repeatable method to assess volitional and

reflex activity of the ankle antagonists. This may be a useful testing paradigm for patients with impaired ankle motor control of peripheral or central origin.

The results of this study showed that patients who were established CVA survivors differ greatly in their manifestations of abnormal motor control at the ankle antagonists, from being relatively normal to a complete disruption. It appears that repetitive ankle dorsiflexion (during open chain or swing) exercise in standing, assisted with EMG biofeedback may have contributed to the recovery of the neuromotor interplay at the ankle muscles in the majority of the patients. Recovery of inhibition of soleus H-reflex amplitude during heel-contact (HC) and swing (SW) postures appeared to be more associated with the recovery of EMG activity in the tibialis anterior (during HC or closed chain posture), implying an improved volitional control, than to the level of soleus spasticity. Lack of recovery of inhibition of the soleus H-reflex amplitude during heel-contact (HC) and swing (SW) appeared to relate more to those who showed higher spasticity scores associated with low EMG activity of the tibialis anterior muscle during HC posture, implying a severely impaired volitional control. Gait speed improvements, however, were achieved in some patients although they retained persistent spasticity and poor soleus H-reflex inhibition recovery. In addition, gait speed improvements may also depend on performance at other levels of the lower limb different from the ankle, especially strength of the hip flexors, and on the integrity of sensation.

Due to the complexity of the underlying factors involved in a CVA and the disparity of its clinical and functional manifestations, individual differences in recovery

profiles may not be seen if patients are assessed with methods using group analyses. Research using single-case format seemed justified to continue seeking further understanding of the abnormal motor control in stroke.

CLINICAL RELEVANCE

The results obtained in Experiment 1 provided evidence supporting the test-retest reliability of the soleus H-reflex amplitude, as measured in the experimental conditions set forth in this investigation. This was demonstrated in both, healthy subjects and patients who were CVA survivors. Clinically, it is important to use an assessment tool that is repeatable and consistent. This would assist in the finding of true differences in the excitability of the soleus muscle motoneurone pool.

The results obtained in Experiment 2 showed that the profound changes in the pattern of modulation of the soleus H-reflex amplitude reported in gait studies can be reproduced by using gait-simulating postures. Clinically, this offers a practical (i.e., shorter testing duration, need of a lesser number of stimuli, minimal displacement of stimulating and recording electrodes) and uniform testing and evaluative paradigm to investigate the excitability of the soleus motoneurone pool in healthy subjects and in individuals with disordered motor control such as CVA survivors.

The results obtained in Experiment 3 showed, as it was reported in previous studies, that the pattern of changes in the soleus H-reflex amplitude is profoundly disrupted in patients with stroke. The disruption was reflected mainly as a lack of

inhibition of the soleus H-reflex amplitude in the stance and swing phases of the gait cycle. This disruption of the inhibition of the soleus H-reflex amplitude varied from being minimal in some cases to its complete lacking in others. Clinically, having a uniform testing paradigm with a known normal profile from healthy subjects will assist in the evaluation and treatment of patients with CVA allowing for comparisons within and between patients.

The results obtained in Experiment 4 suggested that recovery of the inhibition of the soleus H-reflex amplitude may be influenced by repetitive exercise of ankle dorsiflexion activity in standing (open chain). Biofeedback training of ankle dorsiflexion in standing may also help enhance the recovery of the interplay of ankle antagonists, possible enhancing motor control and gait function. The recovery appears to be related to the degree of spasticity and to the level of volitional control over the tibialis anterior muscle. Clinically, these results appears to add support to the present rehabilitation practice of having patients to start intensive stepping in standing and gait activity to reinforce motor control and functional recovery.

LIMITATIONS OF THE STUDY

The main limitation of this investigation was the relatively small sample size. In the experiments of this study 21 healthy subjects and nine patients with CVA were assessed. Therefore, it would preclude the generalizability of the results to other populations.

In this study, self-sustained gait-simulating postures were used to simulate key points of the gait cycle. Although the pattern of changes in the soleus H-reflex amplitude and soleus/tibialis anterior muscles EMG recorded from the gait-simulated postures closely reproduced those from gait studies, they were cross-sectional and did not reflected temporal aspects of the gait cycle.

In this study, healthy subjects and patients were not matched. The difference in the averaged age between the two groups may have added variation to the electrophysiological measures.

In the exercise training experiment, spasticity and functional status were assessed at the beginning only. In future studies, it may be useful to re-assess these aspects after the intervention to evaluate possible changes.

SUGGESTIONS FOR FUTURE STUDIES

Since the small number of patients investigated in this study, further studies would need to increase the sample of participants. To improve generalizability of the results, subgroups based on age and time from CVA onset, to assess maturation effect, as

well as patients with different levels of volitional control over the tibialis anterior, should be considered.

In this study spasticity was assessed once in the patients that participated in the training exercise. It is suggested that spasticity should be assessed before and at the end of the training period, with more than one method, to improve accuracy.

In the exercise training intervention experiment, the frequency of training was 3 times/week for 4 weeks, emulating the attendance of an outpatient physical therapy setting. In future studies, the training method should test longer frequencies (e.g., 3 times/week for 4, 6, and 8 weeks or more) and perform the repeated exercise with alternative number of repetitions, with and without EMG biofeedback.

In the training exercise experiment strength was inferred from EMG activity data. It is suggested that strength be assessed by more than one method to improve accuracy. Muscle strength testing should include not only the ankle muscles but the knee and hip groups as well. In addition, studies should further explore the open/closed chain mechanisms in the disordered motor control of the upper limb.

Due to the complexity of the underlying factors involved in stroke and due to the disparity of its clinical and functional manifestations, individual differences in recovery profiles may not be seen if patients are assessed with methods using group analyses. Research using single-case format seemed justified to continue seeking further understanding of the abnormal motor control of CVA disorders.

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APPENDICES

APPENDIX

A. BARTHEL INDEX

BARTHEL INDEX

Activity	Score
Feeding 0 = unable 5 = needs help cutting, spreading butter, etc., or requires modified diet 10 = independent	0 5 10
Bathing 0 = dependent 5 = independent (or in shower)	0 5
Grooming 0 = needs to help with personal care 5 = independent face/hair/teeth/shaving (implements provided)	0 5
Dressing 0 = dependent 5 = needs help but can do about half unaided 10 = independent (including buttons, zips, laces, etc.)	0 5 10
Bowels 0 = incontinent (or needs to be given enemas) 5 = occasional accident 10 = continent	0 5 10
Bladder 0 = incontinent, or catheterized and unable to manage alone 5 = occasional accident 10 = continent	0 5 10
Toilet Use 0 = dependent 5 = needs some help, but can do something alone 10 = independent (on and off, dressing, wiping)	0 5 10
Transfers (bed to chair and back) 0 = unable, no sitting balance 5 = major help (one or two people, physical), can sit 10 = minor help (verbal or physical) 15 = independent	0 5 10 15
Mobility (on level surfaces) 0 = immobile or < 50 yards 5 = wheelchair independent, including corners, > 50 yards 10 = walks with help of one person (verbal or physical) > 50 yards 15 = independent (but may use any aid; for example, stick) > 50 yards	0 5 10 15
Stairs 0 = unable 5 = needs help (verbal, physical, carrying aid)	0 5 10
Total (0 - 100)	
Patient Name: Rater: Date:	

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APPENDIX

B. ASHWORTH SCALE

MODIFIED ASHWORTH SCALE

Score	**Ashworth Scale** Ashworth (1964)	**Modified Ashworth Scale** Bohannon & Smith (1987)
0 (0)	No increase in tone	No increase in muscle tone
1 (1)	Slight increase in tone giving a catch when the limb was moved in flexion or extension.	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension.
1+ (2)		Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the reminder (less than half) of the ROM (range of movement).
2 (3)	More marked increase in tone but limb easily flexed.	More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved.
3 (4)	Considerable increase in tone - passive movement difficult.	Considerable increase in muscle tone passive, movement difficult.
4 (5)	Limb rigid in flexion or extension.	Affected part(s) rigid in flexion or extension.

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APPENDIX

C. CONSENT FORM - HEALTHY PARTICIPANTS

TEXAS WOMAN'S UNIVERSITY
School of Physical Therapy

Soleus H-Reflex and EMG Measures of the Tibialis Anterior/Soleus Muscles of Healthy Subjects during Five Standing Postures.

CONSENT FORM

I have been asked to voluntarily participate in this research project. I understand that to participate in this study I need to be in good general health and be free of pain symptoms or injuries/diseases that may affect the normal use of my lower limbs.

I will be tested for soleus H-Reflex and surface electromyography (EMG) of one of my lower legs (tibialis anterior and soleus muscles).

Soleus H-reflex. I will be tested in neutral standing, toe standing (plantarflexion), heel standing (dorsiflexion), and right/left single leg standing. After skin preparation (light sanding and alcohol swabs use), a recording surface electrode will be attached with tape to the skin over the soleus (centrally, below the edge formed by the two gastrocnemii) and aligned with the Achilles tendon. The H-reflex will be evoked via electrical impulses delivered to the popliteal fossa (tibial nerve) via a secured stimulating surface electrode. A ground surface electrode will be placed between the stimulation and recording sites. Electrical stimulus will be delivered every 2-5 seconds. The time to complete the H-Reflex testing session will be approximately of 30 minutes.

Surface electromyography (EMG). I will be tested in the same postures as for the H-Reflex testing. After skin preparation, 5 disposable electrodes will be placed over the skin covering the soleus and tibialis anterior muscles of one of my lower legs. EMG measures will be obtained from 3 seconds during each of the 5 standing postures. Three trials will be recorded in each of the postures. The time to complete the EMG testing session will be approximately of 30 minutes.

Subjects may participate in one of the components of this study (H-Reflex or EMG), or both. There are some risks by participating in this testing:

a. There is risk to develop skin reactions to the components of the electrodes, alcohol, light sanding and tape use. The risk for skin reaction to the testing materials is minimal. In the event of noticeable skin reaction a skin lubricant or cream may be recommended. Strong skin reactions will impede me from participating in the study.

b. There is a risk to present intolerance to electrical stimulation (H-Reflex testing). To ensure my confidence and comfort upon the delivered electrical stimulation, a gradual increment in the intensity of the pulses will be assured.

c. There is a risk for falls or loosing balance. To help prevent falls or loosing balance, I will hold onto a walker during the testing. A research associate will also stand by me during the testing.

I understand that I can stop my participation in this study at any time. I understand that the information will be kept confidential and for research purpose only. I understand that my name will not be used in the processing of data since I will be assigned a code to maintain confidentiality. I understand that I will receive no monetary compensation for my participation in this study. I understand that there are may be no direct benefits from my participation in this study.

I was given the opportunity to ask questions, and if I have concerns about my participation in this study I can contact Mohamed Sabbahi, PT, PhD, ECS at 713-7942309 or Enrique Pineda, PT, MS, OCS at 713-7167625.

By signing below I consent to participate in this study.

_____ _____

Signature Date

APPENDIX

D. CONSENT FORM – PATIENTS

TEXAS WOMAN'S UNIVERSITY - School of Physical Therapy

Evaluation and Treatment of Patients with Stroke: An EMG and H-Reflex Feedback Study.

Consent Form

1. Purpose of study and how long it will last: The purpose of this study is to examine the effects of electromyography and reflex feedback training on motor control (gait quality) of subjects who are stroke survivors. The methods used in this study have been frequently used in clinical settings and in past research. This study will take about two hours for the initial and final (after 4 weeks) testing sessions, and about one hour for each of the 12 training sessions (three times per week for four weeks).

2. Description of the study including procedures to be used: I understand that the purpose of this study is to investigate the effect of an exercise training program on the ability to walk with my weakened leg.

I will be asked to provide information about my medical history. Also, my weight and height will be measured. Before the exercise training period, I will be tested on my ability to rise stand on my heels and toes, as well as walking as fast as I safely can and also at my usual pace over 10 meters (about 30 feet) each. This walking will be videotaped. To assess and compare the effect of the exercise training, these tests will be repeated after the training is completed. The initial and final testing sessions should last about 1 1/2 to 2 hours.

The training program will be as it was in the initial testing, that is, rising to toe and heel standing. These movements will be performed several times within one training session, to a certain required level, 3 times/week for 4 weeks. Training sessions should last about one hour.

During the initial and final testing, as well as during the training sessions, a set of probes will be placed over the skin of my lower legs to record how the muscles work. Also, I will feel some mild electrical pulses on the back of my knees. The electrical pulses will occur once every 5 seconds. The investigator will do this while I am standing and when asked to rise to toe and heel standing. I will do these movements several times for about 5 seconds. I will be watching a monitor in front of me where the investigators will explain what to do to control my ankle muscles better.

During all testing and training sessions, I will be able to ask for additional resting times, which can be repeated or extended if I feel I need to.

3. Description of any procedures that may result in discomfort or inconvenience: I understand that the methods and procedures described above involve possible risks or discomfort. I could have a reaction to the tape used to secure the probes on my skin, or to the coupling gel used with the probes. And, there may be unforeseeable risks associated with participation I this study. It may be that I can not tolerate the electrical stimulation on the back of my knees or, I could have a fall during walking. But I agree to wear a safety belt guided by the investigator to prevent falls. I could also have muscle soreness due to the movements that I will perform.

4. Expected risks of study: Risks of participating in this study include those mentioned above, that is, I could fall, I could have a reaction to tape/gel used to secure probes and joint markers, and I could have muscle soreness. The investigators will be monitoring my participation throughout this study to prevent the occurrence of any of these risks.

5. Expected benefits of study: I understand the possible benefits of this study are: potential increase in my ability to walk and control of my weakened leg; potential contribution to scientific knowledge, which may benefit rehabilitation of future patients. I understand also, that it may be no benefits from participating in this study.

6. Other treatment available: The assessments and exercise training I will receive by participating in this study is not meant to substitute for traditional treatment already offered to me for my condition.

7. Use of research results: My participation in this study may result in benefits to other people with problems like mine. Long-term benefits may exist by adding to physical therapy training methods tested in this study. It may improve my gait quality and overall mobility. The confidentiality of the data will be maintained within legal limits.

8. Special circumstances: I understand that my involvement in this study is completely voluntary, and if I am unable or unwilling to complete this study at any time, I may stop taking part in this study without penalty.

RESEARCH SUBJECTS' RIGHT: I have read or have had read to me all of the above information. Enrique Pineda or a qualified associate has explained the study to me and answered all of my questions. I have been told of the risks and discomforts and possible benefits of the study. I have been told of other choices of treatment available to me. **I understand that I do not have to take part in this study, and my refusal to participate will involve no penalty or loss or rights to which I am entitled. I may withdraw from this study at any time without penalty or loss of VA or other benefits to which I am entitled. My participation will not affect the way I now pay for medical care at the VAMC.**

The results of this study may be published, but my records will not be revealed unless required by law.

In case there are medical problems or questions, I have been told I can call the Baylor College of Medicine, Office of Research at 713-798-6970 and Dr. Eugene Lai at 713-798-7262 during the day and I can also page Dr. Lai at 713-798-7262 after hours. In the event of injury resulting from this research, Baylor College of Medicine and/or Methodist Hospital is/are not able to offer financial compensation nor to absorb the costs of medical treatment. However, necessary facilities, emergency treatment and professional services will be available to research subjects, just as they are to the community generally. (My/your) signature below acknowledges (my/your) voluntary participation in this research project. Such participation does not release the investigator(s), institution(s), sponsor(s), or granting agency (ies) from their professional and ethical responsibility to (me/you).

I understand my rights as a research subject, and I voluntarily consent to participate in this study. I understand what the study is about and how and why it is being done. I will receive a signed copy of this consent form.

_____ _____

Signature of Subject Date

_____

Signature of Investigator