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**UNIVERSITY OF ALBERTA**

**THE BEHAVIOUR OF SPINAL REFLEXES  
EVOKED BY CUTANEOUS STIMULI DURING WALKING  
IN INCOMPLETE SPINAL CORD INJURED SUBJECTS**

**BY  
CATHERINE ALLYSON JONES**



**A thesis submitted to the Faculty of Graduate Studies and Research in partial  
fulfillment of the requirements for the degree of MASTER OF SCIENCE.**

**DEPARTMENT OF PHYSICAL THERAPY**

**Edmonton, Alberta**

**FALL 1992**



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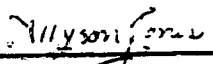
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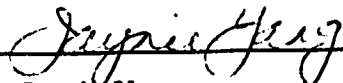
  
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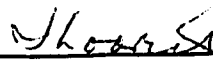
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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled THE BEHAVIOUR OF SPINAL REFLEXES EVOKED BY CUTANEOUS STIMULI DURING WALKING IN INCOMPLETE SPINAL CORD INJURED SUBJECTS submitted by CATHERINE ALLYSON JONES in partial fulfillment of the requirements for the degree of MASTER OF SCIENCE.

  
Dr. Jaynie Yang

  
Mrs. Joan Loomis

  
Dr. Richard Stein

DATED: Oct 5, 1992

## **DEDICATION**

**This thesis is dedicated to those patients with whom I have had the privilege of working with, and who have overcome insurmountable physical disabilities to return to their community lives.**

## **ABSTRACT**

The behaviour of reflex responses evoked by cutaneous stimuli during walking was investigated in 10 subjects with incomplete spinal cord damage. Low intensity stimuli (1.5 to 1.7 times the motor threshold) were delivered to the posterior tibial nerve during treadmill walking. Reflex responses were recorded by surface electromyography from the tibialis anterior and soleus muscles. Responses were evoked at a medium latency of 50 to 80 ms after the first stimulus. The reflex activity examined in both muscles was cyclically modulated in all of the subjects during walking, but the degree of modulation was different from that seen in normal subjects. Only 1 subject showed reflex reversal in the tibialis anterior muscle that was similar to the pattern found in normal subjects. This subject exhibited the least neurological impairment and the fastest walking speed. In spite of an adequate level of background muscle activity at the transition from swing to stance phase, most spinal cord injured subjects did not show inhibitory responses in the tibialis anterior. Inhibitory responses were typically evoked in the soleus muscle during the stance phase, but abnormal excitatory responses developed during the swing phase. Two subjects showed excitatory responses during both the stance and swing phases in tibialis anterior and soleus. The degree of reflex modulation was not correlated with the spinal cord injured subjects' functional walking ability nor the clinical findings of muscle tone. The results demonstrate that most spinal cord injured subjects retained the ability to modulate cutaneous reflexes during walking. However, cutaneous information was not normally gated during the step cycle in most of the spinal cord injured subjects.

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## **CHAPTER 1**

### **INTRODUCTION**

#### **1.1 Statement of the Problem**

Locomotion can occur without sensory input (reviewed in Rossignol et al, 1988; Grillner, 1981); however, sensory feedback is needed to make purposeful adjustments in response to environmental demands (reviewed in Grillner & Dubuc, 1988). When walking is unexpectedly disrupted, spinal reflexes are evoked to generate quick reactions (reviewed in Rossignol et al, 1988). The present study explores the behaviour of a spinal reflex evoked by non-noxious cutaneous stimuli during walking in subjects with spinal cord damage. The research addresses the behaviour of cutaneous reflexes elicited in 2 ankle muscles of incomplete spinal cord injured subjects to determine whether spinal cord injury interferes with the transmission of sensory feedback during walking.

Under normal walking conditions, sensory input interacts with central mechanisms to produce changes in walking speed and direction (Grillner, 1975). When walking is unexpectedly disrupted, sensory information processed at the spinal level (Forssberg et al, 1975; Andersson et al, 1978a) produces rapid compensatory reactions (Forssberg, 1979). When the identical sensory input is applied at different times of the step cycle, different types of reactions are evoked (reviewed in Rossignol et al, 1988). These varied reactions are referred to as modulated responses. An adaptable reflex system is advantageous because quick reactions can be made in response to diverse demands.

Modulated reflex responses may be functionally significant in maintaining the stability of walking (Duysens et al, 1992; Wand et al, 1980; Forssberg, 1979; Forssberg et al, 1977). For instance, if walking is suddenly disrupted during the stance phase, a prolonged stance phase ensures stability. If an obstacle obstructs the swing through of a limb, an enhanced flexor response quickly clears the limb from the obstacle to avoid tripping (Forssberg, 1979).

The behaviour of reflex responses evoked by sensory stimuli during locomotion has been extensively examined in the cat (reviewed in Rossignol et al, 1988). The findings from studies which examined spinalized, decerebrate and intact cats agree that cutaneous responses are modulated with respect to the step cycle (Forssberg et al, 1975, 1977; Duysens & Pearson, 1976; Duysens & Stein, 1978; Forssberg, 1979; Duysens & Loeb, 1980; Wand et al, 1980; Abraham et al, 1985). Cutaneous stimuli applied to the distal hindlimb generally elicit an excitatory response in the flexor muscles during the swing phase and a more varied, weaker excitatory response in the extensor muscles during the stance phase (Forssberg et al, 1975, 1977; Duysens & Pearson, 1976; Duysens & Stein, 1978; Forssberg, 1979; Duysens & Loeb, 1980; Wand et al, 1980; Abraham et al, 1985). Such reflex behaviour is referred to as **phase-dependent reflex reversal** (Forssberg et al, 1975). Another type of reflex reversal less commonly reported in animals occurs within a single muscle. Depending upon the phase of the step cycle, the evoked electromyographic (EMG) activity will be either excitatory or inhibitory (Drew & Rossignol, 1987; Schomburg et al, 1981; Schomburg & Behrends, 1978; Miller et al, 1977).

Recent studies have found that cutaneous responses elicited during walking in humans are similar to those in cats (Duysens et al, 1990; Yang & Stein, 1990). In humans, cutaneous stimuli evoked strong flexor activity in the ankle flexor muscles during the swing phase and a weaker excitatory response in the ankle extensor muscles during the stance phase (Duysens et al, 1990). Moreover, reflex reversal has been reported within single leg muscles during human locomotion (Yang & Stein, 1990). Excitatory responses recorded in leg muscles during one phase of walking were reported to reverse direction and become inhibitory responses when the stimulus was applied at other times within the step cycle. Thus, the evidence obtained from both cats and humans suggests that reflex responses evoked by cutaneous stimuli are modulated during locomotion.

Although cutaneous input is strongly modulated during walking in normal individuals, it is unclear whether spinal cord injury affects the modulation of the cutaneous reflex. Spinal cord damage interrupts ascending and descending fibres, and

often produces a variety of sensory and motor deficits (Eidelberg, 1987). Clinically, spinal cord injury is characterized by spasticity, variable degrees of paresis and altered sensation, all of which impair the individual's locomotion (Ashby & McCrea, 1987; Dimitrijevic & Nathan, 1967a; Hussey & Stauffer, 1973). However, preliminary evidence supports the possibility that basic neural networks responsible for rudimentary locomotion exist in spinal cord injured (SCI) patients (Bussel et al, 1989; Roby-Brami & Bussel, 1987), which is similar to cats (Grillner & Zangger, 1975; Forssberg et al, 1980). In spite of this evidence, SCI patients spend considerable time and energy learning to walk again (Waters & Lunsford, 1985; Hussey & Stauffer, 1973).

Spinal cord damage in humans affects the transmission of sensory input during walking. The Hoffmann (H) reflex, a spinal reflex which measures the excitability of muscle afferent fibres, was abnormal in SCI subjects during walking (Yang et al, 1991). Like the cutaneous reflex, the H-reflex of the soleus (SOL) muscle is deeply modulated as a function of the step cycle in normal subjects (Capaday & Stein, 1986). An abnormally high H-reflex gain appeared to reduce the degree of H-reflex modulation during walking in spinal cord injured subjects (Yang et al, 1991). Therefore, spinal reflexes evoked by cutaneous stimuli may also be altered by spinal cord injury.

If the pattern of reflex modulation evoked by cutaneous stimuli during walking in SCI subjects is different from the pattern of response seen in normal subjects, the abnormality may be directly related to the subjects' functional walking problems. Conversely, the reflex responses recorded in SCI subjects may behave similarly to normal subjects. If this is true, the walking difficulties experienced by SCI individuals would not likely be associated with abnormalities of the modulation of cutaneous reflex.

In this study, electrical stimuli applied to a mixed nerve, the posterior tibial nerve near the ankle, will be considered to elicit a cutaneous reflex. Although low intensity stimuli delivered to a mixed nerve will also stimulate other low threshold afferent fibres, comparisons of reflex activity evoked by stimulation of the sural



nerve, a sensory nerve, and the posterior tibial nerve, a mixed nerve, elicited similar responses (Yang & Stein, 1990; Duysens et al, 1992). The low intensity stimuli applied to the posterior tibial nerve in this study will presumably excite primarily large cutaneous afferents to evoke a cutaneous reflex.

## **1.2 Purpose**

The objective of this thesis is to determine whether a spinal reflex evoked by cutaneous stimuli is modulated during walking in subjects with incomplete spinal cord injuries. A comparison will be made with the findings obtained from normal subjects in an earlier study (Yang & Stein, 1990). The thesis will also determine whether the behaviour of this cutaneous reflex in walking is related to the subjects' functional walking abilities. Findings from this study will provide further insight into how spinal reflexes are modulated during walking in subjects with spinal cord damage. The hypothesis is that the behaviour of the cutaneous reflex response evoked during walking in incomplete spinal cord injured subjects is abnormal. Moreover, the degree of modulation of the reflex will likely be less than that seen in normal subjects.

## **CHAPTER 2**

### **LITERATURE REVIEW**

This literature review discusses the neural control of locomotion and the effect that cutaneous stimuli exert on the spinal circuitry during locomotion in animals and humans. Since no studies have examined the behaviour of reflexes evoked by cutaneous stimuli during walking in SCI subjects, findings from similar conditions and reflexes are discussed. The final section of the review focuses on the methodological issues of evoking cutaneous reflex responses during walking.

#### **2.1 Generation of locomotion**

Purposeful walking requires the ability to a) generate a basic locomotor pattern, b) compensate for external disturbances, and c) maintain balance (reviewed in Grillner, 1975). The neural mechanisms associated with the generation of locomotion have long been investigated by researchers. Extensive investigation of vertebrates has found that the neural networks responsible for basic locomotor patterns are situated in the spinal cord. The neural network is referred to as the locomotor **central pattern generator (CPG)**. Although the specific networks have not been identified for mammals, studies of cats have shown that locomotor capabilities are inherent to the spinal cord (Grillner & Zangger, 1974, 1975; Grillner, 1981).

Descending input and movement-generated afferent feedback are not required to generate rhythmicity (Grillner & Zangger, 1975, 1979). When descending input was removed in cats, the locomotor pattern was similar to the pattern seen in intact cats. Although equilibrium reactions were impaired, spinalized kittens could walk on treadmills in a coordinated manner (Forssberg et al, 1980; reviewed in Grillner, 1975, 1973) and with training, spinalized adult cats could also walk without support on a treadmill (Rossignol et al, 1986; Lovely et al, 1986; Barbeau & Rossignol, 1987). Furthermore, cats devoid of descending and peripheral input were able to walk, but with a slightly altered locomotor pattern (Grillner & Zangger, 1979). Changes were seen in the timing of the gait sequence, muscle force (Grillner &

Zangger, 1975) and inter-limb coordination (Grillner & Zangger, 1984).

The fact that the locomotor CPG can operate in isolation does not imply that sensory input is unimportant. Peripheral input can regulate the locomotor CPG. For instance, the position of the hip influenced the initiation of the swing phase in spinal cats (Andersson et al, 1978b; Grillner & Rossignol, 1978). Furthermore, locomotor activity could be entrained by passive movement of the hip (Andersson et al, 1978b; Grillner & Rossignol, 1978; Andersson & Grillner, 1981). The load on the limb during the stance phase is another factor that can regulate the locomotor CPG. Initiation of the swing phase was prevented in thalamic cats when the load on the ankle extensors was too great (Duysens & Pearson, 1976). These findings suggest that the spinal cord can generate walking, but descending input and movement-generated peripheral feedback are necessary to control locomotion and equilibrium.

There is little evidence to support the existence of a spinal locomotor generator in humans. Preliminary reports have suggested that some components of the spinal stepping generator mechanism may exist in paraplegic man (Bussel et al, 1989). The difference between the ability of animals and humans to generate spinal locomotion is suspected to be related to the degree of descending influence exerted over the spinal locomotor mechanisms (Armstrong, 1988). Spinal walking is not seen in humans with complete spinal cord injuries not because the spinal circuitry is different from other mammals, but rather that cerebral influences are thought to employ more control over the spinal locomotor mechanisms in humans than animals (Armstrong, 1988). Although evidence reported in humans does not confirm the existence of a spinal locomotor generator, strong evidence in cats and other vertebrates suggests that basic stepping movements are generated by neural networks in the spinal cord.

## **2.2 Sensory input and locomotion**

In order to adapt walking to external stimuli, the integration of sensory information with locomotor mechanisms is essential. The neural mechanisms responsible for transmission in sensory pathways during walking are not fully understood; however, afferent fibres relay information from the periphery to interact

with central locomotor mechanisms, and modulate the reflex activity (Rossignol et al, 1988). The transmission of sensory signals during walking has been investigated by manipulating sensory input and recording the locomotor responses.

To determine the role that sensory input plays during locomotion, sensory input has been either inactivated or activated during locomotion (Rossignol et al, 1988). Inactivation of afferent fibres is a more invasive technique that selectively or completely removes the afferent input (Grillner & Wallen, 1985). This technique is commonly used with animals (Forssberg et al, 1975; Grillner & Zangger, 1975, 1979; Duysens & Stein, 1978). Selective activation of afferent fibres during walking is a more practical approach to study the effects of sensory input in humans. Electrical stimuli has been commonly applied to evoke compensatory reactions during walking (Yang & Stein, 1990; Duysens et al, 1990; Belanger & Patla, 1984; Crenna & Frigo, 1984). Although perturbations activate various types of receptors (Schomburg, 1990), this method is noninvasive and is feasible for clinical studies.

### **2.2.1 Reflex reversal**

Studies of cats and humans have shown that sensory input can modify the EMG activity (Forssberg et al, 1975, 1977; Belanger & Patla, 1984; Crenna & Frigo, 1984; Duysens et al, 1990), and the duration of the step cycle (Duysens & Pearson, 1976; Duysens & Stein, 1978). When a cutaneous stimulus is applied to the foot during walking, different reflex responses are evoked depending on the time of stimulus delivery within the step cycle (Duysens et al, 1990; Yang & Stein, 1990; Belanger & Patla, 1984; Crenna & Frigo, 1984; reviewed in Rossignol et al, 1988). These responses are described as phase-dependent changes in the reflex (Forssberg et al, 1975).

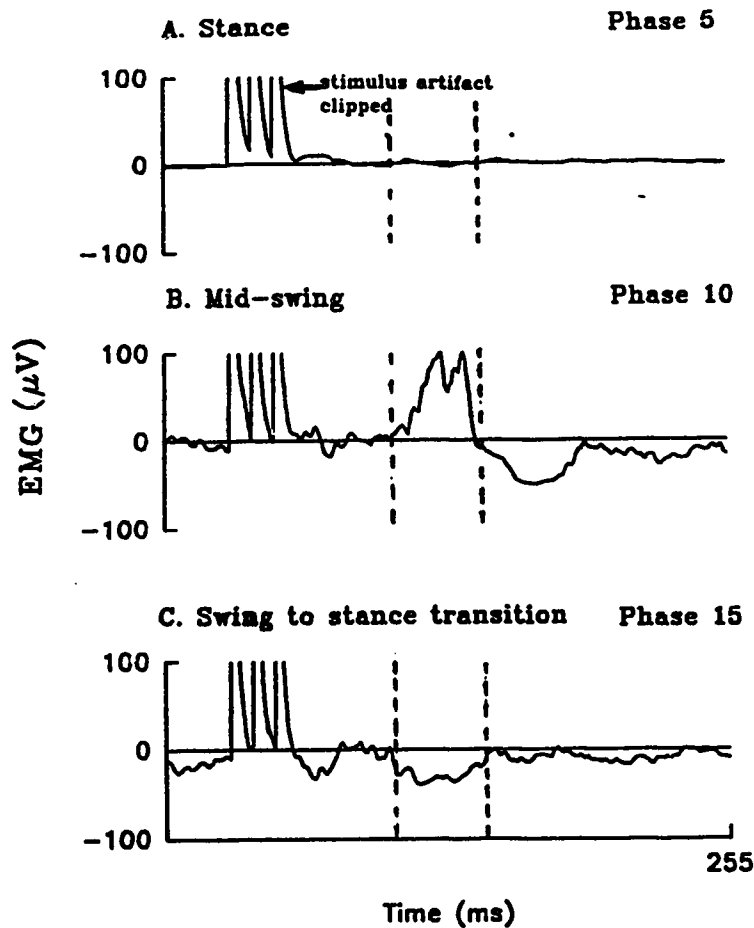
Studies first identified phase-dependent reflex responses in spinal, decerebrate and intact cats (Forssberg et al, 1975, 1977; Duysens & Pearson, 1976; Forssberg, 1979). When a rod was used to tap the dorsum of the hindlimb during the swing phase, the limb flexed to quickly clear the obstacle; when the identical stimulus was applied during the stance phase, the extensor muscle activity was augmented as if to

avoid falling (Forssberg et al, 1975). Moreover, investigators strongly suspected that cutaneous afferent fibres were the principal afferent fibres responsible for these reflex responses. This conclusion was based on the observation that the hindlimb failed to clear the obstacle when a local anaesthetic applied to the dorsum of the paw blocked the tactile input (Wand et al, 1980; Forssberg et al, 1975, 1977).

The duration of the step cycle is also dependent upon when the stimulus is delivered within the step cycle. Cutaneous stimuli applied to the hindlimb of decerebrate, spinal and intact cats during the swing phase increased the duration of the phase by prolonging the flexor burst (Forssberg et al, 1975, 1977; Duysens & Pearson, 1976; Duysens & Stein, 1978). When the stimuli was applied during the late stance phase, the duration of the stance phase was lengthened and the extensor activity increased in decerebrate and spinal cats. Intact cats were able to maintain regular locomotor rhythm when the stimuli were applied during the stance phase (Forssberg, 1979; Duysens & Stein, 1978) by making adjustments with the contralateral limb (Drew & Rossignol, 1987; Duysens & Stein, 1978).

Some features of the phase-dependent reflex reversal reported in animals were observed in humans during walking (Duysens et al, 1990; Yang & Stein, 1990). One type of reflex reversal was identified between antagonistic ankle muscles during walking. Duysens and colleagues (1990) reported that stimulation of either the sural or tibial nerve facilitated the ankle plantarflexor muscles during early stance phase and facilitated the dorsiflexor muscles with a similar latency during the swing phase.

Yang and Stein (1990) identified another type of reflex reversal that was seen within single leg muscles. Stimulation of the tibial nerve evoked an excitatory response at medium latency in the ankle flexor muscles during the swing phase which reversed direction and became an inhibitory response during the transition from swing to stance. Figure 1 displays the reflex response of the tibialis anterior (TA) muscle in a normal subject during walking (obtained from pilot work). The background EMG activity associated with normal walking was subtracted from the response resulting in a zero baseline prior to the delivery of the stimulus. Excitatory and inhibitory responses were identified relative to the prestimulus baseline. During the stance



**Figure 1.** Reflex reversal of tibialis anterior in a normal subject during walking. The reflex response was evoked during 3 phases of the walking cycle. The reflex responses were evoked by triple monophasic pulses, 0.3 ms in duration, and 10 ms apart, delivered to the posterior tibial nerve. Each trace represents an average of approximately 20 responses. The background EMG of normal walking has been subtracted from each trace to reveal the reflex responses. The vertical axis represents the amplitude of the EMG response ( $\mu\text{V}$ ). The type of response (inhibitory or excitatory) was defined relative to the average EMG level over the 30 ms prior to the stimuli. A. Stimuli were applied during the stance phase when the TA was inactive. No response was seen. B. The stimuli applied during mid-swing evoked an excitatory response beginning 72 ms from the onset of the first stimulus artifact (middle latency window defined by grid lines). C. An inhibitory response was observed at the same latency during the transition from the swing to stance phase.

phase the TA muscle was inactive and no responses were seen (Fig. 1A). The medium latency response was typically excitatory during the swing phase (Fig. 1B), but the response reversed direction to become inhibitory when the stimulus was applied during the transition from swing to stance (Fig. 1C). This type of reversal within a single muscle has occasionally been reported in animals (Drew & Rossignol, 1987; Schomburg et al, 1981; Schomburg & Behrends, 1978; Miller et al, 1977).

The functional implications of the cutaneous reflex response are not fully understood in humans, but recent evidence indicates that reflex reversal plays a role in walking stability (Duysens et al, 1992). Because the majority of investigations have examined reactions in the cat, analogies between cat and human locomotion have been commonly accepted. It is difficult to extrapolate from the quadrupedal locomotor demands of cats to the bipedal locomotion of humans since bipedal locomotion requires a more complex control of balance. Nevertheless, some biomechanical and neurophysiological similarities have been identified between the stumbling reactions of the cat and human (Duysens et al, 1990, 1992; Yang & Stein, 1990). Modulation of this reflex response is believed to play a functional role in walking (Duysens et al, 1992; Wand et al, 1980; Forssberg, 1979; Forssberg et al, 1977) by maintaining stability and ensuring continuity of locomotion even in the presence of obstacles (Forssberg et al, 1975).

In summary, the locomotor activity which is generated at the spinal level interacts with cutaneous input to evoke phase-dependent reflex responses. The behaviour of phase-dependent reflex responses has been extensively investigated in the cat (Forssberg et al, 1975, 1977; Duysens & Pearson, 1976; Forssberg, 1979; Wand et al, 1980), and recent studies have identified reflex reversal during walking in humans that may be functionally significant for walking (Yang & Stein, 1990; Duysens et al, 1990, 1992). If reflex reversal is characteristic of normal walking, how does this reflex behave in SCI individuals who experience instability during walking?

## **2.3 Spinal cord injury and cutaneous reflex responses**

### **2.3.1 Clinical features**

Individuals with spinal cord damage experience motor and sensory deficits because ascending and descending neural signals are disrupted (Eidelberg, 1987). This type of injury frequently results in impaired locomotion (Fung & Barbeau, 1989; Waters & Lunsford, 1985). Clinically, a spinal cord injury is categorized as being either a complete or an incomplete injury depending on the sparing of dermatomes and myotomes (Eidelberg, 1987), and is verified by neurophysiological tests (Ashby & McCrea, 1987; Brandstater & Dinsdale, 1976). Disturbances in the spinal circuitry are reflected by exaggerated segmental reflexes, and are frequently associated with spasticity, impaired voluntary movement, paresis and altered sensation (Ashby & McCrea, 1987; Chapman & Wiesendanger, 1982; Dimitrijevic & Nathan, 1967a). Functionally, these problems are manifested in walking difficulties for many SCI individuals (Waters & Lunsford, 1985; Fung & Barbeau, 1989; Hussey & Stauffer, 1973).

### **2.3.2 Reflex responses evoked under static conditions**

Because reflexes evoked by cutaneous stimuli during walking have not been examined in SCI subjects, some insight may be gained by comparing normal and SCI subjects' responses elicited under static conditions. The EMG response of flexor muscles, evoked by non-noxious cutaneous stimuli, consisted of two excitatory responses separated by an inhibitory response in normal subjects (Burke et al, 1991; Yang & Stein, 1990; Aniss et al, 1992)(see Fig. 1A in Yang & Stein, 1990). A certain level of muscle activity was required to evoke reflex activity (Burke et al, 1991; Aniss et al, 1992).

In normal subjects, cutaneous reflexes elicited by non-noxious stimuli are similar to responses evoked by noxious stimuli, except with noxious stimuli there is no inhibitory response between the 2 excitatory responses (Meinck et al, 1985). The reflex response evoked by noxious stimuli is commonly referred to as a flexor reflex, and generates a protective withdrawal of the limb from the stimuli (Schomburg,



1990). The flexor reflex occurs at shorter latencies with increasing stimulus intensities or when the muscle has a minimal level of background activity (Meinck et al, 1985).

The early excitatory response evoked by a noxious stimulus has been extensively investigated under static conditions in subjects with spinal cord injuries. This excitatory response is similar to the responses evoked in normal subjects by noxious stimuli (Roby-Brami & Bussel, 1987; Meinck et al, 1985; Choa & Stephens, 1982; Shahani & Young, 1971; Dimitrijevic & Nathan, 1967b). The long latency excitatory response, however, has a longer latency and lower threshold in SCI subjects than normal subjects (Roby-Brami & Bussel, 1987; Jenner & Stephens, 1982). Contrary to normal subjects, the late reflex response in SCI subjects has a lower threshold than the earlier response. These observations have been reported in both complete and incomplete SCI patients regardless of the level of injury (Shahani & Young, 1971; Meinck et al, 1985). It appears that the early excitatory response evoked by cutaneous stimuli is normal in SCI subjects, while the second excitatory response is not consistently seen under static conditions.

### **2.3.3 The behaviour of other segmental reflexes during walking.**

Although the behaviour of cutaneous reflexes has not been investigated during walking in subjects with incomplete spinal cord damage, other spinal reflexes have been examined. The H-reflex, which is thought to measure the excitability of the Ia monosynaptic reflex arc (Magladery & McDougal, 1950), was evoked in the SOL muscle during walking in incomplete SCI subjects (Yang et al, 1991). Like the cutaneous reflex, the amplitude of the H-reflex is deeply modulated with respect to the step cycle in normal subjects and is thought to be related to the control of locomotion (Capaday & Stein, 1986, 1987). The degree of modulation of the H-reflex recorded in incomplete SCI subjects was less than that reported in normal subjects (Yang et al, 1991). The neural mechanisms responsible for modulation of the H-reflex were thought to be present in SCI subjects, but were masked by saturation of the reflex loop by a high reflex gain (Yang et al, 1991). Under resting

conditions, the H-reflex was also saturated in SCI subjects (Taylor et al, 1984).

In summary, abnormal segmental reflexes commonly occur after spinal cord damage and may interfere with locomotion. For instance, the H-reflex which was exaggerated in SCI subjects under resting conditions (Taylor et al, 1984) showed reduced modulation during walking (Yang et al, 1991). The long latency response evoked by cutaneous stimulation under resting conditions was also altered in SCI subjects (Roby-Brami & Bussel, 1987; Meinck et al, 1985; Shahani & Young, 1971; Dimitrijevic & Nathan, 1967b). It may be that the modulation of cutaneous reflexes is also altered in SCI individuals during walking.

#### **2.4 Possible neural mechanisms responsible for the modulation of reflex behaviour.**

Neural circuitry in the spinal cord can modify sensory information from the periphery (Rossignol et al, 1988). The neural mechanisms that control the sensory pathways during walking have been investigated in both invertebrates and vertebrates (reviewed in Cohen, 1988), although the mechanisms are poorly understood for mammals. Neural mechanisms have not been directly examined in humans because of the technical difficulties associated with examining a complex nervous system with the use of non-invasive techniques. However, phylogenic similarities of the spinal CPG which exist between species suggest that some common principles may apply (Cohen, 1988; Getting, 1989). Evidence from lower vertebrates indicate that sensory information is modulated by a variety of mechanisms at certain sites in the central circuitry (reviewed in Sillar, 1991). Sensory transmission is likely controlled at the motoneuron level, the interneuron level and the afferent terminals.

Modulation of the cutaneous reflex may be affected by the excitability of the motoneuron during locomotion. Intracellular recordings during fictive locomotion in cats reported that the motoneurons were depolarized and hyperpolarized cyclically with locomotor discharges (Andersson et al, 1978a; Schomburg et al, 1981; Shefchyk & Jordan, 1985; Schmidt et al, 1989). Consequently, the motoneuron may be more responsive to synaptic input from peripheral, spinal and supraspinal sources during

specific phases of locomotion. However, complex changes in reflex responses reported during real locomotion in cats cannot be explained by changes in motoneuron excitability alone (Drew & Rossignol, 1987; Duysens & Loeb, 1980; Miller et al, 1977).

Interneurons may be another site responsible for controlling the transmission of sensory information. The involvement of interneurons has not been thoroughly explored because identification of active interneurons that are a part of a reflex pathway is technically difficult (Grillner & Wallen, 1985). Interneurons that discharge rhythmically with locomotor discharges have been identified in the spinal cord of mammals (reviewed in Sillar, 1991). However, the identification of interneurons that receive both sensory and locomotor input has only been reported in vertebrates with simple spinal circuitry (reviewed in Sillar, 1991). Afferent input may be modulated at the interneuronal level by different mechanisms. For instance, sensory information could be gated by postsynaptic modulation of the interneurons. Other evidence has proposed that afferent input could be modulated by the summation of parallel excitatory input from the interneurons onto the motoneuron (Sillar, 1991). Until the specific input to and the projections of interneurons have been identified in mammals, the neural mechanisms that are involved in modulating reflexes at the interneuronal level will be difficult to prove.

Phasic changes of polarization of the primary afferent terminals have been measured during locomotion. Evidence in fictive locomotion of spinal and decorticate cats suggested that sensory input was presynaptically inhibited as a function of the locomotor cycle at the primary afferent terminal (Bayev & Kostyuk, 1982; Dubuc et al, 1988; Gossard et al, 1990). During fictive locomotion, the primary afferents were cyclically depolarized. The primary afferents were more depolarized during the swing than the stance phase (Gossard et al, 1990; Duenas et al, 1990; Dubuc et al, 1988). This would suggest that sensory input was gated more during the swing than the stance phase; however, cutaneous stimuli evoked consistent responses during the swing phase and more variable responses during the stance phase (Dubuc et al, 1988). The functional significance of phasic changes of polarization at the primary afferent

terminals remains unclear.

In summary, sensory afferents relay information via numerous spinal pathways where the information interacts with the CPG and other afferent input to modify walking (Rossignol et al, 1988; Gossard et al, 1990). The exact mechanisms responsible for modulation of spinal reflexes have not been identified, but it is likely that mechanisms at the primary afferent terminal, the interneuronal level, and the motoneuronal level all play a role in the transmission of sensory input.

## **2.5 Methodological considerations of evoking a cutaneous reflex.**

Different methodologies including the type of stimulus and the site of stimulation have been used to study phase-dependent reflex activity in humans. Investigators have commonly applied an electrical stimulus to either a cutaneous or mixed nerve (Duysens et al, 1990; Yang & Stein, 1990; Crenna & Frigo, 1984; Kanda & Sato, 1983), or to the skin on the foot (Belanger & Patla, 1984). Electrical stimuli are preferred because there is better control of the stimulus intensity (Garnett & Stephens, 1980; Forssberg et al, 1975), and easier identification of latencies (Shahani & Young, 1971). However, reflex responses elicited during everyday activities are more likely due to mechanical perturbations not electrical. No comparisons have been made between cutaneous reflex responses elicited by mechanical and electrical stimuli in humans during walking because it is technically difficult to apply a consistent, unexpected mechanical stimulus. Under static conditions in normal subjects, Garnett and Stephens (1980) found no difference in reflex responses elicited in the first dorsal interosseous muscle following electrical or mechanical stimulation. Information gathered from animals also indicated that the 2 types of stimuli elicit similar reflex responses during walking (Forssberg et al, 1975, 1977; Duysens & Pearson, 1976; Wand et al, 1980; Drew & Rossignol, 1987).

The intensity of the stimulus is another factor that may influence the degree of reflex modulation during walking. A noxious stimulus which stimulates the A $\delta$  fibres elicits a more stereotyped flexor response throughout the entire step cycle (Duysens et al, 1990; Belanger & Patla, 1984; Forssberg, 1979; Forssberg et al, 1977) and is

more likely to disrupt the locomotor rhythm (Duysens & Stein, 1978) than a weaker stimulus. Although noxious stimuli applied during walking in normal subjects produced phase-dependent responses in the flexor and extensor muscles (Crenna & Frigo, 1984; Belanger & Patla, 1984), reflex reversal was not observed. Studies that have identified reversal of reflexes (Duysens et al, 1990; Yang & Stein, 1990) used weaker stimuli that excited primarily large cutaneous afferents. There exists some uncertainty as to the exact afferent fibres that are stimulated with a low intensity stimulus to a mixed nerve. Although low threshold cutaneous afferent fibres are excited, other low threshold afferent fibres, such as muscle, tendon or joint afferents, may also be excited (Schomburg, 1990).

Another methodological issue concerns the site of stimulation. Stimulation of peripheral nerves, either sensory nerves (Kanda & Sato, 1983; Crenna & Frigo, 1984; Duysens et al, 1992; Yang & Stein, 1990) or mixed nerves (Yang & Stein, 1990; Duysens et al, 1992), or direct stimulation of the skin of the foot (Belanger & Patla, 1984) have been applied to humans during walking. The disadvantage of applying the stimulus to a cutaneous nerve or to the skin is that the intensity of the stimulus cannot be easily quantified (Meinck et al, 1983). Moreover, the intensity of the stimulus will vary throughout the step cycle because of movement (Capaday & Stein, 1986). The effect of the stimulus can be monitored by the compound muscle action potential (M-wave) when a mixed nerve is stimulated (Meinck et al, 1983; Yang & Stein, 1990). Because the reflex responses evoked by mixed or cutaneous nerve stimulation appear to be similar (Yang & Stein, 1990; Duysens et al, 1992), some investigators have preferred to stimulate mixed nerves so that the strength of the stimulus can be measured throughout the step cycle.

## **2.6 Summary**

Phase-dependent reversal of cutaneous reflexes is characteristic of normal human locomotion (Duysens et al, 1990, 1992; Yang & Stein, 1990). The type of response is dependent upon when cutaneous stimuli were applied in the step cycle. Neural mechanisms responsible for modulation have not been identified, but reflex

modulation can occur at many different sites. Findings from animal studies indicate that sensory input may be controlled at the premotoneuronal or motoneuronal levels (Dubuc et al, 1988; Gossard et al, 1990; Sillar, 1991). The functional significance of modulation of the cutaneous reflex is unclear. It is likely that reflex reversal plays a functional role in maintaining stability during locomotion (Forssberg, 1979; Duysens et al, 1992). Further insight into the functional significance may be gained by examining the behaviour of cutaneous reflexes in subjects with spinal cord damage who experience locomotor deficits.

Individuals with incomplete spinal cord injuries frequently exhibit exaggerated spinal reflexes, poor control of voluntary movement and altered sensation (Chapman & Wiesendanger, 1982; Dimitrijevic & Nathan, 1967a), which often contribute to instability during walking (Waters & Lunsford, 1985; Hussey & Stauffer, 1973). It is unclear whether SCI individuals demonstrate reflex reversal of the cutaneous reflex during walking. Because the behaviour of the cutaneous reflex may affect walking stability, the study of this reflex during walking in SCI subjects is especially important. Should the findings from this study demonstrate that modulation of the reflex is normal, then the mechanisms responsible for reflex reversal can be assumed to be intact in SCI individuals. However, if reflex reversal is not observable in this clientele, the mechanisms responsible reflex modulation may either be absent or altered. Furthermore, the behaviour of the abnormal reflex response may contribute to the walking problems SCI individuals experience.

## **CHAPTER 3**

### **METHODOLOGY**

#### **3.1 Study design**

This was a descriptive study that examined reflex responses evoked by cutaneous stimuli during walking in subjects with incomplete spinal cord injury. Electromyographic responses were examined in the TA and SOL muscles during level walking on a treadmill. The EMG activity from these muscles were later analyzed and compared to another study which examined the reflex responses recorded in normal subjects under similar testing conditions (Yang & Stein, 1990). The extent of neurological involvement of the subjects was obtained by information collected from a clinical assessment (Appendix A).

#### **3.2 Subjects**

The study examined 10 incomplete spinal cord injured adult volunteers who could ambulate. Subjects were recruited from the community through the Canadian Paraplegic Association. The subjects were not stratified according to the level or duration of the spinal cord injury since flexor reflex responses in SCI patients appeared to be similar regardless of the level of spinal injury (Meinck et al, 1985; Shahani & Young, 1971) or duration of the condition (Meinck et al, 1985). Testing protocol required that the subjects have a walking tolerance of at least 2 minutes with or without the use of their assistive walking devices. Subjects must have also demonstrated one or more clinical signs of spasticity, including hyperactive deep tendon reflexes, upgoing plantar response or sustained ankle clonus. Individuals with other medical or neurological complications that may have influenced the behaviour of spinal reflexes or the ability to walk were excluded from the study.

Prior to the day of testing, a screening session was arranged with the subject. Upon agreement to participate, each subject gave his/her informed consent in writing (see Appendix B for consent form). Subject confidentiality was maintained; no data which identified individuals were available to persons or agencies outside of the

project.

### **3.3 Methodology**

#### **3.3.1 Recording parameters**

Electromyographic activity of the TA and the SOL muscles were recorded with bipolar surface disk electrodes (1 cm diameter; Beckman type, silver/silver chloride). The TA was selected because it exhibited the most consistent reversal during walking in normal subjects (Yang & Stein, 1990). Under static testing conditions in normal subjects, the TA muscle was also reported to have the lowest reflex threshold and highest stability of response in leg muscles (Meinck et al, 1985). The recording electrodes were securely attached over the TA muscle, approximately 5 cm below the tibial plateau and slightly lateral to the anterior border of the tibia. The recording electrodes for the SOL were positioned lateral to the midline, below the lateral gastrocnemius muscle. The SOL muscle was selected because reflex reversal had previously been reported in this muscle (Yang & Stein, 1990), and recordings from both the TA and SOL muscles would provide information about the behaviour of an agonist/antagonist pair. The ground electrode was positioned along the anterior aspect of the lower limb between the stimulating and recording electrodes of the TA (Fig. 2). To measure the step cycle, footswitches were placed under the heel and great toe of the ipsilateral shoe. Each footswitch generated a different voltage level. The stance phase was designated as the time from heel contact to toe-off, whereas the swing phase started from toe-off to the next heel contact.

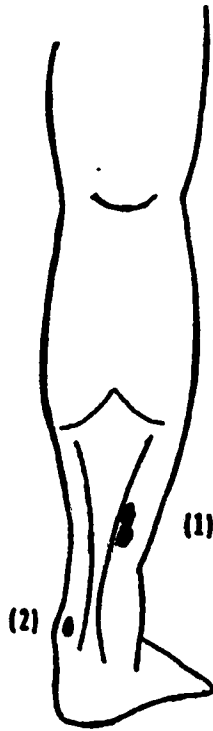
#### **3.3.2 Stimulation parameters**

The posterior tibial nerve was stimulated with a 1-cm disk electrode (the same type of electrode that was used for recording) attached posterior to the medial malleolus while the indifferent (anode) electrode was attached to the lateral aspect of the ankle (Fig. 2). The posterior tibial nerve was selected because it is a mixed nerve which contains predominantly cutaneous afferents from the sole of the foot with some efferent fibres supplying the small intrinsic foot muscles. Therefore, the direct motor

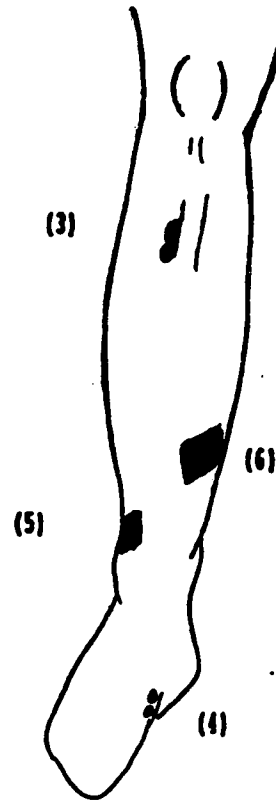


**A**

Posterior View

**B**

Anterior View

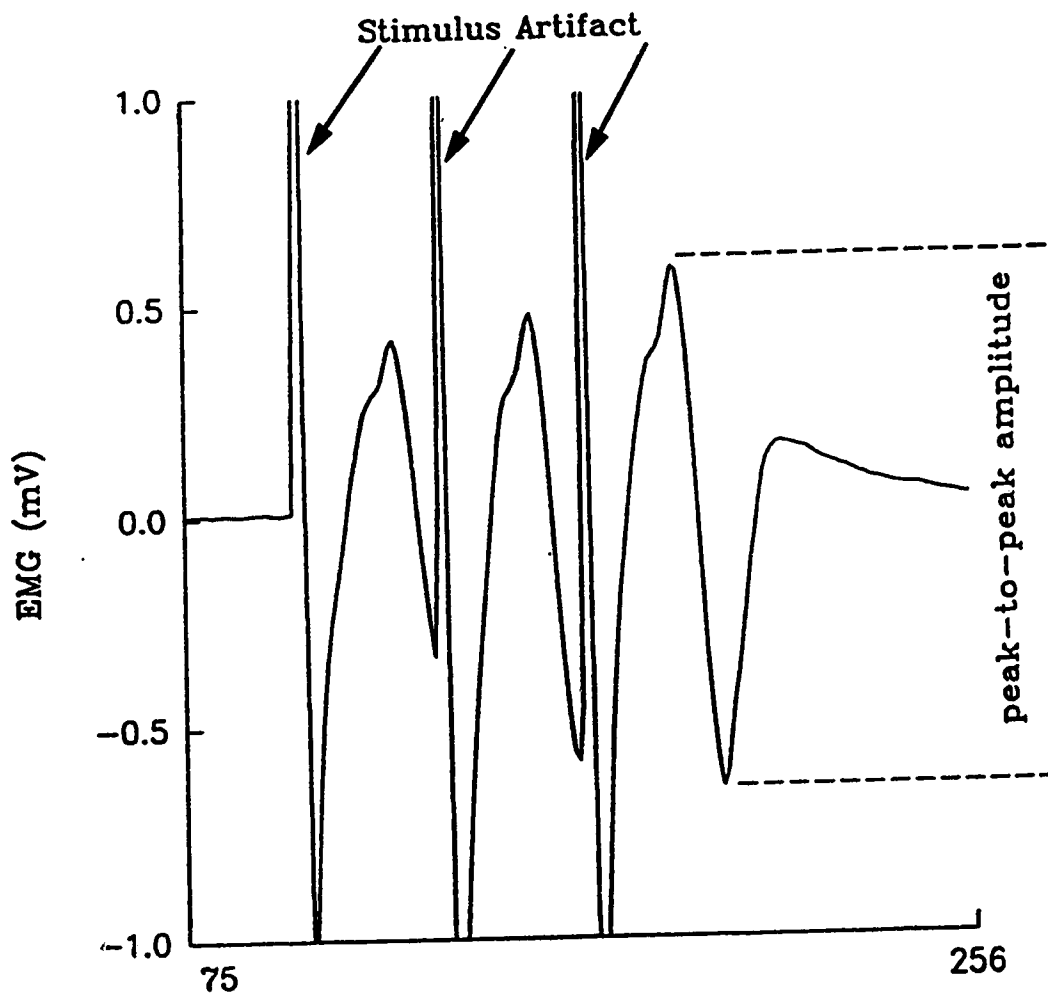


**Figure 2.** Placement of the recording and the stimulating surface electrodes on the lower limb. **A:** recording electrode positioned over the SOL muscle (1) located below the gastrocnemius, lateral to the Achilles tendon. The stimulating electrode (cathode) (2) is positioned over the posterior tibial nerve near the medial malleolus. **B:** recording electrode for the TA muscle (3) is positioned approximately 5 cm below the tibial plateau and 1 cm lateral to the anterior border of the tibia. The M-wave is measured by the surface electrodes (4) placed over the abductor hallucis muscle. The indifferent electrode (anode) (5) is placed on the lateral side of the ankle. The ground electrode (6) is positioned along the anterior aspect of the lower limb between the stimulating cathode and the recording electrodes over the TA muscle.

response of the abductor hallicus muscle could be used as a measure of the effective stimulus strength. Recording electrodes were securely attached over the abductor hallicus muscle to record the direct motor response from this muscle (Fig. 2). A set of triple monophasic pulses, 0.3 ms in duration and 10 ms apart, was delivered to the posterior tibial nerve. The amplifiers and stimulator connected to the subject were electrically isolated to prevent unwanted electrical shock.

The stimulus intensity was expressed as a function of the motor threshold, that is, the minimum stimulus voltage required to elicit the compound action potential (M-wave) in the abductor hallicus muscle. The motor threshold was identified by applying a single monophasic pulse, 0.3 ms in duration, to the posterior tibial nerve under resting conditions. The stimulus voltage was gradually increased until the M-wave was first seen on the oscilloscope. Yang and Stein (1990) found that the middle latency response which reversed in direction during walking, was consistently evoked at a stimulus intensity between 1.5 and 2.0 times the motor threshold. Therefore, the strength of the stimulus was kept within 1.5 to 2.0 times the motor threshold to stimulate the large cutaneous afferents. The maximum M-wave, which represents the lowest voltage required to recruit 100% of the motoneuron pool, was also identified. The intensity of the stimulus was kept well below the maximum M-wave because once the maximum M-wave was reached, the response was saturated and the M-wave no longer accurately reflected the intensity of the stimulus.

During walking the movement between the stimulating electrode and the nerve will cause the strength of the stimulus to vary throughout the step cycle (Stein & Capaday, 1986). However, a constant stimulus throughout the step cycle is necessary for comparison of reflex behaviour over the cycle. The effective stimulus strength was measured by the peak-to-peak amplitude of the M-wave in the abductor hallicus muscle to ensure that a constant stimulus was delivered (Fig. 3). A constant M-wave meant that the same proportion of efferent and presumably afferent fibres were stimulated (Yang & Stein, 1990). A target level for the amplitude of the M-wave was selected based on the first trial of walking in which stimuli were applied. When the amplitude of the M-wave varied more than 20% from the selected target level, the

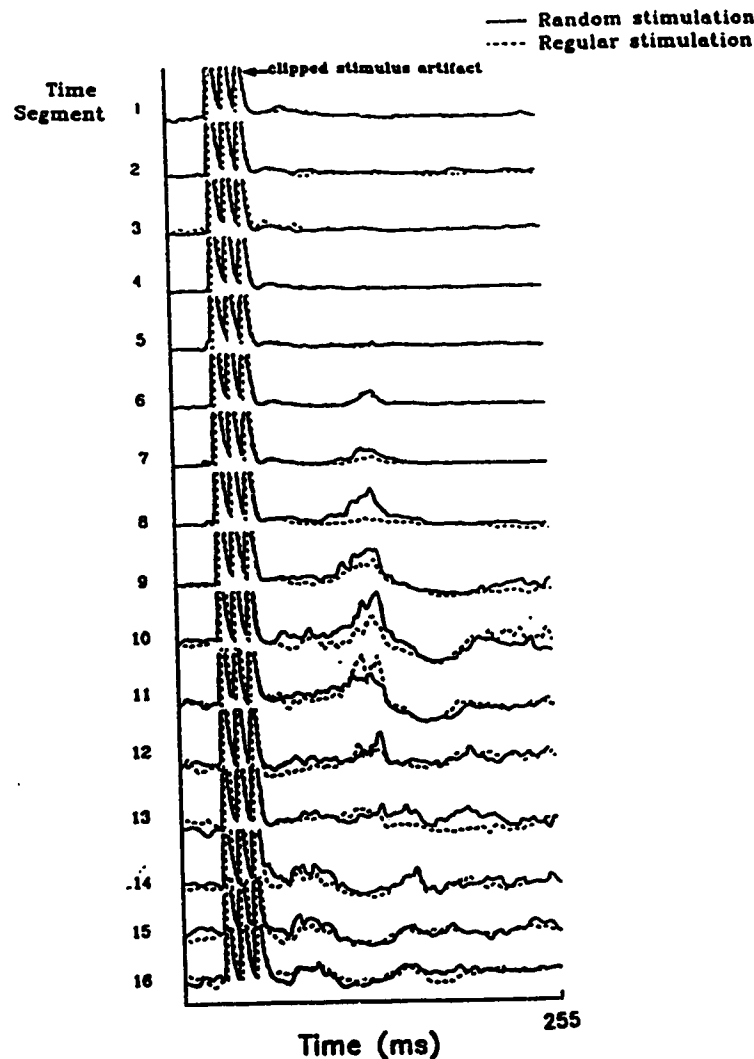


**Figure 3.** Averaged M-wave response of the abductor hallucis muscle to tibial nerve stimulation in a normal subject during one time segment in the gait cycle. A train of 3 pulses (the stimulus artifacts are clipped) elicited 3 M-wave responses ( $n=25$ ). A preset time delay of 30 ms allowed the prestimulus EMG activity to be recorded in other muscles. The third M-wave was measured by the peak-to-peak amplitude to assess the effective stimulus strength. The third M-wave was selected for measurement because it was the only response that was not contaminated by subsequent stimulus artifacts.

trial was repeated at a different stimulus intensity until a comparable match was obtained for all time segments of the step cycle (Yang & Stein, 1990; Capaday & Stein, 1986). The number of trials that could be repeated was dependent on the subject's walking tolerance. Should the subject fatigue and the M-wave amplitude for a time segment still fall outside of the range, that time segment was discarded from the final analysis.

The stimulus was delivered by one of 2 methods which was determined by the subject's walking tolerance. The preferred method of stimulus delivery required that the subject walk a total duration of 20 minutes. Nine of the 10 SCI subjects were tested by this preferred method. During the trial, stimuli were randomly applied approximately 2 to 5 sec apart by a random pulse generator. No more than 1 stimulus per step cycle was delivered. The step cycle was divided into 16 equal time segments because this provided good temporal resolution of the responses within the step cycle (Capaday & Stein, 1986). Approximately 10 to 20 stimuli were averaged for each of the 16 time segments (or phases). Should the subject fatigue during the trial, a rest period was allowed. Previous studies have used this method of random stimulus delivery during walking for both normal and neurologically impaired subjects (Yang & Stein, 1990; Capaday & Stein, 1986; Duysens et al, 1990; Yang et al, 1991).

An alternative method of stimulus delivery was used for 1 subject (GB) who had a walking tolerance of only a few minutes. Stimuli were delivered by the investigator to target only one of the 16 time segments in the step cycle per trial. Although the stimuli were applied at a constant time within the step cycle, the investigator arbitrarily selected the step cycle to apply the stimulus. This method consisted of shorter but more frequent walking trials, and allowed more rest periods. One trial typically took 2 minutes to complete. Although some of the randomness of the stimulus delivery was sacrificed with this method, the subject's walking tolerance was not compromised. Pilot data obtained from normal subjects compared both methods of stimulus delivery and found that there were no major differences in the subjects' responses (Fig. 4).



**Figure 4.** Comparison of 2 methods of delivering stimuli to the posterior tibial nerve during walking in a normal subject. The reflex responses (background activation has been subtracted) were recorded from the TA: A) randomly in time by a pulse generator (solid lines), or B) regularly by the investigator (dotted lines), for each of the 16 phases of the step cycle. In method A, the stimuli generated by the pulse generator were randomly applied throughout the step cycle. One trial required approximately 20 minutes of continuous walking to gather the data from the 16 phases. In method B, stimuli arrived at a constant time within the step cycle for a single trial. Although the stimulus was delivered at a regular time within the step cycle, the investigator arbitrarily selected the step cycle to be stimulated. One trial using method B took approximately 2 minutes of walking to collect the data from one time segment in the step cycle. This method required 16 trials to gather all of the data. In this subject both methods elicited similar reflex responses, no major differences were observed between the 2 methods of application.

### **3.3.3 Experimental Procedures**

Prior to the day of the experiment, a practice session was scheduled to acclimatize subjects who were unfamiliar with walking on the treadmill. The subject walked at a self-selected speed for both the practice and experimental sessions. To prevent falling, the subject had use of a) handrails, b) a switch to stop the treadmill, and if necessary c) a body harness system.

On the day of the experiment, prior to data collection on the treadmill, an assessment of muscle tone and ambulation was completed to obtain a clinical profile of the subject's ambulatory status. The assessment consisted of 2 sections; the first part was a self-report of the subject's functional capabilities, while the second part included common clinical measures of muscle tone (Chan, 1986) and ambulatory status (Nelson, 1974) (see Appendix A). Muscle tone was described by 3 parameters: deep tendon reflexes of the ankle and knee, ankle clonus and plantar response. The deep tendon reflexes and ankle clonus were graded on a modified nominal scale and the plantar response was described by the direction of toe movement (Chan, 1986). Because walking speed is a simple yet useful indicator of gait (Andriacchi et al, 1977) and is often compromised in patients with spinal cord injury (Waters et al, 1989), the maximum walking speed was used as a descriptor of functional status (Nelson, 1974). Subjective information from the assessment provided further detail about the subject's functional mobility.

The experiment was conducted in 4 stages. During the first stage, the reflex responses were recorded when the subject was standing to ensure that the selected stimulus intensity elicited a visible response. Three pulses, 1.50 to 1.70 times the motor threshold, were applied to the posterior tibial nerve while subjects maintained a submaximal isometric contraction of the ankle dorsiflexors. Approximately 20 stimuli were averaged. Because a certain amount of tonic activity is required to detect any reflex responses (Meinck et al, 1983; Dimitrijevic & Nathan, 1967b), the SOL did not show a distinct response during contraction of the ankle dorsiflexors. Therefore, 5 subjects were subsequently asked to contract the SOL submaximally while stimuli were applied to the posterior tibial nerve.

The remaining stages entailed walking on the treadmill. Walking trials which involved the application of stimuli were referred to as **disturbed walking**, and those walking trials in which no stimuli were applied were referred to as **undisturbed walking**. During the second stage of the experiment, one trial of undisturbed walking was recorded. The TA and SOL EMG activity, and the footswitch signal were recorded for approximately 30 strides. The data were collected and averaged on-line to determine the average EMG profile for one step cycle. The duration for a step cycle was determined at this point, and the time parameters calculated for applying the stimuli during the disturbed walking trials. During the third stage of the experiment, the disturbed walking trials were recorded as previously described. The fourth and final stage of the experiment consisted of another trial of undisturbed walking to determine whether the subject had altered his/her walking pattern over the course of the experiment.

### **3.4 Data Analysis**

#### **3.4.1 On-line Analysis**

The raw EMG signals for the TA, SOL and abductor hallicus muscles were amplified and high pass filtered at 10 Hz before they were recorded on VHS tape for off-line analysis. The footswitch and stimulus marker were also recorded on tape. The abductor hallicus EMG and footswitch signals were observed on an oscilloscope without further processing, while the TA and SOL signals were full-wave rectified and smoothed (low pass filtered at 100 Hz) before being displayed on the oscilloscope.

On-line analysis of the **undisturbed walking** consisted of the computer averaging the rectified and smoothed (low pass filtered at 100 Hz) EMG signals from the SOL and TA muscles, and the raw footswitch signal. The footswitch signal also triggered the initiation of averaging by the computer. Because no stimuli were applied during this trial of walking, the EMG from the abductor hallicus muscle was not recorded. The average footswitch signal was used to calculate the exact duration of the step cycle.

For the disturbed walking trials, a random pulse generator signalled a second pulse generator (Master-8) to produce two signals. The first signal was a short pulse which triggered the computer to commence averaging. The second signal consisted of a set of delayed triple pulses which were sent to a Grass SD9 Stimulator to stimulate the posterior tibial nerve.

Because the stimuli were applied randomly in time, the computer needed to determine in which of the 16 time segments of the step cycle the stimuli arrived. The assignment of the stimulus to the appropriate time segment was determined by its relationship to the footswitch signal. The footswitch signal at heel contact triggered a ramp generator to produce a voltage signal in the shape of a ramp. The amplitude of the ramp corresponded to the time of the step cycle. Based on the amplitude of the ramp signal, the computer then determined which of the 16 time segments a response occurred in (see Fig. 5 for block diagram of experimental set-up summarizing on-line analysis).

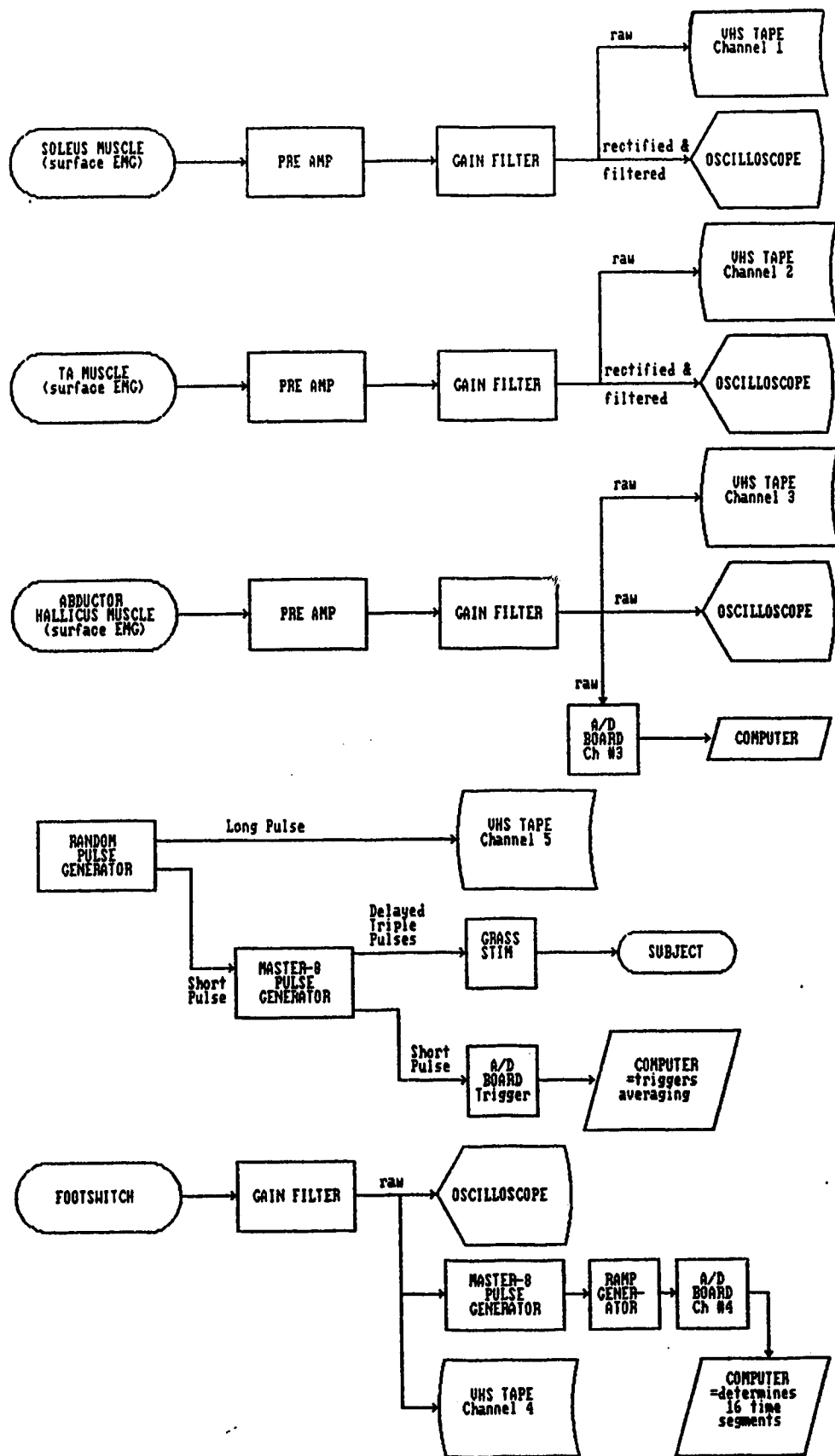
On-line analysis of the disturbed walking involved averaging the EMG signal from the abductor hallicus muscle to determine the M-wave amplitude for each of the 16 time segments in the step cycle. When the stimulus was delivered, the computer averaged the EMG data from the abductor hallicus muscle for a duration of 60 to 70 ms, at a sampling rate of 3 kHz. By having quick access to the M-wave amplitude, the strength of the stimulus was adjusted from trial to trial. The adjustment allowed for a good match of the M-wave amplitude in all parts of the step cycle. Electromyographic signals from the SOL and TA muscles were not averaged on-line because the sampling rate required for these responses differed from that for the abductor hallicus muscle.

### **3.4.2 Off-line Analysis**

Raw EMG activity and the footswitch signals were played back from the VHS tape. The TA and SOL EMG signals were high pass filtered at 10 Hz, full-wave rectified and then low pass filtered at 100 Hz to smooth the signals. Data from the undisturbed walking trials were analyzed in the same manner off-line as was done on-



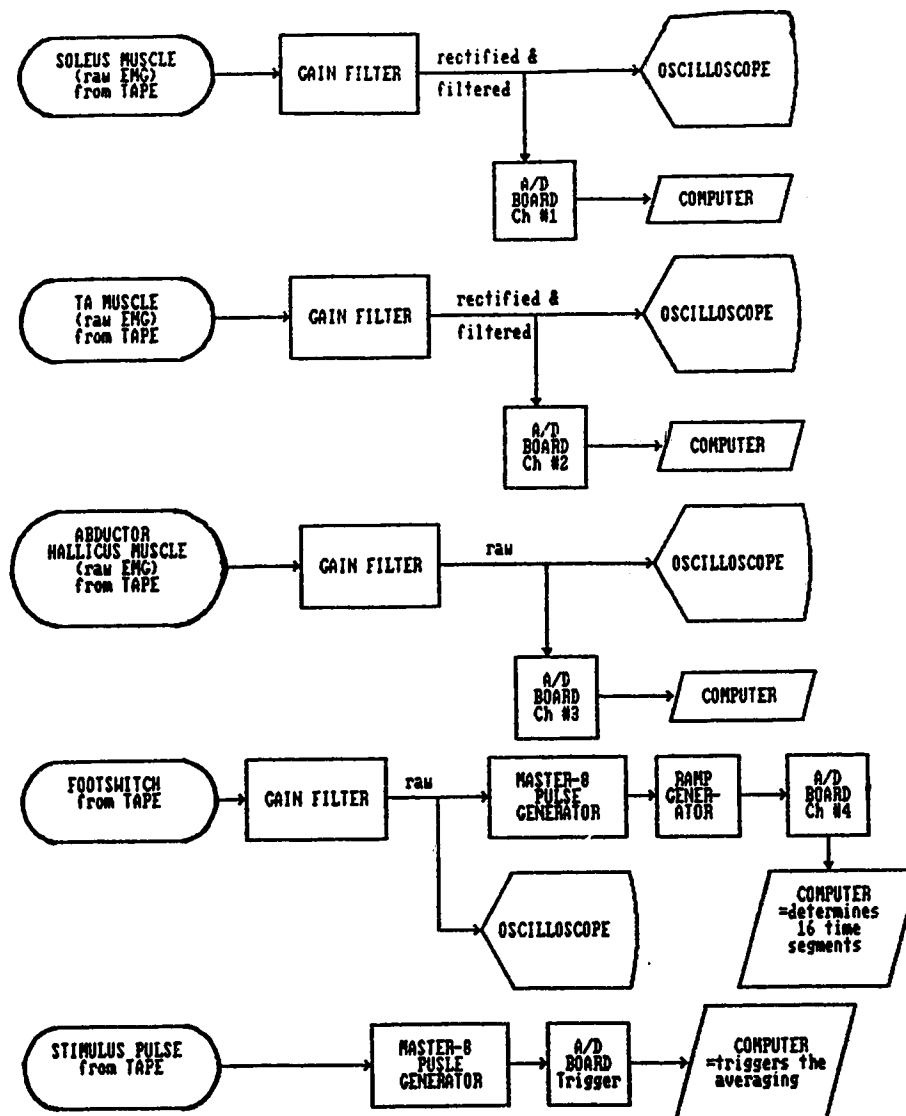
**Figure 5.** Experimental set-up (on-line analysis). The EMG signals were amplified and filtered. The raw signal was recorded on tape while either the raw or rectified signal was monitored on the oscilloscope. The raw signal from the abductor hallucis muscle was also forwarded to the computer for analog/digital conversion and averaging of the responses. For the disturbed walking trials, the random pulse generator triggered the Master-8 to generate triple pulses to the Grass Stimulator with a delay. The Grass Stimulator then delivered 3 pulses to the subject. A long pulse from the random pulse generator which coincided with the beginning of the stimulus was recorded on tape. A shorter pulse, also coinciding with the beginning of the stimulus (generated by the Master-8 unit) was used to trigger the computer to average. The footswitch signal which was also recorded on tape, triggered the Master-8 to generate a short pulse to the ramp generator. A signal whose amplitude corresponded to the time in a step cycle, was produced by the ramp generator. Based on the amplitude of the ramp signal, the computer calculated when in a step cycle a stimulus occurred.



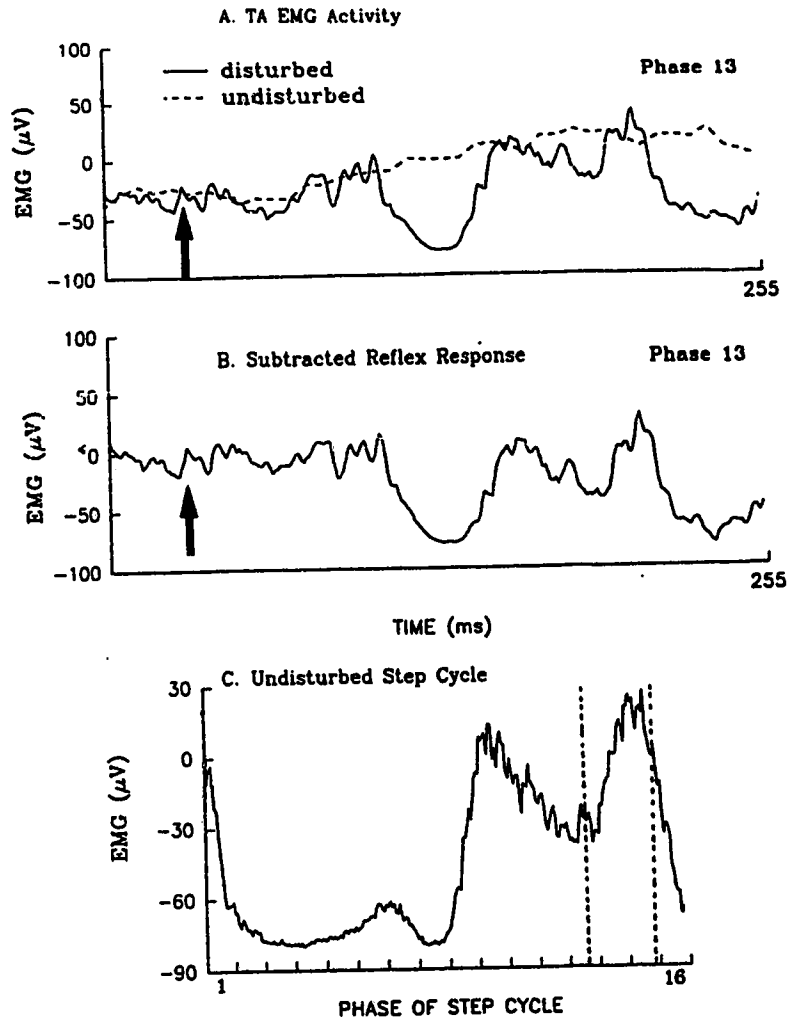
line. For the disturbed walking trials, the stimulus marker triggered the computer to start averaging the EMG signals. For each trial, the computer sampled the TA and SOL data at 1 kHz over a duration of 256 ms for each of the 16 segments in the step cycle. This duration was long enough to record both the short, medium and long latency responses of the reflex. Approximately 10 to 20 responses were averaged for each of the 16 time segments. Because the computer program assumed that the step cycle was constant, and whereas the subject's step cycle may have varied slightly over a trial, the time segments at the end of the step cycle did not always have an average of 10 or more trials. In the final analysis, those time segments of the step cycle that had less than an average of 10 responses were discarded. The filtering parameters described for the on-line analysis of the abductor hallucis EMG and footswitch signals remained unchanged for the off-line analysis. The M-wave was sampled off-line at 3 kHz over a duration of 60 to 70 ms. The footswitch signal was processed in the same manner as for the on-line analysis (see Fig. 6 for block diagram of experimental set-up summarizing off-line analysis).

To identify the true reflex behaviour, the background EMG activity associated with undisturbed walking was subtracted from the disturbed walking trials (Duysens et al, 1990; Yang & Stein, 1990). Prior to the subtraction, the undisturbed walking trials recorded from the beginning and the end of the experiment were compared for differences in timing and amplitude. The undisturbed walking trial that produced the better agreement with the disturbed walking trials was used in the final analysis.

The subtraction process consisted of matching the reflex responses for each of the 16 time segments from the disturbed walking trials to the EMG activity from the corresponding time of the undisturbed step cycle. Because the subtraction removed the background EMG activity of the undisturbed walking from the disturbed response, a baseline near zero should have been seen prior to the arrival of the stimulus if the subtraction was accurate (Fig. 7). In some cases, the subtraction was not ideal because the EMG activity from the undisturbed walking trials did not match the background EMG from the disturbed walking trials. There were a number of possibilities for this discrepancy; for instance, the SCI subjects may have experienced



**Figure 6.** Experimental set-up (off-line analysis). Raw EMG and footswitch signals were played back from the tape, amplified, and the EMG signals filtered. The raw or rectified EMG signals were monitored on the oscilloscope and were converted from analog to digital signals before being averaged by the computer. The stimulus marker from the tape triggered the computer to commence averaging to obtain the reflex responses. The ramp generator generated a voltage signal whose amplitude corresponded to the time in the step cycle. Based on the amplitude of the ramp signal, the computer determined the segment of the step cycle when a stimulus occurred. The computer then averaged the responses that occurred within each time segment.



**Figure 7.** Subtraction process (off-line analysis). **A:** The rectified and smoothed EMG response ( $n=24$ ) of the TA muscle which was evoked by 3 stimuli (arrow depicts the beginning of the stimulus artifacts) delivered in the 13th segment (mid-swing) of a step cycle in a healthy subject. The EMG pattern included the normal EMG associated with undisturbed walking and the reflex response. The EMG pattern from undisturbed walking (dotted line) was subtracted from the total (solid line). Both the disturbed and undisturbed EMG patterns occurred at the same time during the step cycle (between the grid lines in C). **B:** EMG pattern of the true reflex response after the background activity was subtracted. Note that the subtraction resulted in a near zero baseline in the EMG prior to the arrival of the stimulus (30 ms before the first stimulus artifact). **C:** The average EMG pattern ( $n=38$ ) in the TA during undisturbed walking for the duration of a step cycle. Horizontal axis is divided into 16 equal time segments. The vertical axis is the amplitude of the EMG activity ( $\mu V$ ). The EMG activity between the grid lines, which corresponded in time to the reflex response, was used for subtraction.

some muscle fatigue during the testing session or the step cycle may have varied amongst the trials. For the subtraction to be acceptable, the average prestimulus value (0 to 30 ms) after subtraction had to fall within a predefined range. The acceptable range was designated arbitrarily to be plus or minus 25% of the peak EMG value recorded during the undisturbed walking. The responses whose average prestimulus values fell outside of this range were discarded.

The reflex response consisted of 3 components at early, middle and late latencies. Because reflex reversal has been identified during the middle latency response in normal subjects (Yang & Stein, 1990), this response was specifically inspected for reflex modulation. The reflex component occurring at a medium latency was defined by inspection of each data set because the conduction time to and from the spinal cord varies amongst individuals (Roby-Brami & Bussel, 1987; Duysens et al, 1990). The variation in latency is related to the length of the peripheral nerves which varies with an individual's height. Investigators have reported that the latency of the middle response occurred between 50 and 90 ms after the first stimulus in the lower limb of normal subjects (Yang & Stein, 1990). The documented latencies were used as guidelines only. For every subject the latency of the middle response was measured from the first stimulus artifact to the time when the response fell or rose above the zero baseline. The value of the response was described as the average amplitude over a defined window of time. In SCI subjects from this study, this window for the middle latency response was approximately 60 to 110 ms for the TA and 50 to 90 ms for the SOL.

The middle latency response was classified as being excitatory, inhibitory or no response. For each subject an error range was defined above or below which a response was considered to have occurred. The error range was determined by the mean, plus or minus one standard deviation, of the 16 prestimulus values. Those responses falling above the uppermost limit were considered excitatory, those falling below the lowermost limit were inhibitory and those within the range were judged as no responses.

Reflex responses were omitted from the analysis if a) the effective stimulus

intensity, as measured from the M-wave amplitude, fell outside of the target range, b) there were fewer than 10 reflex responses averaged for a time segment, and c) the subtraction between the matched undisturbed EMG and the disturbed EMG fell outside of the acceptable range. The responses from the time segments that met the criteria were then inspected to determine whether reflex reversal had occurred. Reflex reversal was defined as a change in direction of the middle latency response during the step cycle. For example, a reflex reversal would have occurred in the TA if the excitatory response, which is normally seen during the swing phase, reversed in direction to become an inhibitory response at another time within the step cycle. Subjects were later categorized into groups based on the pattern of reflex modulation.

The findings from the functional and clinical measures were reported to provide further information about locomotion and spasticity. A Pearson's Correlation Coefficient ( $r$ ) was calculated to determine whether there was a relationship between the classification of reflex modulation recorded in the TA and SOL, and the clinical findings (Champion, 1981).

### **3.5 Limitations of the study**

The limitations of this study included external and technical constraints of the study design. One external limitation concerned the representation of the subjects. The SCI subjects recruited for this study were not representative of the total SCI population. All of the subjects were community walkers and had achieved a certain level of functional walking. No dependent walkers were examined. Dependent walkers are individuals who walk in controlled conditions and require physical assistance to ambulate (Hussey & Stauffer, 1973). Another external limitation of the study concerned the sensitivity and specificity of clinical measures. A sensitive clinical measure of neurological involvement and functional walking ability has not yet been developed.

One technical limitation of this study concerned the type of stimulus used to evoke the cutaneous reflex. Although an electrical stimulus has technical advantages (Schomburg, 1990), an electrical stimulus is not representative of the unexpected

**mechanical stimuli encountered in daily activities. Therefore, the true functional implications of mechanical perturbations cannot be directly examined by electrical stimuli.**



## **CHAPTER 4**

### **RESULTS**

#### **4.1 Clinical findings**

The subjects examined in this study experienced incomplete traumatic spinal cord injuries, 8 cervical lesions and 2 lower thoracic lesions. All subjects reported that they were in good health and denied any neurological change over the past few months. The clinical findings showed varying degrees of deep tendon reflexes, ankle clonus, and plantar responses. At the time of testing only 2 subjects (BL, KB) were taking medication for spasticity yet they still demonstrated hyperactive reflexes. Relevant demographic and ambulatory information is reported in Table 1 while the clinical findings are presented in Table 2. The data collected from the SCI subjects were not stratified because no differences were seen between subjects who sustained different levels of neurological injury nor chronicity of onset.

All subjects could walk independently with or without assistive walking devices and were considered community walkers (Hussey & Stauffer, 1973). Self-reported walking tolerance ranged from 2 blocks to 5 kilometres. The maximal walking speed over 6 m ranged from 0.61 to 1.67 m/s. The comfortable walking speeds selected by the subjects on the treadmill ranged from 0.19 to 0.63 m/s. The comfortable walking speeds for the SCI subjects were considerably slower than that reported by other authors for normal subjects, which ranged from 1.26 m/s to 1.59 m/s for a comparable age group (Murray et al, 1966, 1969, 1970; Himann et al, 1988). Only 1 subject (LB) used an assistive walking device and the body harness during treadmill walking. The harness was used for safety purposes, rather than a means of supporting body weight. Although some of the other subjects normally used assistive devices to walk, the handrails provided adequate support for walking on the treadmill.

**TABLE 1:** Neurological and ambulatory profiles of spinal cord injured subjects. The maximum walking speed was tested overground for 6 m. The self-selected speed was the comfortable walking speed selected on the treadmill.

SUBJECT (n=10)	AGE (YRS)	SEX	BONY LEVEL OF LESION	ONSET (YRS)	MAXIMUM WALKING SPEED (m/s)	SELF- SELECTED WALKING SPEED (m/s)	ASSISTIVE WALKING DEVICE
IJ	25	M	C4-5	2	1.67	.63	nil
BL	44	M	C3-4	3	.94	.19	nil
KM	47	M	C4-5	3	.91	.55	1 cane
LB	34	M	C4-5	17	.61	.27	2 Canadian crutches
KW	33	M	C4-5	4	1.18	.28	AFO
WF	49	M	T10	5	1.50	.43	nil
LM	34	F	C4-5-6	15	1.15	.29	nil
GS	32	M	C4-5	13	.91	.45	1 Canadian crutch
KB	39	M	C4-5-6	1	1.07	.36	nil
GB	28	M	T12	3	1.54	.31	knee brace

**TABLE 2: Clinical findings of spinal cord injured subjects.**

SUBJECT (n=10)	TENDON JERK*		CLONUS+	PLANTAR RESPONSE^
	KNEE	ANKLE		
IJ	3	3	1	1
BL	4	4	2	1
KM	4	3	0	1
LB	3	1	3	1
KW	4	4	3	1
WF	4	2	0	1
LM	4	3	1	1
GS	4	2	2	1
KB	3	4	1	1
GB	3	3	1	0

\* tendon jerk scores: 0- no response; 1- hyporeflexic; 2- normal;  
3- brisk; 4- hyperreflexic

+ clonus scores: 0= no response; 1= 2-3 beats; 2= 4-10 beats;  
3= >10 beats

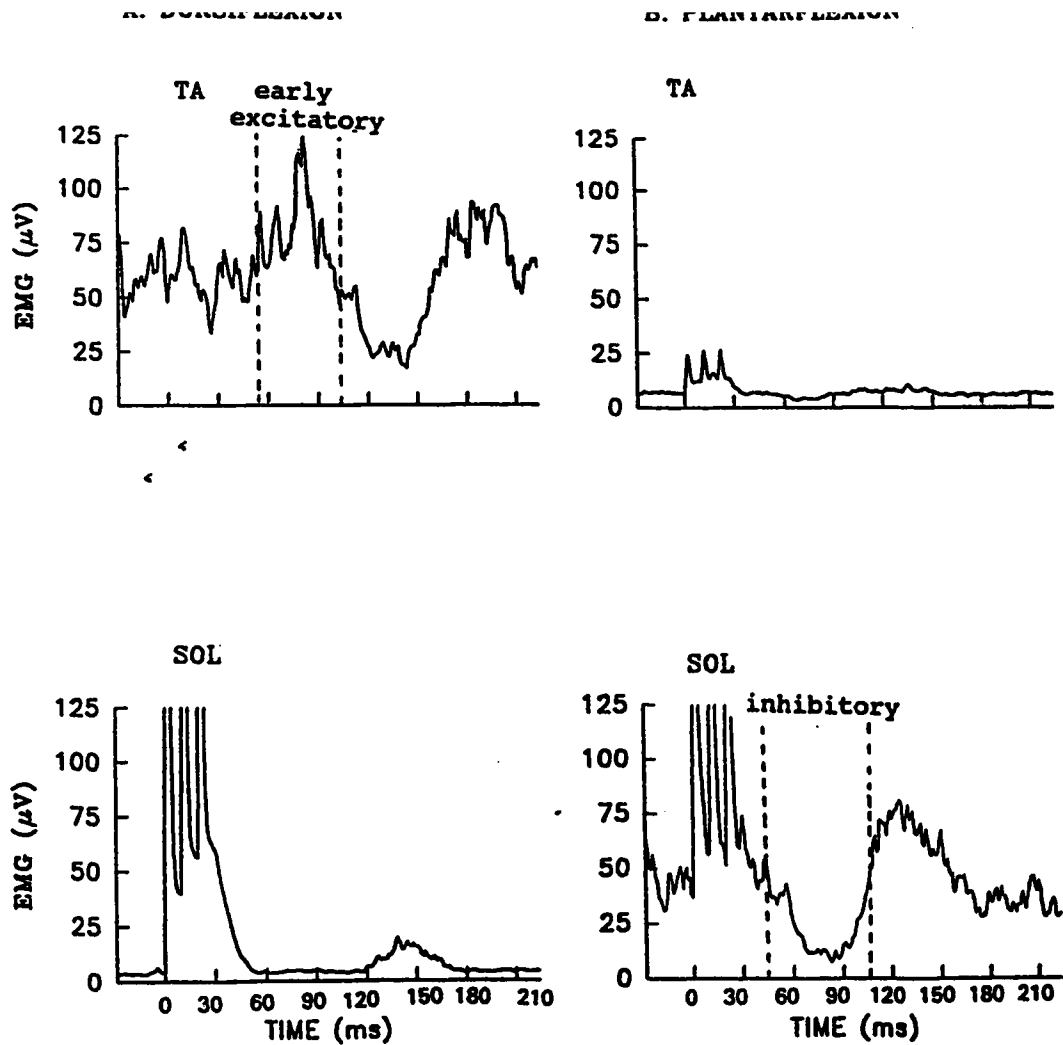
^ plantar response scores: 0= no response or downgoing;  
1= upgoing or withdrawal

## **4.2 Reflex responses evoked in standing**

Stimuli were applied to the posterior tibial nerve while subjects contracted their dorsiflexor muscles when standing. Reflex responses were evoked in the TA muscle in all 10 subjects. An excitatory response with latencies (ranging from 50 to 70 ms after the stimulus) similar to normal subjects, was seen consistently. In normal subjects an early excitatory response occurred 45 to 60 ms after the stimulus onset, and was then followed by inhibition (Burke et al, 1991; Aniss et al, 1992). The inhibitory response (50 to 90 ms latency) was the most prominent and consistent response seen in normal subjects under static conditions (Yang & Stein, 1990). In contrast, only 4 SCI subjects exhibited an inhibitory response with a somewhat longer latency of 100 to 130 ms. A late excitatory response, which occurred 160 ms after the stimulus, was seen in 3 SCI subjects. The inconsistency of the late excitatory response exhibited by the SCI subjects agreed with the findings reported in normal subjects (Yang & Stein, 1990). In summary, the early excitatory response evoked in the SCI subjects had a similar latency range as those reported in normal subjects, but the inhibitory response was less pronounced in the SCI subjects than normal subjects.

Subject KM showed responses at all 3 latencies in the TA during standing (Fig. 8A). The stimulus evoked an early excitatory response 55 ms after the stimulus artifact. An inhibitory response was subsequently seen 105 ms after the stimulus, considerably later than the inhibitory response reported in normal subjects (latency of 50 to 90 ms) (Aniss et al, 1992; Burke et al, 1991; Yang & Stein, 1990). A small late excitatory response was recorded in the SOL with a latency of 120 ms.

Three of the 5 subjects who were asked to contract their plantarflexor muscles when standing showed inhibitory responses, at a latency of 45 to 50 ms, in the SOL muscle. The other 2 subjects did not show any responses. Subject KM demonstrated the inhibitory response seen in the SOL muscle during standing, 45 ms after the first stimulus artifact (Fig. 8B). An excitatory response subsequently occurred with a latency of 105 ms after the stimulus artifact. No responses were recorded in the TA during plantarflexion.



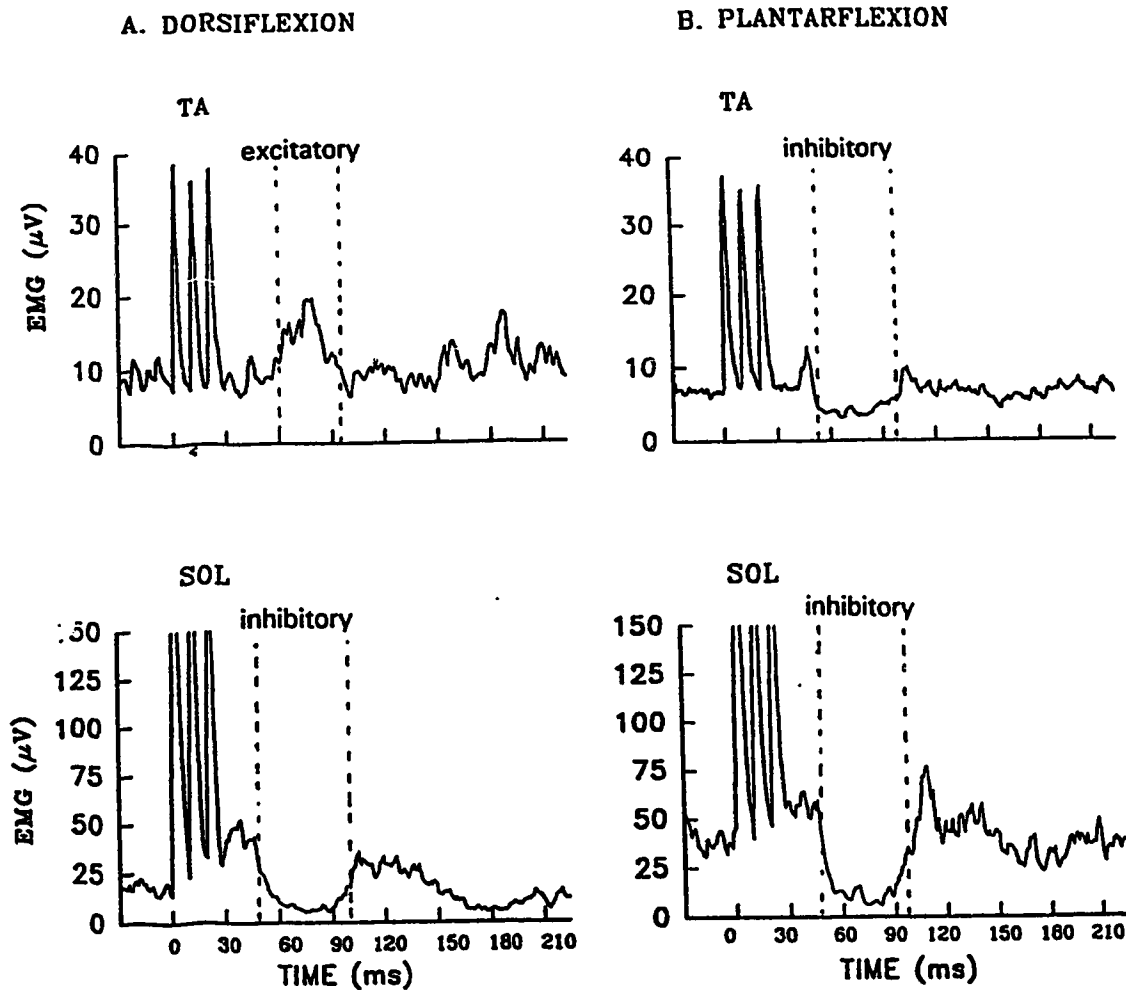
**Figure 8.** The average reflex response of the TA and SOL muscles in a spinal cord injured subject (KM) when standing. Responses were evoked by triple pulses (1.65 times the motor threshold) applied to the posterior tibial nerve. The vertical axis represents the amplitude of the response ( $\mu\text{V}$ ). A. The average responses ( $n=20$ ) of the TA and the SOL during isometric contraction of the ankle dorsiflexors. In the upper trace, an early excitatory response was evoked in the TA with a latency of 55 ms. This subject showed an inhibitory response 100 ms after the first stimulus, and a small late excitatory response at a latency of 160 ms. A small late excitatory response with a latency of 120 ms was observed in the SOL during the contraction of the dorsiflexors (lower trace). B. The average responses ( $n=20$ ) of the SOL and TA were evoked by stimuli during isometric contraction of the plantarflexors of the ankle. An inhibitory response was seen 45 ms after the first stimulus in the SOL (lower trace). An excitatory response then followed with a latency of 105 ms. In the upper trace, no responses were seen in the TA because the muscle was inactive.

Figure 9A illustrates the reflex response observed in subject LB, which was representative of the responses seen in the TA of 6 SCI subjects when standing. A small early excitatory response was evoked in the TA (60 ms latency) while the subject held an isometric contraction of the dorsiflexors. Like the 5 other SCI subjects, LB did not show an inhibitory response afterward. While the ankle dorsiflexors were contracting, subject LB exhibited an inhibitory response (50 ms after the stimulus) in the SOL. The TA and SOL were co-contracting during this trial. The amplitude and latency of the inhibitory response, seen in the SOL of subject LB during dorsiflexion, was similar to the inhibitory response seen during contraction of the plantarflexors (Fig. 9B). An inhibitory response was also seen at the same latency in the TA during contraction of the SOL, but the response could have been due to cross-talk.

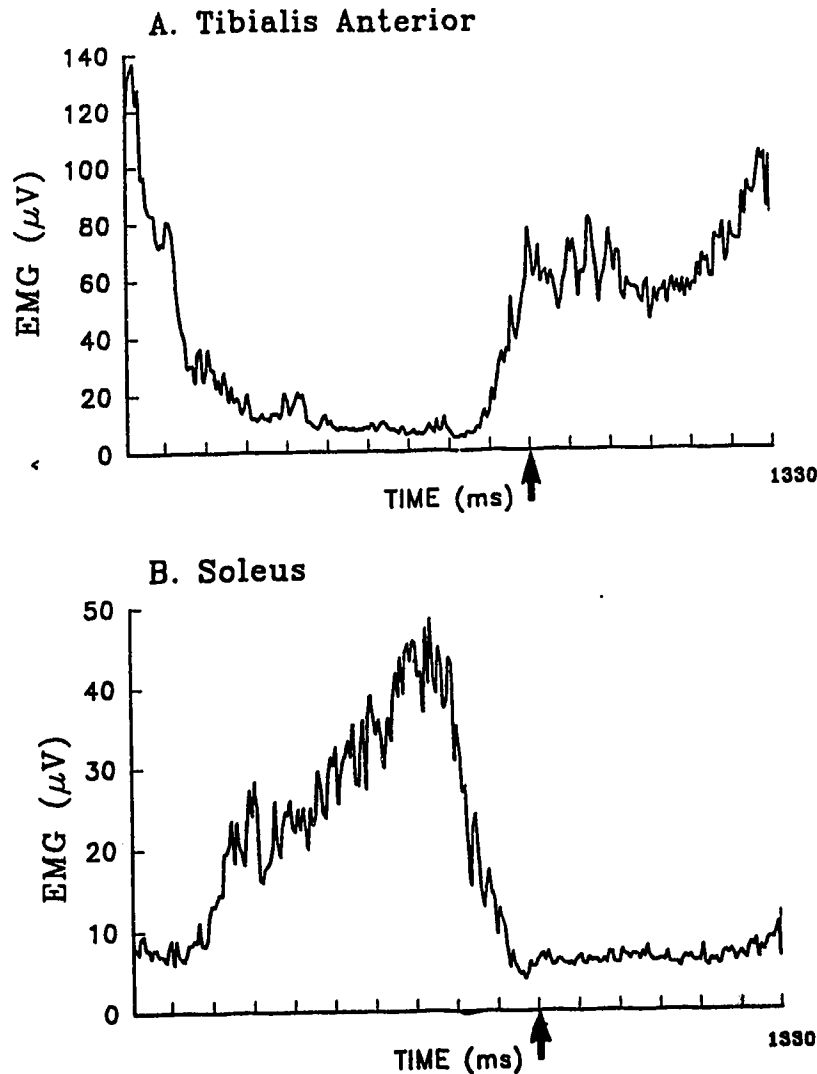
### **4.3 Undisturbed walking**

#### **4.3.1 Activation pattern of the TA and SOL muscles in a normal subject.**

The EMG patterns of the TA and SOL muscles are shown for a normal subject during treadmill walking (data obtained from pilot work) to serve as a comparison for those recorded from the SCI subjects (Fig. 10). The normal subject walked at a deliberately slower speed (0.67 m/s) to emulate the slower walking speeds of the SCI subjects. In Figure 10, 2 bursts of activity can be seen in the TA; the longer burst of TA activity occurred during the swing phase to clear the foot of the ground. During the latter half of the swing phase the activity in the TA helped to maintain the ankle at approximately 90° in preparation for heel contact (Winter, 1991). The second burst which was larger in amplitude occurred at heel contact to control lowering the foot to the ground (Winter, 1991). The EMG activity of the SOL began at heel contact and increased gradually throughout two-thirds of stance phase, peaking near the time of push-off and abruptly decreasing at toe-off.



**Figure 9.** The average reflex response of the TA and SOL muscles in a spinal cord injured subject (LB) when standing. Responses were evoked by triple pulses (1.65 times the motor threshold) applied to the posterior tibial nerve. **A.** The average response ( $n=15$ ) of the TA and SOL during isometric contraction of the ankle dorsiflexors. In the upper trace, a small early excitatory response was seen in the TA 60 ms after the first stimulus. Subject LB did not show a medium latency inhibitory response. No late excitatory response was seen. Unlike subject KM (Fig. 8), subject LB exhibited a small inhibitory response in the SOL during contraction of the dorsiflexors 50 ms after the first stimulus (lower trace). The TA and SOL were co-contracted in this trial. **B.** The average response ( $n=14$ ) of the SOL and TA during activation of the plantarflexors. In the lower trace, an inhibitory response in the SOL was seen at a latency of 50 ms. A small excitatory response was observed afterward. The inhibitory response seen in the TA during plantarflexion (upper trace) could have been due to cross-talk since it occurred at the same latency as the inhibitory response in the SOL.



**Figure 10.** The average EMG pattern of the TA and SOL muscles in a normal subject during undisturbed walking. The average responses ( $n=50$ ) are shown for the duration of a step cycle (1330 ms) in subject DB (obtained from pilot data). The horizontal axis is divided into 16 equal time segments. The arrow depicts when the swing phase commences at toe-off. **A.** The TA is primarily active during the swing phase. A burst of activity seen in the first half of the swing phase clears the foot of the ground, while another burst of activity at the end of the swing phase prepares the foot for the stance phase. At heel contact the TA is active when the foot is lowered to the ground. During the remainder of the stance phase the TA is inactive. **B.** The SOL muscle is most active during the stance phase and peaks near the end of the stance phase as the foot pushes off. Minimal activity in the SOL is seen during the swing phase. The TA and SOL muscles are reciprocally activated.

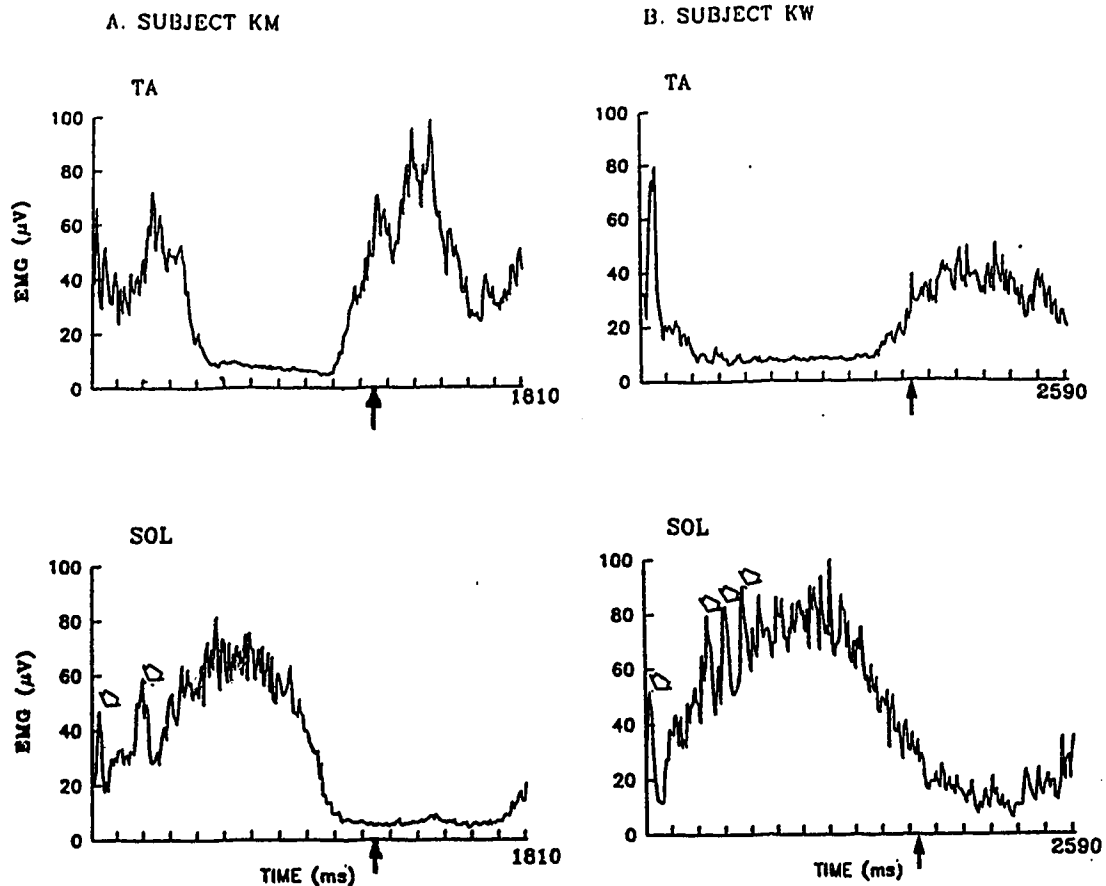


#### **4.3.2 Activation of the TA and SOL muscles in spinal cord injured subjects.**

All of the SCI subjects displayed reciprocal activation of the TA and SOL muscles during walking except for subject WF, who did not show any activity in the SOL muscle. The EMG profile for the TA and SOL displayed some abnormalities, although the timing of activation was generally similar to normal subjects.

There were 2 types of EMG profiles seen in the TA during the swing phase. The first pattern was exhibited by 4 SCI subjects who showed two bursts of activity in the TA. Figure 11A (upper trace) illustrates the pattern of one such subject. The EMG pattern of subject KM was similar to the profile seen in normal subjects. The second pattern of activity is demonstrated in the upper trace of Figure 11B (subject KW). The burst of activity in the TA muscle occurred during mid-swing and then gradually decreased through the latter part of the swing phase. The level of activity at the end of the swing phase never returned to resting levels. All of the subjects showed activity in the TA during the transition from swing to stance when reflex reversal is normally seen. Shortly after heel contact, a short burst of activity was seen in the TA in some subjects (Fig. 11B; upper trace) which could have been a stretch induced response as the foot was lowered to the ground. This large spike was seen in 3 subjects (KW, WF, BL) and has been reported in other spastic paretic subjects (Fung & Barbeau, 1989; Yang et al, 1991).

The SOL muscle activity in most SCI subjects was relatively normal as displayed by subject KM in Figure 11A (lower trace). During the stance phase the SOL muscle activity gradually increased and then abruptly decreased at the end of the stance phase. Another type of SOL EMG pattern, seen in 3 subjects (BL, LB, KW), is shown in Figure 11B (lower trace). The EMG profile of subject KW (Fig. 11B) exhibited a more gradual decrease at the end of the stance phase as compared to subject KM (Fig. 11A). Small rhythmical bursts of activity that were seen primarily during the early stance phase, were likely due to ankle clonus. Functionally, the subjects who showed the second type of SOL EMG pattern were the slowest walkers (0.19 to 0.28 m/s), and 2 of the subjects (KW, LB) were the only ones who demonstrated sustained ankle clonus under resting conditions.



**Figure 11.** Average EMG patterns of the TA and SOL muscles over the duration of a step cycle during undisturbed walking in 2 spinal cord injured subjects (KM, KW). The horizontal axis is divided into 16 time segments but the step cycle duration is different for each subject. A. The upper trace displays the EMG profile for the TA ( $n=50$ ) in subject KM. The step cycle was 1.81 s. in duration. Two bursts of activity were seen in the TA during the swing phase. The burst of activity in the late swing phase extends into early stance when the foot was lowered to the ground. The lower trace shows that the EMG activity of the SOL in this SCI subject was similar to a normal subject's EMG profile (Fig. 10) except for the bursts of activity during early stance (depicted by open arrows). B. The average EMG pattern of the TA and SOL ( $n=40$ ) in subject KW. The step cycle was 2.59 s in duration. The upper graph displays a single burst in the TA during the swing phase. The TA was still active at the end of the swing phase. A large spike seen at the beginning of the stance phase was likely a result of stretch activation of the TA. The lower trace represents the SOL activity. Small rhythmical bursts were seen during the stance phase and were likely related to ankle clonus during the stance phase (depicted by open arrows). This subject also exhibited sustained ankle clonus under resting conditions.

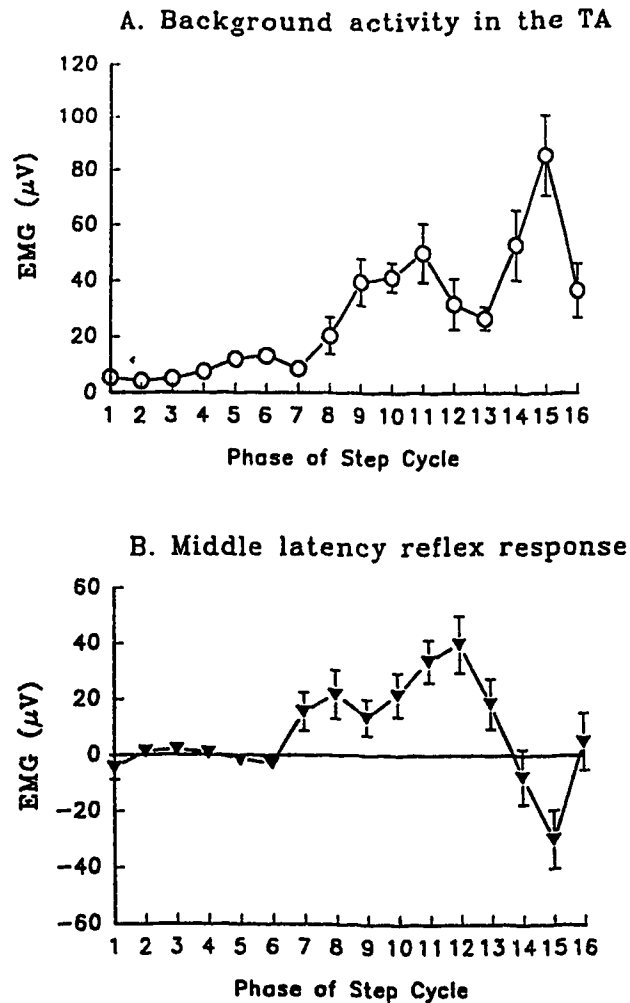
#### **4.4 Reflex responses in walking**

Stimulation of the posterior tibial nerve during walking evoked reflex responses in the TA muscle of all subjects and in the SOL muscles of all except one subject. Subject WF was the only subject who exhibited no background activity and reflex responses during walking in the SOL. The middle latency response will be discussed because this response showed the strongest modulation throughout the step cycle. This observation concurred with the findings reported in normal subjects (Yang & Stein, 1990). Although the comfortable walking speed for normal subjects was considerably faster (1.11 m/s) than that of SCI subjects (0.19 to 0.63 m/s), pilot data have shown that reflex reversal was observed in normal subjects at slower walking speeds (0.67 m/s). Therefore, the data obtained from normal subjects (Yang and Stein, 1990) will be presented for comparison with data from the SCI subjects.

##### **4.4.1 Middle latency reflex responses in normal subjects.**

Data reported by Yang and Stein (1990) were recalculated for comparison with the current results. The averages of the middle latency response in both muscles for normal subjects (n=7) are presented.

TA muscle: Figure 12 displays the behaviour of the reflex at a middle latency in the TA throughout the step cycle for normal subjects. The middle latency response was plotted for each of the 16 time segments of the step cycle (Fig. 12B). The background EMG values obtained from undisturbed walking, corresponded in time to the times that the middle latency responses were recorded, are shown in Figure 12A. Minimal reflex activity was seen during the stance phase when the background muscle activation of the TA was low (time segments 1 to 7). An excitatory response developed prior to the swing phase and continued to increase as the background TA EMG increased during the swing phase. The excitatory response abruptly reversed direction to become an inhibitory response at the transition from the swing to stance phase.



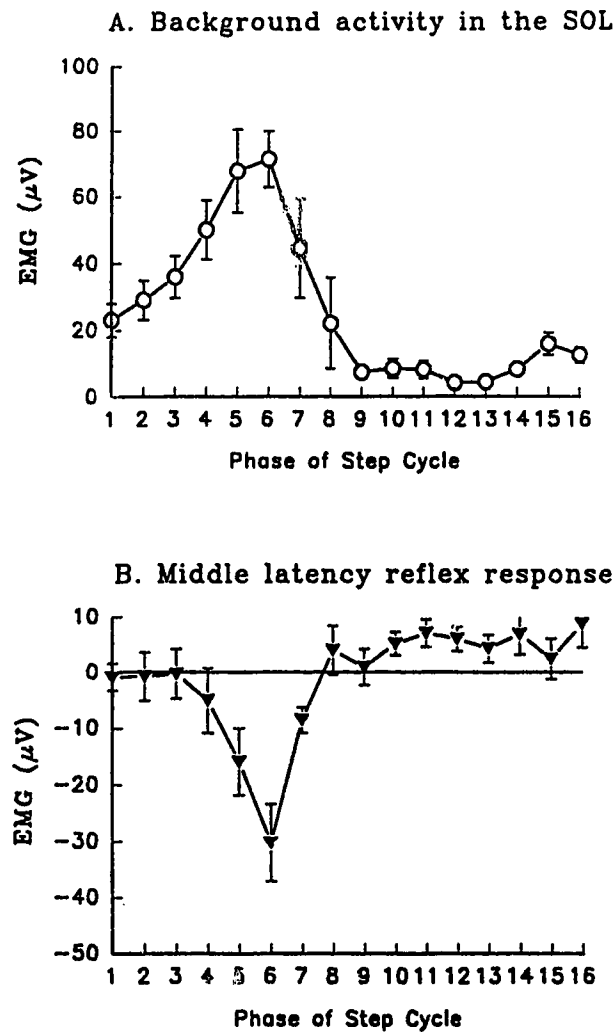
**Figure 12.** Reflex modulation of the TA muscle during walking in normal subjects. Data replotted from Yang and Stein (1990). The average EMG activity of the TA muscle during undisturbed walking and the reflex responses evoked during walking in normal subjects ( $n=7$ ) over the 16 phases of a step cycle. Double or triple pulses were applied to the posterior tibial nerve (1.5 to 2.0 times the motor threshold) to evoke reflex responses throughout the step cycle. The vertical bars represent one standard error about the mean. A. The average background EMG during undisturbed walking, which corresponded in time to the middle latency reflex responses (as seen in B). The TA was minimally active during most of the stance phase. Two bursts of activity were seen; the first burst cleared the foot during early and mid-swing and the second burst occurred at the transition from swing to stance. B. The middle latency response in the TA muscle recorded during disturbed walking trials for each of the 16 phases. No responses were seen during the stance phase when the muscle was normally inactive. An excitatory responses was first seen at the end of the stance phase prior to the rise in background muscle activity and continued through most of the swing phase. During the transition from the swing to stance phase the reflex response reversed direction to become an inhibitory response.

**SOL muscle:** The middle latency response of the SOL muscle in normal subjects was reported to be less consistent than the response measured in the TA muscle (Yang & Stein, 1990). Reflex reversal was more commonly seen in muscles that had 2 bursts of activity during the step cycle, such as the TA, than muscles with single bursts of activity, such as the SOL (Yang & Stein, 1990). The average reflex responses in the SOL muscle are shown in Figure 13. An inhibitory response was seen throughout mid to late stance phase. The depth of inhibition increased with increasing activation level of the background EMG. In a few subjects a small excitatory response was seen in the early part of stance phase. During the swing phase there was minimal reflex activity.

#### **4.4.2 Middle latency reflex responses in spinal cord injured subjects.**

**TA muscle:** The middle latency response was observed in the TA muscle with a latency of 50 to 80 ms in the SCI subjects. This range was comparable to the latencies reported in normal subjects (latency of 60 to 90 ms) (Yang & Stein, 1990). All of the SCI subjects showed excitatory responses at a medium latency during the swing phase. Although this excitatory response was modulated in the SCI subjects during walking, the degree and time course of modulation differed somewhat from the responses found in normal subjects. The behaviour of the reflex response observed in the SCI subjects was divided into 3 groups depending on the degree of modulation over the course of the step cycle. The first group displayed a reflex reversal. An inhibitory response occurring anywhere within the step cycle constituted a reversal from the excitatory response seen during the swing phase. The second group showed only an excitatory response during the swing phase while no response was observed during most of the stance phase. The third group exhibited an excitatory response throughout the step cycle. These groupings of reflex modulation for the TA are presented in Table 3 for each of the 10 subjects.

Although all 3 subjects (IJ, KM, BL) in the first group displayed reflex reversal, only subject IJ showed an inhibitory response during the transition from swing to stance which was similar to normal subjects. Clinically, subject IJ had the



**Figure 13.** Reflex modulation of the SOL muscles during walking in normal subjects. Data replotted from Yang and Stein (1990). The average EMG activity of the SOL muscle during undisturbed walking and the reflex responses evoked during disturbed walking are shown for the duration of a step cycle in the same normal subjects ( $n=7$ ) as described in Figure 12. The vertical bars represent one standard error about the mean. **A.** The background EMG level during the 16 phases of the step cycle corresponded in time to the time of the reflex responses. The SOL was active during the stance phase (time segments 1 to 9) and peaked at the end of the stance phase as the foot pushed off. Minimal EMG activity was seen during the swing phase. **B.** The middle latency response of the SOL was inhibitory for most of the stance phase. During the swing phase when the SOL was inactive, no middle latency responses were seen.

**TABLE 3:** Categories of the modulation pattern seen in the TA and SOL muscles.  
The behaviour of the middle latency response of the SCI subjects was classified into 1 of the 3 basic patterns.

SUBJECT (n=10)	MODULATION PATTERN of TA*	MODULATION PATTERN of SOL+
IJ	1	1
BL	1	1
KM	1	2
LB	2	1
KW	2	2
WF	2	N/A
LM	2	2
GS	3	2
KB	3	3
GB	3	3

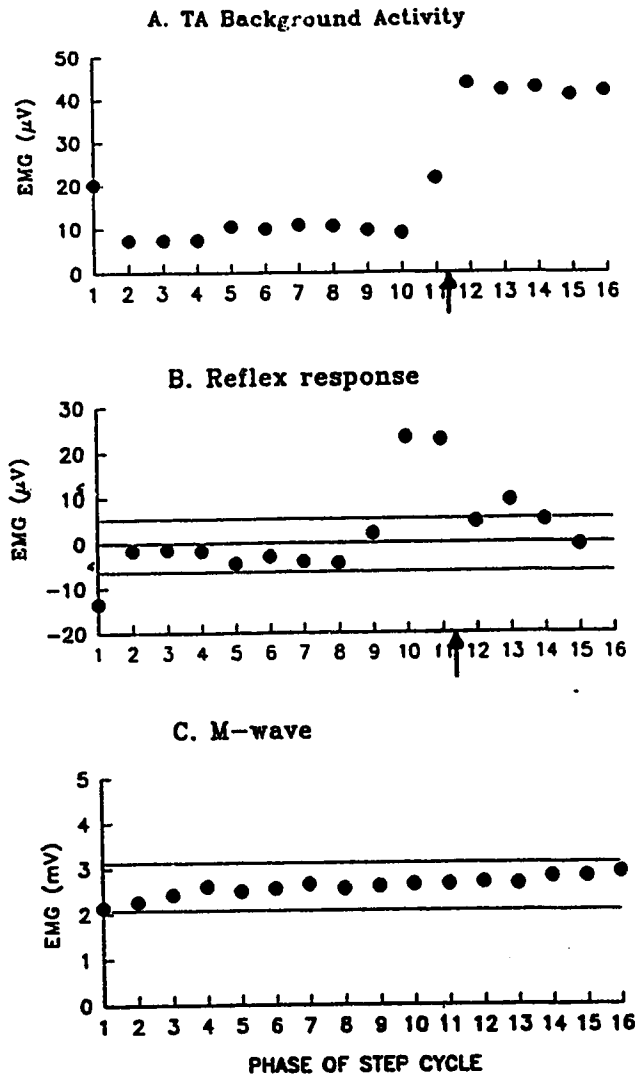
- \* Category 1 for the TA indicates that inhibitory and excitatory responses were observed.  
Category 2 indicates that excitatory responses were seen during the swing phase and no responses were seen during the stance phase for the TA.  
Category 3 for the TA indicates that excitatory responses occurred in both phases of the step cycle.
- + Category 1 for the SOL indicates that inhibitory responses were observed during the stance phase and little reflex activity was seen during the swing phase.  
Category 2 indicates that inhibitory responses occurred during the stance phase and excitatory responses were seen during the swing phase in the SOL.  
Category 3 for the SOL indicates that excitatory responses occurred in both phases of the step cycle.

least neurological impairment and walked at the fastest speed (maximum walking speed of 1.67 m/s overground; self-selected walking speed of 0.63 m/s on the treadmill). Subjects BL and KM also exhibited reflex reversal; however, the timing of the inhibitory response with respect to the step cycle was slightly different from that reported for normal subjects. Figure 14 illustrates the average middle latency response occurring 65 to 115 ms after the first stimulus, and the corresponding background EMG activity recorded from subject BL for 15 time segments of the step cycle. Figure 15 shows the time course of reflex responses during the step cycle for subject BL. Subjects BL and KM showed excitatory responses during the swing phase which reversed in direction to become inhibitory responses during early stance (phases 1 to 3), somewhat later than those observed in normal subjects (phase 14, 15, 16 or 1). Other SCI subjects who exhibited similar background activity during the early stance phase did not exhibit the inhibitory response like subjects BL and KM. Functionally, subjects BL and KM walked at similar maximum walking speeds of 0.94 m/s and 0.91 m/s, respectively, but exhibited different clinical findings. For example, subject KM did not exhibit ankle clonus nor ankle hyperreflexia, whereas subject BL did (Table 2).

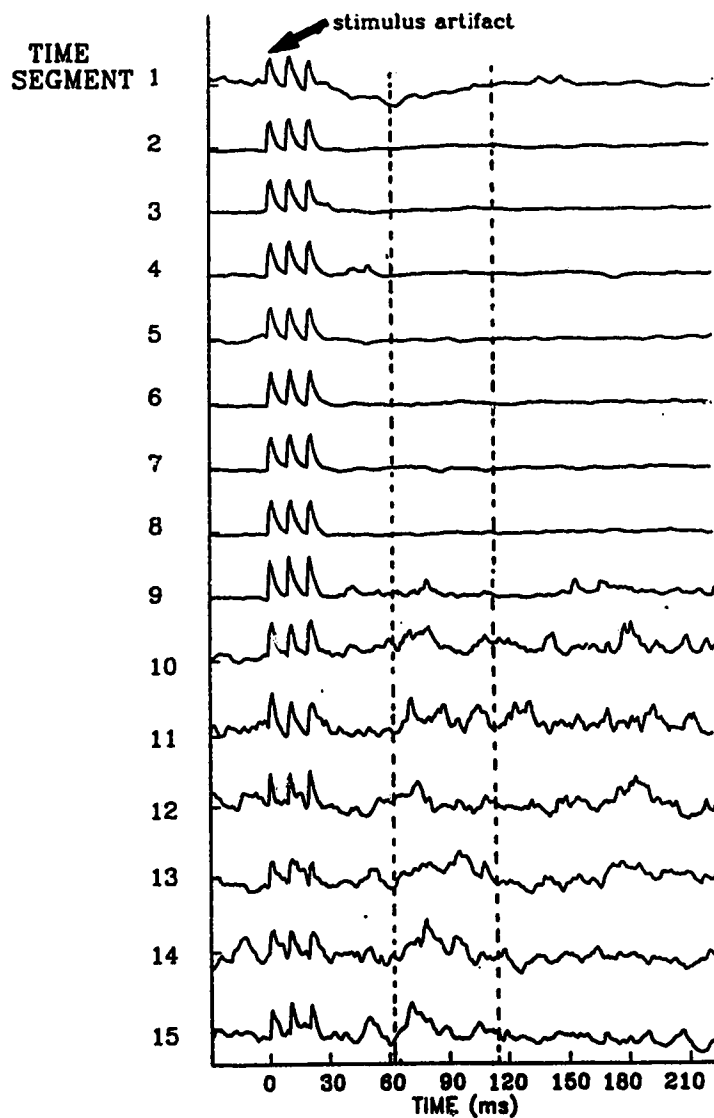
Subject KW represented the typical pattern of reflex response that was seen in the 4 subjects (LB, LM, KW, WF) of the second group (Fig. 16). No inhibitory response was seen in group 2, only an excitatory response, which occurred during the swing phase. There were no responses observed during the stance phase when the TA muscle was inactive. The time course of modulation was dependent on the phase of the step cycle. Three of the subjects in the second group walked at similar treadmill speeds of 0.27 to 0.29 m/s while the fourth subject (WF) walked at a faster rate of 0.43 m/s. Interestingly, no one in this group generated an inhibitory response when standing. The clinical findings of these 4 subjects were diverse; some subjects showed sustained ankle clonus and hyperactive reflexes while others did not (Table 2).

Figure 17 shows reflex responses of subject KB which are representative of the third group (subjects GB, GS, KB). Unlike the other 2 groups, this group exhibited

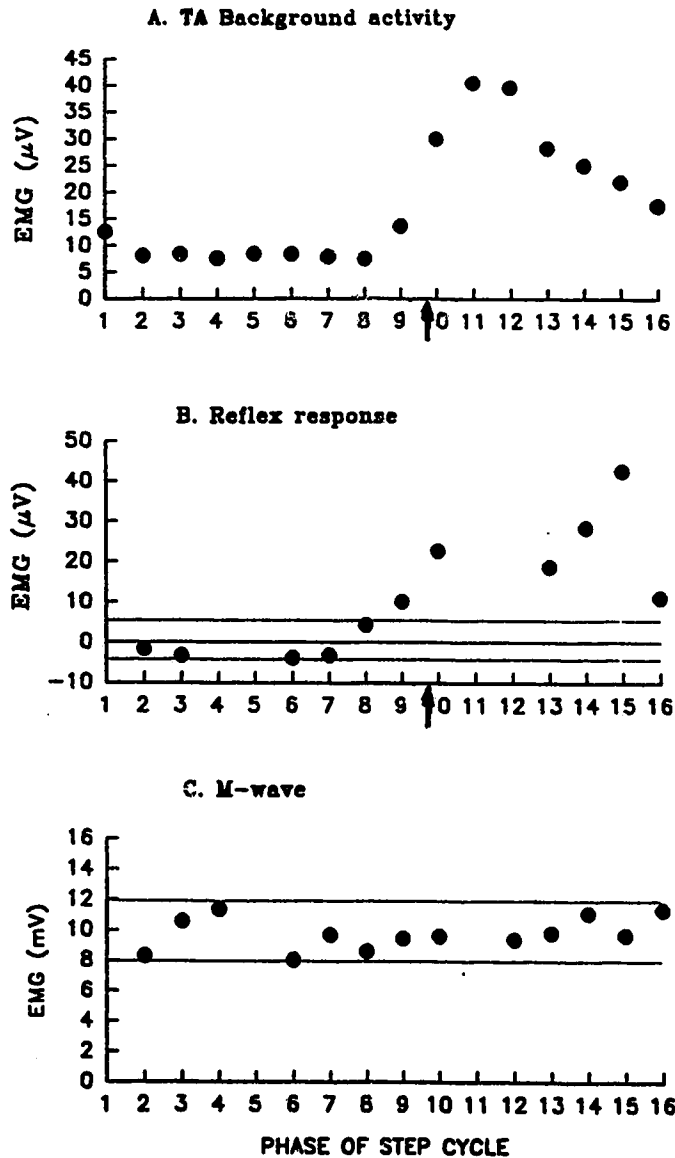




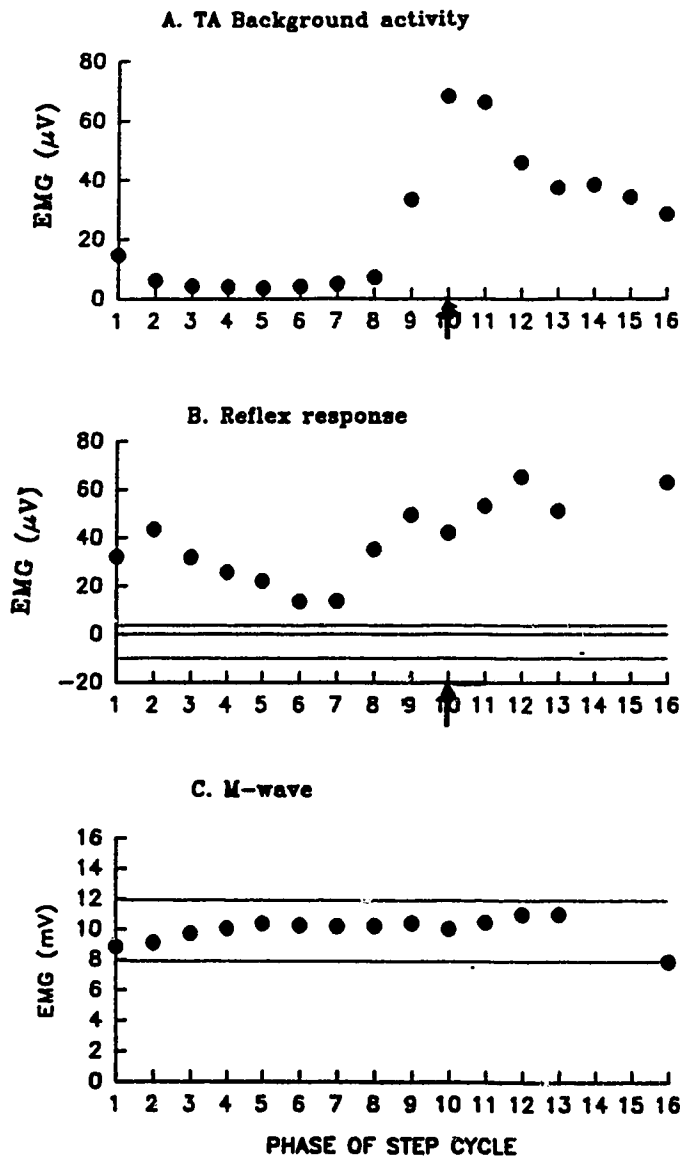
**Figure 14.** Reflex reversal of the TA in a SCI subject (BL) during walking which is representative of the subjects in the first group. **A.** Background activity of the TA during undisturbed walking in subject BL. The EMG values correspond in time to the times of the middle latency reflex response. A relatively constant level of EMG activity was seen in the TA during the swing phase. **B.** Middle latency reflex response averaged over 65 to 115 ms after the first stimulus artifact during disturbed walking. Responses falling outside of the error range (depicted by grid lines) defined excitatory and inhibitory responses. This subject exhibited excitatory responses during the swing phase. The excitatory response reversed in direction to become an inhibitory response in early stance (time segment 1). The inhibitory response occurred later in the stance phase than the response in normal subjects (see Fig. 12 for description of normal pattern). The response from time segment 16 was omitted because there were less than 10 responses averaged for this segment. **C.** The M-wave, which indicated the effective strength of the stimulus, had to fall within a certain range (depicted by the grid lines) for the reflex response to be considered for analysis. In this subject, all of the responses were evoked by stimulus intensities that fell within the range.



**Figure 15.** The reflex response recorded in the TA of a spinal cord injured subject (BL) during walking. Each time segment represents data obtained from a specific time in the step cycle. Time segments 1 to 10 represent the stance phase while time segments 11 to 15 represent the swing phase. The latency of the responses are measured from the first stimulus artifact (depicted by arrow). The middle latency response was defined by the average EMG amplitude between 65 to 115 ms (between the grid lines). This subject showed an excitatory response during the swing phase which reversed in direction to become an inhibitory response in the first time segment. Modulation of the early latency response (30 to 65 ms) and late latency response (115 to 190 ms) was also observed over the step cycle.



**Figure 16.** The middle latency response of the TA in a spinal cord injured subject (KW) during walking. The response pattern is typical of group 2. **A.** The background activity of the TA during undisturbed walking. One burst of activity was seen during the swing phase, but the TA was active during the transition from the swing to stance phase (see Fig. 11B for details). **B.** The middle latency response was averaged over 70 to 100 ms after the first stimulus artifact. An excitatory response was seen during the swing phase. No responses were seen in the stance phase. This subject did not exhibit reflex reversal because no inhibitory response was seen during the step cycle. **C.** M-wave amplitudes were well matched except for 3 time segments of the step cycle. The data corresponding to these 3 time points were discarded.



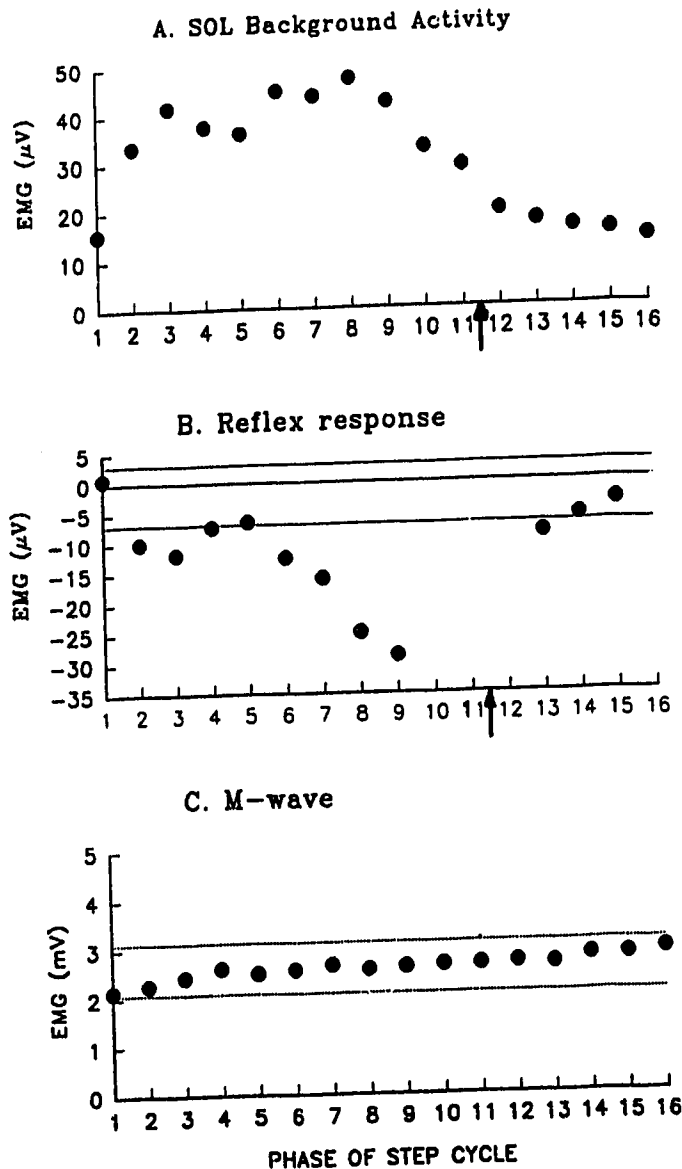
**Figure 17.** The middle latency response of the TA muscle in a spinal cord injured subject (KB) during walking. This subject is representative of the third group of SCI subjects, who showed an excitatory response throughout the step cycle. A. The background TA EMG during undisturbed walking. No activity was seen during most of the stance phase (time segments 1 to 9). During the swing phase, only 1 burst of activity was seen. B. Middle latency response which was averaged over 80 to 130 ms after the first stimulus, was excitatory throughout the stance and swing phases. Two peaks of reflex activity were seen, 1 during early stance, and the other during mid-swing. The minimum response occurred in late stance phase. Reflex responses appeared to be independent of the background activity. C. The M-wave amplitudes were well matched except for 2 time segments. Reflex responses corresponding to the 2 time segments were omitted.

excitatory responses in the TA during the swing and stance phases of the step cycle. The peak excitatory responses during the stance and swing phases were 65% and 101%, respectively, of the peak EMG activity during the undisturbed walking. The minimum response occurred in late stance just prior to the swing phase. Since excitatory responses were observed during the stance phase, this would suggest that the reflex response changed independently of the background EMG activity. The subjects in group 3 did not generate an inhibitory response in the TA during walking but 2 subjects (GB, GS) displayed an inhibitory response (latency of 110 ms) under static conditions.

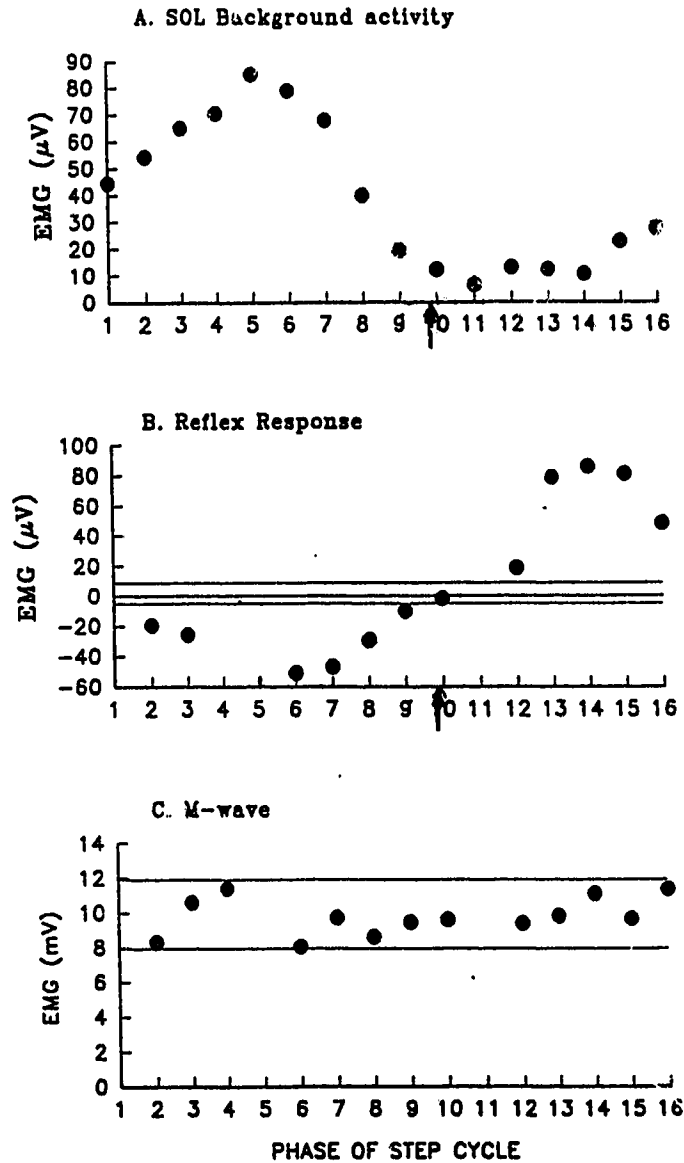
Functionally, the subjects in group 3 showed great variation in walking speed (maximum walking speed 0.91 to 1.54 m/s; self-selected walking speed of 0.31 to 0.45 m/s). The clinical tests showed a variety of responses amongst the subjects in this group (Table 2). For example, deep tendon reflexes varied from normal to hyperactive scores, and not all subjects showed an upgoing plantar response.

SOL muscle: The middle latency response (50 to 60 ms latency) of the SOL muscle was observed in 9 SCI subjects. Three reflex patterns were observed during walking in the SOL. The subject groupings for the pattern of reflex modulation for the SOL are seen in Table 3. Figure 18 illustrates an example of the reflex pattern seen in the first group (subjects IJ, BL, LB). The inhibitory response increased through the stance phase when SOL was activated. During the swing phase there was minimal background and reflex activity. This pattern of reflex response was similar to the response pattern of normal subjects (Fig. 13). No reflex reversal was seen in the SOL. Interestingly, 2 of the subjects from this group (IJ, BL) also showed rather normal reflexes in the TA.

The second reflex pattern which was demonstrated by subjects KM, LM, GS, and KW, consisted of an inhibitory response during the stance phase, which was similar to the first group. An excitatory response, however, developed during the swing phase. Figure 19 is an example of the behaviour of the reflex pattern observed in the SOL, which was characteristic of the second group. The peak excitatory



**Figure 18.** The middle latency response of the SOL muscle in a spinal cord injured subject (BL) during walking. The response pattern was characteristic of the first group which was similar to the responses seen in normal subjects. A. The background muscle activity of the SOL during undisturbed walking. The SOL is active during the stance phase and showed minimal activity during the swing phase. B. The middle latency response occurred 50 to 90 ms after the first stimulus. The reflex was inhibitory during the stance phase when the muscle was active. Minimal reflex activity was seen during the swing phase when the muscle was inactive. C. The M-wave amplitudes from all 16 time segments of the step cycle were well matched.



**Figure 19.** The middle latency response of the SOL muscle in a spinal cord injured subject (KW) during walking. The response pattern was representative of the subjects in the second group. **A.** The background EMG showed a typical increase in activity during the stance phase and minimal activity during the swing phase (see Fig. 11B for details). **B.** The reflex response at a medium latency, 55 to 85 ms after the stimulus artifact. An inhibitory response was seen during the stance phase which reversed in direction to become an excitatory response during the swing phase. **C.** Thirteen of the 16 M-wave amplitudes were within the target range (depicted by grid lines). Reflex responses from time segments 1, 5 and 11 were discarded.

response seen in subject KW during the swing phase was 101% of the peak EMG value from undisturbed walking.

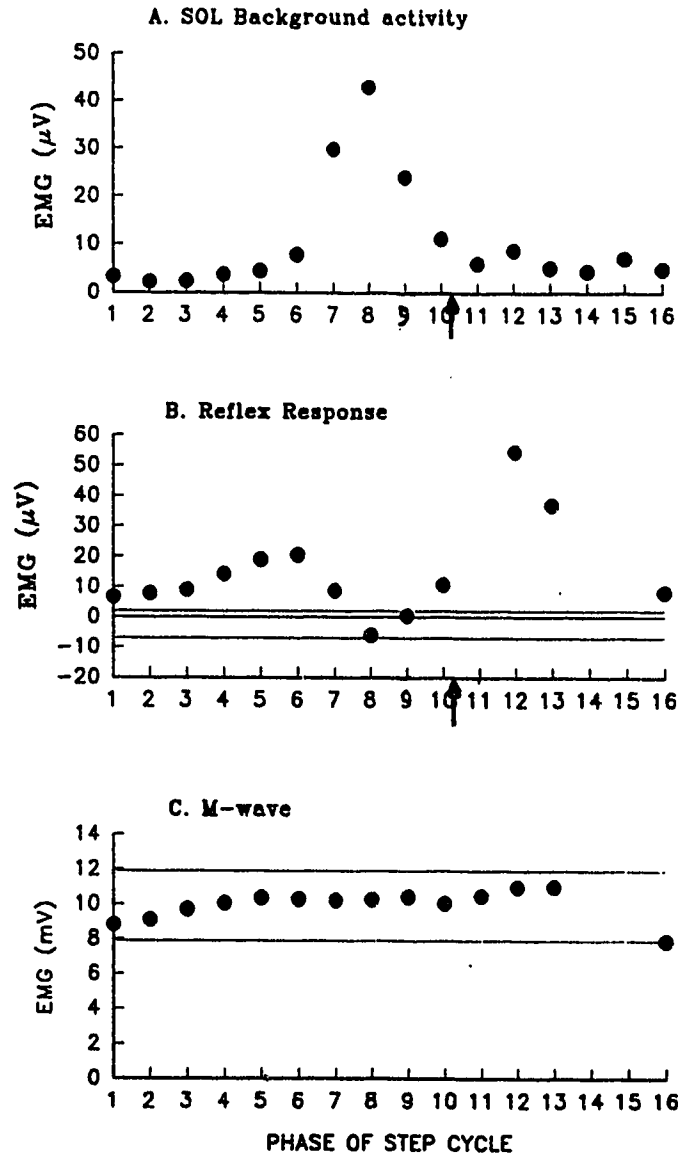
The third reflex pattern was seen in 2 subjects (GB, KB) who exhibited excitatory responses throughout the step cycle in the SOL. Interestingly, these 2 subjects also showed excitatory responses throughout the step cycle in the TA. The responses recorded from subject KB are shown in Figure 20. The peak excitatory responses during the swing and stance phases were 128% and 48%, respectively, of the peak EMG value from the undisturbed walking.

The 3 types of reflex modulation observed in the TA and the SOL did not bear any relationship to the clinical findings. Correlations between the clinical measures and the categories of reflex modulation were low ( $r = -0.3885$  to  $0.3198$ ,  $p > 0.05$ ; Table 4). The correlation between the plantar response and categories of reflex modulation was not calculated since all subjects except one (GB) showed an upgoing plantar response. Qualitatively, subject BL exhibited a slow walking speed, and sustained ankle clonus, but showed relatively normal reflex activity in the SOL muscle. Whereas subject GB who had the second fastest walking speed over 6 m, exhibited excitatory responses throughout the step cycle in both the TA and the SOL muscles. This result concurred with the findings reported by Fisher and colleagues (1979) who found that the plantar response and deep tendon reflexes were not correlated with the changes of the flexor reflex under static conditions in patients with cerebrovascular lesions.

#### **4.5 Summary**

All of the subjects generated reflex responses in the TA muscle under static conditions. Although some subjects exhibited responses at early, middle and late latencies, only the early excitatory response was consistently seen. During walking, 3 types of reflex behaviour were seen in the TA and SOL muscles. The responses were dependent on the phase of the step cycle, but the degree of modulation was different from that seen in normal subjects. The first pattern of response was a reflex reversal in the TA. Two subjects from this group also showed reflex behaviour in the SOL





**Figure 20.** The middle latency response of the SOL muscle in a spinal cord injured subject (KB) during walking. This response pattern was representative of the third group. **A.** The undisturbed walking pattern of the SOL corresponded in time to the time of the reflex response. The SOL was activated in the latter half of the stance phase. Minimal EMG activity was seen during the swing phase. **B.** The reflex response was averaged over 50 to 90 ms after the stimulus artifact. Excitatory responses were seen at a medium latency during early stance when the SOL showed little background activity. No responses were seen in the late stance phase. The peak excitatory response was observed during mid-swing. **C.** All of the M-wave amplitudes were well matched except for 2 time segments. Responses from these time segments were not included.

**TABLE 4.** Pearson's correlation of the clinical findings and the patterns of modulation seen in spinal cord injured subjects. None of these correlations were significantly different from zero at the 0.05 level.

Clinical Measure		TA MODULATION PATTERN	SOL MODULATION PATTERN
Deep Tendon Reflex	knee	-0.2635	-0.1348
	ankle	-0.1368	0.3198
Clonus		0.1266	-0.3855
Walking Speed	Maximum	0.0000	0.2425
	Comfortable	-0.2474	-0.0553

that was similar to the pattern seen in normal subjects. The second pattern of reflex activity in the TA was an excitatory response seen during the swing phase and no response occurred during the stance phase. Most of these subjects also showed abnormal excitatory responses in the SOL during the swing phase. The third pattern of reflex activity consisted of excitatory responses throughout the step cycle in both of the TA and SOL muscles. No clear relationship was found between the clinical findings and the behaviour of the reflex during walking.

## **CHAPTER 5**

### **DISCUSSION**

The results indicate that reflex responses evoked by cutaneous stimuli were modulated in SCI subjects during walking, but the degree of modulation was altered as compared to normal subjects. One subject with minimal neurological and walking deficits demonstrated reflex reversal similar to normal subjects in both the TA and SOL muscles. The remaining SCI subjects who had greater neurological impairment exhibited abnormalities with respect to the degree of modulation. All of the subjects showed an excitatory response in the TA during the first half of the swing phase. Three subjects showed an inhibitory response in the TA that occurred at either the transition from swing to stance or in the early part of stance phase. The remaining 7 subjects did not show a reflex reversal in the TA. Most subjects who did not generate an inhibitory response in the TA also showed abnormal patterns of modulation in the SOL. An unusual excitatory response was seen in the SOL during the swing phase which was not observed in normal subjects. Normal subjects typically showed an inhibitory response during the stance phase and no response during the swing phase. When an excitatory response was elicited in normal subjects, the response occurred in the early part of the stance phase, never in the swing phase (Yang & Stein, 1990) (Fig. 14). Thus, the normal mechanisms responsible for modulating sensory input during walking appeared to be altered in some subjects with spinal cord damage.

#### **5.1 Technical considerations**

Over the course of an experiment, 2 to 3 different stimulus intensities were usually applied because the M-wave amplitude varied within a trial. The variation of the effective stimulus strength was likely due to movement of the ankle during walking. In spite of using different stimulus intensities, the amplitude of the M-wave did not always fall within the predefined target range for all 16 segments of the step cycle. At least one of the 16 responses was discarded in 7 of the 10 subjects because

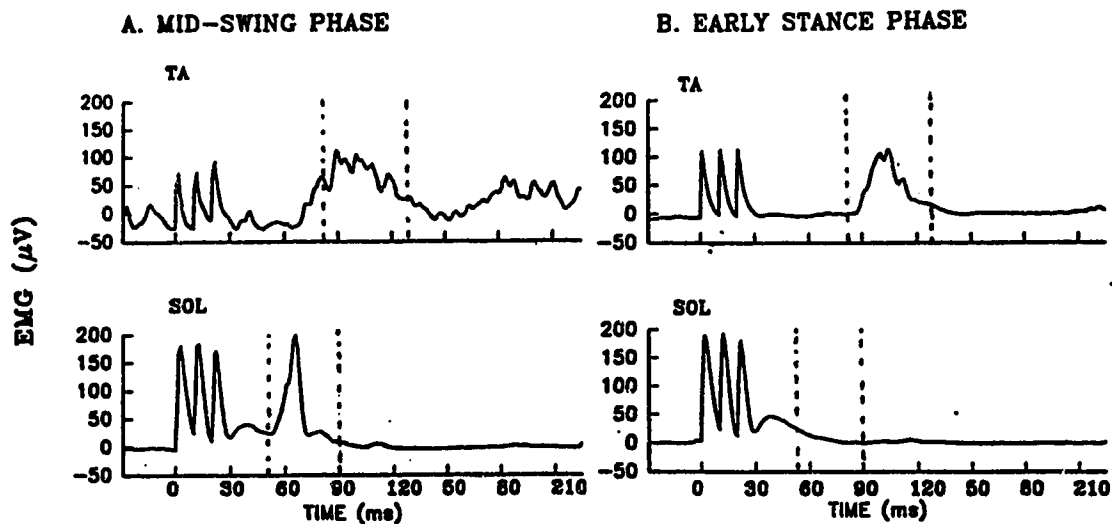
the M-wave amplitude was not within the appropriate range.

An inhibitory response is normally seen in the TA during the transition from the swing to stance phase (time segments 14, 15, or 16). It was unlikely that an inhibitory response was missed because it occurred in one of the time segments that was discarded. Only 3 subjects who did not show reflex reversal had one or two traces omitted during the transition from swing to stance phase.

Sufficient muscle activity at the transition from the swing to stance phase was needed for an inhibitory response to be seen in the TA. It was unlikely that the inhibitory response was not identified because the background muscle activity was inadequate in this group of subjects. Although most subjects did not exhibit a double burst of activity in the TA during the swing phase, the muscle activity was sufficient during the transition from the swing to stance phase for an inhibitory response to be identified in all subjects. The amplitude of the muscle activity at the end of the swing phase in all subjects except one was above 10  $\mu$ V and was at least 40% of the peak EMG value from undisturbed walking.

Some subjects showed an excitatory response in the SOL during the swing phase. Although the SOL is in close proximity to some flexor muscles, the excitatory response seen in this muscle was probably not due to cross-talk from the ankle dorsiflexors. Of the 6 SCI subjects who generated an excitatory response in the SOL, only 1 subject (LM) exhibited a response that had a similar latency and shape to the response recorded in the TA. Such similarities in the responses of the two muscles suggest that cross-talk may have been present in this subject.

Figure 22A compares the reflex responses evoked in the TA and SOL during mid-swing from subject (KB). Two different excitatory responses with different latencies and shapes were seen in the TA and SOL muscles. During the swing phase when the SOL was inactive, an excitatory response occurred at an earlier latency than the excitatory response in the TA. The shape of the response in the TA was different from the response seen in the SOL. Furthermore, during the early stance phase, no response was seen in the SOL while an excitatory response was seen in the TA (Fig. 22B). Clearly, cross-talk between these two muscles could not account for the



**Figure 21.** Comparison of reflex responses evoked in the TA and SOL muscles in a spinal cord injured subject (KB) during two time segments of the step cycle. This subject demonstrated no inhibitory response at a medium latency in either muscle. **A.** The middle latency response of the TA during mid-swing (time segment 12) occurred 80 to 130 ms after the first stimulus (depicted by the grid lines of top trace). A late excitatory response was also observed. The middle latency response of the SOL occurred at a different latency, 50 ms after the first stimulus. The single peak of the SOL response was a different shape than the response seen in the TA. **B.** An excitatory response was evoked in the TA during early stance (time segment 2) when the muscle was inactive during walking (top trace). No response was seen in the SOL during the corresponding time segment. The different shapes and latencies suggest that the responses were not likely a result of cross-talk between these 2 muscle groups.

responses observed.

## **5.2 Possible neural mechanisms**

The results suggest that some features of reflex modulation remain intact in SCI subjects while other features are altered. The amplitude of the middle latency response in SCI subjects was dependent upon when the stimulus was applied within the step cycle. This phase-dependent characteristic of the cutaneous reflex suggests that some parts of the mechanisms responsible for modulating sensory information during walking likely remained intact in these SCI subjects. However, the pattern of modulation over the step cycle was altered in most SCI subjects and would suggest that either a) the mechanism responsible for modulating the sensory information during specific phases of the step cycle was impaired, or b) the mechanism responsible for modulation of the cutaneous reflex was intact but was masked by abnormal reflex gains or altered thresholds. The basis for the altered modulation observed in SCI subjects can only be speculated since the neural mechanisms for modulating the cutaneous reflex during walking are unknown.

Two mechanisms have been proposed to explain reflex reversal observed in single leg muscles during walking (Yang & Stein, 1990). The first possibility suggests that a switching mechanism exists between 2 parallel interneuronal pathways. Parallel inhibitory and excitatory pathways are known to converge onto motoneurons from cutaneous afferent fibres (Holmqvist & Lundberg, 1961). Limited evidence from animal and human work indicate that the transmission of these parallel pathways may be alternately activated during walking (Schomburg et al, 1981; De Serres et al, 1992). If there was a switching mechanism that was impaired in SCI subjects, then this may account for the altered pattern of modulation seen in the TA and SOL muscles. The reflex abnormalities described in this study frequently involved excitatory responses. For example, excitatory responses were seen in the SOL during the swing phase when usually no responses are seen. The appearance of excitatory responses during the swing phase would suggest that excitatory pathways were activated at inappropriate times. Likewise, no inhibitory responses were seen in the

TA during the transition from the swing to stance phase. The reduced modulation described in the TA may have been related to changes of the switching mechanisms that disrupted the normal gating of the excitatory and inhibitory pathways.

The other possible neural mechanism for generating a reflex reversal is that 2 distinct motoneuron pools within a muscle are activated by different reflex pathways at different times within the step cycle (Yang & Stein, 1990; Pratt et al, 1991). For example, one group of motoneurons in the TA may be activated during the swing phase when excitatory responses typically occur, and another group of motoneurons may be activated during the transition from the swing to stance phase when inhibitory responses are normally seen. Limited evidence reported in cats have identified different subpopulations of motoneurons within a muscle that generated either excitatory or inhibitory responses at different times within the step cycle (Pratt et al, 1991). But these motoneuron subpopulations have been identified only in bifunctional muscles (recruited at least twice during the step cycle), and not in unifunctional muscles (recruited only once during the step cycle)(Pratt et al, 1991).

The altered reflex modulation seen in the TA and SOL may have been related to changes which targeted a specific subpopulation of motoneurons. For instance, excitatory responses were identified in the SOL of some SCI subjects when the muscle was inactive. The excitatory responses could possibly have been related to a heightened excitability of a particular subpopulation of motoneurons in the SOL or the cutaneous pathways to this group of motoneurons.

It is conceivable that the mechanisms responsible for modulating the cutaneous reflex during walking were not impaired, but rather modulation was affected by an abnormal gain of the reflex loop. This possibility appears to be true for the behaviour of the H-reflex in SCI subjects. The degree of modulation of the H-reflex was reduced in the SOL during walking in spastic paretic subjects, and in some subjects the degree of modulation was increased at lower stimulus intensities (Yang et al, 1991). The mechanism responsible for the modulation was suspected to be masked by saturation of the reflex loop.

It could be that the reflex gain of the cutaneous reflex in SCI subjects may be



abnormal. Most SCI subjects exhibited excitatory responses in the TA that were modulated within the step cycle, while inhibitory responses were not readily seen. If the gain of the inhibitory pathways was too low during all or some parts of the step cycle, then inhibitory responses would not be readily expressed in the TA. The exaggerated excitatory responses observed in the SOL during the swing phase could also be related to abnormally high reflex gains in the excitatory pathways. If the reflex gains could be artificially changed during walking, then a normal pattern of reflex activity may be revealed in SCI subjects.

The threshold of the reflex may also be altered in the SCI subjects during walking and could be related to the abnormal responses described in this study. Other studies have reported that the threshold for the flexor reflex was altered in SCI subjects under static conditions (Roby-Brami & Bussel, 1987; Shahani & Young, 1971; Dimitrijevic & Nathan, 1976b). It is possible that the excitatory pathways had a lower threshold and were more readily evoked in the TA and SOL muscles. The inhibitory responses, which were not commonly seen in the TA of the SCI subjects, may have had a higher threshold for activation.

The abnormal modulation of the reflex activity may have been related to altered peripheral input. During walking the differences in weight-bearing and limb position between normal and SCI subjects may have contributed to the abnormal responses described in some SCI subjects. The effect of weight-bearing on cutaneous reflexes has not been investigated in humans. But most of the SCI subjects had observable gait deviations, such as knee hyperextension, that could have altered the weight-bearing. Weight-bearing could also have been altered because the handrails provided additional support for those subjects who were unable to properly weight-bear. Results from animal studies suggest, however, that weight-bearing is not essential for modulating the cutaneous reflex during rhythmic movements, since reflex reversal was found in spinal cats during air stepping (Forssberg et al, 1977).

Limb position was not examined during walking in this study, but findings from other human studies indicate that limb position was not a primary factor affecting the behaviour of the cutaneous reflex under static testing conditions.

Different static limb positions did not modify the flexor response in a complete SCI patient (Dimitrijevic & Nathan, 1967b). Moreover, the reflex response evoked by non-noxious cutaneous stimuli, was not altered by different static hip and knee positions in normal subjects (Kanda & Sato, 1983). Because the current study did not examine limb positions during walking, differences in limb position between SCI and normal subjects cannot be disregarded as a possible factor affecting reflex modulation.

### **5.3 Functional significance**

The relationship between the neurophysiological and biomechanical responses to cutaneous stimuli was recently examined in humans (Duysens et al, 1992). Small changes in the ankle and knee movements were correlated with the inhibitory and excitatory responses evoked in the TA by low intensity stimuli in normal subjects (Duysens et al, 1992). Increased dorsiflexion was seen during the swing phase when the excitatory response was observed. At the transition from swing to stance when an inhibitory response is evoked in the TA, increased plantarflexion was measured in the ankle. The plantarflexion was suspected to assist in placing the foot for the stance phase.

In this study, ankle and knee joint movements were not measured during walking, but the absence of an inhibitory response in the TA at the transition from the swing to stance phase would suggest that reflex induced ankle plantarflexion likely did not occur at that time. During the transitional period, the excitatory response demonstrated by many SCI subjects (group 2 & 3) in the TA could have facilitated ankle dorsiflexion. Functionally, dorsiflexion of the supporting limb prior to heel contact could compromise the walking stability. Furthermore, the excitatory responses seen in the SOL of many SCI subjects could also cause walking instability. If the excitatory response in the SOL was associated with plantarflexion during the swing phase, then the foot could not be cleared and possibly cause the individual to trip. Interesting, 5 out of the 9 SCI subject who demonstrated abnormalities of the reflex behaviour used a lower extremity brace and/or an assistive walking device for balance purposes.

Although the modulation of the cutaneous reflex may have functional significance, the degree of modulation observed during walking did not appear to be related to the functional walking ability in this group of SCI subjects. Maximal walking speed was presumed to be a good measure of a subject's functional walking ability (Brandstater et al, 1983), but this clinical measure was not significantly correlated with the degree of reflex modulation. For instance, some of the slower walkers (BL, KM) exhibited reflex reversal in the TA, and some of the faster walkers (GB, GS) showed primarily excitatory responses. Another example of a poor correlation between reflex modulation and clinical measures was the deep tendon reflex of the knee. Subjects who showed reflex reversal in the TA, exhibited brisk and hyperactive deep tendon reflexes of the knee similar to the subjects who showed excitatory responses throughout the step cycle (Table 2).

There are a few possible reasons why the clinical measures did not correlate with the reflex behaviour. First, the clinical measurements used in this study may not bear any relationship to the behaviour of the cutaneous reflex. For example, ankle clonus was induced by sudden muscle stretch, not cutaneous stimuli. Furthermore, the maximum walking speed may be dependent on many neurological and biomechanical factors (Winter, 1991), not just the modulation of cutaneous reflexes. Second, the clinical measurements assessed tone under resting conditions, but the behaviour of the reflex activity was examined during walking; the two testing conditions may not have been directly comparable. Lastly, the functional significance of the cutaneous reflex is still unclear and, therefore, may not be directly related to one's functional walking ability.

#### **5.4 Clinical implications**

The degree of modulation of the cutaneous reflex is a descriptive measure of how particular sensory information is processed during walking. Although the mechanisms and the functional significance of reflex reversal are not clearly understood, it is apparent that reflex reversal is characteristic of normal gait. The results from this study demonstrated that subjects with spinal cord damage exhibited

reflex modulation which was different from those in normal subjects. In contrast, spinalized cats (Forssberg et al, 1975) showed a pattern of reflex modulation similar to the behaviour described for intact cats (Forssberg 1979). Findings from this study indicate that there may be differences between the incomplete SCI human and the complete spinal cord transected cat. Furthermore, the findings from this study stress the importance of human studies to compliment animal work.

It will be of interest to determine what methods, pharmacological or physical interventions, could restore the reflex modulation seen in SCI individuals to a more normal pattern. Although the combined effects of pharmacological intervention and gait retraining have provided limited success in retraining spastic SCI subjects to walk (Fung et al, 1990), it is unknown whether the change in functional walking is accompanied by a change in behaviour of the cutaneous reflex. Further investigation needs to address the neural mechanisms involved in the transmission of cutaneous reflexes and the functional role of this reflex during walking.

## **CHAPTER 6**

### **CONCLUSIONS and RECOMMENDATIONS**

The findings from this study identified abnormalities of reflex responses evoked by cutaneous stimuli during walking in SCI subjects. The results suggest that transmission of cutaneous information through the reflex pathways was altered. Moreover, excitatory rather than inhibitory responses were easier to evoke. The excitatory responses recorded from the TA during the transition from swing to stance may have evoked ankle dorsiflexion rather than plantarflexion when the foot was preparing for heel contact. Dorsiflexion at this time could compromise walking stability. Likewise, plantarflexion associated with excitatory responses in the SOL during the swing phase could hinder the foot from clearing the ground and possibly cause tripping. The functional walking ability of the SCI subjects was assessed by commonly used clinical measures; however, no relationship was identified between the clinical measures and the pattern of reflex modulation.

There remains a great amount of uncertainty about how sensory information is processed during locomotion. The effects that central and peripheral factors exert on the behaviour of cutaneous reflexes during walking need to be ascertained. Direct investigation of the neural mechanisms responsible for controlling reflex activity during walking will continue to involve animal work. However, differences and similarities between animal and human work will ultimately need to be identified.

Future research is needed to determine whether a direct relationship exists between the behaviour of cutaneous reflexes during walking and the stability of walking in humans. Information gained from such studies may shed some light on the ambulatory difficulties many individuals experience with spinal cord damage. To identify a relationship between reflex modulation and walking stability, more quantitative information about walking stability is required. A biomechanical description of stumbling reactions in bipedal locomotion would identify normal compensatory reactions during walking. It would be of further interest to determine whether normal reflex modulation could be restored in subjects who demonstrate

abnormal reflex patterns, and to see if this change is accompanied by any improvement in walking stability. Pharmacological and physical interventions may be a feasible method of manipulating sensory input and examining the behaviour of the cutaneous reflex during walking in humans.

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**APPENDIX A**  
**NEUROLOGICAL ASSESSMENT**

**SUBJECT:** \_\_\_\_\_

**DATE:** \_\_\_\_\_

**DIAGNOSIS:** \_\_\_\_\_

**REFERRAL SOURCE:** \_\_\_\_\_

**ONSET:** \_\_\_\_\_

**DOB:** \_\_\_\_\_

**ADDRESS/PHONE:** \_\_\_\_\_

**MEDICATIONS:** \_\_\_\_\_

**RELEVANT PMH:**

**A. SELF-REPORTED MOBILITY:**

**1. REGULAR MODE OF LOCOMOTION:**

**2. WALKING TOLERANCE:**

**3. STAIRS:**

**4. FUNCTIONAL LIMITATIONS:**

**B.**      **PHYSICAL ASSESSMENT**

**1.**      **MUSCLE TONE -**

**ANKLE CLONUS:**

(0= no response; 1= 2-3 beats; 2= 4-10 beats; 3= > 10 beats)

**PLANTAR RESPONSE:**

(downgoing or upgoing)

**DEEP TENDON REFLEXES:**

(0= no response; 1= hyporeflexic; 2= normal; 3= brisk; 4= hyperreflexic)

	KNEE JERK	ANKLE JERK
RIGHT		
LEFT		

**2.**      **LOCOMOTION -**

**WALKING DEVICES / ORTHOSIS:**

**MAXIMAL WALKING SPEED OVER 6 M (sec):**

## **APPENDIX B**

### **PROJECT SUMMARY**

**Project title:** Neural Control of Human Locomotion

**Principle investigator:** Jaynie F. Yang  
Assistant Professor, Department of Physical  
Therapy & Division of Neuroscience  
University of Alberta  
(403)492-3112

**Assistant investigator:** Allyson Jones  
Graduate Student, Physical Therapy

Many reflexes are known to be important to walking. The purpose of this study is to determine the behaviour of these reflexes during sitting, standing and walking in spinal injured individuals. The results of this study should help us understand how the nervous system controls walking and what aspects of this control are affected by spinal injury.

The experiments will take approximately 3 to 4 hours, and will involve applying surface electrodes on the skin to record the activity of the muscles. Minor skin irritation may result from the surface electrodes. Electrical stimuli will be applied to the nerves using surface electrodes at the ankle. These stimuli may cause slight discomfort. There is a remote chance of unwanted electrical shock because of the use of electrical equipment, but this chance is minimized by using electrically isolated stimulators and by equipment testing according to the Canadian Standards Association.

The electrical stimuli will be applied randomly while you are sitting, standing and walking. Because of the small disturbances associated with these stimuli, you may be more prone to fall. The following safety measures will be taken. All experiments involving standing or overground walking will be performed in the parallel treadmill has a start-stop switch which you will control. Railing is available on the sides of the treadmill for support. In addition, for subjects that feel unstable on the treadmill, a body harness will be worn to support your weight in the event that you should stumble.

Your identity will be kept confidential. All reports resulting from these studies will refer to the subjects by a number or code. You are free to ask any questions at any time, and you may withdraw from the experiment at any time without any adverse consequences to you.



## CONSENT FORM

**Project title:** Neural Control of Human Locomotion

I have read the description of the experiments on the attached page, and am satisfied with the information provided to me regarding the procedures and potential risks. My questions have been answered to my satisfaction, and I know that I am free to ask further questions at any time. I understand that my personal records will be kept confidential. I agree to participate in these experiments voluntarily, and am fully aware that I may withdraw from the experiments at any time with no adverse consequences to me in any way.

**Subject's name:** \_\_\_\_\_

**Subject's signature:** \_\_\_\_\_

**Investigator's signature:** \_\_\_\_\_

**Witness' signature:** \_\_\_\_\_

**Date:** \_\_\_\_\_