

Biomechanical Analysis of Stretch Reflex Responses: An Approach to Spasticity Measurement

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**TEXT
BOUND INTO THE
SPINE**

Declaration

This Thesis describes work carried out by the author in the School of Mechanical and Systems Engineering of the University of Newcastle upon Tyne, United Kingdom, during the period September 1999 to November 2004, under the supervision of Professor G.R. Johnson.

This Thesis describes original work which has not been submitted for a higher degree at any other University and is the work solely of the undersigned author, except where acknowledged in the text.

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Abstract

Spasticity is a clinical condition that may develop in people with central nervous system injuries. It is believed that spasticity results from changes in the excitability of the stretch reflex pathways manifesting clinically as a velocity dependent increase in resistance to passive movement (RTPM) and exaggerated tendon jerks. Stretch reflex excitability is influenced by neural (e.g. feed-forward and feedback mechanisms) and biomechanical components (e.g. muscle length). The objective of this work was to quantify the stretch reflex parameters of the biceps brachii under different initial conditions (amplitude of applied torque, initial muscle length, initial voluntary activity, head position) in non-impaired (NI) volunteers and stroke patients (SP) with diagnosed upper limb spasticity and objectively evaluate their differences. A biomechanical device was designed to provide a 90 ms initially applied torque controlled stretch to the biceps brachii. The stretch reflex response was recorded using surface electromyography and angular displacement with a potentiometer. Stretch reflex characterisation was done on EMG data collected 150 ms before and to complete 450 ms after the perturbation. The outcome measures were the amplitude of the rectified reflex response and, the latency, rise time and duration reflex response. Lower amplitudes, shorter latencies and longer durations were observed in the post-stroke population when compared to the non-impaired volunteers. Amplitude results were unexpected. However latencies and durations suggest increased stretch reflex excitability. Significant differences dependent on the initial conditions were found within the non-impaired volunteers. No differences were found in the post-stroke population. These latter results suggest lack of modulation of the stretch reflex excitability after stroke. More research is necessary to understand the relationship between the changes in the stretch reflex excitability and the clinical concept of spasticity and the importance of their quantification to improve the quality of life of people with neurological lesions.

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1. Introduction

1.1. Foreword

This investigation examines the concept of spasticity, a neurological disorder present in people who have suffered lesions to the central nervous system (CNS) at any level (brain, brainstem, spinal cord, etc.). This phenomenon is believed to cause disability, pain and discomfort, and is therefore considered as an impairment affecting quality of life (Bhakta et al. 1996).

The concept of spasticity has captured the interest of health care professionals, including clinicians, nurses, physiotherapists, neurophysiologists and rehabilitation engineers.

Different techniques have been conceived to understand its pathophysiological mechanisms, assess its degree of severity and the evolution of its treatment with two main objectives: a) to improve the quality of life and b) to comprehend the CNS adaptability mechanisms to such injuries (plasticity).

These objectives are not far from each other. In order to improve quality of life it is necessary to understand the neurophysiological mechanisms in the intact CNS and the changes after injury, how the patient adapts to them, and how their disability can be minimised (physiotherapy, drugs, surgery).

The techniques selected when attempting to evaluate spasticity reflect the different professional backgrounds, training and experience. Some groups rely on obsolete techniques because that is how they have been taught, others in not yet fully proven ones because of fashion and/or convenience and finally others try to develop their own techniques which can either be original or based on pre-existing ones. All these approaches have their advantages and disadvantages. It is to the clinician, therapist, engineer or scientist to decide which one to follow, according to what they feel more obliged to know, prove or discover. However, the adoption of a proper measurement technique will depend on a great extent on the definition used to describe spasticity. This definition must be based on the physical manifestation of what is understood as spasticity, thus providing the variable to be measured.

The intention of this work is to provide a technique to characterise the stretch reflex as a measuring tool capable of discerning the stretch reflex properties under different conditions and study any variations between non-impaired and spasticity diagnosed post-stroked populations and the variations within them.

1.2. Etymology and definitions of spasticity

The word “spasticity”, from the Greek “spastikos” (σπαστικός) meaning to tug or draw (Shapiro 2001), was probably adopted to describe the resistance felt when a clinician passively stretches a joint. Nevertheless its origins are not well known and the term spasticity can have different meanings to different people (O'Brien, Seeberger, & Smith 1996). The term *spastic rigidity* was used in early case study reports from the nineteenth century where two cases of general spastic rigidity are described (Duckworth & Toth 18?? A.D.). The author defines the term rigidity as an increase in muscle tone but the terms spastic and spasticity are not properly defined. In these reports the muscle condition was assessed by applying an electric current and qualitatively assessing its response.

In further experiments, Sherrington used the term ‘rigidity’ to describe changes in neuronal activity in decerebrated cats and associated it with sustained and continuous muscle activity (Sherrington 1898). However the phenomena observed in these cases differ from those described in the clinical definition of spasticity. This clinical definition states spasticity to be “... a motor disorder characterised by a velocity dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks resulting from hyper excitability of the stretch reflex as one component of the upper motor neurone syndrome” (Lance JW 1980). Even though this definition attributes spasticity as an increased muscle tone resulting from abnormalities in the stretch reflex, muscle tone has two components: (1) neural dependent on the electrical activity of the muscle that can be reflexive or voluntary and (2) biomechanical dependent on the muscle fibres and soft tissue properties (Johnson 2002) and not every measurement protocol can differentiate them.

Later on, authors including Lance himself have narrowed the definition. For instance Lance, ten years after this definition was originally proposed, responded in a letter that

“... spasticity does not include impaired voluntary movement and an abnormal posture” (Lance 1990) and attributed these symptoms to other features of the upper motor neuron syndrome that can be associated with spasticity but do not help to define it. Furthermore, in this letter, he stated the differences between decerebrated rigidity and spasticity.

Decerebrated rigidity is defined as the increased reflex resistance to passive muscle stretch with lengthening of muscle fibres, whereas spasticity is when the muscle resistance to passive stretch subsides once the muscle is lengthened sufficiently to excite group II and smaller afferent fibres that inhibit the stretch reflex by a central mechanism, thus producing the “clasp –knife” effect.

Chapman and Wiesendanger (1982) described spasticity as a change in muscle tone characterised by hyperreflexia (brisk and irradiating tendon jerks or phasic stretch reflexes), hypertonia (increased resistance to rapid passive stretch), and clonus (a series of repetitive muscle contractions elicited by a rapidly applied but maintained stretch). In this definition, a redundancy is evident when describing increased muscle tone since hypertonia and the described clonus can be related to the tonic component of the stretch reflex (Chapman & Wiesendanger 1982).

Burke (1988) defined spasticity more broadly as a disorder of spinal proprioceptive reflexes manifested clinically as tendon jerk hyperreflexia and an increase in muscle tone that becomes more apparent the more rapid the stretching movement and considered it as an adaptation to pyramidal tract injury. According to him, spasticity is a common but not inevitable consequence of a lesion in the central nervous system (Burke 1988). In 1988, Botte and colleagues described spasticity as a phenomenon of pathologically increased muscle tone and hyperactive reflexes mediated by a loss of upper motor neuron inhibitory control (Botte et al. 1988). Young (1989) suggested to expand the definition to include paresis, synkinesia, lack of dexterity and fatigability, which characterise the upper motor neurone syndrome in its totality which, according to him, is appropriately described as spastic paresis (Young RR 1989). Stefanovska and colleagues (1991), simply defined spasticity as an impaired control of the stretch reflex loop resulting in exaggerated activity (Stefanovska et al. 1991). Delwaide (1993) updated the definition by saying that it is “...a motor disorder characterised by brisk tendon jerks (sometimes accompanied by clonus) and a velocity-dependent elastic muscle hypertonia during

stretch, affecting certain muscle groups preferentially. It results from hyper excitability of the Ia pathway to motor neurons combined with abnormal processing at the spinal cord level of other peripheral afferent inputs (tonic stretch reflex)” (Delwaide, Pepin, & Maertens de 1993). However, in a study by Wilson et al in 1999 there was no evidence to suggest that the spindle afferents were affected in spasticity (Wilson et al. 1999). They also suggested considering hyperreflexia and increased tone separately, restricting the term spasticity to velocity-sensitive stiffness felt during mobilisation. This however does not address the dissociation between neural and non-neural components in muscle tone.

Young (1994) considered these definitions as narrow and restrictive, and went further describing the so called spastic paresis adding to the clinical definition the following factors (Young 1994):

- (1) other positive symptoms of the UMN syndrome such as exaggerated cutaneous reflexes, autonomic hyperreflexia, dystonia and contractures
- (2) negative symptoms, such as paresis, lack of dexterity and fatigability

Although these additions are obviously relevant for clinical practice and to the development of new treatments, they describe the whole UMN syndrome and not spasticity alone. Lin and Rymer (1991) and Katz (1994) used the term spastic hypertonia to define the abnormal limb resistance to passive stretch (Lin & Rymer 1991), (Katz RT 1994). Nevertheless, they also failed to distinguish between neural and biomechanical components.

According to these interpretations, stretch reflex excitability prevails as the main factor contributing to spasticity. Nevertheless, it has been reported that some patients with spasticity do not have hyperreflexia (O'Dwyer & Ada 1996) and in other cases, patients with hyperreflexia do not present with spasticity (Sherman, Koshland, & Laguna 2000). Although it is true that the neurophysiology of the stretch reflex is the simplest circuitry within the nervous system, it is also true that its final outcome can vary according to other structures and mechanisms influencing its modulation. Furthermore, the complexity of evaluating the presence of spasticity is dependent on the reliability of the

available technology and its capacity to dissociate the stretch reflex input from intrinsic biomechanical characteristics of the muscle.

1.3. Hypotheses

A sudden perturbation stretching the flexor muscles of the elbows will result in a stretch reflex response that can be recorded using EMG surface electrodes.

The stretch reflex excitability is enhanced in people with spasticity.

1.4. Objective

The objective of this work is to design a biomechanical device capable of eliciting a stretch reflex response from the elbow flexors and analyse and compare the response of non-impaired volunteers with that of people who have been diagnosed with spasticity at the elbow flexors as a result of stroke.

2. Introduction to motor control

2.1 Introduction

Activities of daily living (e.g. gait, reaching, grasping, etc.), rely on the coordination of the sensori-motor system to provide balance and posture. Sensory inputs from specialised organs within the muscles, joints and skin give feedback to the central nervous system (CNS) through the dorsal part of the spinal cord (figure 1) and inform about postural or environmental changes, modifying muscle activity.

Muscles are the body actuators; their main purpose is to produce force and movement. Electrical stimuli travelling from the motor neurones in the anterior part of the spinal cord (alpha motor neurones) (Figure 1) to the neuromuscular plate generate muscle contraction.

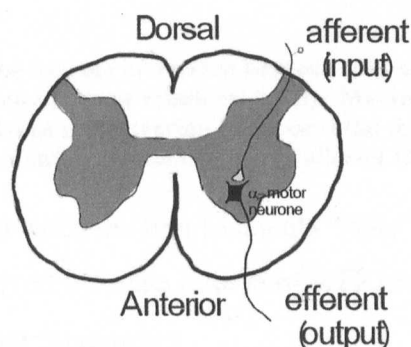


Figure 1: Scheme of the spinal cord (axial view) showing the afferent and efferent pathways and the α -motor neurones.

2.2 Muscle contraction

Contraction occurs at a molecular level in the so called *sliding mechanism*. Muscles are formed by groups of muscle fibres containing myofibrils which are contractile elements. The functional units (i.e. smallest unit within an organ capable of performing all of the organ's functions) of the myofibrils are the sarcomeres which are formed by arrays of thick (myosin) and thin (actin) filaments arranged in parallel (Figure 2A). The maximum tension a muscle can produce at any given length depends on the relative overlap between the actin and myosin filaments within each sarcomere (Figure 2B).

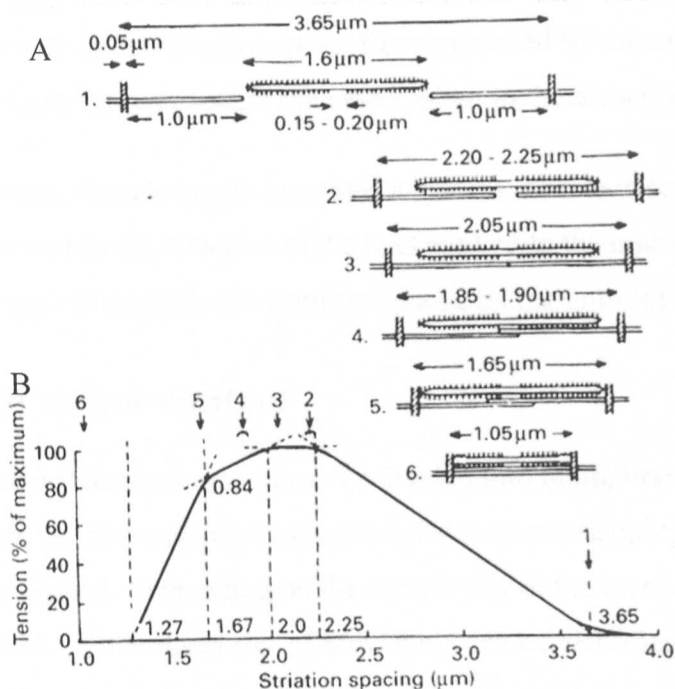


Figure 2: Relationship between the degrees of overlap between thin and thick filaments of skeletal muscle fibres (A) and their force-length curve relationship (B). Maximum tension is generated with maximum overlap. The tension drops if the overlap is less or if the thin filaments collide with each other (minimum muscle length) (Rothwell 1994).

Muscle contraction is produced when the thin filaments “slide” within the thick filaments effecting mechanical work derived from the release of ATP (energy) caused by the electrical stimuli from the α -motor neurones.

The sliding mechanism is described in more detail in “Control of Human Voluntary Movement”, (Rothwell 1994).

There are two types of muscle contraction that can be observed whether it is during activities of daily living or in a clinical environment. These are as follows:

- Isometric: Muscle length remains fixed while the muscle tension is increased.
- Anisometric: Muscle length is variable depending on the activation of the muscle or imposed passive movement. These can be subdivided in two categories:

- Concentric: Muscle length decreases with muscle activity producing movement in the direction of the force produced by the muscle (i.e. when the muscle torque exceeds the load applied to the muscle).
- Eccentric: Muscle length increases while the muscle is active producing movement in the direction of the load applied to the muscle (i.e. when the load applied exceeds the torque produced by the muscle).

2.3 Levels of motor control

Anatomically, the central nervous system can be divided into brain, brainstem and spinal cord. These areas are interdependently responsible for three overlapping levels or hierarchies in motor control. Depending on the complexity of the level, more anatomic structures are involved. Three categories of movement can be identified (Ghez & Krakauer 2000):

Reflexes: Mediated in the spinal cord, they are automatic responses or changes in initial static (e.g. posture) or dynamic (e.g. walking) conditions. These responses are different when the environmental, external (secondary) conditions and/or verbal instructions are changed (Misiaszek et al. 2000). They are necessary to maintain the integrity and stability of a particular organ (e.g. the stretch reflex maintains muscle tension with respect to other muscles due to joint angle changes using a feedback mechanism) and/or the body as a whole (e.g. the flexor-withdrawal and crossed-extensor reflexes avoid a noxious stimulus (tripping) by withdrawing the site from the stimulus and avoiding falling by extending the contralateral limb respectively). In order to do so, the body relies on “proprioception” (sense of position) and kinaesthesia (sense of movement) provided by the muscle receptors and interpreted by the CNS (Gardner et al. 2002).

Voluntary movements: Generated in the motor cortex the signals travel down the brain stem and spinal cord achieving muscle contraction. Depending on the task, resulting actions can range from dexterous (e.g. writing, pinching, etc.) to strong movements (e.g. lifting heavy objects, punching, etc.).

Automatic postural adjustments (rhythmic): Generated in the spinal cord and brain stem, these adjustments are a compromise between reflexes and voluntary movements. They

are more flexible than reflexes but more constrained than voluntary movements. Their main action is to provide and maintain balance. This is evident during gait when the body weight shifts from one side to the other depending on the limb supporting the body weight.

These three levels are interdependent; reflexes are always present during voluntary movement and automatic postural adjustments but their excitability or threshold is different depending on the task being performed at a particular moment.

2.3.1 Feedback and feed-forward mechanisms

Depending on the level of activity of the α -motor neurones, muscle contraction can be controlled based on feedback and/or feed-forward mechanisms.

Changes in the environment (light, temperature, movement, sound, etc.) are detected by bio-sensors which send electrical impulses to the nervous system “updating” the body state to the new environment through the muscles. These reactions are mediated through a **feedback mechanism**. For example, changes in light will adjust the diameter of the pupil; an extreme sound will cause the body to bend forward and use the hands to cover the ears.

2.3.1.1 Feedback and feed-forward mechanisms during muscle stretch

All systems depending on their requirements and degree of functionality under different conditions rely on feedback and feed-forward mechanisms (Lin & Rymer 2001; Oddsson 1990).

Feedback mechanisms require a sensory component that informs the controller whether it has reached any desired value (position, temperature, velocity, etc.) or has reached a higher or lower value informing the controller to increase or decrease the input to the actuator.

Feed-forward mechanisms set the system to a certain level of activity and/or responsiveness increasing or decreasing the threshold at which the controller responds to the feedback input.

The following is a brief description of the stretch reflex to illustrate the feedback and feed-forward mechanisms during a stretch. The stretch reflex itself will be described in more detail later in this chapter.

If a sudden perturbation stretches one of the muscle groups of a joint (e.g. elbow flexors) the muscle spindle acts as a feedback sensor activating the stretch reflex causing the muscle to contract with the aim of maintaining the original position/and or muscle tension (Figure 3A). The muscle spindle is sensitive to changes of length (position) and rates of change in length (velocity) of the muscle. The actuator is the muscle group (biceps brachii, brachio radialis) opposing to the movement trying to maintain the initial position or desired state mediated by supraspinal signals (voluntary activity). The equivalent control diagram for the feed-back mechanism (Figure 3B) consists of one input signal (desired length) for the muscle processed in the summation point which would anatomically correspond to the spinal cord, more specifically the α -motor neurone pool. Signals from this summation point activate the muscle and produce a force. This force, depending on the insertion point on the joint, will generate a moment attempting to maintain or reach the desired state. An external moment may or may not be present contributing to the final levels of muscle activation necessary to produce the force maintaining the position. The sum of muscle and external moments and the physical characteristics of the limb (moment of inertia) will cause an angular acceleration, velocity and movement of the joint associated with linear acceleration, velocity and length changes of the muscle related by the respective muscle's moment arm. Changes in muscle length and the velocity of the stretch are interpreted by the muscle spindle providing with a positive feedback for muscle activation (negative feedback for muscle length).

Before an external stimulus is produced, muscle receptors are set to a particular activation level depending on the task being performed (shooting a gun, carrying a cup of tea, walking, etc.). Previous information of a probable perturbation to come is also taken into account (verbal instruction, visual stimulus, etc). These initial conditions are part of the **feed-forward mechanism**. Visual information, given instructions, previous experience, environmental conditions, etc., set the CNS activity to modify reflex excitability (i.e. sensitivity), so that, when a sudden stretch occurs, the overall effect will vary according to the conditions (Figure 4A). The equivalent control diagram for the

feed-forward mechanism (Figure 4B) illustrates the influence of inhibitory and excitatory mechanisms added to the previously described feedback mechanism. These mechanisms increase or decrease the sensitivity of the controller to the muscle spindle signals (i.e. reduce the threshold for the feedback signal to activate the α -motor neurone pool) (Figure 5).

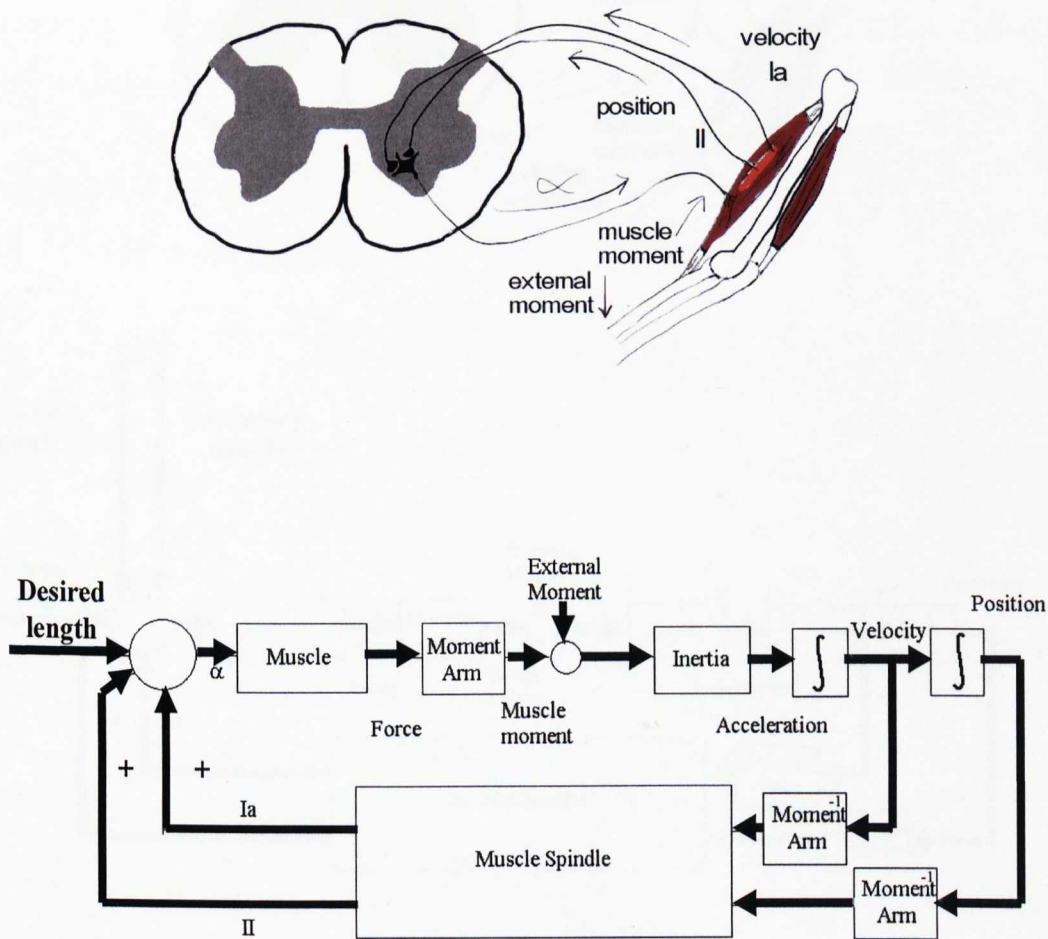


Figure 3: A) Anatomical diagram of the stretch reflex illustrating a feedback mechanism. B) Control diagram for the feedback mechanism. The stretch reflex attempts to maintain the desired length of the muscle when an external perturbation (moment) acts on the forearm. The muscle spindle senses the length changes caused by such perturbation (length and speed of lengthening) increasing the α -motor neurone firing to the muscle.

In summary, the lack or presence of visual, auditory stimuli and the nature of the activity being performed before a perturbation influence reflex excitability resulting in a different pattern of motor control.

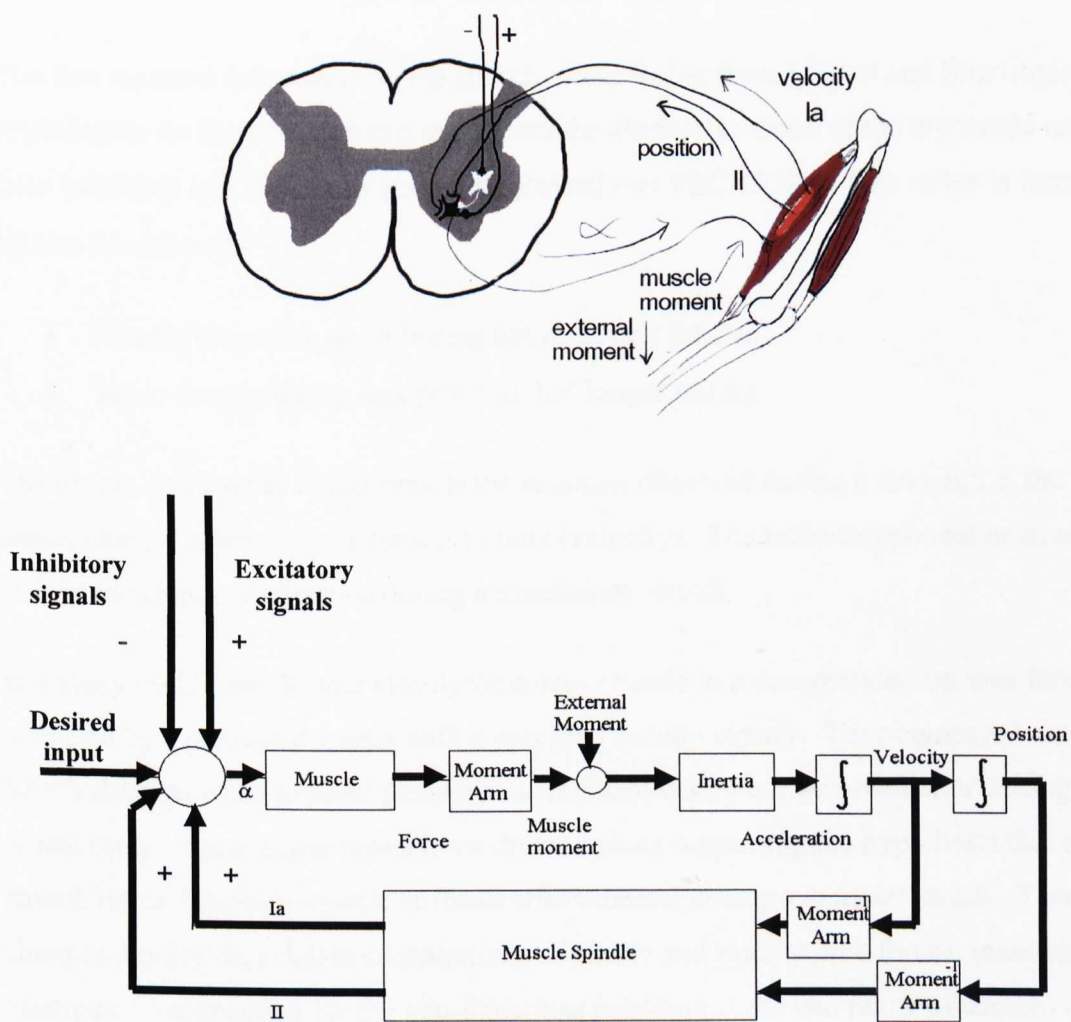


Figure 4: A) anatomical diagram of the stretch reflex and how it is influenced by supraspinal and cortical signals (feed-forward mechanism) that can be excitatory or inhibitory. B) Control diagram for the feed-forward mechanism. The stretch reflex is modulated by supraspinal and cortical signals changing its excitability.

2.4 The stretch reflex

From the definition of spasticity proposed by Lance (1980) it can be inferred that spasticity can be measured by direct or indirect quantification of the excitability of the

stretch reflex. Thus, it is important to understand the variability of the stretch reflex response and its behaviour under different conditions.

2.4.1 Stretch reflex components

The first reported documents on the stretch reflex dating from Liddell and Sherrington's experiments on decerebrated cats gave them the alternative name of the myotactic reflex ($\mu\iota\omicron$ (muscle)- and $\tau\alpha\kappa\tau\iota\kappa\omicron\varsigma$ (extended))(Matthews PBC 1972a). This reflex is formed by two components:

- Phasic (transient): short lasting but relatively intense
- Tonic (steady state): less powerful but longer lasting.

The phasic component or transient is the response observed during a stretch, i.e. the actual change in length with respect to time (velocity). The tonic component or steady state is the response observed during a maintained stretch.

In a study by Lin and Rymer (1998) the soleus muscle in a decerebrated cat was forcibly stretched by a simulated inertia with a specified initial velocity. They compared muscle length changes when afferent pathways were intact with those recorded after cutting the dorsal roots. These experiments have shown results supporting the hypothesis that the stretch reflex increases muscle stiffness with minimal changes in mean length. These changes modify the relative contributions of elastic and viscous-like forces, maintaining elasticity compensating for the non-linearities (yielding at the end range of stretch) of the elastic properties of the muscle (Lin & Rymer 1998).

2.4.2 Biosensors and conduction pathways

Change in muscle length and rate of change (muscle stretch) provide the necessary stimuli to activate the muscle spindles causing them to fire; the impulses are conducted by two groups of afferent fibres (Ia and II) to the dorsal roots of the spinal cord where the α -motor neurones are excited monosynaptic, and polysynaptically, (Homma 1976;Matthews PBC 1972b;Misiaszek, de Serres, Stein, Jiang, & Pearson 2000) contracting the muscle being stretched via efferent fibres (α -motor neurone axons).

2.4.2.1 Muscle Spindle

Structure: Muscle spindles are sensors sensitive to the length and changes of length of the muscle. They consist of encapsulated intrafusal muscle fibres (inside) arranged in parallel within the muscle (Figure 5). Two types of sensory neurones innervate the muscle spindles. Group Ia afferents are large nerve fibres and have a diameter of 12-20 μm and conduct impulses at velocities 80-120 m/s. Group II afferents are smaller (6-12 μm in diameter) at velocities 22-57 m/s (Schafer, Schuppan, & Dadfar 1999).

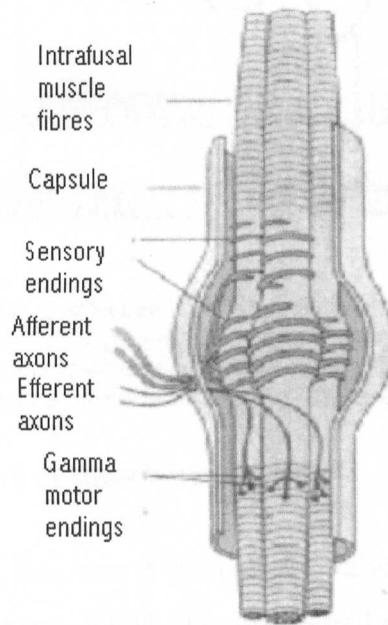


Figure 5: Intrafusal muscle fibres, afferent sensory fibre endings and efferent motor fibre endings are the main components of the muscle spindle. The sensory fibre endings spiral around the central region which is not contractile and are sensitive to stretch of the intrafusal fibres. Gamma motor neurones innervate the polar (extreme) regions which are contractile changing the sensitivity of the sensory fibres endings to stretch after a change in length (Pearson & Gordon 2000).

Physiology: When a muscle is stretched, impulses are generated in both types of sensory fibres with a clear difference in the characteristics of the discharges in the two endings. The properties of the fibres are directly responsible for the stretch reflex components (transient and steady state).

Primary endings (Ia) are sensitive to the rate of change of stretch (they are position, velocity, and possibly acceleration sensitive since there are points of acceleration and deceleration before and after the stretch). The frequency of discharge is maximal during

the transient component of the stretch (Misiaszek, de Serres, Stein, Jiang, & Pearson 2000).

Secondary endings (II) are sensitive to the level of static tension or sustained stretch (position). Their frequency of discharge is maximal during the tonic component of the stretch (Figure 6).

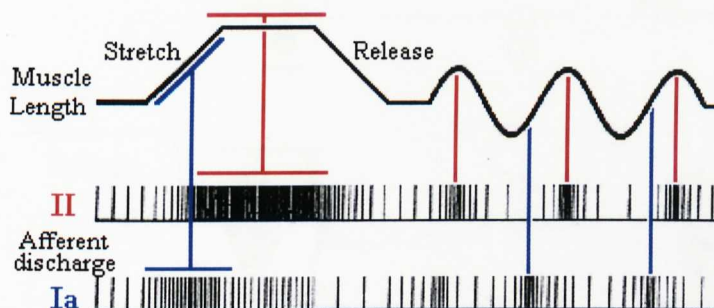


Figure 6: Frequency of discharge of Ia and II afferent fibres due to different levels of stretch. Ia afferent fibres frequency of discharge is maximal during the phasic component of the stretch (velocity sensitive) whereas the II afferent fibres frequency of discharge is maximal during a sustained stretch (position sensitive) (Taylor & Durbaba 2003).

2.4.2.2 Fusimotor system (γ motor neurones)

It is important to remember that muscle spindles are embedded within the muscle, and their length is affected by muscle length changes. Small motor fibres (2-8 μm in diameter) known as γ fibres innervate muscle spindles forming the fusimotor system. During large muscle contractions γ -motor neurones make the spindle contract, maintaining its tension, sensitivity and length relative to the muscle length (Pearson & Gordon 2000) (Figure 7).

2.4.2.3 Beta innervation

Also known as skeletofusimotor innervation (i.e. innervates intra and extrafusal fibres) (Kakuda & Nagaoka 1998) β fibres form a positive feedback loop to the muscle spindle in response to a stretch, augmenting spindle firing (Grill & Rymer 1985), activating the spindle during contraction (Kakuda, Miwa, & Nagaoka 1998). It is suggested that

β innervation and dynamic γ innervation are support each other in fusimotor dynamic innervation (Laporte & Emonet-Denand 1976).

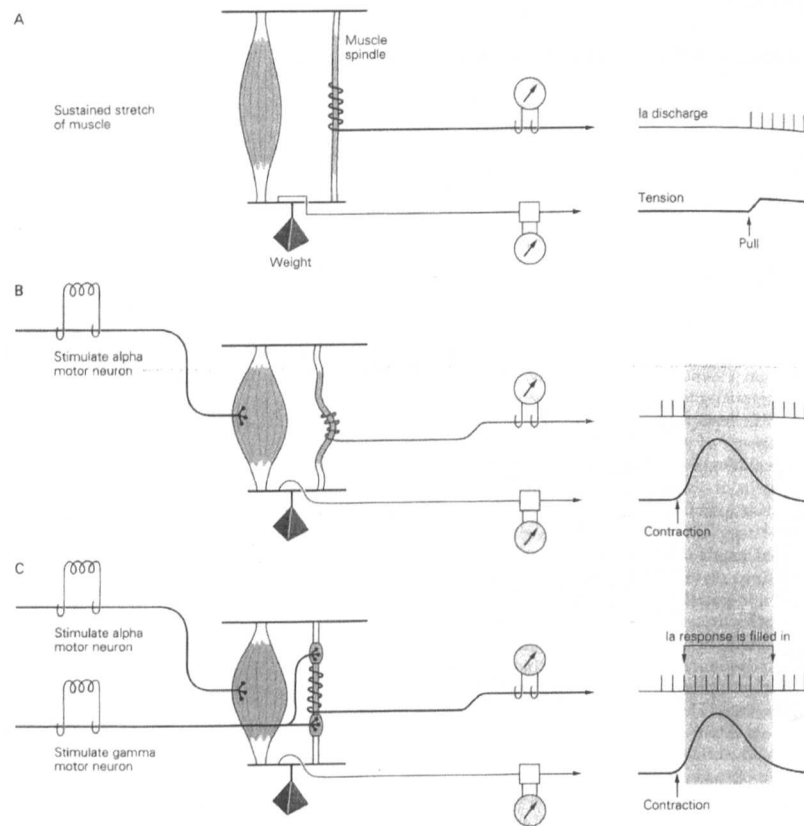


Figure 7: Contraction of muscle spindles via gamma motor neurones during active muscle contraction enables them to continue sensing changes in muscle length. A) sustained tension elicits steady firing in the Ia sensory fibre. B) When the α -motor neurone alone is stimulated, there is a pause in the discharge of the Ia fibre because the spindle is unloaded by the resulting contraction. C) If α and γ motor neurones are stimulated at the same time; the spindle is not unloaded during the contraction, filling the pause in the discharge of the Ia (Pearson & Gordon 2000).

2.4.2.4 Higher centres modulating the stretch reflex (inhibitory and excitatory pathways)

The stretch reflex, like all spinal reflexes, can be modulated at three possible sites in the spinal cord (Pearson & Gordon 2000) (Figure 8A):

- Alpha motor neurones
- Interneurones in polysynaptic pathways
- Presynaptic terminals of the afferent fibres via presynaptic inhibition.

This modulation affects the stretch reflex activation by changing the background activity (Figure 8B) and /or the task and behavioural state (Evarts & GRANIT 1976) and might be regulated via supraspinal signals from higher centres, inhibitory interneurons, Golgi tendon organ (GTO), etc., making connections at these three sites (Misiaszek, de Serres, Stein, Jiang, & Pearson 2000). Furthermore, all these organs, neurones and general structures are also modulated by each other (Windhorst 1996) resulting in a highly non-linear system.

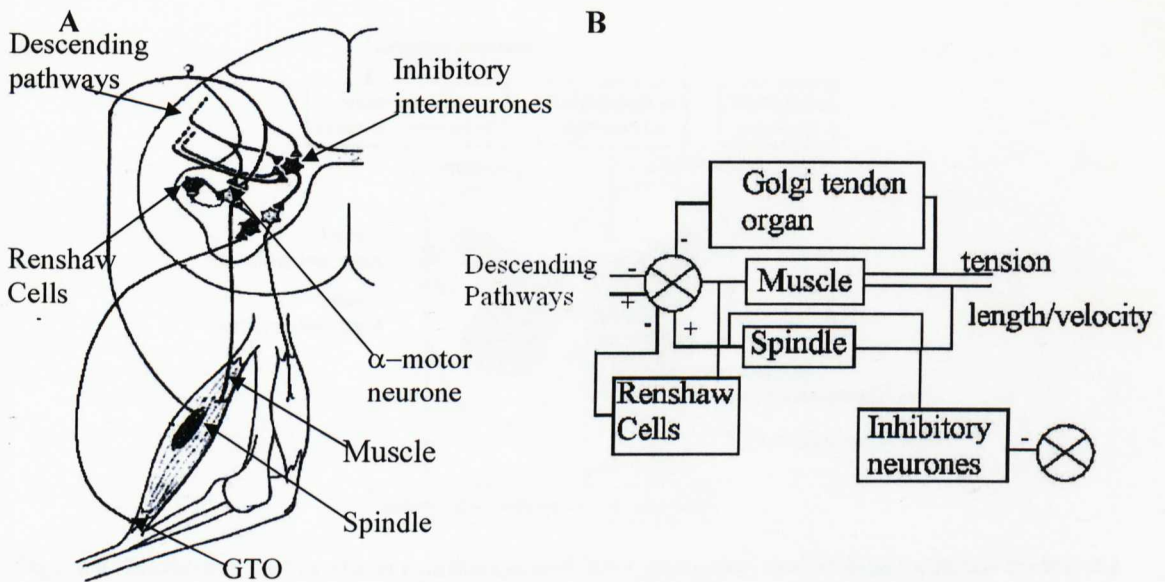


Figure 8: A more complex circuitry, showing most of the variables involved to change the α -motor neurone excitability. A) Descending pathways change voluntarily the α -motor neurones excitability which is self regulated via the Renshaw Cells and influenced by the inhibitory interneurons activated by the contralateral α -motor neurones. The Golgi tendon organ (GTO) senses the changes in tension reducing the motor neurones excitability (negative feedback). Other signals (not shown) come from cutaneous receptors (Pearson & Gordon 2000). B) Schematic representation of the circuitry.

2.4.2.5 Interneuronal pathways

Muscle contraction is possible when an electrical stimulus from the α -motor neurones depolarises the muscle fibres. These electrical impulses result from the integration of signals originating in the cortico-fugal pathways (providing with presynaptic facilitation and inhibition), proprioceptive sensors, inhibitory inter neurones and Renshaw cells. Cortico-fugal pathways also modulate the sensors and interneurones excitability. These modulations suppress or enhance the stretch reflex intensity.

2.4.2.6 Presynaptic facilitation and inhibition

Two balanced systems for control of spinal reflexes arise from the brain stem and descend and run in the spinal cord, one excitatory and one inhibitory. These are anatomically separated and differ with respect to cortical control (Figure 9) (Sheean 2002).

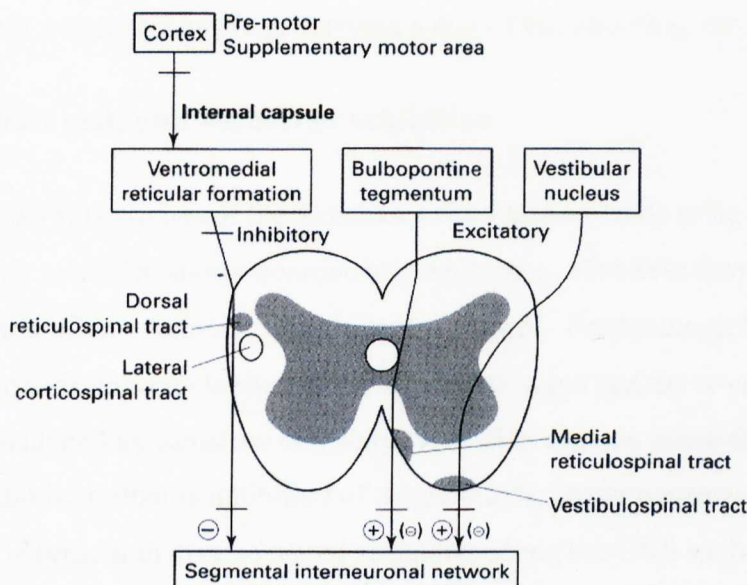


Figure 9: Inhibitory and excitatory pathways and their particular descending locations within the spinal cord. (+) indicates excitatory pathways and (-) inhibitory pathway (excitatory pathways inhibit flexor reflexes). These pathways maintain the balance for feed-forward mechanism (Sheean 2002).

The dorsal reticulospinal tract is the main inhibitory pathway and arises in the ventromedial reticular formation and runs close to the pyramidal tract or lateral corticospinal tract.

There are two main excitatory pathways. The first and most important one arises in the bulbopontine tegmentum and descends in the medial reticulospinal tract. The vestibulospinal fibres originate in the vestibular nucleus descending on the vestibulospinal tract. Signals coming from these pathways modulate the spinal reflexes and are dependent on the awareness or attention via feed-forward mechanisms.

2.4.2.7 Inhibitory interneurons and reciprocal inhibition

Inhibitory interneurons play an important role in the coordination of opposing muscle groups (flexor and extensor or abductor and adductor) of a particular joint. These interneurons inhibit the antagonist group when the agonist is contracting to avoid any opposition to the movement (Figure 10A). This mechanism is known as *reciprocal inhibition* and can be suppressed via volitional activity whenever a task requires activation of both muscle groups (e.g. carrying a cup of tea, shooting, etc.)

2.4.2.8 Renshaw cells and recurrent inhibition

The literature regarding studies of the Renshaw cells function tends to be limited and simplified to their role of α -motor neurone self inhibition. However they have a far more complex and often overlooked role in motor control. Furthermore, the concept of recurrent inhibition is not only limited to the spinal pathways and the α -motor neurone self inhibition mediated by Renshaw cell activity. It also appears in the form of lateral and surround inhibition (that is inhibition of neighbouring motor neurones within the same pool) and is present in diverse neural structures along the CNS such as γ -motor neurones, Ia inhibitory interneurons other Renshaw cells among others (Windhorst 1996). Windhorst comments that the actual function of the recurrent inhibition remains a matter of speculation. Thus for practical reasons the functionality of the Renshaw cell is considered to be producer of negative feedback to the α -motor neurones stabilising them and avoiding saturation (Figure 10B).

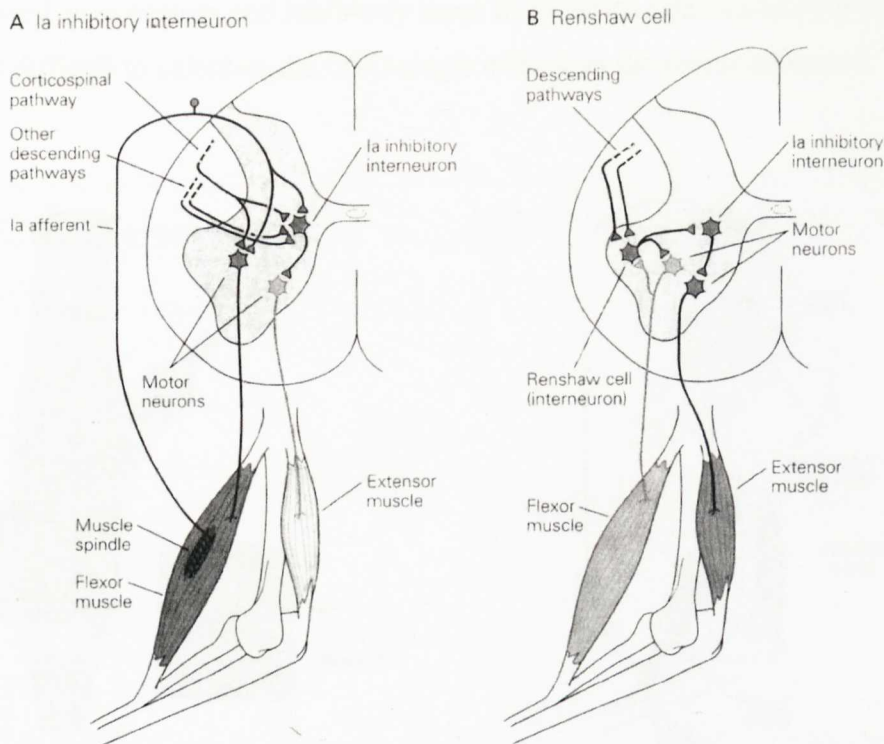


Figure 10: α -Motor neurones regulatory systems: A), inhibitory Interneurons from descendent pathways change the excitability of the α -Motor neurones. B) Renshaw cells are the α -Motor neurones self-regulation mechanism (feedback) (Misiaszek, de Serres, Stein, Jiang, & Pearson 2000).

2.4.2.9 Golgi tendon organ

Structure: The Golgi tendon organs (GTO) are mechanoreceptors whose response increases non-linearly with respect to tension generated within a skeletal muscle (Davies, Petit, & Scott 1995). These sensors are arranged in series with the muscles at the transition from muscle to the tendon (Schafer, Schuppan, & Dadfar 1999). They are slender, encapsulated structures about 1mm long and 0.1mm in diameter and. Each GTO is innervated by a single (group Ib) axon (Figure 11A). Stretching of the GTO straightens the collagen fibres thus compressing the nerve endings and causing them to fire (Figure 11A) (Pearson & Gordon 2000) and so provide information on the muscle tension.

Physiology: The action from the Ib afferent fibres from the GTO is mediated through the Ib inhibitory interneurons (Figure 11B) producing disynaptic inhibition of homonymous motor neurones (autogenic inhibition). However these interneurons also receive input from Ia fibres from muscle spindles, afferent fibres from cutaneous receptors and from

joints as well as excitatory and inhibitory input from various descending pathways making it difficult to calculate the GTO single effects on the motor neurones.

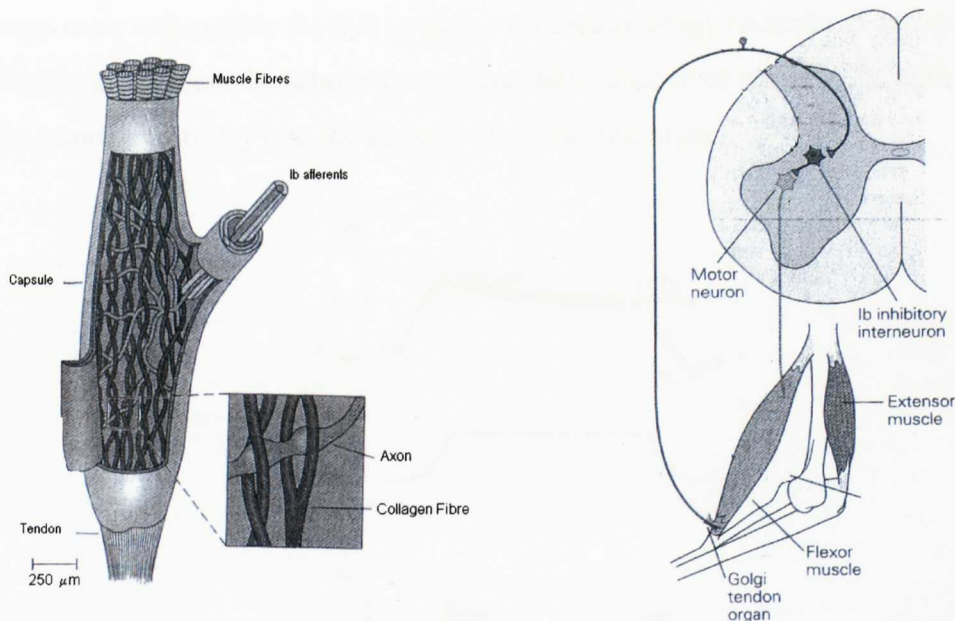


Figure 11: A) When the Golgi tendon organ is stretched, usually because of contraction of the muscle, the afferent axon is compressed by the collagen fibres and its rate of firing increases. B) The Golgi tendon organ changes the excitability of the α -motor neurone via a Ib inhibitory interneurone decreasing the firing rate after a threshold of tension is reached (Pearson & Gordon 2000).

Recent observations

It has been reported that the response is a function of the absolute tension and the change of tension (Davies, Petit, & Scott 1995; Gregory et al. 2002), in other words, there are some GTO that are responsive to static levels of tension (i.e. sensitive to tension at rest) and others sensitive to dynamic tension (i.e. sensitive to changes tension). Figure 12 illustrates the discharge patterns of two Golgi tendon organs recorded under equal experimental conditions (Schafer, Schuppan, & Dadfar 1999) a) the Golgi tendon organ generates an initial firing frequency, b) the Golgi tendon organ has no initial firing frequency with higher static sensitivity than the first group. The dynamic sensitivity did not vary significantly between the two groups.

Furthermore, a change in the sensitivity of the Golgi tendon organs has been observed in their responses to passive and active tension after series of eccentric contractions. It was found that after eccentric contractions nearly all tendon organs commenced firing at a shorter muscle length during slow passive stretch than before (Gregory, Brockett,

Morgan, Whitehead, & Proske 2002) (Figure 13). This study also shows that there was no significant difference between passive and active stretch tension which did not agree with observations from previous studies. They concluded that the different experimental settings were responsible for this as their own methodology considered active contractions produced by whole nerve stimulation instead of motor units with muscle fibres inserting directly into the capsule of the tendon organ.

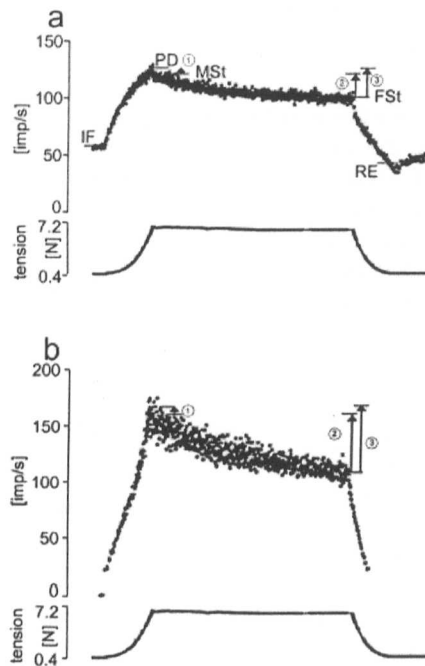


Figure 12: Discharge patterns of two Golgi tendon organs recorded under equal experimental conditions. A) the GTO generates an initial frequency which increases slightly as a result of the ramp stretch and a gentle decline of the discharge frequency to the initial frequency during the release of the stretch. B) the GTO has no initial frequency. A strong burst of discharge frequency originates as a result of the ramp stretch and a steep fall of the discharge frequency to 0 imp/s during the release of the stretch (Schafer, Berkelmann, & Schuppan 1999).

GTOs are not part of the stretch reflex loop however they can modulate the α -motor neurone excitability modifying the levels of muscle activation during a stretch and they should be considered when an experiment is being designed or a model developed.

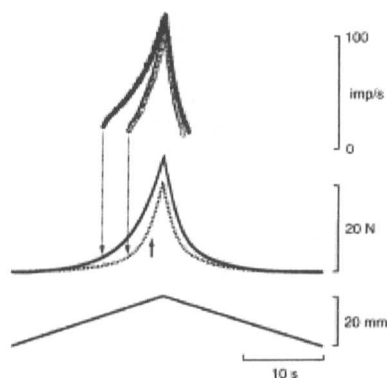


Figure 13: Illustration of the procedure used to test the sensitivity of the GTO to passive stretch. The bottom trace shows the slow triangular stretch and release from a muscle length of maximum physiological length. The traces in the middle show the tension recorded at the tendon before (dashed trace) and after (solid trace) the eccentric contractions, the tension is higher at all muscle lengths after the eccentric contractions. The upper traces show the response of one tendon organ as instantaneous impulse frequency before (open symbols) and after (filled symbols) the eccentric contractions (Gregory, Brockett, Morgan, Whitehead, & Proske 2002).

2.5 Discussion and conclusion

The Nervous System is adaptable, versatile and complex. The structures forming the stretch reflex feedback mechanism and the ones influencing the feed-forward mechanism are inherently non-linear and their activity and overall function are not entirely understood as yet. Each of the structures mentioned in this chapter contributes to the final motor control output of the stretch reflex. The stretch reflex response can be approached from an engineering perspective by assuming certain general function of each component of the circuitry and the time delays between them in order to develop a model.

Once the model is validated against the experimental data it can allow simulation of different neuropathies and help clinicians to choose the appropriate approach for patient treatment. The measurement protocol to obtain the experimental data should be designed to consider different conditions assessing the main physical variables involved in the stretch reflex loop such as velocity of the stretch, amplitude of stretch and feed-forward mechanisms (background activity of the involved muscles). This will help to understand the mechanisms modulating the stretch reflex and the overall motor control activity.

3. Upper Motor Neurone Syndrome and Spasticity

3.1 Introduction

The term upper motor neurone (UMN) syndrome describes a complex condition resulting from lesions disrupting some or all of the cortico-fugal pathways including the pyramidal tract at any level (cortex, internal capsule, brain stem or spinal cord) (Figure 1) causing lost of inhibitory control (Botte et al. 1988). These lesions may be a result from stroke, head injury, multiple sclerosis, cerebral palsy or spinal cord (Burke 1988).

3.2 Upper motor neurone features

The UMN syndrome presents different features (Table 1) that can be grouped as follows:

Negative features

These features are related to motor control or performance deficits and or reduction, causing weakness, loss of dexterity and easy fatigueability (Barnes 2001) and are more related to disability.

Positive features

These are increased or exaggerated abnormal behaviour like abnormal posture, exaggeration of proprioceptive reflexes producing “spasticity”, and exaggeration of some exteroceptive (cutaneous) reflexes of the limbs, producing flexion withdrawal spasms, extensor spasms, and the Babinski response (Byrne et al. 1998).

3.3 Spasticity

3.3.1. Introduction

The UMN syndrome features, especially spasticity, are generally associated with various degrees of paresis. The presence and degree of these features will depend upon the location and extent of the lesion and the pathways affected (Botte, Waters, Keenan, Jordan, & Garland 1988; Chapman & Wiesendanger 1982).

Chapter 3

Upper Motor Neurone Syndrome and Spasticity

Table 1: Features of the upper motor neurone syndrome

Negative	Positive
Muscle weakness	Increased Tendon reflexes with radiation
Loss of dexterity	Clonus
Fatigueability	Positive Babinsky sign
	Spasticity
	Extensor spasms
	Flexor spasms
	Mass reflex
	Dyssnergic patterns of co-contraction during movement
	Associated reactions and other dyssnergic and stereotypical spastic dystonias

Stroke is one of the most common causes for people to develop UMN syndrome. It can occur due to cerebral haemorrhage, blood clots, traumatic injury among others. The consequence of suffering a stroke is a condition known as hemiplegia or hemiparesis manifesting as loss of motor control of the opposite side of the body where the stroke occurred.

There are three possible stages in the hemiparetic subject (Bobath 1990; Bobath 1978; Sommerfeld et al. 2004):

- Initial flaccid stage

Also referred to as a period of shock or depression of reflexes (Sheean 2002), shock is a condition where paralysis/paresis and hyporreflexia are found in a person due to damage of pyramidal tract fibres, it may last typically from 1 to 6 weeks.

- Stage of spasticity

It is now recognised that spasticity may or not may be present in hemiparesis (O'Dwyer & Ada 1996). When it does, abnormal proprioceptive reflexes (related to the sensation of position, location and orientation of body parts) are the sign that the subject is starting in the spastic stage. The transition from the flaccid stage to the spasticity stage is gradual and occasionally the patients attribute some reflexive movements to voluntary activity.

- Stage of relative recovery

During the third stage the patient starts recovering voluntary movement, however the muscles are weak, contractures may be present, and some clinicians think that remaining

spasticity, may be the cause of lack of movement, but as we will see later this is not probable.

Sheean (2002) has divided the positive symptoms of the UMN syndrome into three subcategories, separating the clinical features into neat pathophysiological groups in a way that could help to determine therapy:

1. Abnormal processing of spinal reflexes (afferent-dependent)
2. Efferent drives depending on reflex activity in higher centres
3. Disorders of voluntary muscle movement

3.3.2. Spasticity features depend on the site of lesion

Some doctors tend to talk about two types of spasticity depending on the site of the lesion, cerebral and spinal. Both types have a slow time course of development (weeks or months) after the lesion occurs (Chapman & Wiesendanger 1982). The common view is that cerebral spasticity is characterised by an increased tone in the so called antigravity muscles (flexors in the arm, extensors in the leg, whereas in spinal spasticity it is usually increased in flexor muscles and flexor spasms are often associated). This concept of antigravity muscles is vague from the scientific point of view and results in a generalisation that instead of helping to understand the concept of spasticity, it oversimplifies it.

Nevertheless it is important to realise that the site of the lesion does determine what muscles and/or functions will be affected.

Lesions involving the middle cerebral artery affect the sensory motor functions of trunk, upper extremity, face and the function of speech producing the typical hemiplegic posture, affecting more the upper extremity and face than the lower extremity.

Lesions to the anterior cerebral artery mainly affect the sensory motor functions of the lower extremity resulting in a hemiplegic patient with relatively strong spasticity involved in such extremity.

Patients sustaining anoxic brain injury have diffuse loss of cerebral upper motor neurones and the spasticity involved is often bilateral and affecting both upper and lower extremities.

Lesions to the basal ganglia can give rise to severe increases in muscle tone resulting in rigidity.

Spinal cord injury may disrupt many of the descending pathways of the spinal cord, producing bilateral limb spasticity below the level of the injury. Furthermore, if the injury occurs at the level of the extrapyramidal structures it may result in rigidity.

3.3.3. Pathophysiology and clinical manifestation of spasticity

The definition proposed by Lance specifies the velocity dependent increase in stretch reflex hyperexcitability as the main characteristic of spasticity. However, clinical examination does not often involve any direct and objective test measuring such excitability. Ashworth scales and the tendon tap might be used but their reliability and validity are limited.

Young (1994) has ironically remarked that some clinicians think they can recognise spasticity when they see it and considers that Lance's definition is restrictive and simplistic (Young 1994). This is an unexpected and relevant statement, specially coming from one of the researchers co-authoring Lance's paper (Lance 1980). In fact, Young considers spasticity to comprehend the positive and negative features. Other authors suggest that spasticity should only consider the positive features and the more rigorous ones only consider the stretch hyperreflexia and increased muscle tone as indicative of spasticity (chapter 1). This makes spasticity difficult to describe and therefore to measure.

Some authors consider spasticity as to occur due to an imbalance of the tonic activity of the brain stem centres, i.e. a release of stretch reflex activity from normal inhibitory control coupled with an increase in descending excitation (Chapman & Wiesendanger 1982).

Chapter 3 Upper Motor Neurone Syndrome and Spasticity

Because of the above, there is no absolute agreement on the pathophysiological mechanisms of spasticity are. Therefore for different clinicians a patient might or might not have spasticity depending on their concept of it and the clinical signs presented by the patient.

For those accepting Lance's definition these clinical signs will be tendon jerk hyperreflexia (Burke 1988; Carr, Shepherd, & Ada 1995; Katz RT 1994; Lance JW 1980; Lehmann et al. 1989; Stefanovska et al. 1988); velocity dependent increased resistance to passive movement as an assessment for muscle tone (Burke 1988; Katz RT 1994; Lance JW 1980; Stefanovska, Gros, Vodovnik, Rebersek, & cimovic-Janezic 1988) and presence of the clasp knife phenomenon (Burke 1988; Lance JW 1980; Lance 1990). For some other authors, spasticity will also be accompanied by clonus and will affect preferentially certain muscle groups (Delwaide 1989). In some cases it is necessary to distinguish between spasticity (hyperreflexia), hypertonia (increased resistance to rapid passive stretch) and clonus (series of repetitive muscle contractions elicited by a rapidly applied but maintained stretch) (Chapman & Wiesendanger 1982).

It is important to mention that it is possible for all these symptoms to co-exist, but they also can be found independently from each other (O'Dwyer & Ada 1996; O'Dwyer, Ada, & Neilson 1996; Sherman, Koshland, & Laguna 2000).

The neural component of muscle tone is controlled via descending pathways by forebrain and brain stem structures. Changes in these structures due to UMN syndrome presence may affect some of the segmental mechanisms determining the excitability of the stretch reflex arc (Chapman & Wiesendanger 1982; Rymer WZ & Katz RT 1994) as a result or a combination of :

- a) Increased alpha motor neurone excitability. - considered to exist if motoneuronal recruitment and/or increased discharge are elicited with smaller than normal levels of excitatory input (motor neurones are excited with smaller stretch amplitude or slower than normal stretch velocity) (Katz RT 1994) as a result of a reduced threshold of excitation (i.e. constantly depolarised) (Botte, Nickel, & Akeson 1988; Harburn & Potter 1993; Katz RT 1994) resulting from perturbations in the balance of excitatory and inhibitory inputs (chapter 2).

- b) Enhanced Ia afferent responses or excitatory interneurons within the neural circuit were more responsive to muscle afferent input (Katz RT 1994) due to collateral sprouting, denervation supersensitivity or reduction in presynaptic inhibition of the muscle afferent (Burke 1988; Carr, Shepherd, & Ada 1995; Chapman & Wiesendanger 1982; Rymer WZ & Katz RT 1994; Sherrington & Denny-Brown 1979).

A third possibility is related to an increased fusimotor drive (γ -motor drive) (Harburn & Potter 1993). Some authors supported the idea of increased fusimotor activity driving the spindle (chapter 2) and increasing its sensitivity consequently increasing Ia afferent activity (Chapman & Wiesendanger 1982). This assumption although probable is not feasible as has been observed by Burke (1983) (Burke 1983) and Wilson and colleagues (1999) (Wilson et al. 1999) where no evidence of increased fusimotor drive was found, but this is open to interpretation. The prevailing view is that the reflex circuits involve neurones under supraspinal control increasing the circuit gain or the threshold originating from an abnormal processing of proprioceptive information.

Despite all the above physiological bases and theories, spasticity may or may not be present if a lesion in the brain has occurred and even in the presence of spasticity, not all signs and symptoms associated with it will be externally observable (Burke 1988).

3.3.4. Biomechanical Changes associated to spasticity

Biomechanical changes in the muscle properties may be indirect consequences of the UMN syndrome (due to immobilisation and/or neglect from the patient) and lead to contractures. These consist of the pathological changes of the mechanical properties of the soft-tissues (muscles, skin, subcutaneous tissue, tendon, ligament, joint capsule, vessels and nerves), for instance the non-contractile properties of the muscle and the characteristics of the active muscle length-tension curve are altered due to loss of sarcomeres (Botte, Nickel, & Akeson 1988; Harburn & Potter 1993). They are characterised by increased stiffness (demonstrated by an increase of torque required to extend the joint) and usually associated with loss of elasticity and compliance and fixed shortening of the involved tissues resulting in loss of motion of the surrounding joints, and occur as a secondary sequel of other disease or injuries (Botte, Waters, Keenan,

Chapter 3 Upper Motor Neurone Syndrome and Spasticity

Jordan, & Garland 1988; Harburn & Potter 1993; Lehmann, Price, deLateur, Hinderer, & Traynor 1989; O'Dwyer & Ada 1996). It has also been said that the duration and position of immobilization significantly alter the rate of formation of contractures more than the cause of the immobilization itself (Botte, Nickel, & Akeson 1988; Harburn & Potter 1993). Despite these observations, recent studies have suggested that biomechanical changes do not need to be fully developed to the stage of being considered as a contracture to affect resistance to passive movement (Singer et al. 2003).

There is a level of disagreement about the relationship of spasticity and contracture formation. Some authors (Bobath 1990; Harburn & Potter 1993) suggest that spasticity is the origin of contractures but they do not give an explicit definition of spasticity and they do not give any information describing the mechanisms responsible for the formation of contractures. Other authors (Botte, Waters, Keenan, Jordan, & Garland 1988), hypothesise that spasticity may be responsible for the immobilisation of the joint by preventing movement which then could lead to soft tissue fixation and the formation of contractures. However, Botte et al have highlighted the need for experimental evidence to support this hypothesis.

Furthermore, it has been observed by Singer and colleagues (2003) that also the biomechanical changes are velocity dependent, as Lance's definition of spasticity. This suggests that the phenomenon of spasticity, as it is observed in the clinical environment, may be masked by these changes and that the stretch reflex activity, although hyperexcitable may not be solely responsible for the clinical observations. Nevertheless, Singer experiments (2003) focus on the ankle joint where the connective tissue is relatively more abundant than, for instance in the elbow or the wrist, making the contribution of soft tissue changes to RTPM more evident in the ankle joint. The contribution of the biomechanical changes to RTPM however, needs to be explored in detail for a better understanding of all phenomena associated with neurological impairment and spasticity.

3.4 Conclusion

Lesions to the upper motor neurones cause the so called upper motor neurone (UMN) syndrome. These lesions cause an imbalance between inhibitory and excitatory signals

to the muscles affecting motor control stability. Spasticity is only one of the features of the upper motor neurone syndrome. Although Lance's definition has been accepted for over 20 years, the mechanisms and ultimate effects of spasticity towards disability are still under debate. There are indeed neurological changes affecting motor control but also the muscles affected change their inherent properties. These two components (neural and non-neural) should be dissociated and evaluated separately in order to provide with a proper treatment to avoid or reduce disability. An accepted approach to distinguish between neural and non-neural components in post-stroke hemiplegia is the use of EMG recordings. It has been reported that in a resting and relaxed hemiplegic limb there were no signs of electrical activity in the resting muscles (Basmajian & DeLuca 1985). This would imply that posturing is possibly independent of α -motor neurone activity and therefore cannot be considered as a result of spasticity. However, they have also observed that the threshold of excitability of the α -motor neurones was much lower than that observed in non-impaired subjects and that the hemiplegic subjects would respond to sensory stimuli (non-stretch) whereas a non-impaired person would not respond. There are also reports that in the presence of a lesion in the CNS just about any sensory motor perturbation could trigger increased α -motor neurones activity (Katz RT 1994).

It is also important to acknowledge all other features of the UMN syndrome that are affecting the overall condition of the people presenting it and that might overlap and confound each other.

4. Spasticity Measurement techniques

“When you can measure what you are speaking about and express it in numbers, you know something about it; but when you cannot measure, when you cannot express it in numbers, your knowledge is of a meagre and unsatisfactory kind; it may be the beginning of knowledge, but you have scarcely, in your thoughts, advanced to the stage of science”.

Lord Kelvin (1824-1907)

4.1 Introduction

Some concepts tend to differ from discipline to discipline. Furthermore daily use of words causes them to lose their original meaning and the abuse of synonyms creates misconceptions or misuse of some of these words, such is the case of measurement, assessment and evaluation.

Before reviewing objectively the currently available techniques to measure spasticity and their validity it is necessary to review the notion of measurement.

To measure has been defined as “assignment of numbers to objects according to a rule (Stevens 1946).

Measurements can be categorised by the type of information communicated by the symbols or numbers assigned to the variables in question. Thus, measurements are divided in different levels denominated scales of measurement (Stevens 1946):

4.1.1 Scales of measurement

1. Nominal Scale

- Non-quantitative measurement scale.
- Used to categorise, label, classify, name, or identify variables. It classifies groups or types.
- Numbers can be used to label the categories of a nominal variable but only as markers, not as indicators of amount or quantity (e.g., marking the gender with 1= female and 2= male)

- Examples:
 - Country of origin
 - Personality type
 - Experimental group (e.g., experimental group or control group)

2. Ordinal Scale

- Used to make ordinal judgements (i.e. rank order)
- Used in variables where the levels can be ranked (but the distance between the levels is not necessarily the same)
- Examples
 - Order of finish position in a marathon
 - Rank in class

3. Interval Scale

- Characteristics of rank order and equal intervals (i.e., the distance between adjacent points is the same)
- Does not possess an absolute zero point
- Example:
 - Celsius temperature

4. Ratio Scale

- True zero point.
- It has all the key characteristics of each of the lower level scales (i.e. equal intervals (interval scale), rank order (ordinal scale), and ability to mark a value with a name (nominal scale))
- Examples:
 - Weight
 - Height
 - Response time

4.1.2 Measuring impairment

For obvious reasons, different levels of impairment need to be standardised to evaluate whether a rehabilitation process is successful. To do so, it is necessary to agree which variables are to be measured, how they will be measured and standardise the “normal” levels at which these variables can be found.

Some physical variables are relatively easy to measure due to their “accessibility” and already standardised parameters (e.g. respiratory frequency (inspirations per minute), cardiac rhythm (beats per minute); leucocytes count in blood samples (compared to a standardised range in healthy subjects); body temperature (Celsius or Fahrenheit)).

The main constraint in the proper evaluation of neurological disability is the difficulty to access the relevant organs or their activity i.e. nerves, brain, etc. Imaging techniques, EEG and evoked potentials are currently available but the procedure is complex and time consuming and the knowledge limited to be carried out in a clinical environment. Furthermore in some cases, these techniques are not yet standardised for every type of impairment.

Thus, determining the appropriate variables to measure is of extreme importance to avoid any erroneous interpretation of the magnitude of the impairment as it can depend on confounding factors. Having this in mind, such variables should be within the definition of the impairment, in this case spasticity according to Lance’s definition (i.e. stretch reflex excitability).

“It is inadequate measurement, more than inadequate concept or hypothesis that has plagued researchers and prevented fuller explanation of the variances with which they are confounded.”

Phil Haueser, 1969

4.2 Spasticity measurement (state of the art)

A variety of techniques have been designed intending to evaluate spasticity. Due to their nature and characteristics they can be divided in the following categories:

- Clinical
- Neurophysiological
- Biomechanical

A description of these categories along with their advantages and disadvantages is detailed next.

4.2.1 Clinical Techniques

These techniques are used for routine assessment. They are relatively easy to apply, non-time consuming and do not need complex instrumentation and processing. Nevertheless they generally are subjective and lack standardisation and reliability. Furthermore, in some cases their readings or results are confounded by other features of the upper motor neurone syndrome.

4.2.1.1 Ashworth scales

These are the most commonly used techniques for spasticity measurement, erroneously used as the gold standard and the one used for comparison of new techniques (Fowler, Nwigwe, & Ho 2000; Harburn et al. 1995; Le et al. 2002; Lee et al. 2004). The original scale consists of 4 nominal levels which are assigned to the subjects by the examiners. Later on a modified version was proposed (Bohannon & Smith 1987) where an intermediate level between 1 and 2 was added with the aim to estimate the delay and magnitude of resistance felt during the movement (Damiano et al. 2002) (Table 1).

Table 1: Comparison between the Ashworth and Modified Ashworth scales, subjective nominal scales based on the description of the amount and type of resistance felt by the examiners when stretching a joint.

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

The technique consists in stretching the spastic joints by moving them passively through their range of movement and “feeling” the resistance to the passive movement. This is virtually applicable to every joint at the limbs.

The full methodology used when assessing spasticity at the elbow joint is as follows:

- The subject is comfortably seated and instructed to be as relaxed as possible so no voluntary activity will interfere with the assessment
- The examiner holds upper arm at the joint level whilst holding the wrist with his other hand
- Maintaining a horizontal position, the examiner flexes the elbow and extends it twice, first with low and then a high velocity
- The examiner then evaluates the resistance felt according to the scores in table 1

The theory behind these techniques is simple and easy to understand (their validity though is a matter of debate). The concept of the stretch reflex being hyperexcited and velocity dependent in the presence of spasticity leads to the assumption that the muscle contraction caused by the stretch reflex response will alone be responsible for the

opposition to passive movement or at the very least that this burst of muscle activity will change the mechanical properties of the muscle sufficiently to increase its elastic behaviour.

However there is in fact a misconception of cause and effect, in other words, what it is really being measured is not spasticity *per se*. This scale measures levels of resistance to passive movement (RTPM) which is also influenced by other confounding factors such as soft tissue changes (Johnson 2002) (Figure 1) and probably other positive components of the UMN syndrome. Furthermore, mechanical properties of the muscles are also velocity dependent (Singer et al. 2003) and tend to increase after a neurological injury. In figure 2 these soft tissue velocity dependent properties are observed compared with the non-affected side of a subject with post-stroke hemiplegia. No EMG activity was observed during this trial, yet higher resistance was found when the velocity of the stretch was increased.

Another important factor affecting this technique's validity, standardisation and reliability is its subjective nature. Pandyan and colleagues (1999) concluded from the data provided by two different studies from Bohannon and Smith (1987) and Bodin and Fisher (1991) that the MAS was reliable for classifying the resistance to passive movement at the elbow and wrists flexors. In 2001 Pandyan and colleagues described a system developed to instrument the Ashworth scales and measure the angular range of movement and force applied. In a further study using this technique they, Pandyan and colleagues (2003) have shown that the MAS does not provide a valid measurement of spasticity, particularly at lower grades (1, 1+ and 2) where there is an overlap between the grades. They conclude that the MAS is at the most, a valid measurement of resistance to passive movement (Pandyan et al. 2001; Pandyan et al. 2003). These results are supported by a study comparing clinical and laboratory measurements of spasticity (Vattanasilp & Ada 1999). Pandyan's technique will be described in more detail in the biomechanical measurements section. Nevertheless, regardless of this evidence, many studies still rely on the Ashworth scales to evaluate spasticity and corroborate the effectiveness of certain therapies (Ashworth, Satkunam, & Deforge 2004; Bohannon & Smith 1987; Brashear et al. 2004; Childers et al. 2004; Deltombe et al. 2004; Miscio et al. 2004; Stampacchia, Bradaschia, & Rossi 2004; Tsai et al. 2001; Turner 2003; Watkins et al. 2002; Yang et al. 2003).

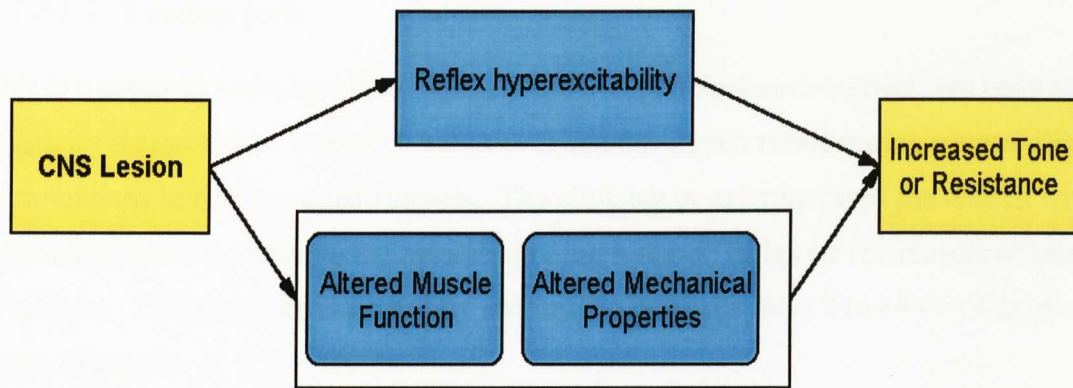


Figure 1: Diagram representing the different factors and its origins (neural and non-neural) contributing to increased tone or resistance to passive movement after a CNS lesion (Johnson, 2002).

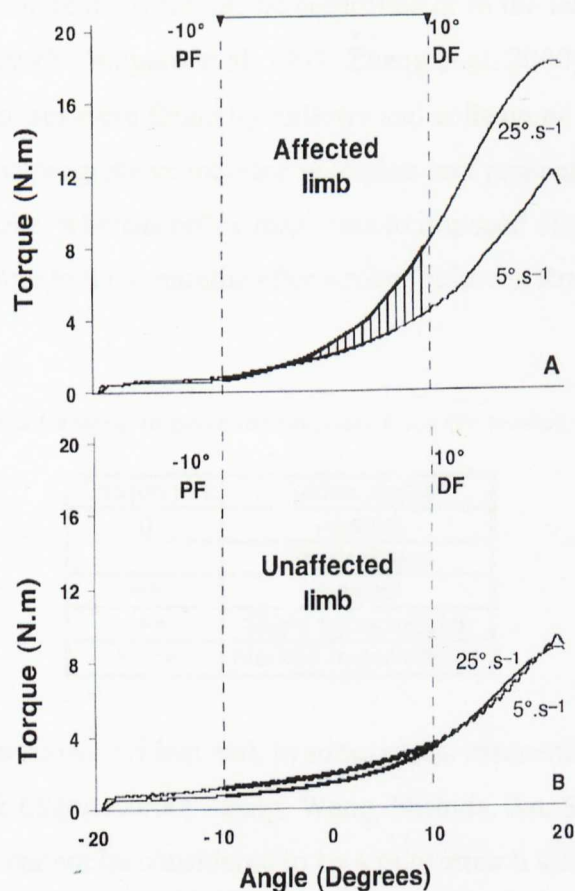


Figure 2: Torque Angle relationship during a passive movement of the affected (A) and unaffected (B) Ankles at two different stretch velocities. No visible EMG suggests that non-neural muscular changes due to neural impairment show a velocity dependent resistance to passive stretch. From (Singer, Dunne, Singer, & Allison 2003).

4.2.1.2 Tendon jerk

This is a common technique also routinely used in a clinical environment, not only as a spasticity measurement technique but to evaluate the stretch reflex during routine clinical examinations in non-impaired subjects. The clinician or examiner taps the tendon with a hammer to cause a stretch to the muscle eliciting a stretch reflex on the muscle of interest (Figure 3). This response is graded by scoring the response from 0 to ++++ (Ada et al. 1998) (Table 2).

The main flaws in this technique are the evident lack of standardisation to the strength of the tap, the material of the hammer, area of contact and exact place of tendon tap. Some researchers have used EMG recordings to measure the muscle response and an instrumented hammer where the force can be controlled or in the least of cases measured (Pagliaro & Zamparo 1999; Zamparo et al. 1997; Zhang et al. 2000). Further limitations of the tendon jerk responses were found by Fellows and colleagues (1993). Their findings showed that the progressive increase in tendon jerk responses occurred over the first year following stroke, whereas reflex responses to imposed displacement reached their peak excitability one to three months after stroke (Fellows, Ross, & Thilmann 1993).

Table 2 Grading to score the response from the tendon tap.

Tendon jerk	Reflex status
0	Absent
+	Hyporeflexia
++	Normal
+++	Slight hyperreflexia
++++	Marked hyperreflexia

Although this technique shows evident and, in some cases, measurable responses (Sherman, Koshland, & Laguna 2000; Zhang, Wang, Nishida, Xu, Sliwa, & Rymer 2000), the perturbation cannot be considered to be a pure stretch since the tap would affect nociceptive and other joint receptors.

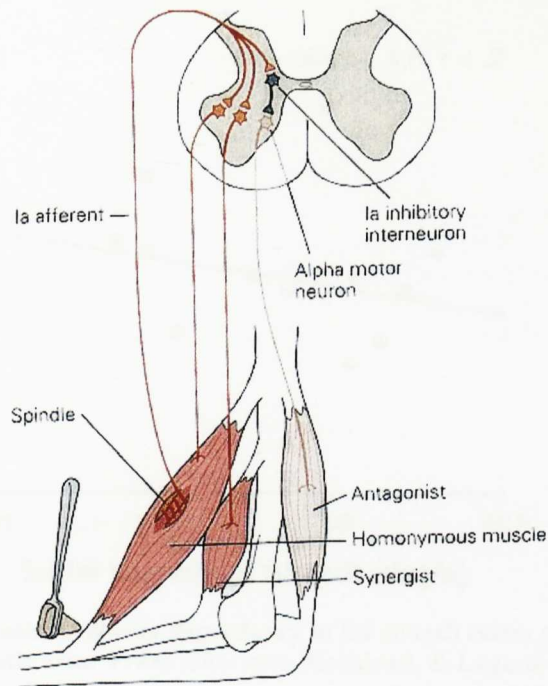


Figure 3 Schematic diagram of the tendon tap on the biceps. The percussion stretches the biceps activating the muscle spindle and eliciting a stretch reflex on the biceps while inhibiting triceps (Misiaszek et al. 2000).

Furthermore, a previous study (Sherman, Koshland, & Laguna 2000) describing a patient presenting hyperreflexia but with no evidence of having spasticity shows the dissociation between spasticity and hyperreflexia using biomechanical techniques and instrumented tendon taps. Figure 4 shows the data from this study demonstrating the lack of velocity dependency on the stretch reflex. However, the only parameter used here is the integrated area of the EMG activity during the stretch. More parameters determining the excitability of the stretch should have been obtained to evaluate the velocity dependency such as the latency and duration. This paper will be reviewed in full in the biomechanical techniques section.

It was also found in a study by Fellows (1993) that 28 patients with unilateral ischemic lesion in the area of the middle cerebral artery had an increase in tendon jerk response occurring over the first year following stroke, whereas reflex responses to imposed displacements reached their peak excitability one to three months after stroke with subsequent reduction in activity (Fellows, Ross, & Thilmann 1993).

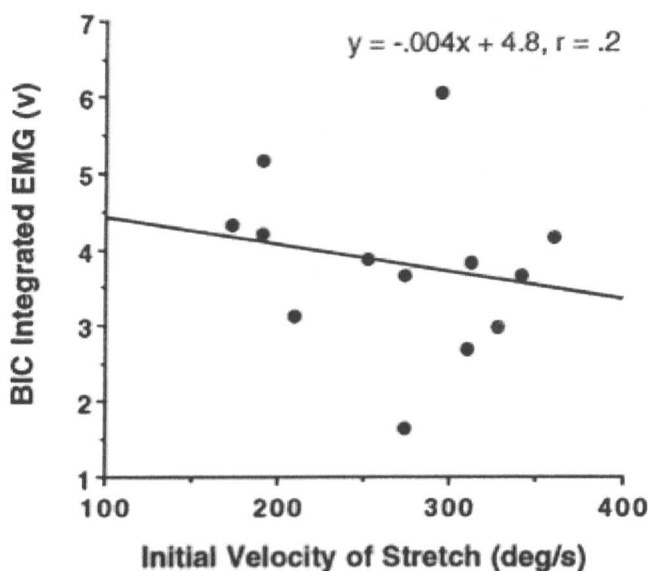


Figure 4 this data shows the lack of velocity dependency in the stretch reflex activity of the biceps EMG on the spastic arm. From (Sherman, Koshland, & Laguna 2000)).

4.2.1.3 Modified Tardieu Scale

This scale is used to evaluate the particular angle at which a joint stops after being subjected to a rapid stretch, defined as R_1 (stopping point). This assumes the stretch reflex to be responsible for this “stop” caused by its hyperexcitability stretch reflex.

This value is related and compared with the angle of maximum extension (i.e. resting angle) obtained by stretching the joint at a much slower rate, defined as R_2 . The relationship between R_1 and R_2 is more important than the individual measurements. A wide difference between R_1 and R_2 indicates the presence of a great dynamic component, while a small difference suggests the presence of a predominantly fixed muscle contracture.

The adapted method of the Tardieu scale was used in a study by Gracies and colleagues (2000) to evaluate spasticity with and without lycra splints on upper limb in hemiplegic patients (Gracies et al. 2000).

The measurement protocol adapted from Tardieu’s method by Held and Pierrot-Deselligny was described and used in a study by Gracies and colleagues in 2000. The grading is always performed at the same time of the day in a constant position of the body for a given limb. Other joints, particularly the neck, must also remain in a constant

position throughout the test and between tests. For each muscle group, reaction to stretch is rated at a specified stretch velocity with 2 parameters, X and Y.

Velocity of stretch:

V1: As slow as possible (minimizing stretch reflex).

V2: Speed of the limb segment falling under gravity.

V3: As fast as possible (faster than the rate of the natural drop of the limb segment under gravity).

V1 is only used to measure the passive range of motion (PROM) only V2 or V3 are used to rate spasticity.

Quality of muscle reaction (X):

0: No resistance throughout the course of the passive movement.

1: Slight resistance throughout the course of passive movement, with no clear catch at a precise angle.

2: Clear catch at a precise angle, interrupting the passive movement, followed by release.

3: Fatigable clonus (<10 seconds when maintaining pressure) occurring at a precise angle.

4: Infatigable clonus (>10 seconds when maintaining pressure) occurring at a precise angle.

Angle of muscle reaction (Y): measured relative to the position of the minimal stretch of the muscle (this angle corresponds to 0 degrees) for all joints except hip where it is relative to the resting anatomic position.

In a review of the treatment of spasticity using botulinum toxin, one of the techniques described to measure spasticity is the modified protocol above described and extends the description to the different joints of the lower limb (Calderon-Gonzalez & Calderon-Sepulveda 2002).

- Pelvic joint (supine position)

Hip: extensor muscles (with extended knee, a fast passive hip flexion movement); abductors (with hip and knee flexed, a fast passive abduction movement); external and internal rotator muscles (with knee flexed at 90 degrees, a fast passive internal or external rotation movement).

- Knee joint

Extensor muscles: With the hip flexed at 90 degrees the leg is then released under gravity released towards flexion

Flexor muscles: With the hip flexed a fast passive knee extension movement is performed by the examiner.

- Ankle joint:

Plantarflexor muscles: with the knee flexed 90 degrees or extended a fast ankle extension passive movement is performed by the examiner.

The muscle stretch angle measurement is from the minimum stretch position (zero) except for hip which is from the anatomical resting position.

The disadvantages of this technique are similar to those of the Ashworth scales. The way of determining the instant when the movement is stopped is qualitative and cannot be considered as an accurate method unless some kind of measurement device, such as a goniometer, is used. Also the confounding effect of the soft tissue changes is still an issue in this technique. EMG recordings are needed to corroborate the presence of the stretch reflex.

4.2.1.4 Assessment of Motor Function

Clinicians and physiotherapists are more concerned about the overall functional recovery after a neurological injury than just one or few components of the UMN syndrome. For this, functional tests are also commonly used in the clinical environment. However, in some cases, researchers have intended to assess spasticity using these techniques and correlate them to the Ashworth scores. This latter approach is not coherent with Lance's

extension to the definition (1990) where he states that spasticity alone does not affect voluntary movement. Furthermore, the sensitivity of these techniques is too low to distinguish the consequences of any deficit in performing the activity.

4.2.1.4.1 The Action Research Arm Test (ARAT)

This test uses a standardised table with dimensions of 92 cm x 45 cm x 83 cm high and with a shelf of 93 cm x 10 cm positioned 37 cm above the main surface of the table and four rods to place the alloy tubes (Figure 5). The test was designed to provide with information about functional recovery in stroke patients (Hsueh, Lee, & Hsieh 2002).



Figure 5: Standard ARAT table used for functional evaluation of motor control

It is designed for evaluation of both arms separately. It consists of 19 test items divided into four subscales, grasp, grip, pinch and gross movement). Items are arranged in a way that by accomplishing the most difficult item, it predicts success with all less difficult subscale items. On the other hand, failure with the easiest item predicts failure with the rest. This allows examiners to assess in a short period of time.

Examiners use a qualitative ordinal scale with four levels ranging from 0 to 3. Maximum scores of the total test, grasp subtest, grip subtest, pinch subtest and gross movement subsets are 57, 18, 12, 18, and 90 respectively.

The equipment required is a specially designed table and a chair, woodblocks, a cricket ball, a sharpening stone, two different sizes of alloy tubes, a washer and a bolt, two glasses, a marble and a 6 mm ball bearing.

Figure 6 shows a post-stroke volunteer during one task of the ARAT test while being assessed with a motion analysis system (Vicon).

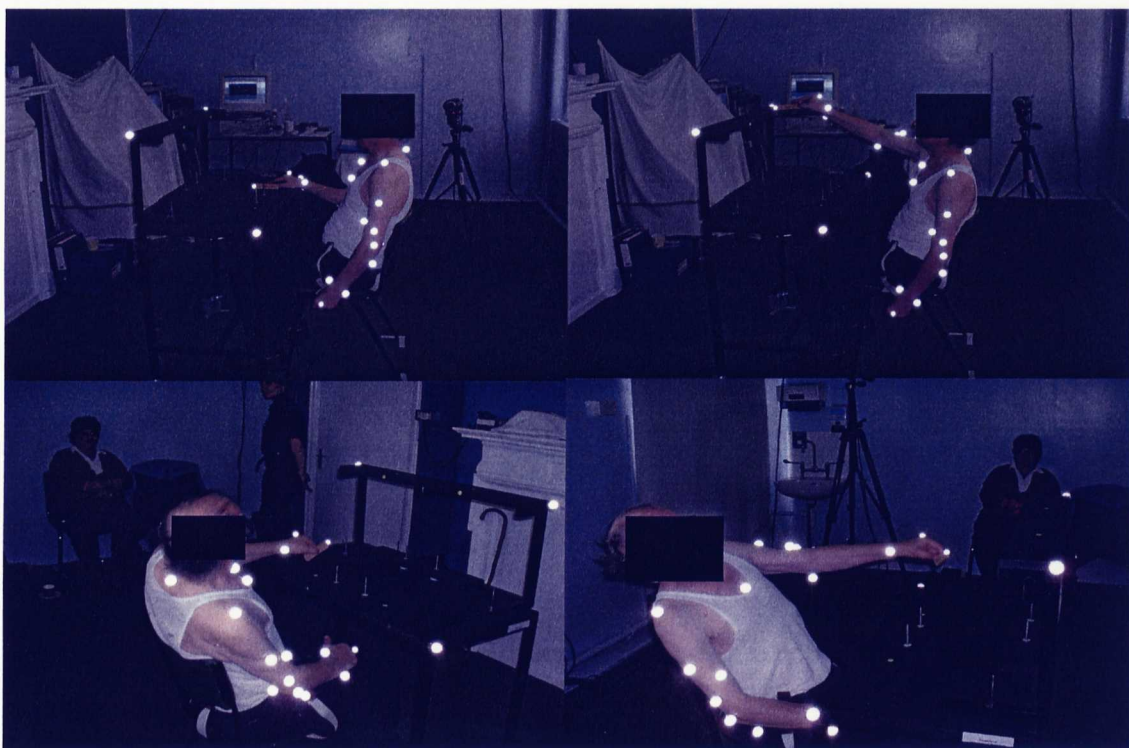


Figure 6: One post-stroke patient during one task of the ARAT with the non-impaired side (upper frames) and (b) the impaired side (lower frames).

This test is useful to assess the motor function of the patients but does not have a direct relationship to spasticity, at least according to the definition. Also, it completely depends on the criteria of the examiner. In an attempt to quantify this test, current research is being carried out using a motion analysis system (Vicon) to correlate the original scoring of the system to the parameters from the motion analysis system.

4.2.1.4.2 Fugl-Meyer scales

This technique assesses voluntary movement, reflex activity, grasp, and coordination (Lamontagne, Malouin, & Richards 2001; Wolf et al. 2001). Performance is measured

on 33 tasks with a 3-point ordinal scale (0 to 2), with a maximum score of 66). The tasks are subdivided in Reflex activity, Flexor synergy, extensor synergy, movements combining synergies, movements out of synergy.

In summary, clinical techniques are useful in the clinical environment as they are relatively easy to use, non-time consuming and no expensive or technical instrumentation is required. This is of particular importance is storage space and the staff technical training is a limitation. However in order to provide an accurate diagnostic of the problem affecting the patients and the future treatment to address their impairments and/or disabilities, it is necessary to consider the design of more objective techniques.

4.2.2 Neurophysiological tests

There are several standard electrophysiological tests for the measuring of spasticity (Katz RT 1994):

4.2.2.1 The Hoffman reflex and the M-wave

In 1918 Hoffmann observed that submaximal stimulation of the tibial nerve produced a delayed response in calf muscles (Braddom & Johnson 1974). It was later concluded that this response is a result of a monosynaptic reflex (Magladery JW & McDougal DB 1950).

These tests are relatively common as a measure of spasticity in a research environment. They consist in applying an electrical impulse to peripheral nerves innervating the muscle of interest and the resulting muscle contraction is recorded. These electrical impulses are transmitted orthodromic (towards the muscle) and antidromic (towards the spinal cord) (Figure 7A). This initiates two separated muscle contractions, the earliest one (M wave) is observed when the stimulus travels on the direction of the muscle, the second one of higher latency is transmitted to the spinal cord and then back to the muscle (H-reflex) (Figure 7B).

The conductivity of the stretch reflex pathways is assessed by observing the relationship between the amplitudes and the time delays with the intensity of the stimulus. The M-wave has a higher threshold for excitation, so it can be elicited only at higher stimulus intensities. At low stimulus intensities, there is no M wave and only a small H-reflex.

Increasing the stimulation level causes the H-reflex amplitude to increase and the M-wave to gradually appear and increase. At higher stimulation intensities the M-wave reaches maximum amplitude while the H-reflex fades away. This property is usually presented in a recruitment curve where the x-axis represents the stimulus intensity (Figure 7C).

The outcome measurements for this test are the latencies and amplitudes of the M-wave and the H-reflex and the H/M ratio of amplitudes (Matthews 1966). It is worth mentioning that in one of the earliest studies, Matthews (1966) did not find changes in the H/M ratio after intravenous injection of chlorproethazine or diazepam while clinically assessed spasticity, electrical response to stretches and ankle jerk were reduced or abolished, suggesting that this measurement may not be related to the clinical signs of spasticity. Nevertheless technological limitations of the time (1966) may have affected the resolution of the measurements.

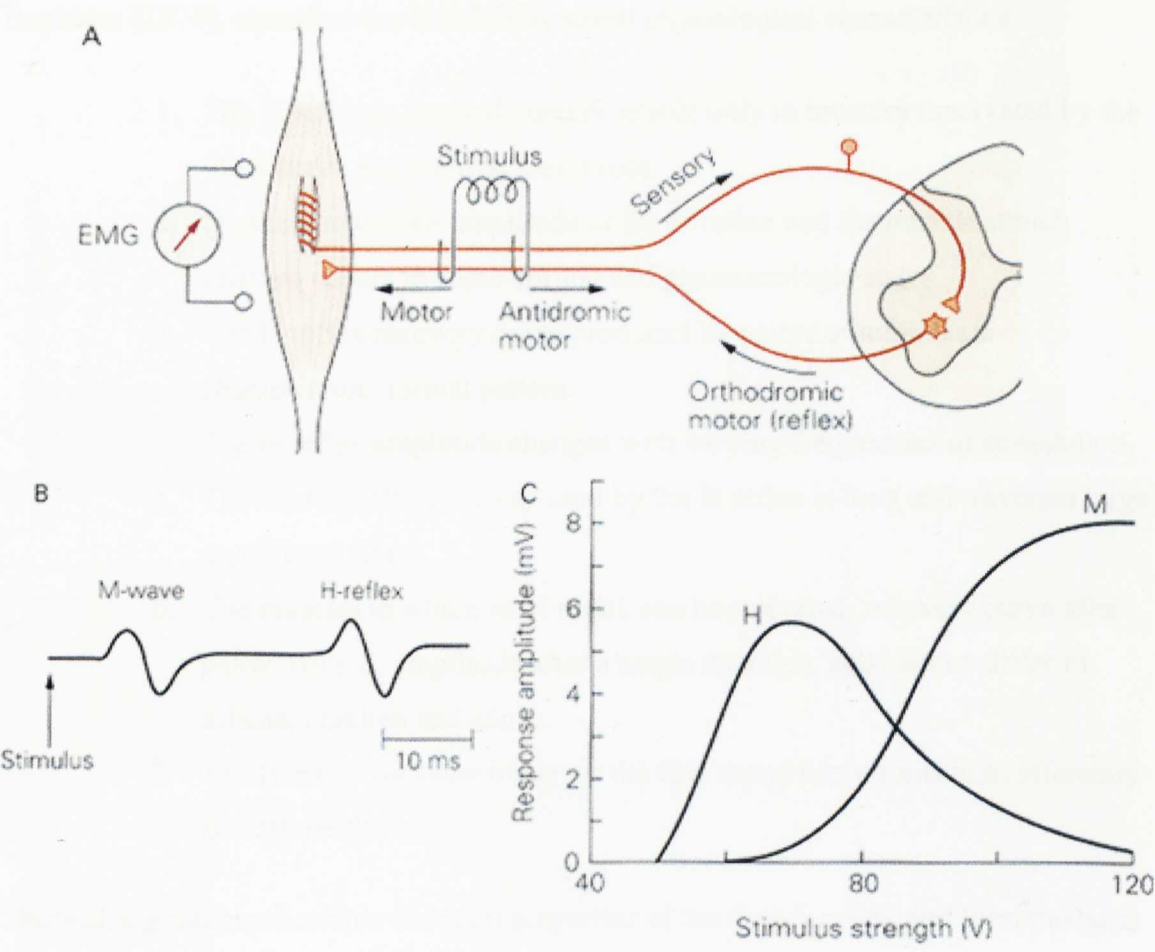


Figure 7: A) Diagram exemplifying the H-reflex pathway showing the antidromic and orthodromic direction. B) M-wave and H-reflex relationship in time, the M-wave has a shorter latency than the H-reflex. C) H-reflex and M-wave response amplitudes against stimulus strength or intensity (Misiaszek, de Serres, Stein, Jiang, & Pearson 2000)

The H-reflex is considered to be analogous to the stretch reflex since the pathways are the same. This statement is however simplistic and overlooks the contribution of the physiological sensors (muscle spindle) to the stretch reflex since it is not a direct response of muscle to stimulation of its corresponding motor nerve, but a reflex similar to (but not the same as) a muscle stretch reflex. The H reflex is usually (but not exclusively) elicited by delivering a submaximal stimulus to the nerve in and recording over the muscle. The generated nerve action potential propagates up to the spinal cord and then, via a predominantly monosynaptic reflex arc, passes down the efferent motor axon. The H reflex is unlike the muscle stretch reflex in that (1) the muscle spindle is bypassed, and (2) the afferent volley is temporally less dispersed, and the tendon jerk involves fewer Ib fibres.

Braddom (1974), classified the H reflex by seven physiological characteristics:

1. The H reflex is normally demonstrable only in muscles innervated by the tibial nerve and the first sacral root.
2. Dissociation of the amplitude of the H reflex and the muscle stretch reflexes occurs in some normal and pharmacologic states.
3. The H reflex recovery curve produced by paired stimuli has a characteristic normal pattern.
4. The H reflex amplitude changes with varying frequencies of stimulation.
5. The neural reflex pathway used by the H reflex is long and traverses large myelinated fibres.
6. The muscles in which an H reflex can be activated, recovery curve after paired stimuli, amplitude after a single stimulus, and latency differ in infants, children and adults.
7. The H reflex pathway traverses the first sacral nerve roots both afferently and efferently.

These characteristics describe the main properties of the stretch reflex and form the basis of what today is the state of the art. However this description is limited to the lower limb as this was the place where H-reflexes were visible with the methodology and equipment available at the time (Braddom & Johnson 1974).

This review also states the most practical and diagnostically helpful clinical uses of the H reflex:

1. An H reflex in the anterior tibial muscles (except in infants) supports the diagnosis of a central nervous system lesion from the mid brainstem down.
2. The H reflex latency is longer or the H reflex may be absent in cases of proximal neuropathy.
3. The H reflex latency is delayed or the H reflex may be absent in cases of first sacral root compromise.

4. An abnormal H reflex recovery curve after paired stimuli and the presence of an H reflex in hand intrinsic after one year of age supports the diagnosis of the central nervous system immaturity

A typical setup for the measurement of the H-reflex is shown in figure 8.

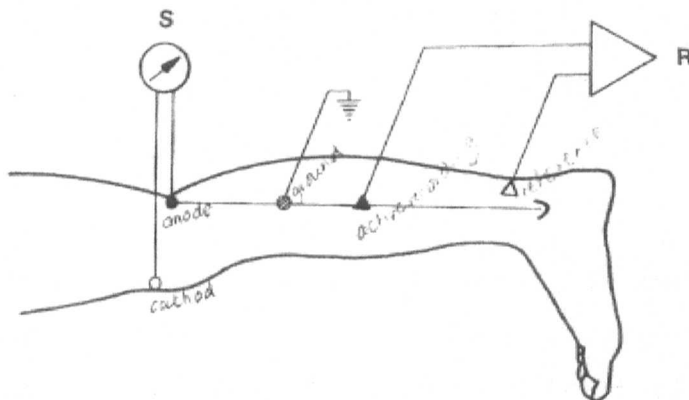


Figure 8: H-reflex typical setup. S represents the stimulating electrodes and R represents recording electrodes (Jankus, Robinson, & Little 1994).

Jankus and colleagues (1994) carried out a study to register the normal limits of side to side tibial H-reflex amplitude variability on 47 healthy volunteers between 21 and 67 years old (35 ± 10 years) with no history of peripheral neuropathy. Two volunteers were excluded because they did not meet an arbitrary minimum side-to-side latency difference of 1.5 ms, with no explanation for this restriction, and a 67 years old woman who had absent reflexes bilaterally. The results of this study are shown in table 3. In this particular study the stimulation duration was 1 ms with a rate of 0.2 Hz. They found that the latency in both sides was of 29.6 ± 2.5 ms, the amplitude ratio of 0.74 ± 0.17 and the side to side difference of 0.45 ± 0.4 ms (Jankus, Robinson, & Little 1994).

Table 3: Mean and standard deviation values for amplitude ratio, absolute amplitude, absolute latency and side-to-side latency difference (Jankus, Robinson, & Little 1994).

	n	Mean± SD
Amplitude ratio		
all subjects	45	0.74± 0.17
<40 years	30	0.70± 0.17
≥40 years	15	0.81± 0.15
Absolute amplitude		
Ramp (mV)	45	8.6± 4.0
Lamp (mV)	45	9.0± 4.6
Absolute latency		
Rlat (ms)	45	29.6± 2.5
Llat (ms)	45	29.6± 2.5
Side to side latency difference (ms)	45	0.45± 0.40

In a similar study in 1999 by Bodofsky, H-reflexes were tested bilaterally in upper extremity muscles in 23 healthy volunteers (4 men, 19 women) aged 19-42 years old. In this case the reflexes were tested during an isometric contraction. His results showed that the mean latencies varied from 9.5 to 27 ms. These latencies were correlated to the arm length and it was found to be of 0.64 ms for the abductor pollicis brevis and 0.7 ms for the abductor digiti minimi. Side to side variation ranged from 1.5 to 2.7 ms. H reflex latencies were predicted given by arm length and were consistent with the mean measured latencies (Bodofsky 1999).

The H-reflex has also been measured on the impaired side and the non-impaired side in people with hemiparesis to obtain the side-to-side amplitude and latency variability as a measure of spasticity (Higashi et al. 2001;Marque et al. 2001;Okuma, Mizuno, & Lee 2002). This measurement however is not advisable as the side regarded as “non-impaired” is also affected by ipsilateral pathways from the affected side thus not comparing the affected side with a “normal” value.

In a different study, Panizza and colleagues (1995) compared the H-reflex recovery curve in the upper limb of a group of 33 patients with different degrees of spasticity secondary to stroke with 25 controls. An increase of the late facilitation part of the H-reflex recovery curve was found. The authors claim that these abnormalities appeared to be related to increased muscle tone but the correlation showed was low ($r^2=0.43$) (Panizza et al. 1995).

More recently Aymard and colleagues (2000) carried out a comparison study of presynaptic inhibition and homosynaptic depression between lower and upper limbs in “normal” human subjects and patients with hemiplegia. H-reflexes were elicited in the wrist and finger flexors and the soleus muscle obtaining the H/M ratio and the amplitude of the H-reflex. Presynaptic inhibition of Ia terminals with primary afferent depolarisation (PAD) was evoked by H-reflexes applied to the nerve supplying antagonistic muscles of the soleus and the flexor carpi radialis and homosynaptic (post activation) depression was explored by varying the time interval between two consecutive H reflexes. The resulting reflex depression was assessed based on its dependency on the excitability of the PAD interneurons: the larger the excitability, the larger the presynaptic inhibition and thus the reflex depression (Aymard et al. 2000). Their results showed no right-left asymmetry in presynaptic Ia inhibition, homosynaptic depression or the H/M ratio. In the hemiplegic side of patients with lesions in the middle cerebral artery, the H/M ratio significantly increased in the soleus but not in the flexor carpi radialis. Presynaptic inhibition was significantly reduced on the hemiplegic side but was unchanged at the lumbar level. Homosynaptic depression was reduced at cervical and lumbar levels on the hemiplegic side but not modified on the affected side. The decrease in post activation depression is likely to contribute to the exaggeration of the stretch reflex characterising spasticity and it might be a consequence of the changes in the pattern of activation of Ia afferents and MN following stroke.

In a series of studies by Brooke and colleagues (1992, 1995 and 2000) it has been seen that the magnitude of the H-reflexes can be significantly modulated by active and/or passive movement. The results from a study of somatosensory evoked potentials and H-reflexes on six volunteer subjects show that these responses are significantly attenuated in the flexor carpi radialis by either active or passive movement at the elbow and wrist (Brooke JD et al. 2000). Similar results were observed in the lower limb when pedalling (De et al. 1992), passive “locomotor like” movements (Brooke et al. 1995a; Brooke et al. 1995b).

Another group studied the H-reflex modulation during voluntary and automatic movements in ten subjects with upper motor neurone damage and twelve non-impaired subjects (Leonard et al. 1998). This study showed soleus H-reflexes inhibition following postural perturbations in the non-impaired group. There was no evidence of inhibition

during either voluntary movements or automatic postural perturbations in the subjects with upper motor neurone syndrome. These results seem to be congruent with those of Kasai and Komiyama (1996) where it was observed that voluntary arm movements caused H-reflex depression in the soleus muscle of non-impaired subjects (Kasai & Komiyama 1996).

4.2.2.2 The F-wave

The F-wave is similar to the H reflex in that it reflects proximal conduction of the peripheral nervous system. It is recorded by supramaximal stimulation of a mixed nerve while recording over a distal muscle innervated by that nerve. It is however different in the way that it is used to demonstrate changes in motor-neurone excitability (Dressnandt, Auer, & Conrad 1995; Rosche et al. 1996). F-waves are low amplitude motor responses (0.2-0.5 mV) produced by backfiring of motor neurones in the anterior horn of the spinal cord (Fisher 1995) (Figure 9).

In a study by Bischoff and colleagues F-wave responses of the posterior tibial nerve were studied in 22 patients with spasticity and 18 normal control subjects. Mean amplitude and mean duration were significantly longer in patients with spasticity than in healthy controls (Bischoff, Schoenle, & Conrad 1992). The results from this study correspond with the clinical definition of spasticity.

Another study by Joodaki (2001) observed H reflexes and F-waves of the soleus muscle in 10 non-impaired subjects and three spastic hemiplegic patients. It was found that the mean peak to peak amplitude of H-reflexes and F-waves, H/M ratios and F/M ratios were significantly decreased after application of transcutaneous electrical nerve stimulation (TENS) in both groups whereas the mean latencies were increased suggesting a reduced motor neuron threshold after application of TENS (Joodaki, Olyaei, & Bagheri 2001).

4.2.2.3 Other electrophysiological techniques

The following techniques can be potentially used to measure spasticity indirectly or its consequences and origin. Although they are not conclusive yet, even in non-impaired subjects, the limited understanding of the Central Nervous System calls for more research at the neurological level as well as for the development of mathematical models.

4.2.2.3.1 Tonic Vibration Reflex

The Tonic Vibration Reflex (TVR) is a technique where a sustained sinusoidal vibration applied to the tendon of human limbs elicits progressive tonic reflex activity (Takata, Nakajima, & Yamada 1996). This technique has been used to assess the status of presynaptic inhibition which may act by limiting the magnitude of calcium current moving into primary afferent terminals, limiting neurotransmitter release (Katz RT 1994).

Tonic vibration stimulates Ia and group II afferent nerve fibres. It exerts inhibitory characteristics by way of an interneurone. Normally, it suppresses the H reflex. The failure of tonic vibration to suppress the H reflex in spastic patients has been cited as evidence for the loss of presynaptic inhibition (Katz RT 1994).

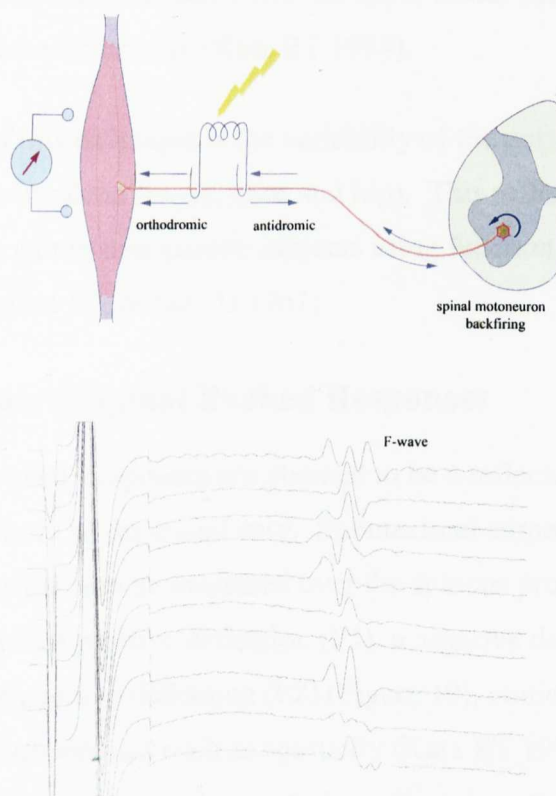


Figure 9: Diagram representing the F-wave pathway (upper diagram). Examples of F-wave recordings, the duration of the wave is representative of the motor-neurone excitability (lower figure).

In a study by Abbruzzese and colleagues in 1982 it was observed that the tonic vibration reflex amplitude was reduced in “cerebellar” patients, particularly in cases with unilateral

hemispheric lesion (stroke). The reflexes were absent or very weak in patients with spinal cord injuries (Abbruzzese et al. 1982).

However, results are very poorly correlated and there is a wide dispersion of values among patients, diminishing its reliability (Katz RT 1994).

4.2.2.3.2 Flexor withdrawal response

The flexor withdrawal response is characterised by ankle dorsiflexion and knee and hip flexion due to a stimulus given in the peroneal nerve.

Latency, amplitude and duration of electromyographic recordings of the automatic withdrawal response of the lower extremity upon electrical stimulation supposedly reflect global interneural activities. EMG recording from tibialis anterior show a low-threshold early response (50-60 ms) that disappears with an upper motor neuron lesion and a later (110-400 ms) high threshold response (Katz RT 1994).

A particular problem of this technique is the variability of the polysynaptic response due to the number of inputs involved (angle, knee and hip). This reflex is used to correct the characteristic foot drop gait in hemiparetic subjects using functional electrical stimulation (Lieberson WT, Holmquest HJ, & Scot D 1961).

4.2.2.3.3 Lumbosacral Spinal Evoked Responses

Lumbosacral Spinal Evoked Responses are claimed to be a reflection of presynaptic inhibition in the dorsal horn of the spinal cord. Submaximal stimulation of the tibial nerve evokes a response that can be measured over the spinous process of T-12. This response has three peaks: an positive deflection (P1), a negative deflection (S) and a second larger amplitude positive deflection (P2) (Figure 10), clinically, the ratio of P2/S is used for neurological assessment such as spasticity (Katz RT 1994). However this technique is difficult to standardise and to apply in a clinical routine environment.

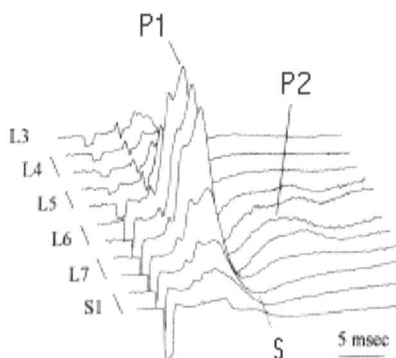


Figure 10: Example of a Lumbosacral spinal evoked response (Adapted from Noga et al, 1995)

4.2.2.3.4 Central Motor Conduction Times

It has been seen that conduction velocities within the central nervous pathways or Central Motor Conduction Times (CMCT) are affected in stroke patients (Heald et al. 1993a); (Heald et al. 1993b). The responses to electromagnetic stimulation of the motor cortex and cervical motor roots were recorded bilaterally in surface EMG.

In this study, the CMCT were obtained by first stimulating the motor cortex and measuring the delay in the EMG response in a particular muscle group, second stimulating the cervical motor roots and measuring the delay in the EMG response in the same muscle group. Subtraction of these two values would give the actual CMCT (Table 4). However, in this study, the assumption that peripheral nerves are intact is made and only final values are reported. Raw data is considered to give more information of the subjects' actual condition

Three different groups were found in the first CMCT assessment within the 72 hours after stroke: those with absent responses, those with delayed responses and those with normal responses. During the first 12 months following stroke some changes occurred: CMCT may remain unchanged, delayed CMCT may return to normal and previously absent responses may reappear and be delayed or normal. Threshold for motor evoked responses were initially high and fell over 12 months.

This study shows a difference within the function of the cerebral pathways following stroke and suggests a way to assess the evolution of patients with Upper Motor Neurone Syndrome.

Table 4: results from the CMCT in ms between non-impaired volunteers and the affected and unaffected sides of the post-stroke volunteers

CMCT (ms)					
Muscles	Normal subjects	Hemiparetic patient-Paretic side		Hemiparetic patient-Non-paretic side	
		within 72 hrs	12 months after	within 72 hrs	12 months after
B brachii	5.1+-0.8	5.0+-1.3	6.2+-2.4	4.9+-0.9	5.1+-0.7
T brachii	5.3+-0.7	6.1+-2.0	6.9+-3.0	5.3+-1.3	5.3+-1.2

The major drawback of these techniques is the lack of standardisation in the equipment used. They are mainly invasive and time consuming, causing discomfort and pain in the patients affecting the measurement by stimulating nociceptive receptors and the complex procedure required to properly stimulating the nerves.

4.2.3 Biomechanical techniques

These techniques are a result from the attempt of objectifying the clinical tests and provide with devices and techniques that can be reliable, repeatable and valid with virtually no variation between different locations. The disadvantages of these methods are, if they are used alone, the same of those of the clinical scales, i.e. confounding factors such as biomechanical muscle changes and soft tissue properties.

Biomechanical techniques can be subdivided in the way movement is produced (Price 1990) as follows:

4.2.3.1 Manual Techniques

These techniques arose as an attempt to objectify the clinical scales. Relatively simple instrumentation is required to adapt the tests to record physical variables such as angular displacement, force/torque, acceleration and EMG activity.

Neilson and McCaughey (1981) developed a technique to record stretch reflex responses from biceps brachii in non-impaired and cerebral palsied subjects. Each subject was instructed to maintain a constant average contraction of the biceps at 10% or 20% of maximum voluntary contraction (Neilson & McCaughey 1981). A goniometer was used to record the angle and EMG signals from the biceps displaying the result on an oscilloscope and recorded at 20 Hz. EMG signals were full-wave rectified and low-pass filtered at 20 Hz. The examiner applied manually at five constant amplitudes (1.67, 2.5, 5, 7.5 and 10 degrees peak to peak) and frequency sinusoidal perturbation (4 Hz.) to the forearm by moving the elbow angle about the ninety degree position (Figure 11). Their findings show that the tonic stretch reflex gain (amplitude) increases with the average contraction level and decreases with the magnitude of the stretch in both non-impaired and cerebral palsied subjects (Figure 12). No significant differences were found between both populations. This at the time was explained by assuming that the linear range of the stretch is exceeded. Nevertheless, no reference is made to the possible contribution of the Golgi tendon organ that could be responsible for the decreased gain at higher magnitudes of stretch. Also an important point in the discussion is the difficulty of attempting to measure muscle tone during voluntary activity. Furthermore, subjects may

anticipate the perturbations and change the feed forward mechanisms of the reflex excitability over time.

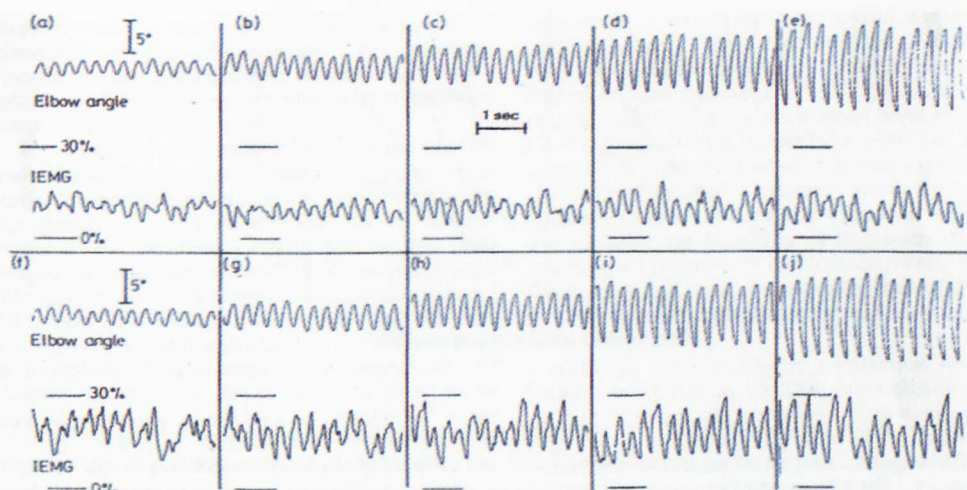


Figure 11: Angular displacements and the integrated EMG responses from a cerebral palsied volunteer voluntarily sustaining a contraction of 10% (upper diagram) and 20% (lower diagram) of maximum voluntary activity. Oscillations were held at 4Hz at five different amplitudes (1.67 (a and f), 2.5 (b and g), 5 (c and h), 7.5 (d and i) and 10 (e and j) degrees peak to peak) (Neilson & McCaughey 1981).

This technique in particular is outdated and much better technology is now available for EMG analysis and controlled displacement perturbations. However it provides a basis for current and future studies. The main concern about this technique is the restrictive inclusion criteria for spastic volunteers as not every person who presents spasticity is able to maintain constant voluntary muscle activity.

In a different study, Marchese and colleagues (2001) designed a manually controlled method for the measurement of spasticity at the elbow joint. The measurement protocol describes the subjects lying or in a sitting position while the arm was supported by the device. The examiner used a handle to move the patient's arm in an oscillatory motion following the rhythm of a metronome at three different speeds (45, 90 and 120 degrees/s). The outcome measurements included angular position, torque and EMG signals using a sample frequency of 25 samples per second. However EMG recordings were not used to assess the reflex activity but to assess the muscle activity before and during the trial, hence no results from the EMG were presented (Marchese et al. 2001).

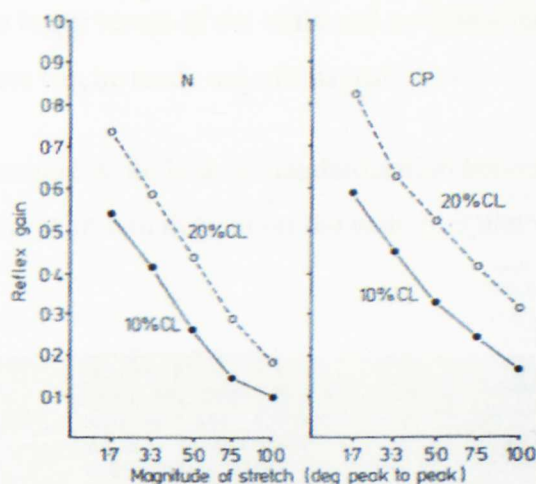


Figure 12: Profiles of the mean Tonic stretch reflex gains across the five normal subjects (left side) and five cerebral palsied subjects (right side) for the five different peak to peak magnitudes of sinusoidal stretching at the two sustained voluntary contraction levels (10% and 20% of maximum voluntary contraction). Changes of gain contraction level and magnitude of stretch were significant for both subject groups ($p < 0.001$) (Neilson & McCaughey 1981).

In their work, Marchese and colleagues introduce a polynomial fit to quantify the contribution of individual muscle considering their moment arm in relation to the elbow angle. This work is mostly directed at the measurement of resistance to passive movement and does not convey with the clinical definition proposed by Lance.

Generally speaking, this technique has potential to be used to measure spasticity if some modifications could be used such as increasing the sampling frequency and relating the outcome measurements to the muscle activation.

Pandyan and colleagues (2001) developed a device capable of measuring resistance to passive movement and angular changes at the elbow joint and EMG activity of elbow flexors and extensors. Their protocol allowed having the Ashworth scores and the recordings of the angular displacement and the necessary force to achieve such displacement along with the EMG activity of the elbow flexors and extensors simultaneously. The therapist was blinded to the biomechanical parameters and the Ashworth scales were not revealed to the person analysing the data (Pandyan et al. 2001).

The device consists of a load cell where the examiner applies a force to stretch the elbow flexors while the elbow angle is measured by a flexible goniometer (Figure 13). They concluded that the MAS was useful as a measurement of resistance to passive movement, but its usefulness as a measure of spasticity was not sufficient as no difference can be

distinguished between the lower levels of the scale and no dissociation between neural and non-neural components can be made out of this test.

The drawback of this technique is the lack of standardisation between examiners. The required force to stretch the joint will depend on the examiner and variations within trials are expected.



Figure 13: Instrumented Ashworth score measuring Force Vs angle to evaluate resistance to passive movement.

Kong and Chua (2002) developed a manual method using a standard goniometer to measure passive range of movement to be used in addition to the MAS. In this case the joint of interest was the proximal interphalangeal joint of the 2nd and 5th digits. They compared the differences between before and after intramuscular neurolysis with alcohol into the fingers flexors in stroke patients with severe spasticity. This technique however cannot be considered to be a measurement of spasticity per se as only passive ROM is measured (Kong & Chua 2002).

A single-case study was mentioned previously where a patient presented hyperreflexia but not spasticity (Sherman, Koshland, & Laguna 2000)). A range of different manual methods were used, including the tendon tap, using an instrumented hammer with an in-built load cell to measure the force of the tap and EMG recordings to test hyperreflexia, and the measurement of EMG responses to manual rapid extension of the elbow joint from rest to 90 degrees flexion to elicit stretch reflexes. This technique was used on a single case study where a patient presented with a unilateral infarct to the medullary pyramid.

In this case, the tendon taps were assessed using EMG recordings from the biceps after tapping its tendon. A one to one stimulus-response relationship was observed (Figure 14).

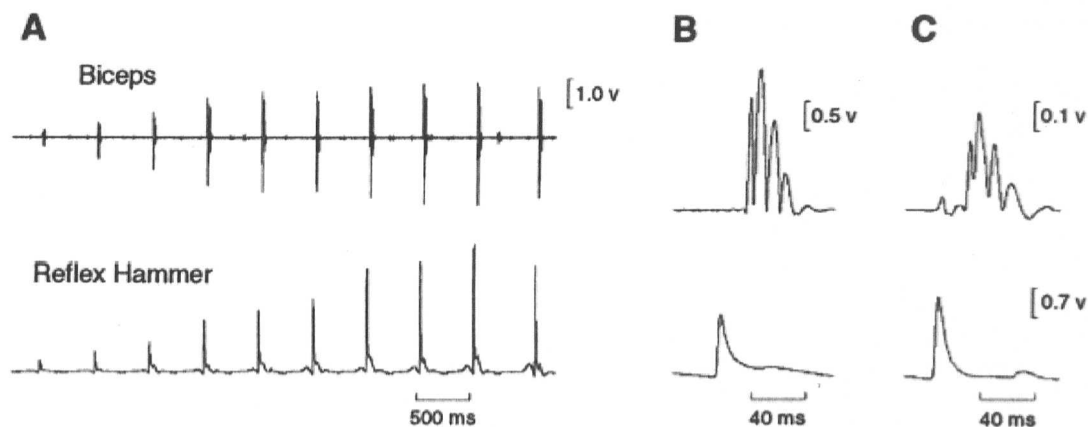


Figure 14: A) Recordings of a set of 10 tendon taps with gradually increasing the tap intensity on the involved arm. Latencies of 24-26 ms were found between the tendon tap and the EMG burst. No differences in this value were found between the involved and the uninvolved arm. Tendon taps and their respective EMG bursts are shown for the (B) involved and (C) uninvolved arms (Sherman, Koshland, & Laguna 2000).

Figure 15 shows the EMG signals obtained from the elbow stretches from the involved and uninvolved arm at different velocities of stretch. Although the authors claim that there was no evident difference between the uninvolved (Figure 15A) and involved (Figure 15B) arm, the stretch velocities recorded for the involved arm are lower than for the uninvolved arm. This suggests that the examiner used lower force to move the involved arm or that due to soft tissue changes this arm had more resistance to passive movement. In any case the stretch reflex amplitudes are lower in the non-impaired arm.

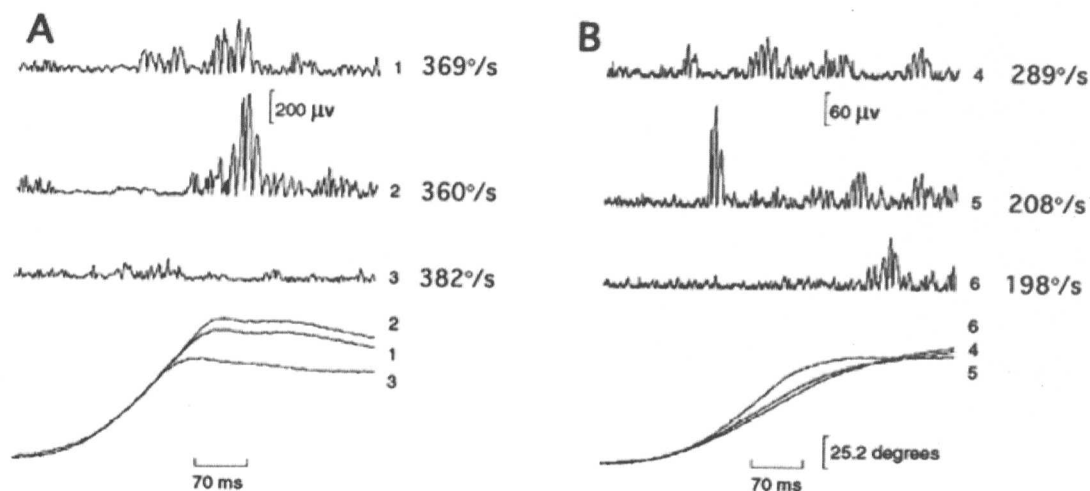


Figure 15: Comparison between the recordings from the involved (B) and uninvolved (A) arm. Lower stretch reflex amplitudes have been observed in the involved arm, although also lower velocities were used (Sherman, Koshland, & Laguna 2000).

Nevertheless, the main concern with this type of comparison is that there is no information about the resolution of the tests (i.e. intensity of stretch). In both cases a stretch reflex was elicited, however, the mechanical perturbations applied to the muscle spindle and Golgi tendon organ may stimulate them differently from a passive stretch. A scatter plot of the normalised EMG against the force of the tendon tap from the affected and unaffected arm is shown in figure 16 and the amplitude of the stretch reflex against the velocity of the stretch showing no stretch reflex dependency. However, when showing the regression line, values higher than 120 units were arbitrarily discarded increasing the correlation factor.

These results showed that there was dissociation between hyperreflexia and spasticity as it is understood clinically. However this work fails to compare results using the same experimental protocol in people with clinically diagnosed spasticity and/or non-impaired subjects with no signs of spasticity and/or tendon hyperreflexia. This information would be very important to evaluate the actual differences in stretch reflex amplitudes between people that have been diagnosed with spasticity and non-impaired people. Torque or force measurements against the angle (stiffness) correlated to the EMG amplitude and latencies are necessary to estimate the contribution of soft tissue and neural components to the resistance to passive movement.

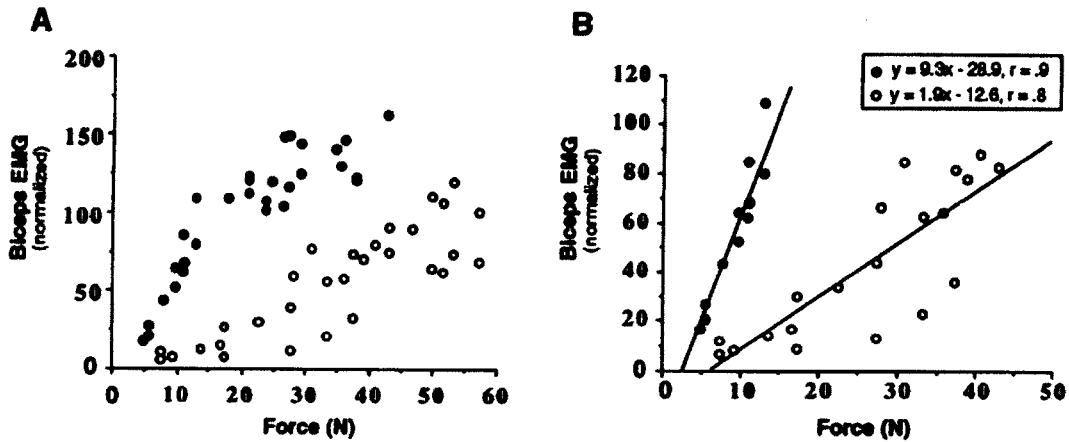


Figure 16: Relationship between the normalised Biceps EMG and the tendon tap intensity (N) are shown. Open circles represent the data from the uninvolved arm and closed circles represent the data from the involved arm. A) All data points from the three sets of ten measurements are shown and (B) the regression lines for the data in the linear range with regression lines fitted to the data (Sherman, Koshland, & Laguna 2000).

The evidence from this study suggests three not mutually exclusive explanations:

- 1) The contribution of the stretch reflex to resistance to passive movements may not be significant.
- 2) Resistance to passive movement with increased muscle activity may have cortical influences or,
- 3) Resistance to passive movement depends mainly on the soft-tissue changes.

Discussion

Manual methods provide easily applicable techniques for a routine clinical assessment. However they are still subject to inter and intra-rater variability as it is difficult for one person to repeat the same type of movement or perturbation every time and for different subjects and different examiners. An alternative to these types of measurements is to gather sufficient enough data to increase the level of analysis and in this way obtain a better estimation of the differences between impaired and non-impaired people, as well as the progress of any therapy involved.

4.2.3.2 Based on the force of gravity (Pendulum Test)

The pendulum test to assess spasticity was first proposed in 1951 by Wartenberg (Vodovnik, Bowman, & Bajd 1984) using gravity as a force generator and observing carefully the movement of the lower limb. This test is easy to apply since the instrumentation is limited to record the movement and velocity and does not need any external device to produce movement. Vodovnik and colleagues (1984) proposed to instrument this test recording the angular position with a goniometer.



Figure 17: Experimental setup for the pendulum test of the lower limb. The examiner holds the leg in a horizontal position and releases letting the lower leg swing freely. (Salazar-Torres and Mayagoitia, unpublished figure).

The methodology for the pendulum test is as follows: the subject is sitting, or lying in a semi-supine (Bajd T & Vodovnik L 1984) or supine (Vodovnik, Bowman, & Bajd 1984) position with both knees hanging free from the edge, the examiner lifts the leg to be tested to a horizontal position (Figure 17). The limb is allowed to fall freely while recording knee angle with an electrogoniometer (Bajd T & Vodovnik L 1984). Figure 18 shows a typical goniogram of a non-impaired subject and the parameters used to evaluate spasticity.

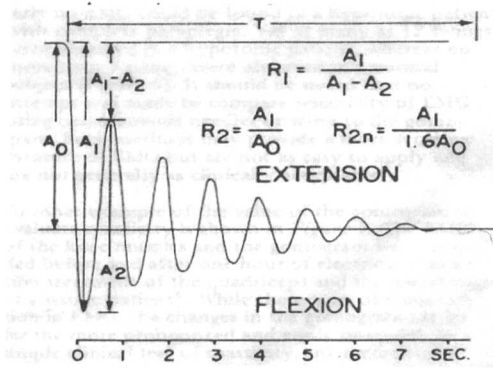


Figure 18: typical goniogram of the pendulum test and its associated parameters to diagnose spasticity. (Bajd and Vodovnik, 1984).

Bajd and Vodovnik proposed a group of parameters that can be measured from the goniometer (Table 5). These parameters are explained as follows:

The relaxation index (R_{2n}) corresponds to the angle at which the spasticity stops the natural backward swing. This parameter is normalised by the difference in angles between the resting and starting position (A_0) to eliminate influence of different resting angles from different patients or the same patient at different testing days. In normal subjects the ratio is around 1.6. This parameter was further normalised by this value ranging from zero where no motion is recorded to a one where a normal swing is recorded and therefore no spasticity is observed.

The second parameter is determined by the counting of the maximums of the goniogram after the release of the lower limb. In normal subjects this number is about 6 or 7 oscillations.

The third parameter is the area between the goniogram and resting angle prior to the first crossing over the resting angle. In Bajd and Vodovnik's study (1984), this parameter is expressed in cm^2 when performing the measurements at sensitivity of 5 deg mm^{-1} and a chart speed of 25 mm s^{-1} . These specifications are no longer valid with the available technology. Therefore this area should be expressed in degrees (or radians) *time units.

The fourth parameter is defined by the first maximum of the goniogram. Bajd and Vodovnik (1984) suggest that this value provides evidence of how strongly the spasticity pushes back the limb towards the starting angle. The values for healthy subjects range from 20 to 35 degrees.

The fifth parameter was defined as a relaxation index from a starting angle between the full extension and the resting angle.

The sixth parameter is the averaged value of ten relaxation indices from 10 consecutive swings.

The seventh and eighth parameters are obtained from the maximal velocity of the first backward and first forward swings respectively. The parameters in healthy subjects range from 11 to 10 rad/s for the seventh parameter and from 9 to 12 rad/s for the eight.

Table 5: Pendulum test parameters as described by Bajd and colleagues

Parameter	Description
p1	Relaxation index
p2	Number of swings
p3	Ara between goniogram and resting angle
p4	First maximum of the goniogram
p5	Relaxation index at the half swing
p6	Average relaxation index of ten successive swings
p7	First maximum of the tachogram
p8	First minimum of the tachogram

In their study, Vodovnik and colleagues (1984) studied in ten non-impaired subjects, ten spinal cord injured patients and ten hemiparetic patients. EMG signals of quadriceps and hamstrings were recorded.

Non-impaired subjects did not show any EMG activity either sitting or supine position. The goniograms were repeatable within experimental error.

Spinal cord injured patients showed goniograms dependent on several factors and were irregular in general. In some cases their goniometers will show lower tonus with 11-12 swings whilst in non-impaired is about 5-7 swings (Bajd T & Vodovnik L 1984;Vodovnik, Bowman, & Hufford 1984). EMG activity had more variability and in some cases the test provoked a massive spasm observed by a large burst of EMG activity extending the leg for several seconds.

Stroke patients showed more consistent results and it was never observed a case of low tonus. However, a change in body position from sitting to supine regularly increased the level of spasticity. EMG activity was observed during the first two swings in mild cases

but strong ones it was difficult to differentiate whether it is caused by the muscle stretch or by continued muscle activation. However, technically speaking, these cases would be considered to be a result of rigidity and not spasticity since the muscle activity is continuous even when at rest (Lance 1990).

The results from Bajd and Vodovnik's study (1984) show that the degree of spasticity according to the relaxation index (R_i) range from severe ($R_i=0.02$) to mild ($R_i=0.71$). Moderate spasticity was found in the R_i 's of five hemiplegic subjects. The number of swings was only counted in the patients (three) with the largest relaxation index. In the rest of the patients the area between the goniogram and the resting angle was determined. The first maximum of the goniogram can display lower or higher values than those encountered in healthy volunteers. In cases of severe spasticity the value of the first maximum is above the one found in normals. This parameter can be in the normal range even when noticeable spasticity is present. The first maximum alone therefore cannot be considered as a reliable measure of spasticity.

Bajd and Vodovnik (1984) suggest that EMG is not necessary for fast routine assessment. Nevertheless this contradicts the discussion of Vodovnik and colleagues (1984) who mention the large variability of goniograms in spastic patients. The lack of knowledge regarding the neurophysiology of spasticity prevents further development of realistic models which should be time varying and nonlinear (Vodovnik, Bowman, & Hufford 1984). However in both cases, the non-neural changes that may not be differentiable from the neural ones, confounding the results, are not mentioned.

This pendular motion has been modelled by different authors to obtain a quantitative analysis for spasticity (Fee, Jr. & Foulds 2004; Le et al. 2001; Lin & Rymer 1991; Vodovnik, Bowman, & Hufford 1984). A differential equation describes the motion of the passive knee using the linear elements of a stiffness, damper and inertia combined to obtain an underdamped second order system (Lin & Rymer 1991) of the form:

$$I\ddot{\theta} + B\dot{\theta} + K\theta + C + K1(e^{-K2\theta} - 1) = m * g * l * \sin(\theta) \quad (\text{eq. 1})$$

In order to apply this equation to the pendulum test measurements, the leg is usually approximated by a cylinder obtaining the moment of inertia (I), mass (m), centre of mass

(l_c) of the lower leg (usually half the length) measured from the knee axis. System identification techniques are used to obtain the damping coefficient (B), the natural frequency (ω_n) and the lumped stiffness (K) from the frequency and ratio of maximums of the goniogram itself (Lin & Rymer 1991; Vodovnik, Bowman, & Hufford 1984).

In a later study by Lin and Rymer (1991) the model is analysed further and it was observed that, in one non-impaired subject, no significant EMG activity was observed and more importantly the values of K and B are not constant throughout the motion indicating significant non-linear behaviour (Lin & Rymer 1991). It was also noted that periods of the goniogram did not differ significantly among consecutive half-cycles but periods over three half-cycles did vary significantly. In this same study, both legs, affected and unaffected of three hemiplegic patients behaved in a different manner from the normal oscillation. The amount of flexion and the number of oscillations were much less in the affected side and EMG activity was present. In the mildly spastic subject, the EMG of quadriceps was much greater in amplitude and duration than that of the hamstrings. In more spastic subjects, the amplitude, duration and number of bursts of both the quadriceps and hamstrings EMG's were greater than that of mildly spastic subject.

The parameters K and B tend to vary with the motion amplitude, velocity and direction of swing. It has been reported that the knee and ankle present hysteresis when oscillated and that K and B increase when muscles are active (Lin & Rymer 1991).

In summary, the pendulum test is a simple technique easy to instrument and it is possible to obtain quantitative measurements from the goniogram. Nevertheless, the movement of the leg does not only depends on the level of spasticity but also on the non-neural properties of the muscles and EMG recordings are not always performed and even if they are with the goniometer alone there is no measurement of the torques generated by the stretch reflex (Lin & Rymer 1991).

More recently, the velocity-sensitive reflex mediated torque (R_f) (i.e. stretch reflex) was added to the second order equation creating a neuromechanical model (Le, Poudens, Chagneau, Carrault, Allain, & Rochcongar 2001). Le and colleagues recorded experimental data from 8 healthy volunteers and 15 CNS injured patients with focal

cerebral or spinal lesions. In this case the subjects were sitting down while performing the pendulum test. EMG electrodes were placed to measure the rectus femoris and semimembranous muscles. Knee angular displacements were recorded with a potentiometer. Computed dynamics of the Rf were derived from previous experiments by Burke and colleagues. These dynamics consist of a linear increase reflex response magnitude (sEMG) in quadriceps femoris muscle as the velocity of passive knee flexion increased, but a decrease of the response when the stretching movement was started with the knee joint partly flexed for a constant velocity of flexion:

$$Rf + M(\ddot{\theta}, \dot{\theta}, \theta) = -mgl_c \sin\theta$$

$$\text{if } (\dot{\theta} - a\theta) \leq 0, \text{ then } Rf = 0$$

$$\text{if } (\dot{\theta} - a\theta) > 0, \text{ then } Rf = R(\theta, \dot{\theta}) \quad (\text{eq. 2})$$

The resulting model was highly non-linear where Rf is the reflex mediated torque in knee extensor muscles. The threshold function (x) is determined by the angular velocity and a multiple of the angular displacement. In this way, the model only produces a reflex mediated torque when the velocity overcomes a multiple of the angular position.

The second order equation was loaded in software for computed and implemented with the physical characteristics of the leg geometrically modelled as a cylinder. Estimation of mechanical parameters (K and B) was performed by fitting computed simulations with the averaged knee goniogram in the control group using a least squared error criterion to generate a “mean virtual leg”.

The physical parameters of the “mean virtual leg” were assumed to be similar in the spastic and healthy groups (i.e. non-neural changes are not considered). This assumption however cannot be considered as true for every volunteer.

The pendulum test responses shown, in the control group a regular, pseudo sinusoidal, damped motion of the knee with no or poor EMG activity, always limited to the first swing when present. In spastic patients exhibited typical restraints in knee goniograms during the stretching phase(s), concurrently with increased EMG activity in the rectus femoris. Semimembranous exhibited most often only low-level myoelectric activity.

They mention no evidence of voluntary contraction observed in spastic subjects and they attributed it to associated weakness, although this is just a hypothesis and cannot be determined from this test. In a few patients, a significant sustained EMG signal was detected in rectus femoris during the resting position, allowing classification of responses in two groups:

Group I: 10 patients only a single EMG burst usually during the first cycle of flexion including the peak velocity.

Group II: 5 patients shown two types of EMG activity: a) an early “dynamic” burst, similar to the features in Group I, followed by b) continuous “static” activity during the resting phase, which was responsible for a less flexed resting position when compared with the control group. Group II pooled the patients with the most severe spastic hypertonia.

Table 6 shows the calculated parameters of the second order equation for the lower limb calculated by Vodovnik (1984), Lin (1991) and Le (2001). This table shows that the moment of inertia (I) and viscous damping (B) do not differ between studies.

Differences in the elastic coefficient can be explained by the modelling technique approach between studies. Furthermore, it was found by Le that the elasticity (K) had minor influence in the computed simulations (Le, Poudens, Chagneau, Carrault, Allain, & Rochcongar 2001) which may account for the variability between the different authors.

Table 6: Mechanical parameters of the lower limb for the second order equation obtained from different studies. Vodovnik: si= sitting, su=supine. Lin, S1=subject 1; S2=subject 2; S3=subject 3

	m (kg)	l (m)	I (N.m.s ² .rad ⁻¹)	B (N.m.s.rad ⁻¹)	K (N.m.rad ⁻¹)
Vodovnik	4.2	0.53	0.4	0.25(si) 0.35 (su)	11
Lin	S1: 3.86	0.389	0.26	0.15-0.7	5.5-8.5
	S2: 4.99	0.54	0.61		
	S3: 4.46	0.485	0.43		
Le	3.95	0.476	0.2983	0.23 (su)	0.3

He (1998) carried out a study using the pendulum test where he assessed the effects of postural and muscle length changes in the stretch reflex sensitivity. Fifty-nine patients with multiple sclerosis were studied, 37 of these were also evaluated using the Ashworth scales. The clinicians were blinded to the results. The test was performed in the supine and the erect sitting postures, although 7 subjects could not lie down comfortably on their back and three could not sit up for the test. Different muscle activation pattern of flexors and extensors may produce erroneous results added to the variations due to testing posture. He concluded that this test cannot reveal useful information about the mechanisms of spasticity (He 1998).

More recently, Fee and colleagues (2004) had the unique opportunity to develop a neuromuscular model of spasticity using the pendulum test in three identical triplets (age 9 years 5 months). Two of them had cerebral palsy with mild spasticity. They used the non-spastic subject as a passive plant (i.e. non-neural parameters without feedback only) for two active models, one of which allows application of external torques and the second provides additional torque as a result of velocity feedback as a result of stretch reflex activity.

Their anthropometric dimensions were:

Subject	Height	Weight
1 (no clinical signs of spasticity)	1.42 m	28.1 kg
2 (clinical signs of spasticity)	1.38 m	27.5 kg
3 (clinical signs of spasticity)	1.40 m	27.9 kg

The pendulum test was carried out while the subjects sat on a cushioned seat allowing the shanks of the legs to swing freely about the knee. EMG was monitored to determine the relaxation. The examiner passively extended the knee to an angle limited by the resistance of the knee joint and then released and allowed to oscillate under the influence of gravity. This oscillatory movement was measured for 10 s six times each subject. Angular displacement was measured using an electromagnetic sensor (3SPACE Isotrak) placed at the lateral malleolus aligning its horizontal axis with the long axis of the lower shank. The authors argue that this type of sensors have advantage on the usual systems (goniometers, tachometers, etc.) as the localisation of the centre of rotation of the knee

joint is not the main concern. Nevertheless the transmitter is located away from the leg and any unexpected displacement of the upper leg in any direction will modify the signal received, affecting the measurement. EMG signals were recorded at 1 kHz.

The model used is described by the second order equation from the models above mentioned, however in this paper a more complex variant is introduced by considering elements representing coulomb friction C and nonlinear stiffness elements. The additional torque due to nonlinear stiffness is defined as:

$$T = K1(e^{k2\theta} - 1) \quad (\text{eq 3})$$

Furthermore another variant was introduced due to the different stiffness and damping coefficients between flexion and extension. Thus separating extensor and flexor values for the second order equation depending on the direction of the movement:

$$I\ddot{\theta} + B_f\dot{\theta} + K_f\theta + C_f + K1_f(e_f^{-K2\theta} - 1) = m * g * l * \sin(\theta) , \text{ when } \theta \geq 0 \quad (\text{eq 4})$$

$$I\ddot{\theta} + B_e\dot{\theta} + K_e\theta + C_e + K1_e(e_e^{-K2\theta} - 1) = m * g * l * \sin(\theta) , \text{ when } \theta < 0 \quad (\text{eq 5})$$

The active element model introduces additional step torque values believed to be related to the stretch reflex. However, these additional torques do not depend on the EMG signals recorded but on the optimisation algorithm to specify the timing and amplitude of torques to provide with the best fitting model.

The equation including this latter element for the extensor part of the equation is:

$$I\ddot{\theta} + B_e\dot{\theta} + K_e\theta + C_e + K1_e(e_e^{-K2\theta} - 1) + T_e = m * g * l * \sin(\theta) , \text{ when } \theta < 0 \quad (\text{eq. 6})$$

Finally, the velocity feedback model is based on the argument that physiologically speaking step torque changes due to the stretch reflex are not possible. The velocity dependent Ia signal does not linearly produce an EMG signal and the feedback does not produce instantaneous muscle force due to delays in signal transmission, muscle stimulation, and muscle activation. This is known as electromechanical delay (Cavanagh & Komi 1979; Stokes 2004).

The final equation introducing the velocity dependent feedback torque of the extensor component of the model can be written as:

$$I\ddot{\theta} + B_e\dot{\theta} + K_e\theta + C_e + K1_e(e^{-K2\theta_e} - 1) + BF_e\dot{\theta} = m * g * l * \sin(\theta) , \text{ when } \theta < 0 \quad (\text{eq. 7})$$

Fee and colleagues' results show how the passive model presents an acceptable fit with the experimental data from the subject without cerebral palsy but failed to represent the other two siblings. In the second model, the step torques appear to be well synchronised with the EMG burst seen in the electrophysiological measurements. The active-feedback model generates the additional torque by means of velocity feedback considering the onset times for changes in gain and delays in the application of torque. The onset times of gain changes coincide with the EMG bursts and are likely to reflect the change in damping coefficients due to muscle activity.

This particular study gives an opportunity to understand the contribution of the stretch reflex excitability to the additional torques that modify the kinematical behaviour of the pendulum test that are different between the non spastic sibling (i.e. passive plant) and the spastic siblings. The added torques from the active-feedback seem to correspond with the bursts of EMG simulating the muscle spindle activity and Ia conduction times. However a specific study of the EMG signals observed and their relationship to the torques observed could provide a much better insight of the EMG characteristics in eccentric contractions and the relative torque depending on the muscular activity.

Even though this model can predict such active parameters representing the velocity feedback and EMG burst torque contributions, the origin of such muscle activity may not be uniquely attributed to spasticity as some cortical influence (i.e. non-stretch reflex behaviour) may be responsible for such EMG bursts. This can be explored adding a mechanism that can provide a faster stretch ensuring that the only muscle activity present is due to the stretch reflex activity.

Pendulum test in the upper limb

Some authors have applied the pendulum test principle to measure spasticity in the upper limb. The modifications have required developing simple instrumentation to use the gravity to produce elbow joint rotations. In 2001, Lin CC and colleagues introduced a

biomechanical model for modified pendulum test of the upper limb. The device consisted of a shaft connected at the midpoint to a test bed through a pure rotary joint, an electronic goniometer measures the changes in the elbow joint angle, a weight attached to the lower end of the shaft to increase the inertia and counterbalance the weight of the forearm, and a part that fastens to the wrist. EMG recordings were taken from biceps brachii and triceps brachii. However, the EMG is not analysed but used only as an indicator for large or inappropriate time of muscle activity. No reference to the sampling frequency is found in this paper (Lin et al. 2001).

The dynamic characteristics of the whole system (device and arm) were expressed in the following equations:

$$I\ddot{\theta} = -\tau_g + \tau_m - K(\theta - \theta_e) - C\dot{\theta} \quad (\text{eq. 8})$$

$$I = I_a + I_f \quad (\text{eq. 9})$$

$$\tau_g = m_a g L_a \sin(\theta - \frac{\pi}{2}) - m_f g L_f \sin(\theta - \frac{\pi}{2}) \quad (\text{eq. 10})$$

Where θ is the elbow joint angle; τ_g is the torque caused by gravity; K is the stiffness constant; θ_e is the threshold angle; C is the damping coefficient; g is the constant of gravity and I_a , m_a , L_a , I_f , m_f , and L_f are the inertia, mass, and length of the device and forearm (including hand), respectively.

From calculations from the device oscillations alone, the authors determined that the stiffness and damping effects of the device were negligible when estimating the parameters of the forearm oscillations.

Three biomechanical models with different levels of complexity were formulated.

- A simple linear additive stiffness-damping model
- A nonlinear velocity-dependent term was added, representing the effects of velocity-dependent stretch reflex
- Incorporation of both non-linear position and velocity dependent terms for stretch reflex. Model parameters were estimated with the optimization techniques

Three stroke patients and three normal subjects were included in the study. Their data were collected and analysed with the models. The parameters for each subject for the different models are summarized in table (7).

The authors concluded that the simple linear model was able to differentiate spastic from non-spastic subjects and that both, the stiffness constant and the damping coefficient were increased in the stroke patients with spasticity. This technique, however does not dissociate between neural and non-neural characteristics of hypertonia, furthermore no information of EMG measurements and how it can give information on the changes in the model is provided.

Table 7: Results of parameter estimation with each model (Lin et al, 2001). K was the stiffness constant, C the passive or linear damping coefficient, B the non-linear damping coefficient, q_e was the threshold angle of spring element, q_{th} was the threshold angle for the position related torque and q_s was a constant

	Subject	K (N.m/rad)	C (N.m.s/rad)	C (N.m.s/rad)	θ_e (deg)	RMS (deg)	V (Nil)	Kp (N.m/rad)	θ_a (deg)	θ_s (deg)
Model 1	N1	0.72	0.34	NA	53.9	3.42	NA	NA	NA	NA
	N2	0.84	0.55	NA	94.4	1.51	NA	NA	NA	NA
	N3	0.65	0.55	NA	70.5	2.28	NA	NA	NA	NA
	S1	3.88	1.65	NA	101.5	2.23	NA	NA	NA	NA
	S2	2.27	1.31	NA	68.8	1.3	NA	NA	NA	NA
	S3	6.54	2.63	NA	76.8	2.77	NA	NA	NA	NA
Model 2	N1	0.75	0	0.6	39.6	2.16	NA	NA	NA	NA
	N2	0.84	0.55	0.24	94.6	1.24	NA	NA	NA	NA
	N3	0.65	0.55	0.6	61.9	1.13	NA	NA	NA	NA
	S1	3.88	1.65	1.13	98.6	2.14	NA	NA	NA	NA
	S2	2.27	1.31	0.07	68.2	1.31	NA	NA	NA	NA
	S3	6.54	2.63	1.49	75.1	2.54	NA	NA	NA	NA
Model 3	S1	3.6	0.75	0.77	99.7	2.16	0.33	5	0	15.5
	S2	2.55	1.09	0.1	68.8	1.01	0.33	4.04	0	40.7
	S3	5.9	1.11	1.7	75.1	2.47	0.25	0	0	0

Later in 2003, the same authors presented a similar study with the aim of estimating parameters of the elbow biomechanical model as the candidate indicators of spasticity. In this study eleven stroke patients with spasticity and eleven non-impaired subjects were recruited. EMG electrodes were placed on biceps and triceps and recorded with a sampling frequency of 600 Hz, which is lower than the average normally used (1000 Hz) when registering EMG activity for offline analysis (Lin, Ju, & Lin 2003).

Their results show a decreased stiffness constant on the intact side of the stroke patients and increased damping coefficient in the affected side of stroke patients. They claimed that the damping ratio increased with spasticity and should be used as an indicator of spasticity and automate the analysis for broad clinical applications.

The main contribution from this work is the biomechanical model developed from the oscillatory movement caused by the inertial properties of the limb and the viscoelastic properties of the muscle. The latter is of extreme importance to muscle tone assessment. Nevertheless, without EMG measurements it is not possible to determine whether any changes in presumably spastic patients are due to biomechanical changes in the muscle properties or abnormal neural activity.

4.2.3.3 Controlled displacement methods

These methods use feedback-controlled devices allowing the limb to move at a determined velocity and amplitude. The type of movement depends on the waveform input applied to the limb such as ramp, sinusoidal and stochastic (random) and this depends on the type of methodology and device used.

Detrembleur C and Plaghki L (2000) used a technique described by Lehmann and colleagues (1989) where a system is used to assess quantitatively the muscle tone in normal and spastic subjects (Detrembleur & Plaghki 2000) (Lehmann et al. 1989). The method measures muscle resistance to passive low-amplitude sinusoidal displacements of the ankle joint at different frequencies of oscillation. They modelled the calf-ankle-foot system in mechanical terms, as a torsional spring (elasticity of the gastrocnemius-soleus-Achilles tendon), a torsional viscous damper (viscous characteristics of the same tissues) and a rotary mass connected in parallel (mass of the foot rotating about an idealised ankle pivot joint). This passive viscoelastic system will produce a characteristic torque in response to a sinusoidal displacement. Based on the fact that elastic stiffness is in phase with the displacement and viscous stiffness is out of phase by 90 degrees, the 2 components of net muscle stiffness may be computed separately using Fourier analysis. However, spasticity is of neural origin and this method is likely to include information from soft tissue information, which is useful to evaluate muscles and tendons condition due to immobilisation but not for spasticity assessment.

In other study (Given JD et al 1995) torque-angle relations at the elbow and angle joints of relaxed non-disabled controls and hemiparetic stroke patients were compared. In this study, the authors are aware of the importance of dissociating between the soft tissue components and the neural components. Their study is oriented to obtain the passive mechanical properties of muscle and tendon in order to obtain an accurate quantification of spasticity in further studies.

Their methodology consisted of low velocity flexion/hold/extension angular perturbations applied to the joint. The outcome torque-angle profiles described a hysteresis loop with parallel slopes during extension and flexion. They used muscle activation absence to determine passive torque angle responses. No differences in passive stiffness between non-impaired and hemiparetic subjects were found in the elbow joint, contrary to the ankle joint where significant differences in the torque-angle hysteresis loop in all hemiparetic patients tested. However, no EMG measurements were used in this protocol making it difficult to determine whether the passive stiffness is affected by neural components.

Kamper and Rymer (2000) used a servomotor to quantify passive resistance and reflex response to stretch of the extrinsic muscles of the fingers with a ramp and hold waveform. The extrinsic finger muscles were stretched through simultaneous rotation of the metacarpophalangeal joints of the four fingers. Thirteen stroke subjects with chronic unilateral motor deficits with at least 2 years after incident (8 right and 5 left hemiparesis) and two control subjects participated in the study. The Ashworth scale was used to quantify resistance to passive stretch of the elbow, wrist and fingers joints (Kamper & Rymer 2000).

The arm of the subject was mounted on the device and the subject was instructed to relax. The ROM was found by manually rotating the motor shaft. The limits were set by the subject when mild discomfort was felt. Static passive torques were recorded at six different evenly distributed angular positions throughout the ROM by the servomotor moving the joints to each of the six angular positions and holding it for 2 seconds. Dynamic trials consisted of constant-velocity stretches of the extrinsic finger flexors by rotating the MCP joints from the limit of flexion to the limit of extension. Outcome measures were position, torque, velocity and EMG. In this work, the reflex response was

quantified in terms of the reflex threshold and the net work needed to stretch and release finger flexors. The authors argued that the threshold can be estimated from changes in joint stiffness and that the reflex torque can be estimated by subtracting the passive torque. They also mention that the passive elastic forces do not contribute to the amount of work needed to stretch the fingers and then return them to the initial position. All these assumptions do not consider that muscle properties are also changed after a period of immobilisation which can confound the results.

Kamper and Rymer results show that the passive response to stretch increased with the stretch velocity and that significant reflex responses were elicited in response to the MCP joint extension in eight of the thirteen stroke subjects determined from analysis of torque and EMG signals. However, the EMG contribution to the torque changes was not discussed.

Ageranioti and Hayes in 1990 used a strain gauged torque motor system to measure hypertonia and hyperreflexia in twenty nine volunteers with spastic hemiplegia or hemiparesis. The motor displaced their hand periodically through flexion and extension. The outcome measures included the total resistive torque of the wrist, its component non-linear stiffness and damping torques, and the mean energy loss during passive wrist flexion and extension. The total integrated EMG activity of the wrist flexors and extensors during 7.5 cycles of passive displacement of the hand were used as measures of hyperreflexia. Their results showed the torques at extension to be significantly greater than at flexion (Ageranioti & Hayes 1990).

Plots of the resistive torque versus angular displacement of the hand revealed three different forms of hysteresis loops: elliptical (linear stiffness and damping properties), sigmoidal shaped (rate dependent non-linear stiffness and nonlinear damping mechanisms) and loops with pointed ends (rate-independent non-linear systems) two subjects presenting the latter exhibited high volume integrated EMG activity indicative of marked hyperreflexia. The concern of using this kind of technique is that there is no way to ensure that all EMG activity is of reflexive nature and some voluntary contribution might confound the results.

This study was designed as a two group crossover and aimed to investigate the effects of tendon vibration over the wrist extensors. From the hysteresis graphs it can be seen qualitatively that the vibration reduces the hypertonia which tends to return after a certain time (Figure 19). It was also observed that integrated EMG values were reduced after the treatment and the patterns of hyperreflexia were altered from complex combinations of stretch and shortening reflexes to more simple patterns such as exaggerated stretch reflexes in the flexor muscle alone. It can be argued however, that this vibration may affect the intrinsic viscoelastic properties of the muscles and that its effect is mechanical one that will have also effects at a neurological level and that the EMG changes, given the length of the tests, are not solely reflex modulated.

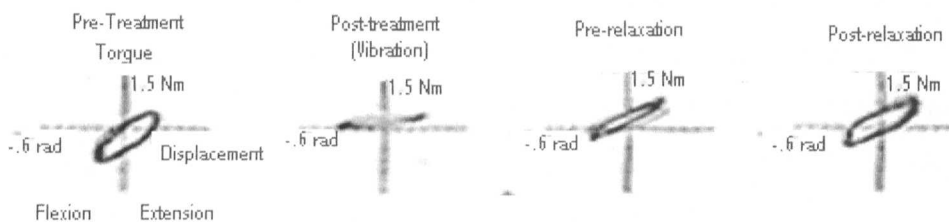


Figure 19: Qualitative comparison of hysteresis loops from one subject for the whole experimental setup. From left to right, pre-treatment, post treatment, pre relaxation, and post-relaxation. After the treatment the area within the loop was reduced but tended to increase after some time (Ageranioti & Hayes 1990).

In a study by Harris and colleagues in 1990 a set of quantitative instruments for monitoring the surgical and rehabilitative progress of children and teenagers with cerebral palsy including biomechanical and electrophysiological equipment, automated clinical test series and functional assessment instrumentation (Pike et al. 1990).

The biomechanical devices aimed at the quantitative evaluation of spasticity and included active and passive motion resistance devices. These devices were motorised and capable of producing multiple constant velocity motion conditions. The authors describe a set of instrumented devices to assess different joints of the body. The custom made device was used to quantitatively evaluate wrist joint control during active and passive motion consists on a computer controlled DC servo motor; torque and displacement sensors. It generated step changes in angular position, constant velocity, and constant acceleration about the wrist joint while monitoring flexion/extension range

of motion, torque and EMG activity of the flexor and extensor groups in the forearm (Figure 20).

Their results support the hypothesis that spasticity in muscles acting across the joint could be detected as higher torque values. Stiffness and damping characteristics were determined using a biomechanical model of the wrist. Stiffness was significantly higher in the hemiplegic (0.31, 0.32 and 0.39 N-cm/deg) wrists as compared to the non-impaired subjects (0.08, 0.03 and 0.06 N-cm/deg) at respective velocities of 20, 60 and 100 degrees/sec. Mean isometric torques were significantly different between controls and cerebral palsy children ($p < 0.01$) with 388 N-cm (+ 36 N-cm) flexion, 270 N-cm (+47) extension for controls while in the cerebral palsy wrists it was only 102 N-cm (+53) flexion, 82 N-cm (+ 56) extension. Harris and colleagues also mentioned that EMG traces showed co-contraction in the cerebral palsy affected limbs. However the authors did not consider the dissociation between neurological and biomechanical changes due to upper motor neurone lesion. Furthermore, the test described does not address the stretch reflex excitability of spasticity but rather considers it as increased muscle activity without taking into account the possibility of voluntary activity confounding the results.

The elbow device consisted of a strain gauged cantilever rotating in the horizontal plane. Elbow torque and angular position are measured during 25 passive cycles. The hip and knee device consisted of a motorised cart dynamometer to which the foot was attached. Linear horizontal displacement of the foot produced coupled hip and knee motion in a parasagittal plane. A DC motor and controller allowed a selection of constant cart velocity while resistive forces at the foot were detected by the dynamometer (3 forces and 2 moments). The authors argue that solution of the systems equations of motion allows determination of hip and knee spastic moments.

A third device was used to determine torques about the knee joint during active and passive limb testing. Strain gauges were used to measure sagittal and coronal torques while potentiometers record angular and axial motion also producing constant angular velocity. Electromyography was used to monitor muscular activity of flexor and extensor groups during upper and lower extremity hypertonicity tests. The authors used this only to document co-contraction periods and investigate the relationship between EMG and joint torque patterns.

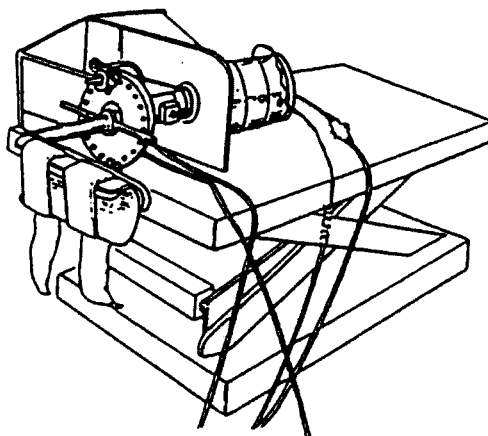


Figure 20: joint test device used to evaluate joint dynamics at the elbow, knee and ankle during active and passive motion.

In a different study, two different approaches were designed to determine the feasibility of Isokinetic Dynamometry by measuring RTPM (Firoozbakhsh et al. 1993): 1) a quantifiable method was developed by determining the summation of the four consecutive resisting torque amplitudes during flexion and extension of the knee at specified speeds and a range of motion and; 2) an assessment was made by finding the slope of the linear regression curve of torque-velocity data.

They found that the values of maximum torque were higher in a spastic population than in the normal group, but the difference was statistically significant only when the sum of the torque amplitudes was considered ($p < 0.0028$). Values of the maximum torque as well as the sum of the torque amplitudes increased in a linear fashion ($r > 0.75$) with increasing velocity. The slopes of the torque velocity curves were greater in spastic subjects than in non-spastic subjects. The sensitivity to the rate of stretch was statistically greater ($P < 0.0004$) for the spastic group. Like other studies, this paper fails to dissociate neurological from non-neurological causes of hypertonia. It may be considered as a reliable measure of RTPM but not of spasticity.

Dvir and Panturin (1993) developed a study to measure spasticity and associated reactions in stroke patients before and after physiotherapeutic intervention used isokinetic measurements (KinCom) (Dvir & Panturin 1993). The seat of the device was positioned so that the patient's arm was placed parallel to the actuating arm of the dynamometer and their centres of rotation are aligned. Twenty women and thirteen men

were included in the protocol. Their shoulder was flexed at 45 degrees throughout the range of elbow motion (90 degrees). Five angular velocities were applied (200, 120, 90, 60 and 30 degrees/s) from high to low speed. They showed that the resistance decreased significantly with lower velocities. The pre and post intervention results showed no differences.

Pisano and colleagues (2000) used a servo-controlled DC torque motor to induce wrist extension at 50 deg at different speeds using a ramp-and-hold wave form input in non-impaired and post-stroke volunteers. EMG activity of flexor carpi radialis was recorded. The outcome measurements were the slope of the torque/position curves (stiffness), the stretch reflex threshold speed (SRTS) and the stretch reflex latency and area (i.e. integrated EMG signal). In their work the authors attempted to distinguish between neural and non-neural components of stiffness. The passive stiffness was calculated at 10 degrees/s and labelled as intrinsic stiffness index (ISI) whereas the stiffness calculated at 200 degrees/s was considered to incorporate passive and neural components and was labelled as total stiffness index (TSI). Ten repeated measurements were obtained from each subject at each condition while the subjects were instructed to relax completely. Figure 21 shows the stiffness indices ISI and TSI in a post-stroke patient (Pisano et al. 2000).

Their results indicated that both, passive and total stiffness indexes were higher in the stroke population than in the non-impaired volunteers. The SRTS was lower in patients than in normals and the area was significantly larger in patients whereas the latency did not differ between the groups. However when the EMG recordings were studied it was observed that even at the slower velocity most of the non-impaired subjects showed EMG activity. This suggests that the stretch reflex activity is more excitable in post-stroke subjects and it is consistent with the definition of spasticity. However there is no information about the levels-of EMG activity prior the perturbation which may explain the amplitude values at lower velocities and furthermore indicate the difference between rigidity and spasticity.

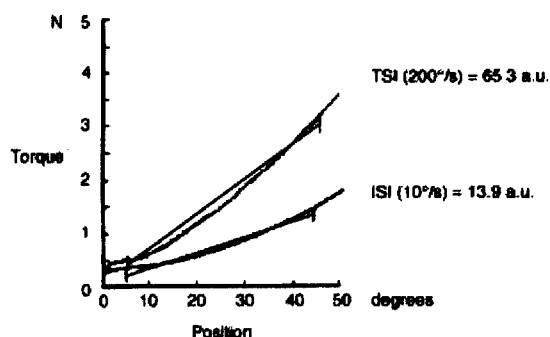


Figure 21: Total and intrinsic stiffness indices in a post-stroke patient. The indices correspond to the slope of the regression lines calculated on the torque/position curves at 10 and 200degrees/s respectively (Pisano, Miscio, Del, Pianca, Candeloro, & Colombo 2000).

In 1999 Lin and Sabbahi attempted to correlate spasticity, stretch reflex hyperactivity and motor dysfunction of the wrist and hand in adults with hemiplegia (Lin & Sabbahi 1999). While spasticity was evaluated with the MAS, the stretch reflexes excitability was assessed with a mechanical device comprised of a computer-controlled microstepping motor. Stretch reflexes were elicited by extending the wrist at different velocities: 100, 200, 300 and 400 degrees/s. These velocities were applied under two preload conditions consisting of instructing the volunteers to a) relax and not resist the stretch or b) increase their background voluntary contraction at 10% of their maximum voluntary contraction levels (MVC) resisting the stretch. Resistance to passive stretch was measured with a force transducer while EMG was measured with surface electrodes over the flexor carpi radialis (FCR). The outcome measures were the amplitude and the integrated EMG activity between 30 and 50 ms associated with the short-latency response M1 were recorded and expressed as a percentage of the MVC. However the assumption for the duration of this activity assumed to be the short latency response is a theoretical value and may vary between different people, furthermore they do not provide a reference for this value although M1 values for Biceps Brachii is between 15-30 ms and lasts up to 50 ms, (Yamamoto et al. 2000). These latencies may be different for the FCR under this study. In their results the authors reported the relationship between the outcome measurements by calculating correlation coefficients. Regarding spasticity measurement the results of interest were the correlation between the MAS and the measurement of stretch reflex activity. In their work, they found a strong correlation between the amplitude of the reflex EMG activity with the MAS in both active and passive background conditions but this relationship was more consistent in the active background

conditions ($r=0.77$ and 0.74 , $p=0.005$ and 0.007 in session 1 and session 2 respectively). Their torque measurements were inconsistent and showed little correlation.

This paper however, does not provide enough information about the instrumentation and certain parameters such as sampling frequency and the filter used to obtain the integrated EMG which will influence the outcome results significantly. Therefore the results from this study should be considered with caution.

Dewald and colleagues (1994, 1996), used a system to assess the passive properties (passive muscle, tendon, and connective tissue) and the stretch reflex response. The system delivered a slow ramp position perturbation. The mechanical parameters measured were torque, velocity and position, the physiological parameter was EMG activity of the elbow. They found that spasticity was reduced after electrical stimulation over the antagonistic muscle, the post-stimulatory torque responses of the elbow joint correspond to a pre-stimulatory response at a significantly reduced stretch velocity. The electrical stimulation was applied for 10 minutes at 20 Hz, with an intensity level below motor threshold but above sensory threshold (Dewald & Given JD 1994). The measurement protocol involved the subjects sitting and their elbow positioned at 60 degrees flexion and the elbow was passively moved through 57.3 degrees at constant angular velocity. The starting position and ramp velocity were not standardised but selected on individual basis in order to obtain a repeatable stretch reflex response. The outcome measurements in this study were the angular position and velocity, peak torque and EMG activity in the elbow flexors and extensors. Later on in 1996 a similar methodology by the same group was used however in this case the starting position was varied between 105 degrees and 130 degrees and velocities from 0.05 to 1.6 rad/s. Repeated measurements were performed pre and post intervention (Dewald, Given, & Rymer 1996).

A different study was designed to investigate the effects of the initial position of the elbow joint and the velocity of the muscle stretch on the stretch reflex threshold angle (angular position of the joint at which the muscle EMG response exceeds the baseline EMG by 2.5 standard deviations) (Wolf et al. 1996). The initial positions were 70 degrees and 90 degrees flexion and the angular speeds were 0.5 and 1 rad/s. The results of this study shown that the threshold angle could be reached independently of the

required speed (i.e. the EMG level increased before the required speed was reached). This suggests that the threshold angle is not affected by the speed, but is affected by the starting position.

In 1996, Lai and colleagues developed a spasticity measurement system with a real time controlled servo motor system with torque sensor, accelerometer and EMG recording electrodes (biceps, brachioradialis and triceps). The control system is programmed in simulink. In their protocol they used five different constant stretching velocities (20, 40, 60, 80 and 100 degrees/s) to elicit the stretch reflex of elbow joint in spastic subjects. The shoulder was positioned in 90 degrees abduction and the elbow in 110 degrees flexion and the range of motion was of 45 degrees. Four subjects were included in their protocol with different etiology, one with multiple sclerosis, two CVA patients and one with Parkinson's disease measured twice, before and after medication. The outcome measurements in the protocol were the stiffness ratio defined as the ratio of dynamic stiffness and static stiffness and the reflex torque defined as the difference between the dynamic torque (DT) and static torque (ST). Their results show a high correlation between the outcome measurements (stiffness ratio and reflex torque (RT)) with the net EMG activity, as well as with the velocity of the stretch. Figure 22 shows the relationship of the outcome measurements with the net EMG activity (Lai et al. 1996).

From the correlation plots it can be seen that although the correlation is high, most of the net EMG activity is clustered near the origin, suggesting that the EMG activity was not always elicited. Furthermore, the time of the stretch is not short enough to assume solely stretch reflex activity but also voluntary activity may be contaminating the results.

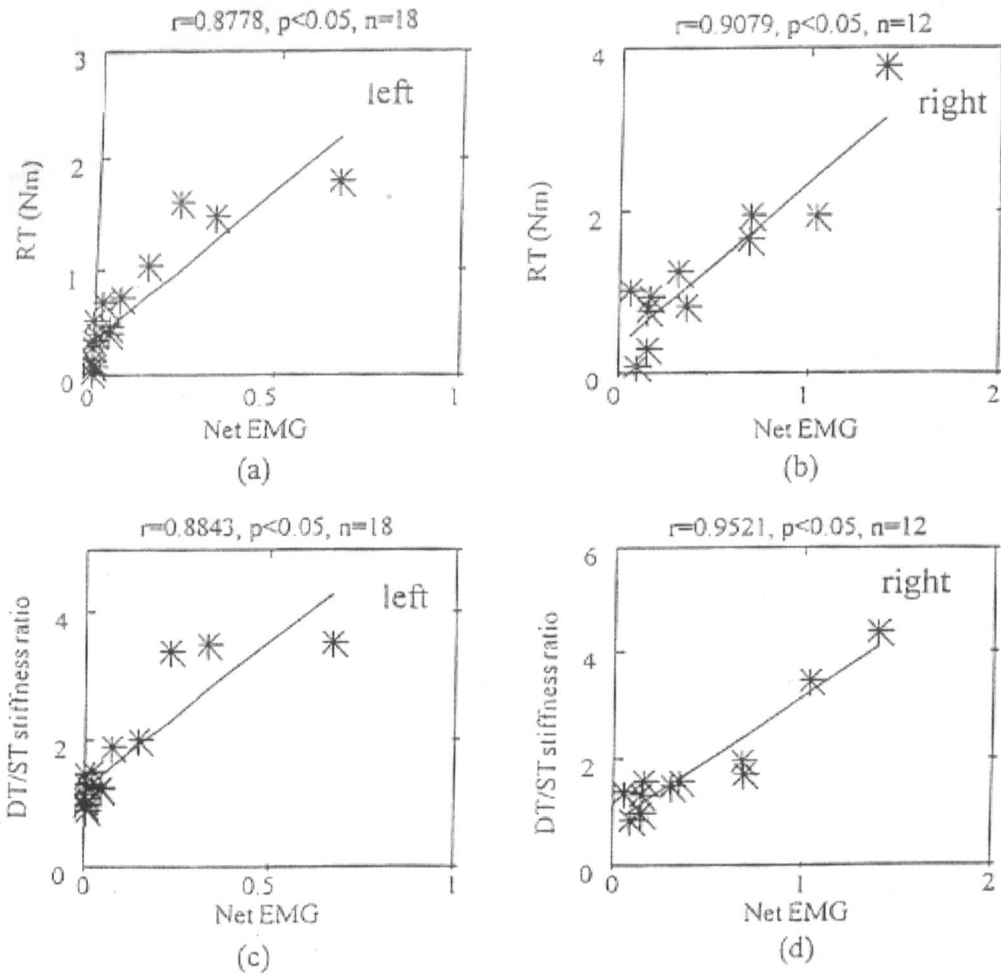


Figure 22: Correlation between spasticity parameters (Reflex Torque (RT) and the Dynamic Torque (DT) - Static Torque (ST) stiffness ratio) and net EMG in affected and intact elbows of a multiple sclerosis subject. (a) (c) left side, (b) (d) right side. (Lai, Gau, Ju, & Chen 1996).

In a similar study from the same group, Ju and colleagues (2000) used different outcome measurements since they argue that the parameters from Lai and colleagues show too much inter-subject variability (Ju et al. 2000). Their outcome measurements are the averaged speed-dependent reflex torque (ASRT), defined as the measured torque deviated from a baseline torque measured at a selected low stretch velocity of 5 degrees/s. Four patients with CVA were recruited. Figure 23 shows the diagram of a typical ramp-and hold position, velocity and torque profiles profile. The figure also shows how the measured torque is segmented in fixed specified periods. By subtracting the baseline torque determined by the start and end positions (P1 and P2 respectively) the ASRT is defined as the area under the baseline torque divided by the angular displacement.

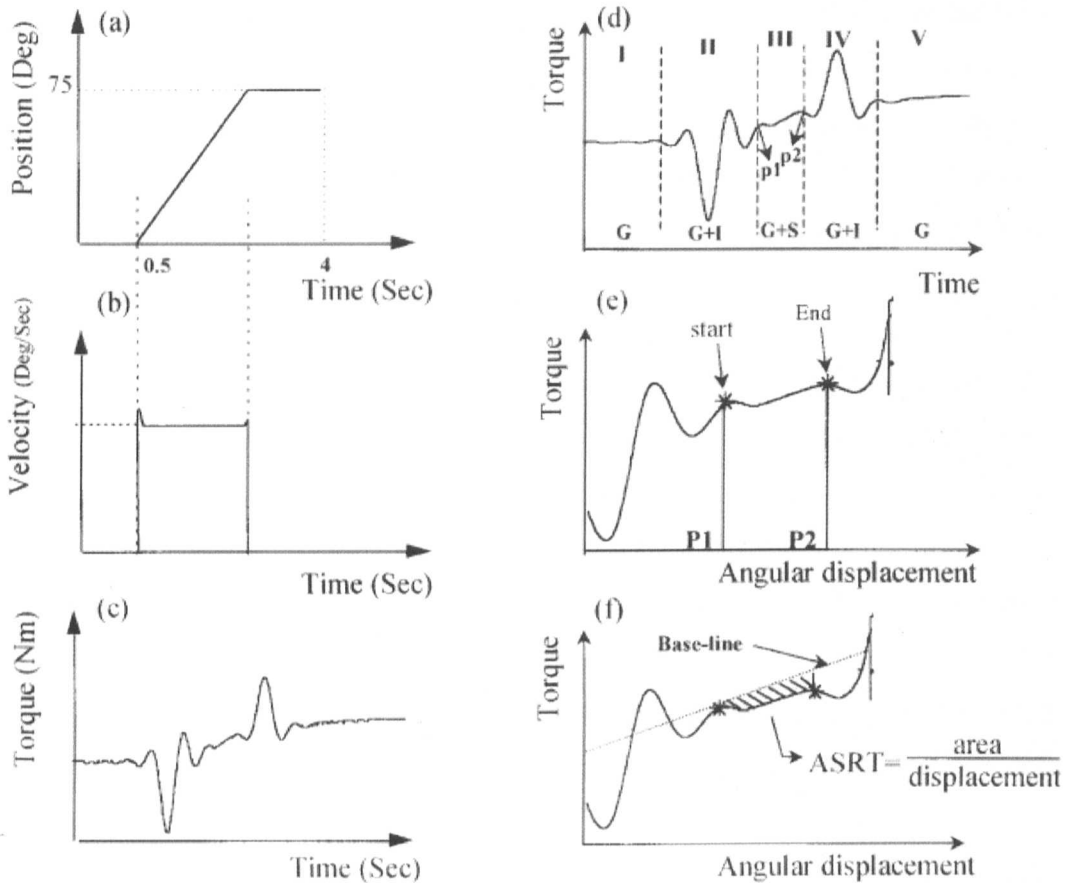


Figure 23: Diagram of the ramp-and-hold position profile, a) angular displacement, b) angular velocity, c) torque pattern, d) reactive torque as a combination of gravity, G, inertial acceleration/deceleration, I, and the measured stretch reflex, S. e) Positions P1 and P2 define base line torque, which is subtracted to calculate the averaged speed-dependent reflex torque (ASRT) defined by the area under the baseline torque divided by the angular displacement (Ju et al. 2000).

This methodology is flawed in the sense that EMG measurements as a result of the stretch reflex activity are not considered. Furthermore, more recent evidence suggests that the soft tissue changes occurring after stroke are velocity dependent and are likely to affect the outcome measurements from these studies. These remarks are supported by another study from the same group (Lee et al. 2002) where, using the same methodology, they attempted to compare the increased muscle tone in spasticity with the one observed in rigidity. Their results found no significant differences in the velocity dependent reflex torque between spasticity and rigidity, suggesting that either the common belief that hypertonia in Parkinson's disease is not velocity dependent or that the theoretical background of this methodology is flawed. Nevertheless, the authors also found that the muscle tone in spasticity increased with increasing joint position. The one found in rigidity was at a higher level that was constant and independent of the position of the

joint. They suggested that this variable (segmented average speed dependent reflex torque) could be used to indicate position dependency of the reactive torque and could be used to differentiate between spasticity and rigidity. This latter argument can be debatable, as it may be an easier approach to observe the EMG variability during the movement and, estimate the differences of muscle activity between prior and during the perturbation to differentiate spasticity from rigidity.

A Biodex Rehabilitation/ Testing system 2 was used in a series of studies from the Rehabilitation Institute of Chicago, aimed to study the behaviour the stretch reflex response of the elbow flexors (1999, 2000 and 2001). In these studies they developed a parametric model of the reflex torque response to large extension amplitudes (90 degrees-100 degrees) at constant angular velocities to help quantify spasticity in eight hemiparetic brain injured subjects (Schmit et al. 1999). They have suggested two specific parameters to what they assume to reflect significant aspects of the underlying pathophysiology of spasticity. These parameters are: a) the angular threshold, defined as the angle at which the passive movement first showed evidence of motor neurone activation reflecting the baseline excitability of the motor neurone, and b) the reflex stiffness, estimated from the slope of the torque-angle relationship of the joint, was associated with the reflex loop gain relating the discharge of the motor neurone and associated muscle force to the level of afferent input. The authors explain that the determination of these parameters is based on the assumption that the reflex response is a linear phenomenon. The initial position of the limb was with the shoulder abducted at 80 degrees and shoulder flexed at 0 degrees- 10 degrees. The minimum elbow angles ranged from 45 degrees to 60 degrees while maximums were 130 degrees- 155 degrees (full extension defined as 180 degrees). The authors estimated the reflex torque of the elbow flexors by subtracting the passive torque measured at slow velocity (6 degrees/s) from the torques measured at the test velocity. They claim this is possible because passive torque responses of flexion/extension are essentially velocity insensitive at the elbow. However there is evidence from the ankle joint that the biomechanical changes of the soft tissue surrounding the joint are velocity sensitive (Singer, 2003). Nevertheless, these two joints have a different anatomical structure and experimental data is needed to corroborate these assumptions. The incremental reflex stiffness was calculated by the first derivative of the elbow torque with respect to the elbow angle. This stiffness was

examined in a case by case manner with the intent to identify a reflex stiffness and angular threshold.

They observed that the torque exhibited a plateau at a mean angular increment of 51 degrees ± 10 degrees s.d. after the initial rise. Two main effects can be responsible for this plateau:

- Effects of moment arm changes
- Effects of muscle activation

The authors found that the plateau could not be explained by decreases in elbow flexor moment arms during elbow extension. They concluded this from the evaluation of the linearity of the reflex response independent of the changing muscle moment arms by converting the measured joint torque to muscle stress. Muscle stress was estimated considering the measured torque, the estimated moment arms and the relative physiological cross-sectional areas (PCSA) of the brachialis, biceps and braquioradialis (Obtained from reported values).

The torque was expressed as:

$$\tau_e = \sigma PCSA_{Bi} d_{Bi} + \sigma PCSA_{Bra} d_{Bra} + \sigma PCSA_{BRD} d_{BRD} \quad (\text{eq. 11})$$

Where τ_e is the measured elbow torque, σ is the muscle stress, PCSA is the physiological cross sectional area of biceps (Bi), brachialis (Bra), or brachioradialis (BRD) and d is the estimated moment arms.

This equation however, makes the assumptions that the activation function (reflected as muscle stress) is identical for all elbow flexors, that the PCSA does not change with elbow angle, and that the moment arm is a function of the elbow angle and can be approximated using a general model. Their results showed that the levelling effect did not appear to be determined by the absolute joint angle and that was not a consequence of the muscle moment arm changes.

They found however that this plateau is attributable to a consistent levelling in muscle activation. This was confirmed using an EMG coefficient model designed to quantify the

reflex torque response accounting for nonlinearities in the muscle activation functions. They used a modelling technique in which joint torque is described by a linear summation of the weighted EMGs of the relevant muscles.

The equation used was:

$$\tau_e = C_0 + C_1 EMG_{Bi} d_{Bi} + C_2 EMG_{BRD} (d_{Bra} + d_{BRD}) \quad (\text{eq. 12})$$

the coefficients of the model (C_0 , C_1 and C_2) were estimated using five ramp stretches of the elbow flexors using a linear, minimum least squares estimation.

This model assumes that the torque can be described by the summation of two activation functions equivalent to the rectified smoothed EMGs.

Nonlinear Parameterisation of the reflex torque response

Schmit and colleagues (1999) established that non-linearities in the reflex torque response resulted primarily from patterns of muscle activation rather than from changes in muscle moment arm with changing joint angle. This prompted them to develop a new model of reflex torque to parameterise the response based on activation functions of the elbow flexors with the activation of each muscle described as a cumulative normal distribution with respect of joint angle.

A variation in the equation to calculate muscle stress was introduced. Two activation functions were used, one based on the activity of the biceps and the second based on the brachioradialis activation. The equation is expressed as:

$$\tau_e = \sigma_1 PCSA_{Bi} d_{Bi} + \sigma_2 PCSA_{Bra} d_{Bra} + \sigma_2 PCSA_{BRD} d_{BRD} \quad (\text{eq. 13})$$

the muscle stresses can be expressed as a function of elbow angle in the following way:

$$\sigma_1 = K_1 \left\{ \frac{1}{2} \operatorname{erf} \left(\frac{\theta - \mu}{\sqrt{2} \beta_1} \right) + \frac{1}{2} \right\} \quad (\text{eq. 14})$$

$$\sigma_2 = K_2 \left\{ \frac{1}{2} \operatorname{erf} \left(\frac{\theta - \mu - \alpha}{\sqrt{2}\beta_2} \right) + \frac{1}{2} \right\} \quad (\text{eq. 15})$$

where

$$\frac{1}{2} \operatorname{erf} \left(\frac{\theta - \mu}{\sqrt{2}\beta_1} \right) = \int_0^\theta \frac{1}{\sqrt{2\pi}\beta} e^{-(1/2)[(x-\mu)/\beta]^2} dx \quad (\text{eq. 16})$$

The authors claim that the cumulative normal distribution represents the probability distribution of many naturally occurring phenomena and it was used in this model to describe the probability of motor unit activation with increasing joint angle.

The authors claimed that the parameters obtained from this activation function model provided useful analogues to reflex stiffness and reflex threshold for each of the two muscle groups. Torque measurements were used to identify the model parameters and EMG data were used to validate the model. A non-linear test was used to estimate the parameters. Overlays of the model activation functions and the corresponding measured activation functions (rectified smoothed EMG) were used to confirm the validity of using torque responses to estimate the activation functions of the stretch reflex.

In 2001 Schmit and Rymer used this model to identify static and dynamic components of reflex sensitivity in spastic elbow flexors in 13 hemiparetic brain injured individuals. Ten randomly applied extension velocities were applied and the resulting reflex torque response was plotted as a function of elbow angle and fitted with a mathematical model designed to depict elbow flexor activation (Schmit 2001).

Their results showed that four of the six model parameters (α , μ , $\beta_{1,2}$ and $K_{1,2}$) were independent of test velocity (static). In 73% of the cases involving the other two model parameters were dependent on velocity of joint extension (dynamic).

The effects of changing activation function model parameters on the torque-angle response are shown in figure 24.

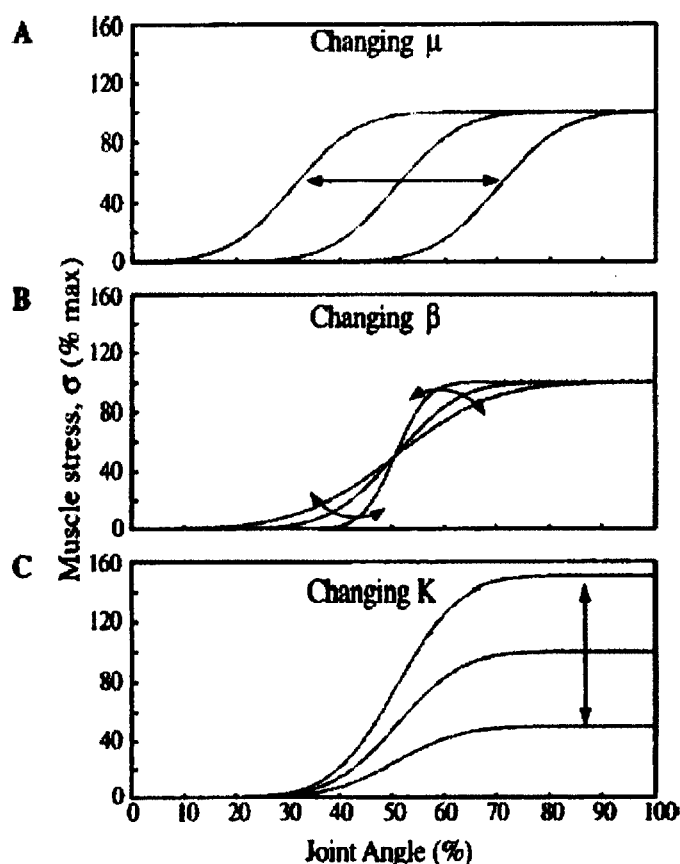


Figure 24: The effects of changing the activation function model parameters on the torque/angle response for each parameter (Schmit 2001).

In 2000, Schmit and colleagues modified their protocol to study the stretch reflex adaptation in elbow flexors during repeated passive movements in unilateral brain-injured people (Schmit, Dewald, & Rymer 2000). Elbow torque position, velocity and EMG of biceps, brachioradialis and triceps muscles were recorded for each flexion and extension movement. The stretch reflex torque was calculated by subtracting passive torque from total elbow torque, as in their previous protocol. Seven hemiparetic brain-injured subjects participated in 2 to 9 sessions. They found that repeated externally imposed sequential flexion extension movements of the elbow decreased the elbow flexor stretch reflex in six of seven subjects with significant variations in the degree of adaptation between subjects. The authors hypothesise three possible mechanisms for the reduction for “spastic hypertonia” with repeated stretches: 1) mechanical creep of muscle and joint connective tissues (i.e. changes in mechanical properties due to repeated or prolonged loading, similar in principle to the thixotropic properties mentioned by other authors); 2) spindle afferent rate adaptation (i.e. changes in the receptor sensitivity

linked to prior intrafusal muscle fibre stretch or contraction); and 3) central neural mechanisms (i.e. progressive reduction of motor neuronal or interneuronal excitability in the spinal cord due to changes in the intrinsic membrane properties after prolonged excitation).

Among the conclusions the authors drawn from these studies are the evident significant short-term beneficial effect of repeated joint movements on spastic hypertonia and, more importantly, the non-constant variability in the adaptation of the reflex torque within and between sessions.

In 2001 Kamper and colleagues used the Biodex to evaluate the effect of muscle biomechanics on the quantification of spasticity. They compared the reflex responses of the elbow and metacarpophalangeal (MCP) flexor muscles in eight hemiplegic subjects following stroke (Kamper, Schmit, & Rymer 2001). Similarly to the study by Schmit and colleagues, they used estimated biomechanical parameters to convert measured reflex joint torque and joint angle into composite flexor muscle stress and stretch. In this study they found that the stretch reflex response for the MCP had a 74% greater mean stiffness modulus than that for the elbow muscle group and that the reflex threshold was initiated at an 80% shorter mean muscle stretch. They concluded that biomechanical parameters of muscle do appear to have an important effect on the stretch reflex in individuals with impairment following stroke. The stretching velocities in this study were 60, 75 and 90 degrees/s for the elbow and 200, 250 and 300 degrees/s for the MCP joints.

During all these studies (Kamper, Schmit, & Rymer 2001; Schmit, Dhaher, Dewald, & Rymer 1999; Schmit 2001; Schmit, Dewald, & Rymer 2000) the authors mention that the reflex torque was calculated by subtracting passive torque measured at 6 degrees/s from total elbow torque. Nevertheless they mention that in some cases muscle activity was elicited even at these low velocities and they discarded such measurements until no EMG activity was evident. These studies failed to determine the reflex torque observed at these slow velocities. This calculation would have validated their methodology and definitely be able to estimate the relationship of the torque and the EMG independently of biomechanical properties of the muscle.

Some of the drawbacks using this commercial equipment are the size for storage and the specialised knowledge required to use and develop the models described. Also, patient selection may be difficult due to the limitations of patient positioning due to reduction in range of movement. Nevertheless, this type of settings is meant to be for experimental purposes and routine clinical techniques may develop from these studies. Furthermore, no data from non-impaired volunteers has been reported, namely because of their theoretical absence of stretch reflex responses at these velocities. However, there is no mention of trials corroborating this fact in any of these papers.

4.2.3.4 Controlled torque methods

These methods consist in using an external device to move the limb with a certain force/torque that it is maintained constantly, regardless of the reaction of the spastic limb. In this method the amplitude of the movement is limited to the reflex activity, which is velocity and length dependent, the higher the spasticity, the lower the amplitude of motion.

This type of device is not very common due to the complexity of the instrumentation and control design required to maintain a constant torque in relation to the angle changes, particularly when the gravity component is variable.

Particular caution has to be taken when using this kind of principle since an applied torque of high magnitude could cause discomfort and pain and, if the applied torque is too low, it may not be necessary enough to generate movement and assess spasticity.

Brown and colleagues (1987) aimed to identify the neurological aspects of hemiplegia related to the child's ability to use the hand for skilled manipulation. For this study, the authors designed a study protocol where 25 hemiplegic children were selected and examined in detail clinically and by neurophysiological techniques. One of these techniques was aimed to assess muscle tone (Brown et al. 1987). The technique consisted in grading from low tone through normal tone to a mild, moderate or severe resistance to stretch was made for five groups of muscles: thumb adductors, long finger flexors, wrist pronators, wrist flexors and biceps, giving a possible total score of 25. Fifteen of these children were further investigated by "sophisticated neurophysiological techniques". These consisted of a device used to apply a given torque at increasing

frequency to the limb. The torque can be increased and the test repeated with different preset forces allowing measurement of resonant frequency that the authors define as the frequency at which the most rapid speed and distance of excursion occurs for a given force and the joint range of the wrist in radians produced at the wrist by a given force. Another variable that can be measured is the maximum speed of voluntary movement at the wrist by getting the child to wiggle the motor as fast as possible. Besides the resonant frequency measurement, EMG electrodes were placed over the origin of the flexor and extensor muscles of the forearm. This allowed observing whether muscles could completely relax during passive movements and whether the stretch reflex activation was phasic or tonic.

In their results, as expected muscle tone was usually increased on the hemiplegic side although occasionally there was a decrease in tone in the affected limb, the authors attributed this increased tone to either spasticity or rigidity. Nevertheless an important observation from the authors was that not all hypertonus is due to spasticity and explains that in some cases there could be severe hypotonia and very brisk reflexes and in others there could be severe hypertonia and absent reflexes. However they do not mention the possibility of other factors influencing the observed hypertonia.

The authors observed a significant increase in the resonant frequency between the normal and hemiplegic sides ($p < 0.01$). Also, a relative good correlation of (0.729) was observed between the resonant frequency and the clinical assessment of muscle stiffness.

It was observed in the EMG studies that 10 children showed phasic responses to sudden stretch and only four showed continuous tonic activity during the period that the stretch was maintained.

In this paper, Brown and colleagues make reference to muscle “thixotropy” which is defined as the resistance to stretch occurring in a normal muscle after a period of rest which fades away after stretching the muscle to the full range of motion. In this study it occurred in 10 out of 15 children although the authors mention that exaggerated thixotropy does not appear to be a significant cause of increased resistance to passive movement.

Although in this study the assessment of muscle tone is vague, the authors address the important issue of changes in the muscle biomechanics and the increased muscle tone due to anxiety and failure to relax as well as the pathological hypertonus that is claimed to be associated to spasticity.

This latter method was used later in 1994 (Iloeje 1994) to measure muscle tone in children with cerebellar ataxia. The outcome measurements in this study were the torque, displacement, velocity and acceleration. The resonant frequency of the wrist, proportional to the muscle stiffness, was used as a measure of muscle tone. Adding EMG measurements to this methodology, it can be adapted to measure spasticity.

A custom built device was used in another study to evaluate wrist control dynamics generated by the wrist flexors and extensors in children with CP (Benson et al. 1990). Their device consists of a computer controlled DC servomotor with torque and displacement sensors, signal conditioning electronics and data acquisition and analysis system. The signals recorded were: flexion and extension torque; angular position; EMG signals from flexors and extensors. The protocol consisted of multiple isometric power tests to obtain the maximum voluntary contraction in flexion and extension (three positions, neutral, 40 degrees palmar flexion and 40 degrees dorsiflexion).

No significant differences were found between the isometric contraction results between the unaffected side of the children with CP and the control children. The affected side however, showed lower torques during both flexion and extension (73% weaker). This abstract does not mention any results of spasticity measurements. Nevertheless the design is potentially usable for the quantification of RTPM.

Another study describes a protocol using a custom design device consisting of a handle which was attached via a cable and a pulley to the shaft of a torque motor (Crago, Houk, & Hasan 1976) (Figure 25). This protocol studies the characteristics of the stretch reflex of the elbow flexor muscles. The subject holds the handle while the shoulder is stabilised against a brace. The background tension of the cable is set at a level that the subject is required to resist. The motor was programmed to deliver random mechanical disturbances while the stretch reflex was evaluated from EMG responses. Increased

force stretched the biceps and produced an increase in EMG whereas decreased force allowed biceps to shorten and produce either an EMG decrease or increase.

This method, although useful and viable to register stretch reflex responses is not likely to be used to measure spasticity since the amount of voluntary interaction may not be suitable for people with neurological impairment. Modifications to the protocol however may be adapted for spasticity assessment.

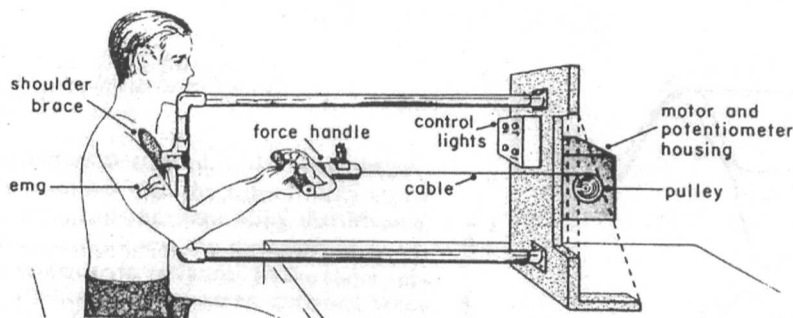


Figure 25: Custom made device designed to elicit stretch reflexes. The subject grasps the handle which is attached to a pulley on the motor shaft by way of a steel cable. A shoulder brace was included to minimise body movement. A computer was programmed to control the motor current and instruction lights. The following variables were recorded: EMG from biceps, arm force from a transducer in the handle, arm deflection from a potentiometer in the motor housing, motor current from the servo amplifier (Crago, Houk, & Hasan 1976).

4.2.4 Discussion

This chapter was aimed at introducing the state of the art techniques used to quantify/assess spasticity. These techniques were divided in clinical, neurophysiological and biomechanical techniques. Table 8 summarises the advantages and disadvantages of each group.

Clinical techniques are easy to implement but are subjective and not standardised. They depend on the level of training of the assessors and their experience. Furthermore, they are mainly directed to measure the resistance to passive movement of the limb without considering certain confounding factors that may mask the neurophysiology of the problem.

Neurophysiological techniques address more theoretical and direct issues regarding the pathophysiology of spasticity. Nevertheless, they are difficult to implement, time consuming and in most cases invasive which may be inconvenient for the patient.

Biomechanical techniques are potentially standard as they produce repeatable movements and/or perturbations. However if only used with physical variables as outcome measurements there is the risk of falling onto the same predicament of the clinical techniques where biomechanical soft-tissue changes will mask the effects of neurological contributions. Most of the papers reviewed in this section use EMG recordings. These recordings are definitely useful for the estimation of the neurological contributions. However when it comes down to presenting the results EMG recordings were only used as a way to ensure that the subjects were relaxed prior the perturbation. Only few studies refer to the EMG signals and their activation during the perturbation analysing latencies, areas and amplitudes. Others go one step further and attempt to model this neurophysiological response and relate it to the torque changes observed. Nevertheless, such models and approximations are generalised responses and may or may not be applied for every subject.

It is evident that a combination of these techniques is necessary to address as many factors contributing to hypertonia as possible. Biomechanical techniques are, in principle, based on the clinical techniques. They, measure, in one way or another the resistance to passive movement around a joint. Adding physiological recordings to ensure that the resistive torque observed is a consequence of muscle activation and not passive properties is imperative as it will define the treatment or therapy to rehabilitate the patients.

More research is evidently necessary in order to address the origins of the pathophysiology of spasticity and dissociate between neural and non-neural components of hypertonia. Also, more general models including other muscle groups, neurophysiological interactions between the different structures and the feed-forward mechanisms are required.

Nevertheless, Dietz (2003) has argued that recent advances in the concepts of spasticity, although well established scientifically, there has been little transfer to clinical practice and suggests that scientific research results should be translated into an understandable and pragmatic format and should initiate the development of new forms of treatment (Dietz 2003). In a more recent study, Sommerfeld and colleagues (2004) did not find a strong correlation between levels of disability and the presence of spasticity in stroke

patients. They concluded that the focus on spasticity in stroke rehabilitation is out of step with its clinical importance (Sommerfeld et al. 2004). These points of view, although valid, fail to acknowledge the importance of basic research towards the understanding of the underlying mechanisms of motor control, which may eventually help to reduce disability resulting from altered motor control. Furthermore, Sommerfeld and colleagues used the Ashworth scores to assess spasticity, which have shown to be unreliable techniques for the measurement of spasticity.

Table 8: Summary of the advantages and disadvantages of the different techniques used to assess spasticity.

Method	Advantages	Disadvantages
Clinical	Easy to apply	Subjective assessment
	Non-time consuming	Neural and non-neural components may be confounded
	Do not require complex instrumentation	
Neurophysiological	Direct measurement	Poor correlation between tests
	Results between different laboratories can be compared	Poor correlation with clinical status
	Quantifiable parameters	Complex procedure
		Override important structures in the reflex pathway (i.e. muscle spindle and Golgi tendon organ)
Biomechanical	Accepted by most neurophysiologists and clinical engineers	Not well-standardised methods
	Good compromise between the clinical and neurophysiological ones	Device implementation
	Objective outcomes	Errors due to device relative position to body
	Easy to apply	Sometimes uncomfortable for the patient
	They evoke natural responses	May also be confounded by non-neural components

5 Materials and Methods

5.1 Feasibility Study

5.1.1 Introduction

Muscle tone has both biomechanical (soft tissue properties) and neural (stretch reflex properties) components which are affected after a CNS lesion and develop, if not at the same time, very close in time with each other (chapter 3).

Previous attempts to assess or measure spasticity have relied on the principle of measuring resistance to passive movement (RTPM) to evaluate muscle tone. Even when this is true in most of the cases of people with upper motor neuron (UMN) syndrome, the contribution of each of the components of muscle tone is not clearly distinguished, reaching the point of neglecting the soft tissue changes (e.g. contractures) and attributing it completely to the stretch reflex hyperexcitability (chapter 4). Thus the importance of characterising the stretch reflex and outline the differences between non-impaired subjects and post-stroke patients.

5.1.1.1 Physiological signals

One way to determine the response of a particular organ is registering its electrical activity. EMG signals are the electrical representation of muscular activity (contraction). This activity can be voluntary or involuntary. Because fewer synapses are involved, reflexes are faster than voluntary movements (chapter 2). The delay between the stimulus and the reflex differs according to the conduction velocities of the nerves, the number of synapses involved in the reflex pathway and the threshold of the motor neurones.

The reflex latencies and rise time may be influenced by the following factors:

- Signal transduction delays at the muscle spindle
- Length and conduction velocities in the afferent pathway
- Conduction delays at the level of the spinal cord
- Length and conduction velocity in the efferent pathways
- Delays at the motor neurone junction

In theory, assuming a conduction velocity of 60 m/s for the forearm muscles (35-50 cm to the spinal cord approx) the delays in the afferent and efferent reflex pathways will be between 12 and 20 ms approximately ($t_a + t_e$). If the signal transduction delays and synaptic delays at the motor neurone junction are ignored, then any further increase in the reflex latency can be attributed to the synapses involved in the spinal cord.

It has been seen in the literature that the latency for the stretch reflexes from the onset of a mechanical stimulus (muscle stretch) (Pisano et al. 2000) or by tendon tap (Faist et al. 1999) is between 20 and 60 ms (Hayashi et al. 2001).

It has also been mentioned that it is unlikely that activity from the monosynaptic pathways alone can contribute to force generation opposing passive movement. Therefore it is likely that such activation will occur at the polysynaptic pathways level (Sheean 1998; Sherman, Koshland, & Laguna 2000).

Based on the above, EMG signals from stretch reflexes resulting from imposed passive movement will be able to be distinguished from voluntary EMG activity. Furthermore, if the stretch reflex excitability is increased then muscular activation patterns might also change.

5.1.1.2 Kinematics

Body motion can be measured by means of the relative changes between joint segments in terms of kinematic descriptors such as angular displacement, velocity and acceleration.

Different technologies are available for measuring these variables ranging from video motion capture to specialised sensors mounted on the body segments. The decision of which one to use depends on which segments will be analysed, the complexity of the movement, cost, etc. For this project the most viable solution due to the required sampling frequency was the use of mounted sensors on the limb segments.

A passive movement of the elbow joint will result in a stretch of its flexors or extensors. As was described previously, the reflex will depend on the starting position of the limb, the magnitude of the stretch and its velocity.

To test the hypothesis based on the principles addressed in chapter two, passive movements of the elbow joint resulting in a flexor or extensor stretch were applied in three non-impaired subjects. The objective was to elicit stretch reflexes from the elbow flexors and extensors in non-impaired subjects with the aim of obtaining an EMG response as a consequence of the stretch reflex activity.

5.1.2 Methodology

5.1.2.1 Equipment

A flexible goniometer (Biometrics) was used to measure the angular changes at the joint and an EMG (DelSys) system was used to record the EMG activity prior, during and after the stretch.

5.1.2.2 Experimental protocol

- Subjects were sitting on a chair
- The each end of the goniometer was placed on the upper arm and the forearm respectively (Figure 1)
- EMG electrodes were placed on the bulk of biceps and triceps
- Random passive flexions and extensions of the elbow with the upper arm aligned with the vertical plane were imposed to the subjects while recording the EMG and angular changes

5.1.2.3 Signal processing

- Data was collected at 1024 samples per second
- All signals were filtered at 50 Hz
- EMG signals were full wave rectified and low pass filtered at 20 Hz

5.1.3 Observations

All subjects presented EMG activity resulting from the stretch perturbation.

After the data was collected and filtered it was possible to determine qualitatively four outcome measures for the stretch reflex signal (Figure 2):

1. Reflex latency
2. Rise time
3. Amplitude
4. Duration

The neurophysiological significance and the detection of these outcome measures will be explained further in the biomechanical analysis section.

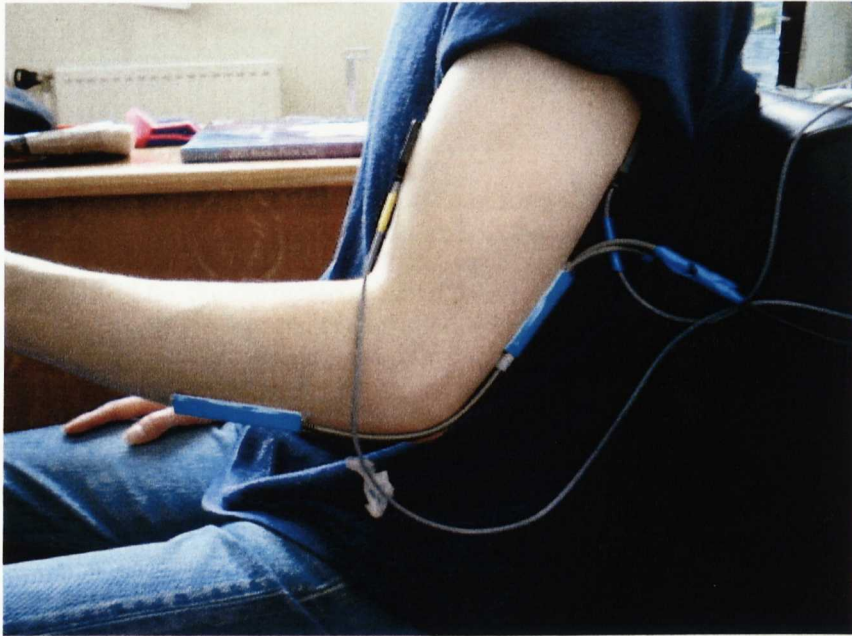


Figure 1: Experimental setup. The flexible goniometer is placed on the arm to measure the angular displacement of the elbow joint when a stretch is imposed in either flexion or extension. EMG electrodes are placed on the bulk of the biceps and triceps to record the electrical activity of the muscles before, during and after the stretch.

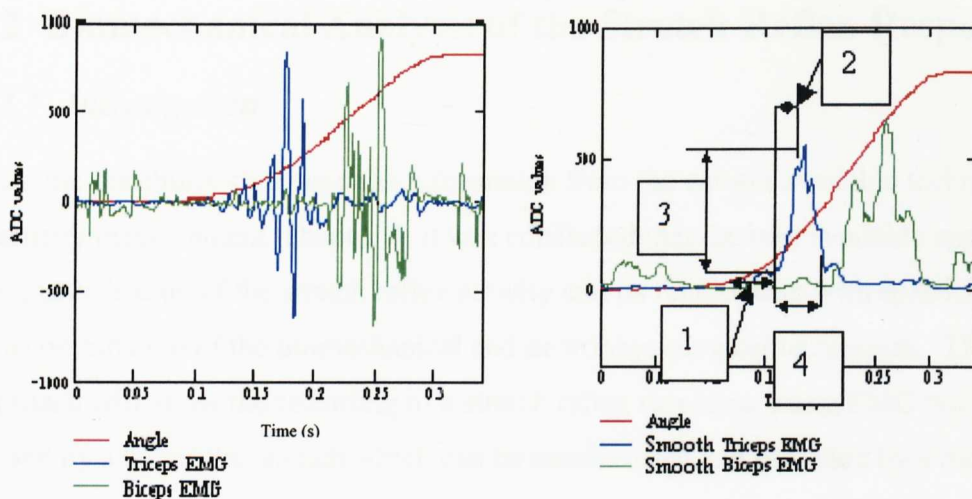


Figure 2: Manual test signals. Elbow joint angle and Raw EMG signals of biceps and triceps in imposed elbow flexion (left) showing a stretch reflex signal for triceps (blue) and a shortening reaction signal for elbow (green). Smooth EMG signals (right) from the raw data, showing the parameters obtained for the characterisation of the stretch reflex (1) latency, (2) rise time, (3) amplitude and (4) duration of the reflex response.

A major factor to consider when observing these signals is the presence of muscle activity as a result of cortical input. Hayashi and colleagues (2001) assigned constant theoretical time segments to the latency components of the stretch reflex (30-60 ms for the short component (M1) and 60-90 ms for the long component (M2)). Any activity starting beyond this time is considered to be influenced by cortical input and will not be considered as a reflex. Nevertheless, these theoretical values must be considered carefully when observing different conditions as they might vary.

5.1.4 Discussion and Conclusion

Rapid stretches to the elbow muscles are capable of eliciting stretch reflexes. EMG signals resulting from such reflex activity can be analysed and parameters related to the excitability of the stretch reflex can be obtained. Nevertheless, when the stretch is applied manually there is no way to ensure the displacement, velocity and accelerations will be always the same. The need to develop a system capable of applying a standard stretch to the elbow muscles is evident. This system ought to be non-invasive and relatively easy to use.

5.2 Biomechanical Analysis of the Stretch Reflex Response

5.2.1 Introduction

From the feasibility study and the information from the current available techniques for spasticity measurement (chapter 4) it was concluded that the best available approach for the quantification of the stretch reflex activity and its relationship with spasticity would be a combination of the biomechanical and neurophysiological techniques. This approach will allow the recording of a stretch reflex response, using EMG recordings, caused by a controlled stretch which can be standardised and provided by a mechanical device rather than a manual stretch.

5.2.2 Design

A mechanical device (Figure 3) was developed to provide a rapid standardised angular perturbation to the elbow joint (appendix E).

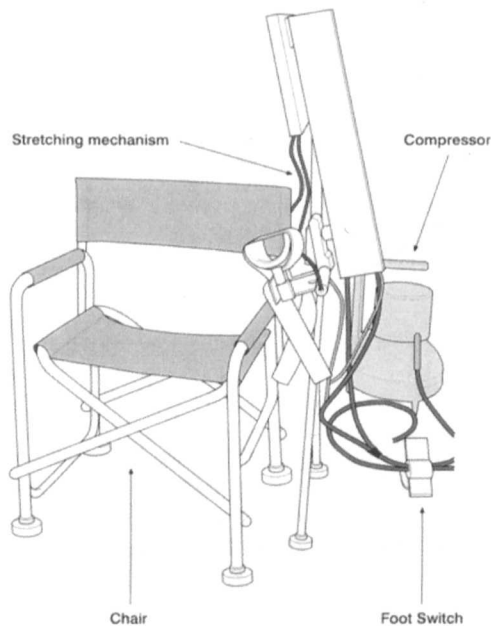
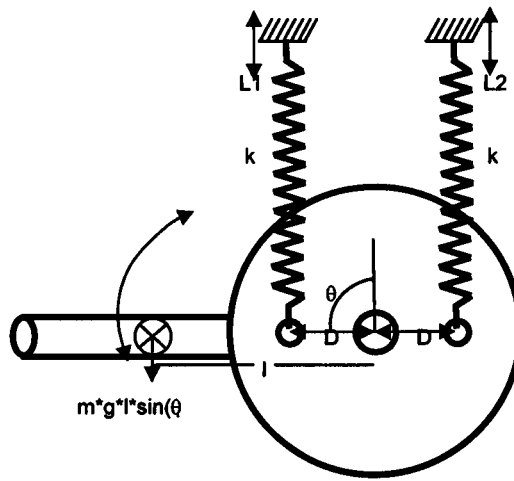


Figure 3: Diagram of the system used in the experimental setup. The footswitch releases pneumatically the locking pin inside the mechanism to apply the stretching perturbation to the elbow joint.

The mechanism consisted of rotational arm driven by two extension springs (SF-DFX; free length 300 mm; $k = 15.2 \text{ N/mm}$). A single turn potentiometer (RS-6187; $1 \text{ K}\Omega$; measured sensitivity 21.08 deg/V) records the angular displacement and an

accelerometer (Analogue devices, ADXL250; range ± 50 g; measured sensitivity 3.54 g/V) measures the angular acceleration. A load cell (RS-632-742; range ± 20 kg; measured sensitivity 2.008 kg/V) is mounted on the lever to measure the moment applied voluntarily by the subjects (appendix C).

The spring tension is set by adjusting their length with two knobs on top of the device. Figure 4 shows the schematic diagram of the spring lever mechanism. A locking pin was added to maintain the angle of the lever after the tension of the springs had been set. A foot-switch was used to release the pin pneumatically. A pressure-switch was used to detect the time when the foot-switch was pressed. The signal from this switch is used as a flag to indicate that the test has started.



$$\tau = k \cdot d \cdot \left(\frac{L1 - L2}{2} \right) \quad (\text{eq. 1})$$

Figure 4: Schematic diagram of the mechanism. The tension of the springs is adjusted by changing their length while a pin maintains the position. A footswitch was used to release the tension pneumatically causing the mechanism to rotate. The equivalent torque is dependent on the constant of the springs ($k=15.2$ N/mm), the length difference between the springs ($L1-L2$ mm) and the distance from the point of rotation ($D=30$ mm).

5.2.3 Device Modelling

The device was tested under no-load conditions (i.e. without subject) at five different initial torque magnitudes (2.28, 3.42, 4.56, 5.7 and 6.84 N.m; obtained from increasing every 5 mm the respective length of one of the springs), five times each to test its reliability and repeatability. The responses from these trials were modelled as a step input using a second order equation of the form:

$$I\ddot{\theta} + B\dot{\theta} + K\theta = -m \cdot g \cdot l \cdot \sin \theta \quad (\text{eq. 2})$$

Where θ is the angle of the lever respect to the horizontal, the subsequent time derivatives are the angular velocity and acceleration.

Assuming $\sin(\theta) \approx \theta$ for small θ the above equation can be rewritten as:

$$I\ddot{\theta} + B\dot{\theta} + (K + mgl)\theta = 0 \quad (\text{eq. 3})$$

Mechanical parameters are the elasticity (K), obtained from the spring arrangement and the moment of inertia (I) and viscous damping (B) by calculation from the oscillatory response using the following procedure:

The half period of the oscillations was calculated in MathCAD using a maximum and minimum detector using numerical differentiation twice and zero crossing detection. If the zero crossing happens from negative to positive values then it is a minimum otherwise a maximum. The time calculated between the first half period and the second half period were averaged and multiplied by two to obtain one period (T).

The damped natural frequency (ω) was obtained using the formula:

$$\omega = \frac{\pi}{T} \quad (\text{eq. 4})$$

The decreasing amplitude values from the initial position to the first maximum were used to determine the damping ratio (D) with respect of the steady state angle.

The damping ratio was then used to obtain the damping coefficient (ζ) using the formula:

$$\zeta = \sqrt{\frac{\ln(D)^2}{4\pi^2 + \ln(D)^2}} \quad (\text{eq. 5})$$

The undamped frequency (ω_n) of the oscillation was calculated using the following formula:

$$\omega_n = \frac{\omega}{\sqrt{1 - (\zeta)^2}} \quad (\text{eq. 6})$$

The moment of inertia (I) of the device was then calculated:

$$I = \frac{K}{(\omega_n)^2} \quad (\text{eq. 7})$$

The viscous element (B) was obtained using the formula:

$$B = \zeta \cdot 2 \cdot \sqrt{I \cdot K} \quad (\text{eq. 8})$$

Physical characteristics of the model were determined from the elements of the mechanism itself: mass (m) of the lever, distance between the centre of mass of the lever and centre of rotation (l), and gravity (g).

The equation describing the model can be expressed as (Ogata 1998):

$$y(t) = \left(1 - \frac{\omega_n}{\omega} \cdot e^{-(\omega_n \zeta)t}\right) \cdot \left(\cos(\sqrt{1 - \zeta^2} \cdot \omega_n)t - a \tan\left(\frac{\zeta}{\sqrt{1 - \zeta^2}}\right)\right) \quad (\text{eq. 9})$$

5.2.4 Biomechanical modelling

Biomechanical models are necessary to attempt to explain the behaviour of a system during determined activities. Biomechanical models should enable the diagnosis of movement disorders and predict the outcome of clinical intervention.

The moment of inertia of the forearm can be calculated using the data from the body mass and the length of the forearm and the distance between its centre of mass and the elbow joint (Winter 1990).

Once the moment of inertia is calculated the viscous damping and elastic parameters can be obtained from the experimental data.

Theoretically, the most important parameter that would be obtained from these measurements is the damping ratio related to the velocity of the movement as it has been proposed by Lin and colleagues (2003).

In this case the overall equation of motion can be expressed as:

$$(I + I_{arm})\ddot{\theta} + (B + B_{arm})\dot{\theta} + (K + K_{arm})\theta = -(m + m_{arm})(l + l_{arm})g \sin \theta \quad (\text{eq. 10})$$

5.2.5 Measurement protocol

5.2.5.1 Subjects

Seventeen non-impaired volunteers (mean age 35 years; range 24-55 years) and 14 patients with stroke spasticity (mean age 67 years; range 52-86 years) were recruited for this protocol. The patients were recruited from a botulinum toxin clinic at a tertiary care neurological Rehabilitation Centre. Measurements were taken on the day of botulinum toxin administration before or within the first fifteen minutes after the injection (i.e. within the window before the toxin could have any influence on the outcome measures), as it is reported that their effects are expected to occur three days after administration (Ghosh & Das 2002; de Paiva et al. 1999; Hughes 1994).

5.2.5.2 Inclusion criteria

Non-impaired subjects inclusion criteria

- No previous history of injuries to Central Nervous System (CNS)
- No previous history of Peripheral Nervous System (PNS) damage
- Normal Range of Movement (ROM) about the elbow and shoulder
- No disease causing peripheral neuropathy
- No cardiovascular abnormalities
- Sitting balance
- Ability to comply with instructions

Post-stroke subjects inclusion criteria

- Unilateral right-sided CVA leading to left-sided hemiplegia
- No previous history of Peripheral Nervous System (PNS) damage

- No upper limb contractures or UL musculoskeletal problems
- Sufficient ROM to take part in the study: Elbow full flexion to 140° extension; Shoulder full adduction to 45° abduction
- No other disease causing peripheral neuropathy
- Good sitting balance
- Ability to comply with instructions
- Ability to give informed consent (directly or via a care giver)

5.2.5.3 Experimental setup

Due to the dependency of the stretch reflex on a different number of conditions such as background activity (BGA), initial position of the limb and velocity of the stretch (chapter 2) the protocol was designed to observe the influence of these conditions.

- The arm of the subject was positioned and strapped to a lever aligning its centre of rotation with the elbow joint (Figure 5)
- EMG Electrodes were placed on the elbow flexors and extensors to register their electrical activity
- The initial position (flexed or extended) was adjusted
- The subjects were asked to maintain one of three levels of background voluntary activity (flex, extend or relax). Visual feedback using a two LED display (Figure 6) is provided for the subjects to ensure a minimum and maximum levels of background activity
- A footswitch attached to an air compressor is pressed to release pneumatically the spring tension
- Two initially applied torque values were used in this protocol:
 - 4.56 N.m
 - 6.84 N.m

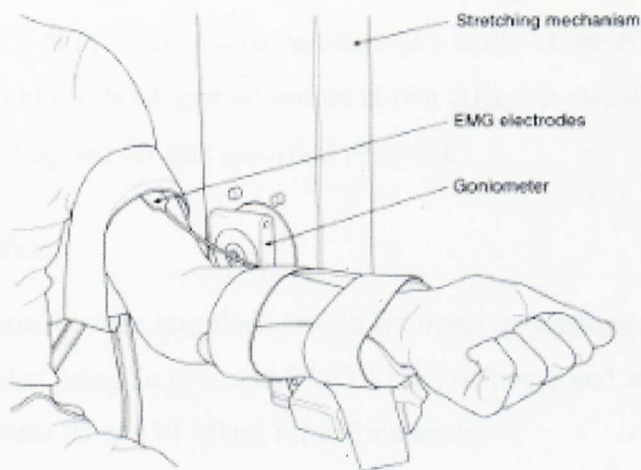


Figure 5: Experimental setup. The elbow joint is aligned with the centre of rotation of the lever. EMG electrodes are placed on the muscle and the upper arm is strapped to avoid movement and misalignment of the sensor and the joint. There are two springs in the mechanisms whose tension is determined prior the test. Once the tension is released an angular perturbation stretches the elbow flexors eliciting a stretch reflex response.

A full factorial experiment was designed to evaluate the contribution of the conditions:

- Starting angle
- Voluntary activity
- Applied torque
- Head position

Head position was an additional variable introduced when the higher torque was applied, the initial position was extended and when the subject was completely relaxed. Figure 7 shows the tree diagram with all the combinations.

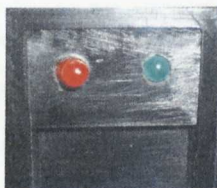


Figure 6: Visual feedback for the subjects used to help them maintain a level of background activity when flexing or extending the arm. The green LED indicates that they have reached the desired level. The red LED indicates that they have gone beyond that level. The instruction given to them was to keep the green LED without lighting the red one. For the relax conditions the instruction was to maintain both lights off.

In cases when the post-stroke subjects were not able to voluntarily activate their arms in either flexion or extension a modification to the protocol was introduced. The subjects

were asked to relax as much as possible and three additional applied torque values were added (2.28, 3.42, and 5.7 N.m). This variation allowed a study of the differences of the stretch reflex at 5 different levels of applied torque at two different initial positions. Figure 8 shows the tree diagram for this modified protocol.

5.2.5.4 Data acquisition

All signals for both protocols were sampled at 4096 Hz using an analogue to digital converter (ADC), recorded using DelSys and EMGWorks software and stored on a desktop computer (Pentium III at 450 MHz) for offline analysis.

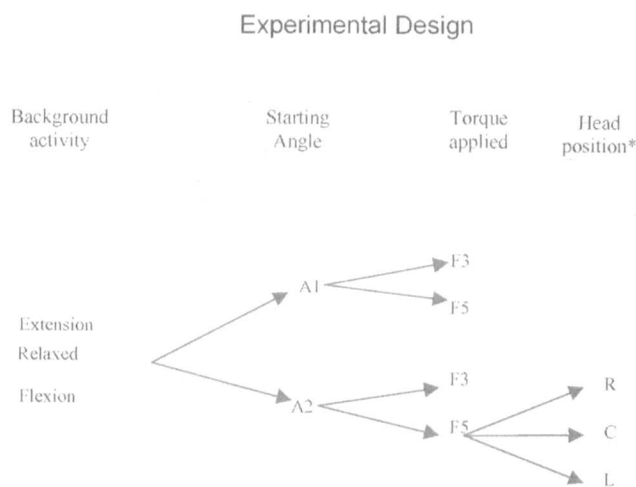


Figure 7: Tree diagram representing the full factorial design involving the conditions considered to affect the stretch reflex excitability for the mechanical device protocol. Background activity performed by the subject takes the values of extension, relaxed and flexion. Imposed movement refers to the direction of the perturbation produced by the mechanical device which can be in flexion or extension. Starting angle refers to the starting angular position of the limb, A1=extended and A2=flexed degrees respect to the vertical. The Force applied is changed by adjusting the length of the springs (F3= 4.56, F5=6.84). *The head position is changed by asking the subjects to keep it centred (C), turn it to the left (L) or to the right (R). This is only for an extension movement in the extended position for the higher applied torque.

5.2.5.5 Data analysis

5.2.5.5.1 Stretch reflex parameters

An automated analysis program in MathCAD was designed to process the obtained signals (appendix D).

EMG signals were full-wave rectified and low-pass filtered at 20 Hz using a 4th order Butterworth filter to obtain the envelope of the signal.

The range of the rectified EMG and the mean and standard deviation of the filtered EMG levels were registered for 100 ms before the perturbation were calculated.

The maximum amplitude of the rectified EMG after the onset of the perturbation was registered and subtracted from the range of the rectified EMG before the perturbation.

A three standard deviation threshold of the mean filtered EMG during the 100 ms prior to the perturbation was used as criterion to determine when the EMG was active (Russell et al., 2002).

The maximum peak of the filtered EMG was detected and the signal was tracked backwards and forwards until the three standard deviation threshold was reached to detect the onset and termination of the stretch reflex activity

The starting of the angular displacement was detected using a threshold of 0.25 degrees relative to the initial position.

From this processing and the angular displacement it was possible to obtain a group of parameters that can be considered to be related to the stretch reflex excitability (Figure 9). These parameters are:

1. Stretch reflex amplitude- obtained from the maximum peak value of the rectified EMG signal can be related to the activity of the α -motor neurone, the intensity of the muscle activity and to the force produced by the muscle
2. Stretch reflex latency- obtained from the start of the stretch to the start of the filtered EMG signal could indicate the conduction velocities of the reflex pathways, presynaptic inhibition and/or the excitability of the alpha motor neurone pool
3. Stretch reflex rise time- obtained from the start to the maximum points in the filtered EMG signal would be proportional to the number of oligosynaptic (i.e. a few synapses more) connections in the circuitry

4. Stretch reflex duration- obtained from the start to the end points in the filtered EMG signal is an indicator (along with the amplitude) of the intensity of the muscle activity and the presence of long latency components

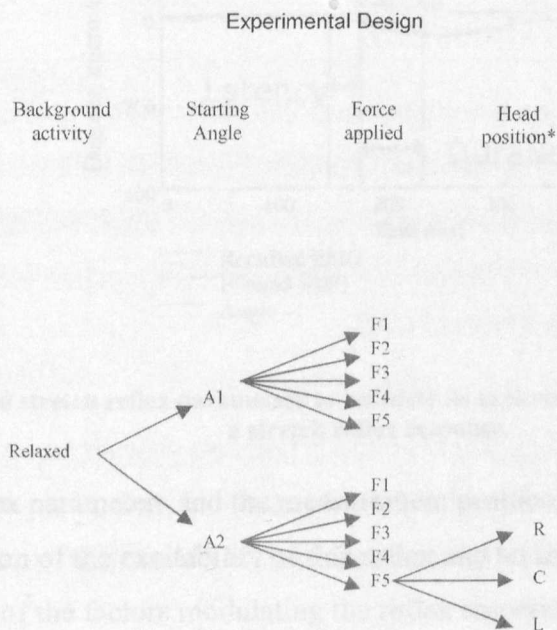


Figure 8: Tree diagram representing for the modified protocol for the post-stroke subjects who were not able to voluntarily activate the elbow flexors and extensors. The modifications include no variation in the background activity performed by the subject (only relaxed). Imposed movement refers to the direction of the perturbation produced by the mechanical device which can be in flexion or extension. Starting angle refers to the starting angular position of the limb, A1=extended and A2=flexed degrees respect to the vertical. The Force applied is changed by adjusting the length of the springs (F1= 2.24, F2=3.42, F3=4.56 F4=5.7 and F5=6.84). *The head position is changed by asking the subjects to keep it centred (C), turn it to the left (L) or to the right (R). This is only for an extension movement in the extended position for the higher applied torque.

5.2.5.5.2 Statistical analysis

Analysis of variance was used to determine the differences and similarities of the stretch reflex parameters above mentioned within and between the populations. In addition, a general linear model was used to determine the influence of the interaction of the initial conditions upon the stretch reflex excitability and determine if a particular condition alone or a combination of them affect such parameters.

5.2.6 Discussion

The methodology described in this chapter uses the information provided in previous chapters and aims to take into account the neurophysiological mechanism that are responsible for the stretch reflex.

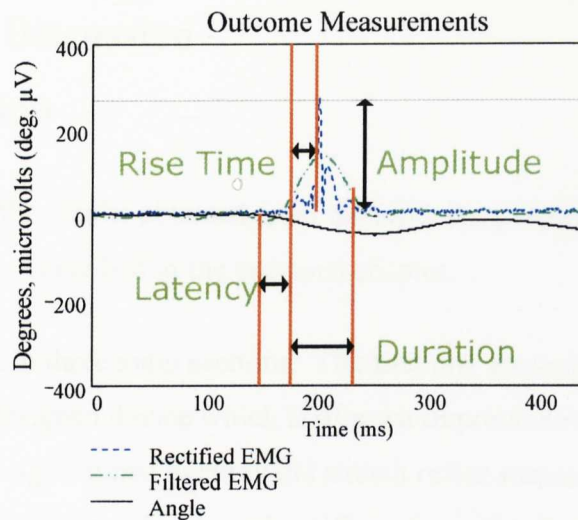


Figure 9: Proposed stretch reflex parameters to estimate its excitability in a typical EMG signal from a stretch reflex response.

The stretch reflex parameters and the measurement protocol proposed are considered to give an estimation of the excitability of this reflex and an insight to its variability by analysing some of the factors modulating the reflex response.

Other factors that may have an influence on the stretch reflex excitability were not considered mainly for the difficulty of measuring or standardising them. These factors, such as attention span and relaxation levels, bladder pressure, temperature, etc., have influence on the feed forward mechanisms.

Further research is still necessary to include the assessment of such factors and better estimations of their role towards stretch reflex modulation.

6 Results and Discussion

6.1 Introduction

This chapter presents the results obtained from the modelling technique and measurement protocols described in the previous chapter.

The chapter is divided in three main sections. The first one presents the mechanical model of the custom designed device which is of great importance for this work and future experimental designs aimed to study the stretch reflex responses and their variability between and within subjects under different experimental conditions and subjects characteristics. The second section uses the biomechanical parameters from experimental data available in the literature and adds them to the original mechanical model to simulate the mechanical response of the whole system. The third section and most important for this work looks at the stretch reflex parameters obtained from a non-impaired and a post-stroke populations and compares the variability between the different conditions between and within each population.

These results suggest that some of the stretch reflex parameters described in the previous chapter are influenced by the factors considered in the measurement protocol up to a certain extent and hypothesises the influence of other factors of physiological origin based on the existing literature. These statistical analysis needs to be taken with caution due to the limitation of the number of subjects and their variability with each other.

6.2 Modelling

6.2.1 Device modelling

The importance of this model was two fold. First to ensure that the mechanical perturbation to be applied to the elbow joint would be consistent for all volunteers and second to suggest the development of a biomechanical model of the elbow joint capable of determining its viscoelastic properties and their interaction with muscle activity evaluating the EMG signals. It is of particular importance to evaluate the viscous damping, proportional to the velocity and study the alleged velocity dependency of spasticity.

The response of the device was modelled using the second order equation previously explained:

$$I\ddot{\theta} + B\dot{\theta} + (K + mgl)\theta = 0 \tag{eq. 1}$$

The mechanical parameters for the device were found to be for the elastic coefficient (K) of 27.3 N.m.rad⁻¹, obtained from the properties and configuration of the springs, the moment of inertia (I) of 0.018 kg.m² and the damping coefficient (B) of 0.075 N.m².s.rad⁻¹, both obtained from the data output model and the experimental data. The value added to the elastic coefficient by the mass and gravity factors was within the 10% of the elastic coefficient and considered to be negligible.

The angular displacements from the experimental trials at 5 initial applied torques (2.28, 3.42, 4.56, 5.7 and 6.84 N.m) and model behaviour for each applied torque value along with their correlation coefficients are shown in Figure 7.1. From this figure it can be observed that the velocity of the stretch is related to the applied torque.

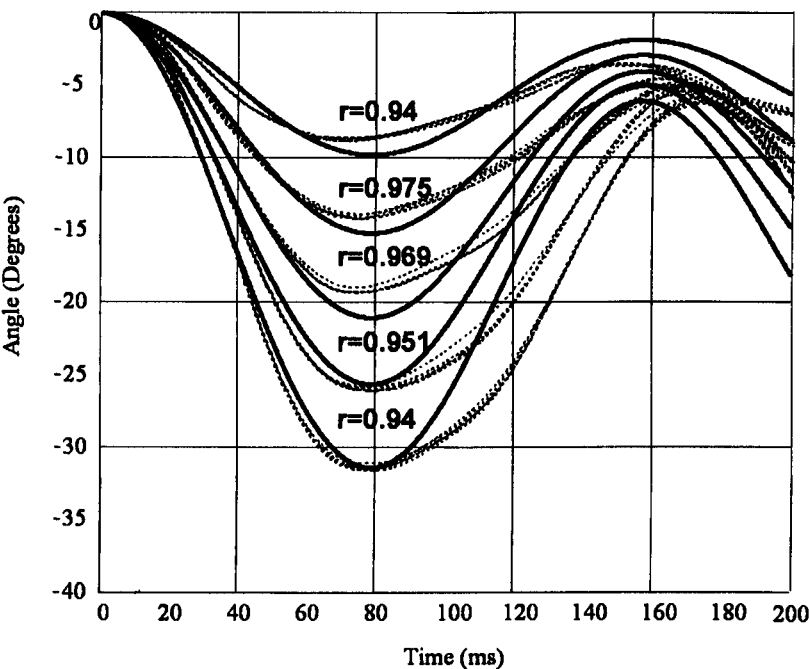


Figure 1: Angular displacements from the five experimental data (dotted lines) and the model (bold lines) for each of the 5 inputs of the device alone. Correlation coefficient values for each input are shown above each line. This model will be useful to develop a complete biomechanical model for the elbow joint given anthropometric values and potentially add the interaction with EMG responses.

The high correlation of the model with the experimental data suggests that the device is useful in a routine research environment since it can provide standardised perturbations dependent on the initial applied torque.

6.2.2 Biomechanical modelling

Since the main objective of this work was to characterise the stretch reflex response by means of the EMG signal recorded after stretching the muscle, the biomechanical modelling of the elbow joint was not of main concern at the time of measurement and anthropometrical data were not registered. Nevertheless, data from other studies can be incorporated into the model of the device simulating tests with these values.

From Lin and colleagues (2001) the range of the stiffness constant of the normal subjects ranges from 1.01 to 4.42 Nm/rad compared to published data ranges from 0.74-2.2 Nm/rad and the damping coefficients from 0.11-0.79 N.m.s/rad against published data of 0.1-0.3 N.m.s/rad for non-impaired subjects. However in this paper the authors do not provide any information about values of the moment of inertia (I) of the forearm used. Therefore a generalised limb is obtained using the anthropometrical approximation described in (Winter 1990) .

This technique uses the following equations:

$$F_m = 0.016 * B_m$$

Where F_m is the Forearm mass and B_m is the Body mass.

$$I_{fa_{elbow}} = F_m * l_{fa}^2 * 526^2$$

Where F_m is the Forearm mass, $I_{fa_{elbow}}$ the moment of inertia about the elbow joint and l is the length between the elbow joint and the elbow centre of mass.

Assuming an individual weighs 81.6 kg with a forearm length of 25.4 cm and the distance between the elbow joint and the centre of mass is of 10.9 cm using the above equations the forearm moment of inertia about the elbow joint will be:

$$I_{fa_{elbow}} = 0.0233 \text{ kg m}^2$$

The elastic stiffness and damping coefficients suggested for this virtual model are obtained from the midrange of values reported by Lin and colleagues¹ (Lin et al. 2001).

Damping coefficient $_{fa} = 0.39 \text{ N.m.s/rad}$

Elastic stiffness $_{fa} = 2.5 \text{ Nm/rad}$

6.2.3 Full model

The model of the device was introduced in VisSim. The response of the model to an initially applied torque of 6.84 Nm and the angular displacement and acceleration obtained from the actual experimental trial using the same initial torque are shown in figure 2.

In a similar way, the parameters from the hypothetical limb and the parameters provided by Lin and colleagues (2001) were added as a system in parallel. The response to the initially applied torque of 6.84 Nm simulating what the response would look like if a subject with the physical properties above shown would be measured is shown in figure 3.

6.2.4 Discussion

For this model to be completely functional it is required to estimate the anthropometric measurements of the volunteers to obtain the moment of inertia of the forearm. Using experimental data and the system identification technique it is possible to obtain the elastic constant and damping coefficient that best describe the forearm displacement. Thus determining the muscles viscoelastic properties and, using more advanced system identification techniques, estimating non-linear properties of the muscle, in particular the influence of muscle activity in the changes in the viscoelastic properties of the muscle where the level of muscle activation and respective electromechanical delay will be time variant.

¹ The relationship between the midrange values is assumed to be physiologically feasible

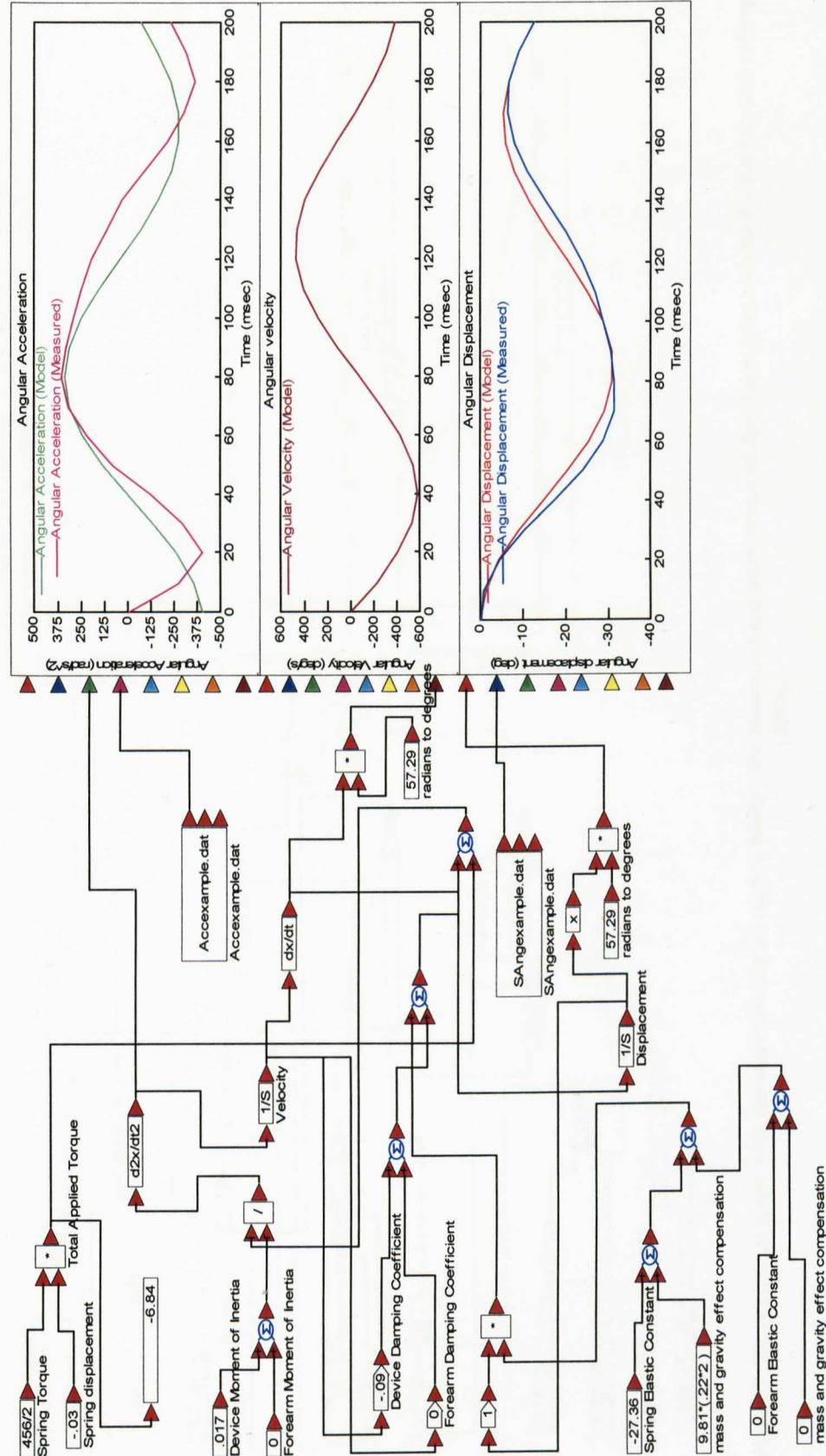


Figure 2: Simulation of a no load test with the parameters obtained from the mechanical model compared with one of the measurements (angular displacement and angular acceleration) the angular velocity is obtained from integrating the acceleration signal.

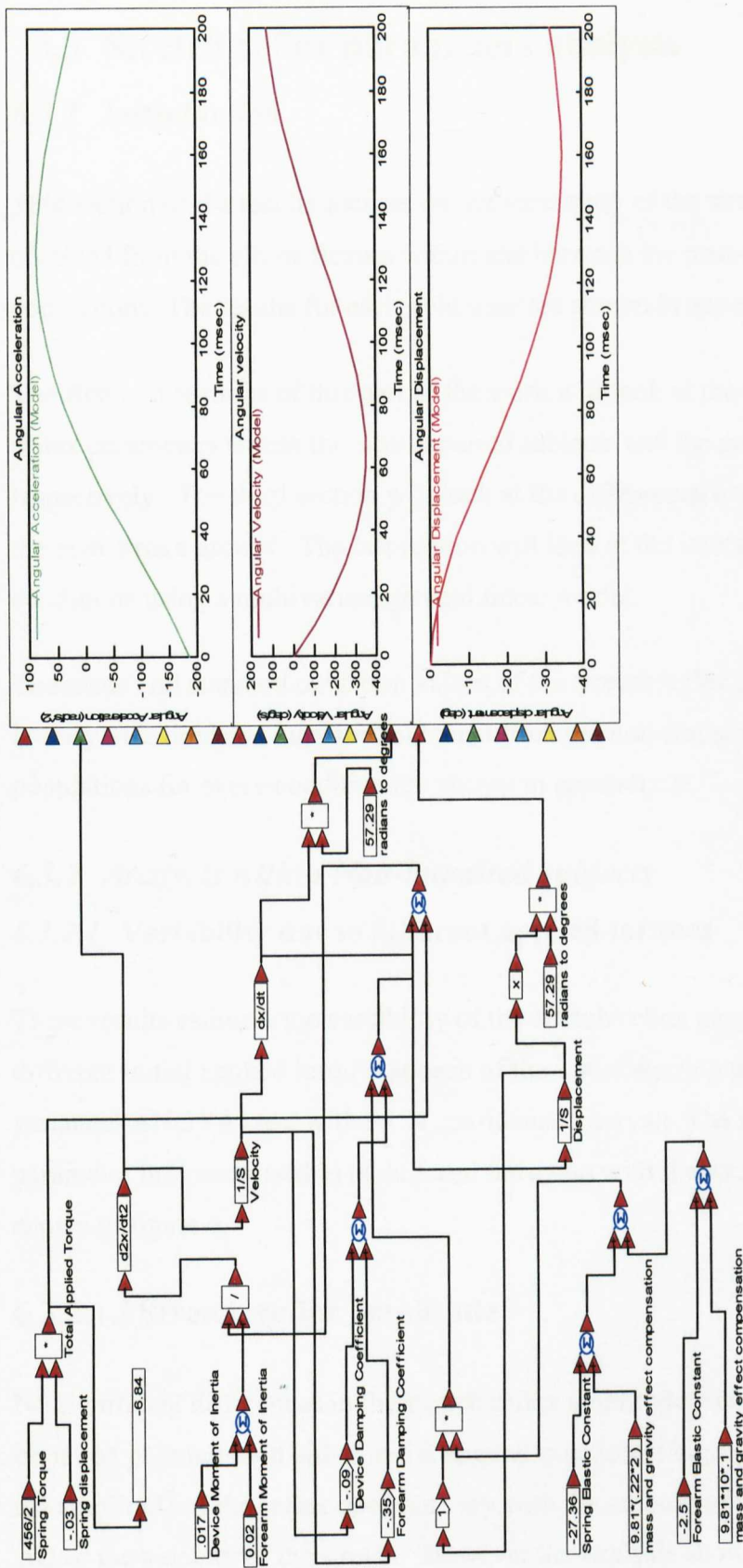


Figure 3: Simulation of a test with the mechanical parameters adding the biomechanical parameters of the elbow joint obtained from Lin and colleagues (2003).

6.3 Stretch reflex parameters analysis

6.3.1 Introduction

This section of the results focuses on the variability of the stretch reflex parameters obtained from the elbow flexors within and between the post-stroke and non-impaired populations. The results for each volunteer are shown in appendix A.

The first two sections of this part of the work will look at the differences of the stretch reflex parameters within the non-impaired subjects and the post-stroke subjects respectively. The third section will look at the differences between the non-impaired and the post-stroke groups. The last section will look at the interaction between the conditions using a multivariate general linear model.

The mean and standard deviation values of the stretch reflex parameters (amplitude, latency, rise time and duration) obtained from the non-impaired and post-stroke populations for every condition are shown in appendix B.

6.3.2 Analysis within Non-impaired subjects

6.3.2.1 Variability due to different applied torques

These results estimate the variability of the stretch reflex parameters between the different initial applied torques at each of the initial starting positions using an analysis of variance (ANOVA) test with 95 % confidence interval. The statistical analysis for each parameter is summarised in table 1 and the mean with the standard deviation plot is shown in figure 4.

6.3.2.1.1 Stretch reflex amplitude

No significant differences in the stretch reflex amplitude were found within the non-impaired population in either initial angular position at high or low torque suggesting that the amplitude of the reflex does not vary with the amount of torque applied to the joint and/or the velocity of the stretch. However the analysis shows a tendency for the amplitude to be higher when the lower torque is applied at the flexed position ($p=0.075$) (Table1) (Figure 4a).

This latter tendency does not seem to be congruent with the stretch reflex properties, however the values are not statistically significant and the difference can be attributed to other factors probably originated by feed-forward mechanisms modulating the stretch reflex response. Such modulation is expected to occur in people with intact nervous systems, unlike to people with spasticity.

6.3.2.1.2 Stretch reflex latency

- Extended position

Significantly shorter stretch reflex latencies were found at the lower torque (23.52 ± 10.31 ms) when compared with the values found at the higher torque (37.58 ± 15.91 ms) ($p=0.008$) at the extended position (Figure 4b).

- Flexed position

Significantly longer stretch reflex latencies were found at the lower torque (41.44 ± 16.53 ms) when compared with the values found at the higher torque (21.64 ± 15.94 ms) ($p=0.004$) (Figure 4b).

The results of the latency at the flexed position seem to be consistent with the velocity dependent property of the stretch reflex. However at the extended position this is not the case and it is even opposite. Nevertheless it is possible to theorise an explanation for these results considering the Golgi tendon organ activity. Golgi tendon organs are sensitive to the level of tension (chapter 2). This level of tension may be relatively higher with the elbow at in the extended position in comparison with at the flexed position (Black-Schaffer, Kirsteins, & Harvey 1999; Davies, Petit, & Scott 1995; Gregory et al. 2002). This tension would be further increased when the higher torque is applied compared to the lower one and would mask the muscle response to the muscle spindle input.

6.3.2.1.3 Stretch reflex rise time

No significant differences in the stretch reflex rise time were found within the non-impaired population in either initial angular position suggesting that the rise time is not

influenced by the initial applied torque and/or velocity of the stretch in a non-impaired population (Figure 4c).

6.3.2.1.4 Stretch reflex duration

- Extended position

Significantly shorter stretch reflex durations were found at the lower torque (65.51 ± 17.32 ms) when compared with those found at the higher torque (90.17 ± 31.12 ms) ($p = 0.013$) (Figure 4d).

- Flexed position

Significantly shorter stretch reflex durations were found at the lower torque (88.59 ± 25.32 ms) when compared with those found at the high torque (131.12 ± 61.58 ms) ($p = 0.019$) (Figure 4d).

The stretch reflex duration is directly related to the tonic component of the stretch reflex (i.e. higher activity at longer and or faster displacements) and as it can be seen in these results, the reflex lasts longer when higher torques (i.e. faster velocities and longer displacements) are applied both in the flexed and the extended positions. These results are consistent with the concept of the stretch reflex and the behaviour of the muscle spindle (chapter 2).

6.3.2.2 Variability due to different initial angular position

These results estimate the variability of the stretch reflex parameters between the different initial starting positions at the each of the initial applied torques using an analysis of variance (ANOVA) test with 95 % confidence interval. The statistical analysis for each parameter is summarised in table 2.

Table 1: Statistical analysis determining the differences of the initial applied torque within the non-impaired population when the initial position is flexed or extended. The red cells represent the significantly different values with 95% confidence. Yellow cells represent the values that tend to be different but do not have statistically significance.

Parameters	Initially extended		Initially flexed	
	F	Sig.	F	Sig.
Stretch reflex Amplitude	0.377	0.544	3.44	0.075
Stretch reflex Latency	8.095	0.008	10.131	0.004
Stretch reflex Rise Time	1.35	0.255	2.405	0.133
Stretch reflex Duration	6.975	0.013	6.282	0.019

6.3.2.2.1 Stretch reflex amplitude

- Low Torque

No statistical differences were found in the stretch reflex amplitude values between the two initial starting positions (Figure 4a).

- High Torque

Significantly higher stretch reflex amplitudes were found at the extended position ($264.79 \pm 158.64 \mu\text{V}$) when compared with those found at the flexed position ($140.76 \pm 140.97 \mu\text{V}$) ($p = 0.039$) (Figure 4a).

The differences in the amplitude at the high torque can be explained from the possible increased sensitivity of the muscle spindle at the extended position, increasing the motor neural drive to the muscle. Stretch reflex values at the low torque, although not statistically significant, show a similar tendency.

6.3.2.2.2 Stretch reflex latency

- Low Torque

Significantly shorter stretch reflex latencies were found at the extended position (23.52 ± 10.31 ms) when compared with those found at the flexed position (41.44 ± 16.53 ms) ($p = 0.002$) (Figure 4b).

- High Torque

Significantly longer stretch reflex latencies were found at the extended position (37.58 ± 15.91 ms) when compared with those found at the flexed position (21.64 ± 15.94 ms) ($p = 0.013$) (Figure 4b).

These stretch reflex latencies differences in the initial angular position analysis are consistent with the argument stated in the analysis of differences in initial applied torque. At lower torques, the Golgi tendon organ activity may not be significant and the latency results can be attributed only to the muscle spindle, thus the shorter latencies at the extended position (i.e. increased muscle spindle sensitivity) than at the flexed position (i.e., reduced muscle spindle sensitivity). At higher torques, the Golgi tendon organ activity may be increased, especially at the extended position, thus delaying the motor neural drive to the muscle. The latencies at the high torque at the shortened position are lower but with lower amplitudes whereas at the extended position the latencies are longer but the amplitude is higher. These results might also suggest that the amplitude of the reflex may have an inverse relationship to the latency. In this sense an EMG signal with shorter latency will affect the muscle stiffness earlier and the yielding will not be significant resulting in lower amplitudes whereas a longer latency will require higher amplitudes to maintain a particular level of stiffness. This latter explanation is supported by Lin and Rymer (1999) experiments. This hypothesis needs to be explored further at a neurophysiological level.

6.3.2.2.3 Stretch reflex rise time

No statistical differences in the stretch reflex rise time between the two initial starting positions in either initial applied torque suggesting that such parameter is not affected

either by initial position or initial applied torque (Figure 4c). However a tendency is seen for the rise time to be higher at the flexed position (44.84 ± 16.76 ms) when compared to the extended position (36.63 ± 7.75 ms) ($p=0.086$).

6.3.2.2.4 Stretch reflex duration

- Low torque

Significantly shorter stretch reflex durations were found in the extended position (65.51 ± 17.32 ms) when compared with those found at the flexed position (88.59 ± 25.32 ms) ($p = 0.008$) (Figure 4d).

- High torque

Significantly shorter stretch reflex durations were found in the extended position (90.17 ± 17.32 ms) when compared with the stretch reflex durations found at the flexed position (131.12 ± 61.58 ms) ($p = 0.026$) (Figure 4d).

Differences in the duration of the reflex, both at the lower and the higher torque are consistent. As previously suggested, this variability can be explained by considering the Golgi tendon organ sensitivity to the change of tension at the muscle. If the Golgi tendon organ activity overrides the spindle activity at the end of the displacement, this would be more evident at the final length at the lengthened position thus inhibiting the motor neuron drive to the muscle, ceasing the stretch reflex activity earlier than at the shortened position.

Table 2: Statistical analysis determining the differences of the initial starting angle at the high and lower torques within the non-impaired population when the initially applied torque is low or high. The red cells represent the significantly different values with 95% confidence. Yellow cells represent the values that tend to be different but do not have statistically significance.

Parameters	Low torque		High torque	
	F	Sig.	F	Sig.
Stretch reflex Amplitude	1.099	0.303	4.702	0.039
Stretch reflex Latency	12.242	0.002	7.042	0.013
Stretch reflex Rise Time	1.858	0.184	3.156	0.087
Stretch reflex Duration	8.232	0.008	5.566	0.026

6.3.2.3 Variability due to different previous voluntary activity

This section presents the analysis of the stretch reflex parameters variation dependent on the voluntary activity prior to the stretch at each initially applied torque and each initial starting position. An analysis of variance with a Bonferroni post-hoc analysis was used to determine the differences between the three initial voluntary conditions (initially relaxed, initially flexing and initially extending).

6.3.2.3.1 Stretch reflex amplitude

No significant differences where found in the stretch reflex amplitude values when comparing between any of the previous voluntary activity conditions with:

- a) The low initially applied torque at the extended position (Table 3) (Figure 5a)
- b) The low initially applied torque at the flexed position (Table 4) (Figure 6a)
- c) The high initially applied torque at the extended position (Table 5) (Figure 5a)
- d) The high initially applied torque at the flexed position (Table 6) (Figure 6a)

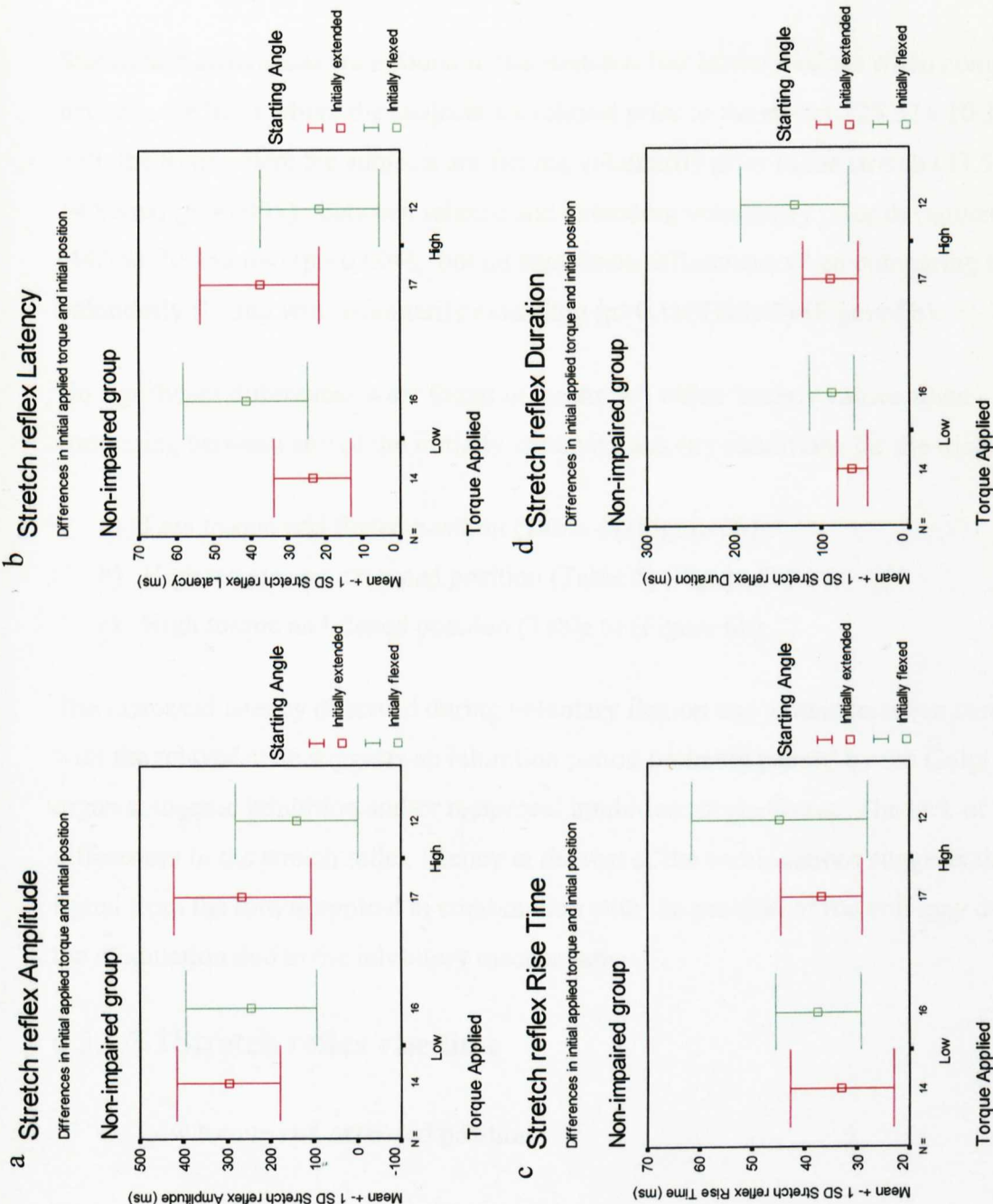


Figure 4: Mean and standard deviation plot of the stretch reflex parameters showing the variability between the initially applied torque and the initial angular position. a) Amplitude, b) latency, c) rise time and d) duration.

6.3.2.3.2 Stretch reflex latency

- Low torque and extended position

Significant differences were found in the stretch reflex latency values when comparing between the trials where the subjects are relaxed prior to the stretch (23.52 ± 10.31 ms) with the trials where the subjects are flexing voluntarily prior to the stretch (43.58 ± 14.89 ms) ($p = 0.005$); between relaxed and extending voluntarily prior the stretch (44.75 ± 20.135 ms) ($p = 0.004$); but no significant differences when comparing between voluntarily flexing with voluntarily extending ($p > 0.1$) (Table 3) (Figure 5b).

No significant differences were found in the stretch reflex latency values when comparing between any of the initially voluntary activity conditions for the trials using:

- a) Low torque and flexed position (Table 4) (Figure 6b)
- b) High torque and extended position (Table 5) (Figure 5b)
- c) High torque and flexed position (Table 6) (Figure 6b)

The increased latency observed during voluntary flexion and extension when compared with the relaxed state suggests an inhibition period probably caused by the Golgi tendon organ autogenic inhibition and/or reciprocal inhibition mechanisms. The lack of differences in the stretch reflex latency in the rest of the combinations suggests that the signal from the torque applied in combination with the position of the arm may override the modulation due to the inhibitory mechanisms.

6.3.2.3.3 Stretch reflex rise time

- Low torque and extended position

It was observed that the stretch reflex rise time values were significantly shorter where subjects are relaxed prior the stretch (32.95 ± 9.87 ms) than the values observed where subjects are extending voluntarily prior the stretch (52.91 ± 23.13 ms) ($p = 0.004$); no significant differences were found when comparing the stretch reflex rise time value between relaxed and voluntarily flexing or voluntarily flexing and voluntarily extending (Table 3) (Figure 5c). This parameter has a slight tendency to increase when the triceps

is active (52.91 ± 23.13 ms) when compared to the values observed when the biceps is active (39.64 ± 5.13 ms) ($p=0.082$).

These results suggest that reciprocal inhibition mechanisms are triggered when a perturbation is applied in the opposite direction of the voluntary activity causing the reflex EMG signal to increase at a slower rate.

- Low torque and flexed position

No significant differences were found in the stretch reflex rise time values when comparing between any of the initially voluntary activity conditions (Table 4) (Figure 6c).

- High torque and extended position

Significantly shorter stretch reflex rise time values were found in trials where subjects are relaxed prior the stretch (36.63 ± 7.75 ms) when compared with the trials where they are voluntarily extending prior the stretch (52.59 ± 22.38 ms) ($p=0.008$); also shorter rise times were found in trials where subjects are flexing voluntarily prior the stretch (31.41 ± 8.86 ms) when compared with the trials where they are voluntarily extending prior the stretch ($p=0$); no significant differences were found when comparing the trials where they are relaxed with the trials where they are extending voluntarily prior the stretch (Table 5) (Figure 5c).

Similarly to the results at the low torque in the extended position reciprocal inhibition may be responsible for the differences between initially relaxed and initially extending conditions. Shorter rise time values when voluntarily flexing at the high torque in the extended position are probably due to the background activity of the elbow flexors facilitating the muscle activity to reach the maximum value earlier. This latter one however could be an artefact in the statistics since the actual difference between both conditions are within the standard deviation values.

- High torque and flexed position

Significantly longer stretch reflex rise time values were found when comparing trials when the subjects relaxed prior the stretch (44.84 ± 16.76 ms) with trials where subjects are flexing voluntarily prior the stretch are (28.65 ± 13.82 ms) ($p = .011$). No significant differences were found when comparing the subjects extending voluntarily when relaxed prior the stretch or when comparing the subjects voluntarily extending prior the stretch with voluntarily flexing prior the stretch (Table 6) (Figure 6c).

These results also suggest the contribution of the Golgi tendon organ inhibiting the signal at an earlier time when the tension of the torque and the biceps flexing is increased.

6.3.2.3.4 Stretch reflex duration

- Low torque and extended position

No significant differences were found in the stretch reflex duration values when comparing between any of the initially voluntary activity conditions (Table 3). However a slight tendency of the stretch reflex duration to increase when comparing trials when the subjects flexing voluntarily prior the stretch (124.399 ± 109.52 ms) with the subjects relaxed prior the stretch (65.51 ± 17.32 ms) ($p = 0.087$) (Figure 5d).

- Low torque and flexed position

No significant differences were found in the stretch reflex duration values when comparing between any of the initially voluntary activity conditions (Table 4) (Figure 6d).

- High torque and extended position

Significant lower stretch reflex duration values were found when comparing trials where subjects are voluntarily flexing (71.35 ± 25.13 ms) and voluntarily extending (107.60 ± 37.70 ms) ($p = 0.007$). No significant differences were found when comparing trials when the subjects are relaxed prior the stretch with voluntarily extending; or when relaxed and voluntarily flexing (Table 5) (Figure 5d).

- High torque and flexed position

Significantly lower stretch reflex duration values were found where subjects are flexing voluntarily prior the stretch (54.28 ± 23.25 ms) when compared with stretch reflex duration values found where subjects are relaxed prior the stretch (131.12 ± 61.58 ms) ($p = 0.001$); also lower values of the stretch reflex duration were found when flexing voluntarily prior the stretch in comparison with voluntarily extending prior the stretch (112.28 ± 55.3 ms) ($p = 0.016$). No significant differences were found when comparing the subjects relaxed and voluntarily extending prior the stretch (Table 6) (Figure 6d).

A possible explanation for the results found at the high torque can be attributed to autogenic inhibition mechanisms that may inhibit the tonic stretch reflex in the flexors when the tension increases due to the applied torque and the voluntary activity.

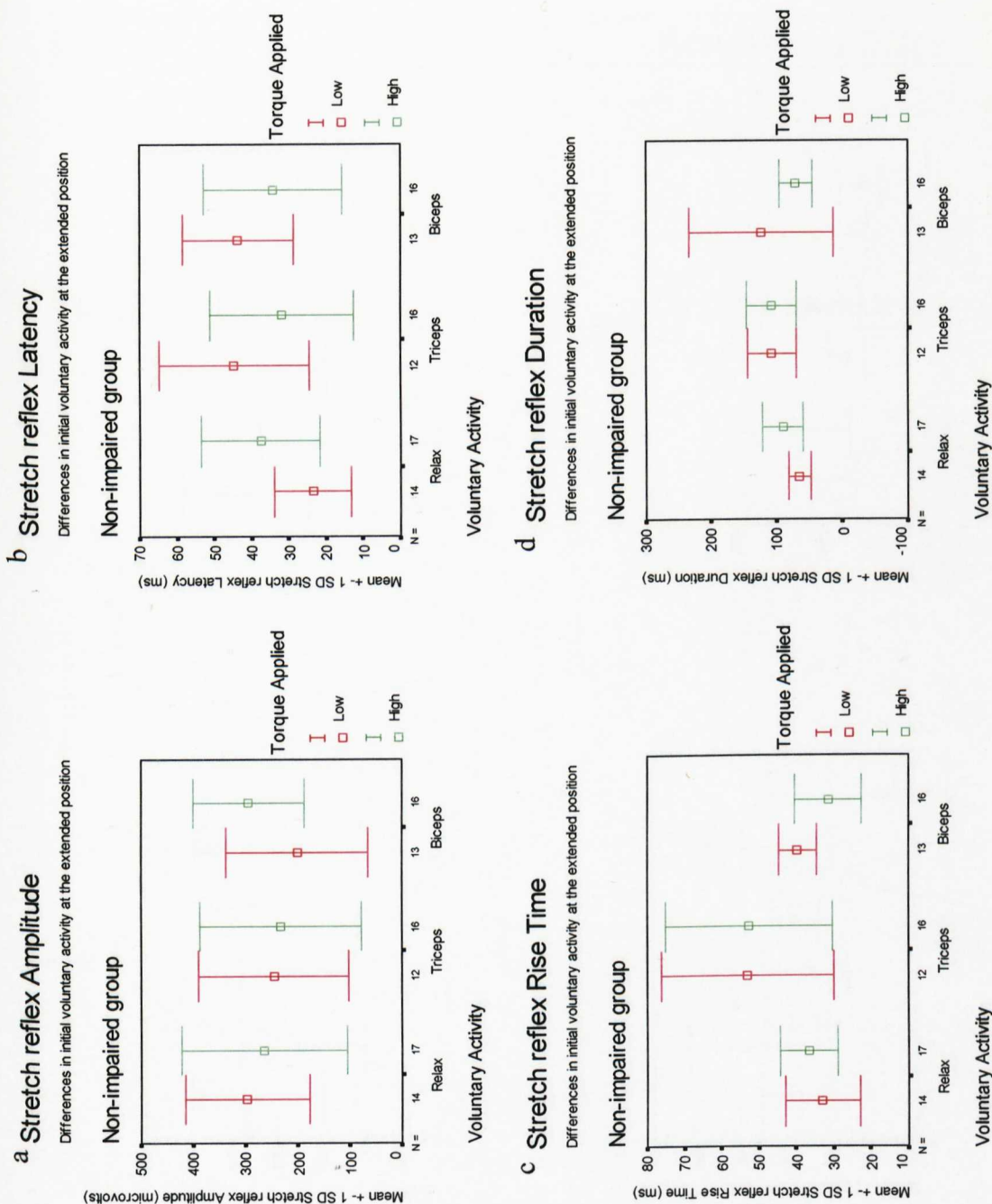


Figure 5: Mean and standard deviation plot of the stretch reflex parameters showing the variability between the voluntary activity previous to the perturbation at the initially extended position at the high and low torques. A) Amplitude, b) latency, c) rise time and d) duration

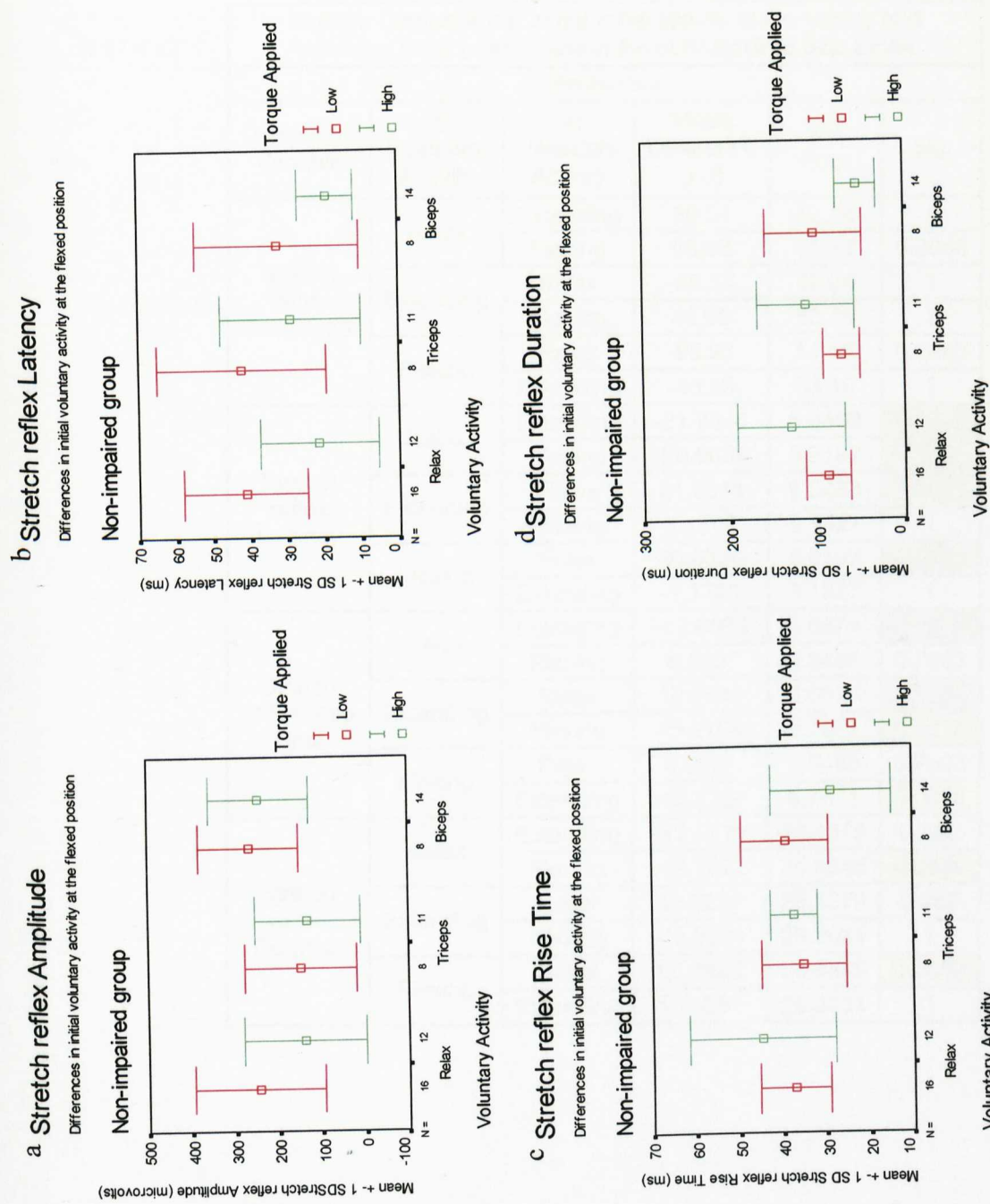


Figure 6: Mean and standard deviation plot of the stretch reflex parameters showing the variability between the voluntary activity previous to the perturbation at the initially flexed position at the high and low torques. A) Amplitude, b) latency, c) rise time and d) duration

Table 3: Bonferroni statistical analysis determining the differences of the initial voluntary activity (relaxed, activating triceps and activating biceps) when the initially applied torque is low and the initial position is extended within the non-impaired population. The red cells represent the significantly different values with 95% confidence. Yellow cells represent the values that tend to be different but do not have statistically significance.

Multiple Comparisons in the initial activity at low torque and extended initial position within the non-impaired population					
Bonferroni					
Dependent Variable	(I) Voluntary Activity	(J) Voluntary Activity	Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Relax	Extending	50.34	52.24	1
		Flexing	95.93	51.15	0.2065
	Extending	Relax	-50.34	52.24	1
		Flexing	45.59	53.16	1
	Flexing	Relax	-95.93	51.15	0.2065
		Extending	-45.59	53.16	1
Stretch reflex Latency	Relax	Extending	-21.2344	6.0463	0.0037
		Flexing	-20.0638	5.9197	0.0051
	Extending	Relax	21.2344	6.0463	0.0037
		Flexing	1.1706	6.1527	1
	Flexing	Relax	20.0638	5.9197	0.0051
		Extending	-1.1706	6.1527	1
Stretch reflex Rise Time	Relax	Extending	-19.9585	5.6674	0.0036
		Flexing	-6.6857	5.5488	0.7083
	Extending	Relax	19.9585	5.6674	0.0036
		Flexing	13.2728	5.7671	0.0818
	Flexing	Relax	6.6857	5.5488	0.7083
		Extending	-13.2728	5.7671	0.0818
Stretch reflex Duration	Relax	Extending	-42.0271	26.4379	0.362
		Flexing	-58.8822	25.8846	0.0869
	Extending	Relax	42.0271	26.4379	0.362
		Flexing	-16.8551	26.9031	1
	Flexing	Relax	58.8822	25.8846	0.0869
		Extending	16.8551	26.9031	1

Table 4: Bonferroni statistical analysis determining the differences of the initial voluntary activity (relaxed, activating triceps and activating biceps) when the initially applied torque is low and the initial position is flexed within the non-impaired population.

Multiple Comparisons in the initial activity at low torque and flexed initial position within the non-impaired population					
Bonferroni					
Dependent Variable	(I) Voluntary Activity	(J) Voluntary Activity	Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Relax	Extending	95.20	59.37	0.3590
		Flexing	-23.08	59.37	1.0000
	Extending	Relax	-95.20	59.37	0.3590
		Flexing	-118.28	68.55	0.2853
	Flexing	Relax	23.08	59.37	1.0000
		Extending	118.28	68.55	0.2853
Stretch reflex Latency	Relax	Extending	-0.9155	8.5053	1.0000
		Flexing	8.7891	8.5053	0.9299
	Extending	Relax	0.9155	8.5053	1.0000
		Flexing	9.7046	9.8211	0.9938
	Flexing	Relax	-8.7891	8.5053	0.9299
		Extending	-9.7046	9.8211	0.9938
Stretch reflex Rise Time	Relax	Extending	2.3346	3.8854	1.0000
		Flexing	-1.7853	3.8854	1.0000
	Extending	Relax	-2.3346	3.8854	1.0000
		Flexing	-4.1199	4.4865	1.0000
	Flexing	Relax	1.7853	3.8854	1.0000
		Extending	4.1199	4.4865	1.0000
Stretch reflex Duration	Relax	Extending	16.8762	14.9320	0.8030
		Flexing	-14.8315	14.9320	0.9864
	Extending	Relax	-16.8762	14.9320	0.8030
		Flexing	-31.7078	17.2420	0.2285
	Flexing	Relax	14.8315	14.9320	0.9864
		Extending	31.7078	17.2420	0.2285

Table 5: Bonferroni statistical analysis determining the differences of the initial voluntary activity (relaxed, activating triceps and activating biceps) when the initially applied torque is high and the initial position is extended within the non-impaired population. The red cells represent the significantly different values with 95% confidence.

Multiple Comparisons in the initial activity at high torque and extended initial position within the non-impaired population					
Bonferroni					
Dependent Variable	(I) Voluntary Activity	(J) Voluntary Activity	Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Relax	Extending	32.17	49.57	1.0000
		Flexing	-28.98	49.57	1.0000
	Extending	Relax	-32.17	49.57	1.0000
		Flexing	-61.15	50.31	0.6913
	Flexing	Relax	28.98	49.57	1.0000
		Extending	61.15	50.31	0.6913
Stretch reflex Latency	Relax	Extending	5.7077	6.2486	1.0000
		Flexing	3.2510	6.2486	1.0000
	Extending	Relax	-5.7077	6.2486	1.0000
		Flexing	-2.4567	6.3425	1.0000
	Flexing	Relax	-3.2510	6.2486	1.0000
		Extending	2.4567	6.3425	1.0000
Stretch reflex Rise Time	Relax	Extending	-15.9616	5.0474	0.0083
		Flexing	5.2176	5.0474	0.9200
	Extending	Relax	15.9616	5.0474	0.0083
		Flexing	21.1792	5.1234	0.0004
	Flexing	Relax	-5.2176	5.0474	0.9200
		Extending	-21.1792	5.1234	0.0004
Stretch reflex Duration	Relax	Extending	-17.4309	11.0510	0.3647
		Flexing	18.8240	11.0510	0.2857
	Extending	Relax	17.4309	11.0510	0.3647
		Flexing	36.2549	11.2172	0.0068
	Flexing	Relax	-18.8240	11.0510	0.2857
		Extending	-36.2549	11.2172	0.0068

Table 6: Bonferroni statistical analysis determining the differences of the initial voluntary activity (relaxed, activating triceps and activating biceps) when the initially applied torque is high and the initial position is flexed within the non-impaired population. The red cells represent the significantly different values with 95% confidence.

Multiple Comparisons in the initial activity at high torque and flexed initial position within the non-impaired population					
Bonferroni					
Dependent Variable	(I) Voluntary Activity	(J) Voluntary Activity	Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Relax	Extending	8.02	0.5253	1.0000
		Flexing	-103.03	0.4950	0.1350
	Extending	Relax	-8.02	0.5253	1.0000
		Flexing	-111.05	0.5070	0.1064
	Flexing	Relax	103.03	0.4950	0.1350
		Extending	111.05	0.5070	0.1064
Stretch reflex Latency	Relax	Extending	-7.3834	6.0522	0.6926
		Flexing	2.2728	5.7039	1.0000
	Extending	Relax	7.3834	6.0522	0.6926
		Flexing	9.6562	5.8418	0.3226
	Flexing	Relax	-2.2728	5.7039	1.0000
		Extending	-9.6562	5.8418	0.3226
Stretch reflex Rise Time	Relax	Extending	7.6201	5.4852	0.5214
		Flexing	16.1888	5.1695	0.0107
	Extending	Relax	-7.6201	5.4852	0.5214
		Flexing	8.5687	5.2945	0.3444
	Flexing	Relax	-16.1888	5.1695	0.0107
		Extending	-8.5687	5.2945	0.3444
Stretch reflex Duration	Relax	Extending	18.8414	20.1631	1.0000
		Flexing	76.8374	19.0026	0.0009
	Extending	Relax	-18.8414	20.1631	1.0000
		Flexing	57.9961	19.4621	0.0159
	Flexing	Relax	-76.8374	19.0026	0.0009
		Extending	-57.9961	19.4621	0.0159

6.3.2.4 Variability due to different head position

This section presents the analysis of the stretch reflex parameters variation dependent on the initial head position at the higher torque at the extended position when the initial voluntary activity is relaxed. An analysis of variance with a Bonferroni post-hoc analysis was used to determine the differences between the three head positions (facing straight, facing left and facing right) (Table 7).

6.3.2.4.1 Stretch reflex amplitude

No significant differences were found in the stretch reflex amplitude values when comparing between any of the initial head position conditions (Figure 7a).

6.3.2.4.2 Stretch reflex latency

No significant differences were found in the stretch reflex latency values when comparing between any of the initial head position conditions. However a minor tendency to increase where they are facing to the right when comparing the values where subjects are facing to the left ($p=0.09$) (Figure 7b).

6.3.2.4.3 Stretch reflex rise time

Significant differences were found in the stretch reflex rise time values when comparing the trials where subjects are facing left (34.09 ± 14.7 ms) with the trials where subjects are facing right (49.27 ± 20.63 ms) ($p=0.017$). No significant differences were found when comparing the trials where subjects are facing straight (36.63 ± 7.75 ms) with the ones where they are facing to the left ($p>0.1$). This value had a tendency to increase where the subjects are facing right when compared with the ones where they are facing straight ($p=0.06$) (Figure 7c).

6.3.2.4.4 Stretch reflex duration

Significant differences in the stretch reflex duration values were found when comparing the trials when the volunteers are facing right (115 ± 48.23 ms) with the values of the

trials where they are facing left (67.74 ± 23.19 ms) ($p=0.001$). No other significant differences were found (Table 7) (Figure 7d).

Rise time and duration results suggest an increased facilitation or reduced presynaptic inhibition when the head is turning to the opposite side of the arm being tested. More experiments regarding body posture and its relationship with the excitability of the stretch reflex need to be addressed to attempt to explain these findings.

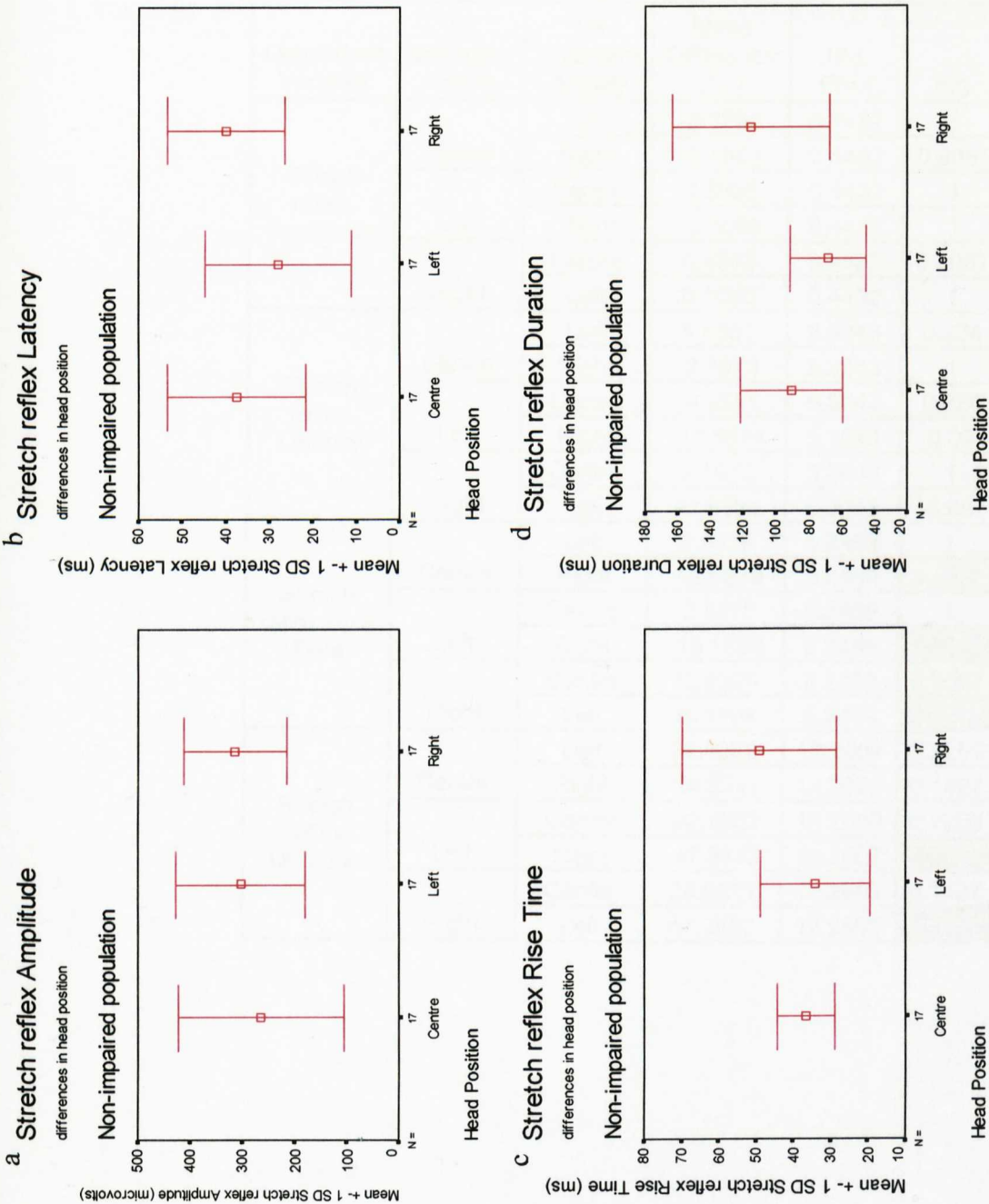


Figure 7: Mean and standard deviation plot of the stretch reflex parameters showing the variability between the head position during the perturbation. A) Amplitude, b) latency, c) rise time and d) duration

Table 7: Bonferroni statistical analysis determining the differences of the head position during the tests (facing straight, left or right) when the initially applied torque is high and the initial position is extended within the non-impaired population. The red cells represent the significantly different values with 95% confidence. Yellow cells represent the values that tend to be different but do not have statistically significance.

Bonferroni					
Dependent Variable	(I) Voluntary Activity	(J) Voluntary Activity	Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Centre	Left	-0.3895	0.4432	1
		Right	-0.4963	0.4432	0.8051
	Left	Centre	0.3895	0.4432	1
		Right	-0.1068	0.4432	1
	Right	Centre	0.4963	0.4432	0.8051
		Left	0.1068	0.4432	1
Stretch reflex Latency	Centre	Left	9.6651	5.3043	0.224
		Right	-2.1973	5.3043	1
	Left	Centre	-9.6651	5.3043	0.224
		Right	-11.8624	5.3043	0.09
	Right	Centre	2.1973	5.3043	1
		Left	11.8624	5.3043	0.09
Stretch reflex Rise Time	Centre	Left	2.5419	5.2469	1
		Right	-12.6379	5.2469	0.0597
	Left	Centre	-2.5419	5.2469	1
		Right	-15.1798	5.2469	0.0172
	Right	Centre	12.6379	5.2469	0.0597
		Left	15.1798	5.2469	0.0172
Stretch reflex Duration	Centre	Left	22.4322	12.2609	0.2206
		Right	-24.9311	12.2609	0.1427
	Left	Centre	-22.4322	12.2609	0.2206
		Right	-47.3633	12.2609	0.001
	Right	Centre	24.9311	12.2609	0.1427
		Left	47.3633	12.2609	0.001

6.3.3 Analysis within Post-stroke subjects

6.3.3.1 Variability due to different applied torques

These results show no significant differences in any of the stretch reflex parameters when comparing the results at the lower torque with the ones at the higher torque for either initial starting position using an analysis of variance (ANOVA) test with 95 % confidence interval (Table 8).

Table 8: Statistical analysis determining the differences of the initial applied torque when the initial position is flexed or extended within the post-stroke population.

Parameters	Initially extended		Initially flexed	
	F	Sig.	F	Sig.
Stretch reflex Amplitude	0.368	0.550	0.388	0.539
Stretch reflex Latency	0.002	0.967	0.390	0.538
Stretch reflex Rise Time	0.077	0.783	0.177	0.678
Stretch reflex Duration	2.169	0.154	0.395	0.536

6.3.3.2 Variability due to different initial angular position

No significant differences in any of the stretch reflex parameters were found when comparing the results at the initial shortened position with the ones at the initially extended position for either initially applied torque with the exception of the stretch reflex duration when the initial low torque was applied. Under this latter condition, significant longer durations were found when the elbow joint is initially flexed (131.57 ± 77.05 ms) when compared with the ones found when the elbow joint is initially extended (75.06 ± 33.27 ms) ($p= 0.036$) and a tendency in the rise time to be lower at the initially extended position (34.26 ± 13.01) when compared with the initially flexed position (44.88 ± 15.31) (Table 9).

6.3.3.3 Variability due to different previous voluntary activity

6.3.3.3.1 Stretch reflex amplitude

No significant differences were found in the stretch reflex amplitude values when comparing between any of the previous voluntary conditions with:

- a) The low initially applied torque at the extended position (Table 10)
- b) The low initially applied torque at the flexed position (Table 11)
- c) The high initially applied torque at the extended position (Table 12)
- d) The high initially applied torque at the flexed position (Table 13)

Table 9: Statistical analysis determining the differences of the initial starting angle when the initially applied torque is low or high within the post-stroke population. The red cells represent the significantly different values with 95% confidence. Yellow cells represent the values that tend to be different but do not have statistically significance.

Parameters	Low Torque		High Torque	
	F	Sig.	F	Sig.
Stretch reflex Amplitude	0.223	0.642	0.431	0.517
Stretch reflex Latency	1.289	0.269	0.685	0.416
Stretch reflex Rise Time	3.177	0.089	0.744	0.396
Stretch reflex Duration	5.038	0.036	0.901	0.351

6.3.3.3.2 Stretch reflex latency

No significant differences where found in the stretch reflex latency values when comparing between any of the previous voluntary conditions with:

- a) The low initially applied torque at the extended position (Table 10)
- b) The low initially applied torque at the flexed position (Table 11)
- c) The high initially applied torque at the extended position (Table 12)
- d) The high initially applied torque at the flexed position (Table 13)

6.3.3.3 Stretch reflex rise time

No significant differences were found in the stretch reflex rise time values when comparing between any of the previous voluntary conditions with:

- The low initially applied torque at the extended position (Table 10)
- The low initially applied torque at the flexed position (Table 11)
- The high initially applied torque at the extended position (Table 12)
- The high initially applied torque at the flexed position (Table 13)

Table 10: Bonferroni statistical analysis determining the differences of the initial voluntary activity (relaxed, activating triceps and activating biceps) when the initially applied torque is low and the initial position is extended within the post-stroke population.

Multiple Comparisons in the initial activity at low torque and extended initial position within the post-stroke population					
Bonferroni					
Dependent Variable	(I) Voluntary Activity	(J) Voluntary Activity	Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Relax	Triceps	-28.79	100.33	1.0000
		Biceps	-62.44	96.54	1.0000
	Triceps	Relax	28.79	100.33	1.0000
		Biceps	-33.65	90.57	1.0000
	Biceps	Relax	62.44	96.54	1.0000
		Triceps	33.65	90.57	1.0000
Stretch reflex Latency	Relax	Triceps	-8.9981	10.5109	1.0000
		Biceps	-2.1774	10.1141	1.0000
	Triceps	Relax	8.9981	10.5109	1.0000
		Biceps	6.8207	9.4879	1.0000
	Biceps	Relax	2.1774	10.1141	1.0000
		Triceps	-6.8207	9.4879	1.0000
Stretch reflex Rise Time	Relax	Triceps	5.7868	8.1356	1.0000
		Biceps	8.5863	7.8285	0.8828
	Triceps	Relax	-5.7868	8.1356	1.0000
		Biceps	2.7995	7.3438	1.0000
	Biceps	Relax	-8.5863	7.8285	0.8828
		Triceps	-2.7995	7.3438	1.0000
Stretch reflex Duration	Relax	Triceps	37.5355	18.1854	0.1840
		Biceps	2.3395	17.4989	1.0000
	Triceps	Relax	-37.5355	18.1854	0.1840
		Biceps	-35.1960	16.4154	0.1596
	Biceps	Relax	-2.3395	17.4989	1.0000
		Triceps	35.1960	16.4154	0.1596

Table 11: Bonferroni statistical analysis determining the differences of the initial voluntary activity (relaxed, activating triceps and activating biceps) when the initially applied torque is low and the initial position is flexed within the non-impaired population.

Multiple Comparisons in the initial activity at low torque and flexed initial position within the post-stroke population					
Bonferroni					
Dependent Variable	(I) Voluntary Activity	(J) Voluntary Activity	Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Relax	Triceps	-50.30	80.14	1.0000
		Biceps	-130.92	76.41	0.3260
	Triceps	Relax	50.30	80.14	1.0000
		Biceps	-80.62	80.14	0.9944
	Biceps	Relax	130.92	76.41	0.3260
		Triceps	80.62	80.14	0.9944
Stretch reflex Latency	Relax	Triceps	0.2759	14.6990	1.0000
		Biceps	-0.4471	14.0149	1.0000
	Triceps	Relax	-0.2759	14.6990	1.0000
		Biceps	-0.7231	14.6990	1.0000
	Biceps	Relax	0.4471	14.0149	1.0000
		Triceps	0.7231	14.6990	1.0000
Stretch reflex Rise Time	Relax	Triceps	3.0036	6.4874	1.0000
		Biceps	5.4525	6.1855	1.0000
	Triceps	Relax	-3.0036	6.4874	1.0000
		Biceps	2.4489	6.4874	1.0000
	Biceps	Relax	-5.4525	6.1855	1.0000
		Triceps	-2.4489	6.4874	1.0000
Stretch reflex Duration	Relax	Triceps	56.6164	48.0715	0.7756
		Biceps	22.3391	45.8344	1.0000
	Triceps	Relax	-56.6164	48.0715	0.7756
		Biceps	-34.2774	48.0715	1.0000
	Biceps	Relax	-22.3391	45.8344	1.0000
		Triceps	34.2774	48.0715	1.0000

Table 12: Bonferroni statistical analysis determining the differences of the initial voluntary activity (relaxed, activating triceps and activating biceps) when the initially applied torque is high and the initial position is extended within the non-impaired population.

Multiple Comparisons in the initial activity at high torque and extended initial position within the non-impaired population					
Bonferroni					
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Relax	Triceps	-52.81	83.11	1.0000
		Biceps	-43.17	75.87	1.0000
	Triceps	Relax	52.81	83.11	1.0000
		Biceps	9.63	83.11	1.0000
	Biceps	Relax	43.17	75.87	1.0000
		Triceps	-9.63	83.11	1.0000
Stretch reflex Latency	Relax	Triceps	2.4483	7.5514	1.0000
		Biceps	4.4289	6.8935	1.0000
	Triceps	Relax	-2.4483	7.5514	1.0000
		Biceps	1.9806	7.5514	1.0000
	Biceps	Relax	-4.4289	6.8935	1.0000
		Triceps	-1.9806	7.5514	1.0000
Stretch reflex Rise Time	Relax	Triceps	10.7570	6.3580	0.3301
		Biceps	4.3594	5.8040	1.0000
	Triceps	Relax	-10.7570	6.3580	0.3301
		Biceps	-6.3977	6.3580	0.9878
	Biceps	Relax	-4.3594	5.8040	1.0000
		Triceps	6.3977	6.3580	0.9878
Stretch reflex Duration	Relax	Triceps	27.9150	18.7480	0.4678
		Biceps	-1.7787	17.1145	1.0000
	Triceps	Relax	-27.9150	18.7480	0.4678
		Biceps	-29.6937	18.7480	0.3984
	Biceps	Relax	1.7787	17.1145	1.0000
		Triceps	29.6937	18.7480	0.3984

6.3.3.3.4 Stretch reflex duration

No significant differences were found in the stretch reflex rise time values when comparing between any of the previous voluntary conditions with:

- The low initially applied torque at the extended position (Table 10)
- The low initially applied torque at the flexed position (Table 11)
- The high initially applied torque at the extended position (Table 12)
- The high initially applied torque at the flexed position (Table 13)

Table 13: Bonferroni statistical analysis determining the differences of the initial voluntary activity (relaxed, activating triceps and activating biceps) when the initially applied torque is high and the initial position is flexed within the non-impaired population.

Multiple Comparisons in the initial activity at high torque and flexed initial position within the non-impaired population					
Bonferroni					
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Relax	Triceps	-35.23	78.09	1.0000
		Biceps	-22.07	74.20	1.0000
	Triceps	Relax	35.23	78.09	1.0000
		Biceps	13.16	80.76	1.0000
	Biceps	Relax	22.07	74.20	1.0000
		Triceps	-13.16	80.76	1.0000
Stretch reflex Latency	Relax	Triceps	-0.1193	10.9097	1.0000
		Biceps	-14.7586	10.3659	0.5249
	Triceps	Relax	0.1193	10.9097	1.0000
		Biceps	-14.6393	11.2822	0.6421
	Biceps	Relax	14.7586	10.3659	0.5249
		Triceps	14.6393	11.2822	0.6421
Stretch reflex Rise Time	Relax	Triceps	11.6139	10.0299	0.7950
		Biceps	4.5337	9.5299	1.0000
	Triceps	Relax	-11.6139	10.0299	0.7950
		Biceps	-7.0803	10.3723	1.0000
	Biceps	Relax	-4.5337	9.5299	1.0000
		Triceps	7.0803	10.3723	1.0000
Stretch reflex Duration	Relax	Triceps	37.6115	29.7718	0.6773
		Biceps	27.2270	28.2876	1.0000
	Triceps	Relax	-37.6115	29.7718	0.6773
		Biceps	-10.3845	30.7882	1.0000
	Biceps	Relax	-27.2270	28.2876	1.0000
		Triceps	10.3845	30.7882	1.0000

These results suggest that the parameters are not dependent on the level of activity prior to the stretch. On the other hand, it is also possible that a combination of factors such as the activity prior the perturbation and the effects of such activity on the kinematics of the movement are compensated.

6.3.3.4 Variability due to different head position

An analysis of variance with a Bonferroni post-hoc analysis showed no significant differences in the stretch reflex rise time values when comparing between any of the

three initial head positions (facing straight, facing left and facing right) at the higher torque at the extended position when the initial voluntary activity is relaxed (Table 14).

Table 14: Bonferroni statistical analysis determining the differences of the head position during the tests (facing straight, left or right) when the initially applied torque is high and the initial position is extended within the non-impaired population.

Multiple Comparisons differences in head position post-stroke					
Bonferroni					
Dependent Variable	(I) Voluntary Activity	(J) Voluntary Activity	Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Centre	Left	-14.74	38.79	1.0000
		Right	-2.85	37.08	1.0000
	Left	Centre	14.74	38.79	1.0000
		Right	11.89	39.44	1.0000
	Right	Centre	2.85	37.08	1.0000
		Left	-11.89	39.44	1.0000
Stretch reflex Latency	Centre	Left	2.5523	3.9833	1.0000
		Right	1.6899	3.8078	1.0000
	Left	Centre	-2.5523	3.9833	1.0000
		Right	-0.8624	4.0501	1.0000
	Right	Centre	-1.6899	3.8078	1.0000
		Left	0.8624	4.0501	1.0000
Stretch reflex Rise Time	Centre	Left	2.9998	4.0830	1.0000
		Right	3.3939	3.9031	1.0000
	Left	Centre	-2.9998	4.0830	1.0000
		Right	0.3941	4.1515	1.0000
	Right	Centre	-3.3939	3.9031	1.0000
		Left	-0.3941	4.1515	1.0000
Stretch reflex Duration	Centre	Left	2.6445	17.4440	1.0000
		Right	6.9811	16.6757	1.0000
	Left	Centre	-2.6445	17.4440	1.0000
		Right	4.3366	17.7368	1.0000
	Right	Centre	-6.9811	16.6757	1.0000
		Left	-4.3366	17.7368	1.0000

6.3.4 Analysis between Non-impaired and Post-stroke subjects

6.3.4.1 Differences at extended position

These results estimate the variability of the stretch reflex parameters between the non-impaired and post-stroke population at the extended position at the high and low initially applied torques using an analysis of variance (ANOVA) test with 95 % confidence interval. The statistical analysis for each parameter is summarised in table 15.

6.3.4.1.1 Stretch reflex amplitude

- Low torque

Stretch reflex amplitudes were significantly lower in the post-stroke population ($97.8 \pm 112 \mu\text{V}$) when compared with the stretch reflex amplitude values found in the non impaired population ($296.27 \pm 118.58 \mu\text{V}$) ($p=0$) (Figure 8a).

- High Torque

Stretch reflex amplitudes values were significantly lower in the post-stroke population ($121.52 \pm 83.150 \mu\text{V}$) when compared with the stretch reflex amplitude values found in the non impaired population ($264.79 \pm 158.64 \mu\text{V}$) ($p=0.005$) (Figure 8a).

These results were unexpected and do not agree entirely with the accepted definition of spasticity as it would be expected to observe higher amplitudes within the stroke population.

The patients were recruited from a botulinum toxin clinic at a tertiary care neurological Rehabilitation Centre. It is unlikely that these injections, given 15 minutes prior to measurements (de Paiva et al. 1999), would have influenced the outcome measures in any way as it is reported that their effects are expected to occur three days after administration (Ghosh & Das 2002; Hughes 1994). Measurements were taken on the day of botulinum toxin administration before or within the first fifteen minutes after the injection, which is within the window before the toxin could have any effect. However it is possible that there could be carry over effects from previous injections or that the toxin could start taking effect within the first hour of administration confounding the results.

6.3.4.1.2 Stretch reflex latency

- Low torque

No significant differences were found when comparing the stretch reflex latencies values in the post-stroke population with those found in the non-impaired population (Figure 8b).

- High torque

Stretch reflex latencies values were significantly higher in the non-impaired population (37.58 ± 15.91 ms) with compared with those found in the post-stroke population (18.06 ± 11.72 ms) ($p > 0.1$, $= 0.001$) when the high torque was applied (Figure 8b).

Lower values in the stretch reflex latency in the post-stroke volunteers do suggest increased excitability in the stretch reflex. Nevertheless this only occurs at the high torque level, which implies that the differences are expected to be in an intact nervous system as a result of modulation of all structures in the stretch reflex loop and other neural pathways and this account more to the lack of modulation of the reflex than for the velocity dependency of the reflex.

6.3.4.1.3 Stretch reflex rise time

No significant differences were found when comparing the stretch reflex rise time values in the non-impaired population with the stretch reflex rise time values found in the post-stroke when either the low or the high torque were applied (Figure 8c).

Lack of differences in the reflex rise time suggests that the behaviour of the synaptic connections and the period of time when the reflex loop is active remain similar after a stroke.

6.3.4.1.4 Stretch reflex duration

No significant differences were found when comparing the stretch reflex duration values in the non-impaired population with the stretch reflex duration values for the post-stroke population when either the high or the low torques were applied (Figure 8d).

This lack of differences in this parameter supports the findings in the stretch reflex rise time, suggesting that the duration of the output signal from the α -motor neurone is active for only a determined period of time.

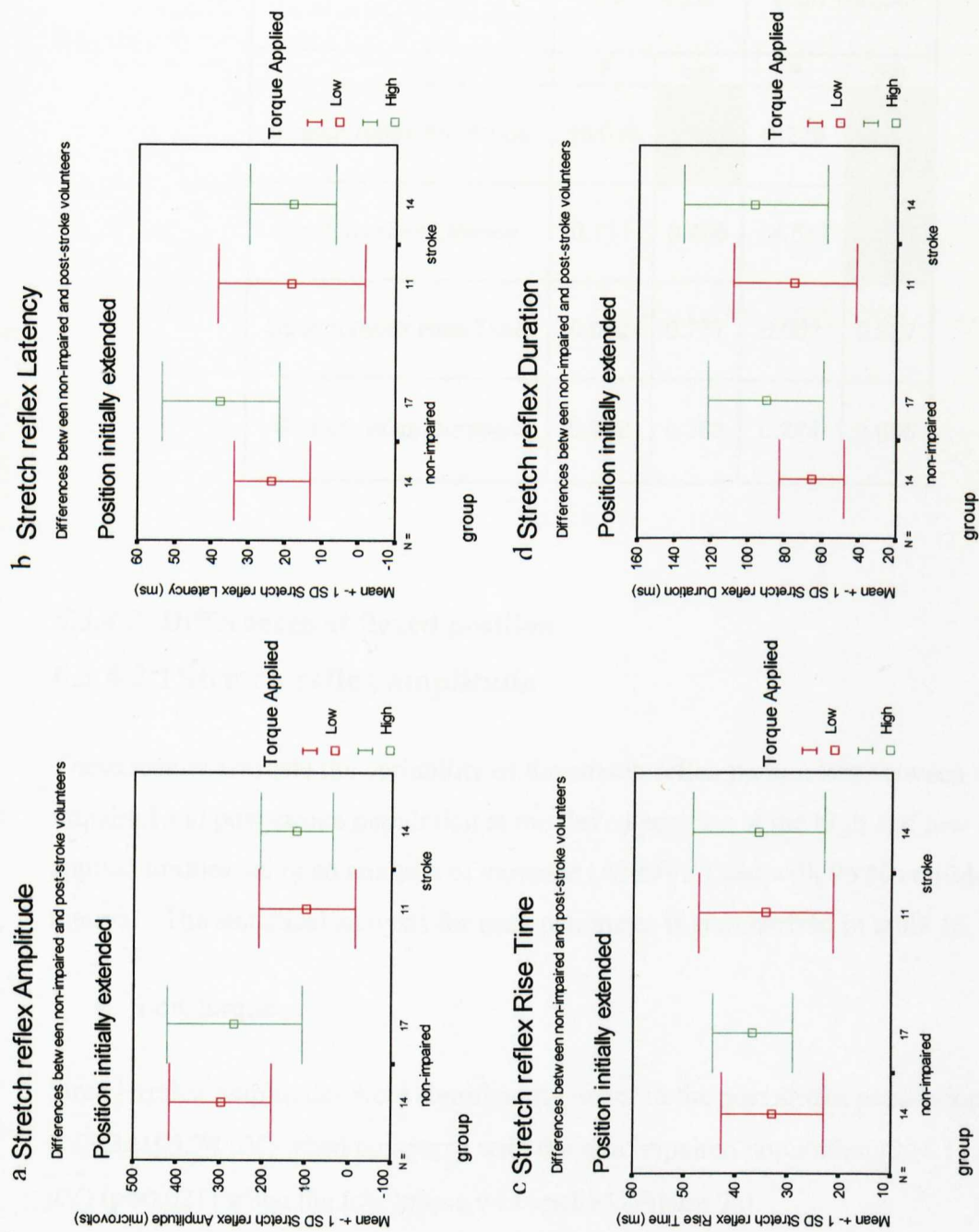


Figure 8: Mean and standard deviation plot of the stretch reflex parameters showing the differences between the post-stroke and non-impaired populations at the high and low torque when the initial position is extended. A) Amplitude, b) latency, c) rise time and d) duration

Table 15: Statistical analysis determining the differences between the post-stroke and non-impaired populations at the extended position with high and low torque. The red cells represent the significantly different values obtained with ANOVA with 95% confidence.

	Low Torque		High Torque	
	F	Sig.	F	Sig.
Stretch reflex Amplitude	18.018	0.000	9.279	0.005
Stretch reflex Latency	0.711	0.408	14.517	0.001
Stretch reflex Rise Time	0.082	0.777	0.061	0.807
Stretch reflex Duration	0.862	0.363	0.274	0.605

6.3.4.2 Differences at flexed position

6.3.4.2.1 Stretch reflex amplitude

These results estimate the variability of the stretch reflex parameters between the non-impaired and post-stroke population at the flexed position at the high and low initially applied torques using an analysis of variance (ANOVA) test with 95 % confidence interval. The statistical analysis for each parameter is summarised in table 16.

- Low torque

Stretch reflex amplitudes were significantly lower in the post stroke population ($119.3\pm105.74\text{ }\mu\text{V}$) when compared with the non impaired population ($244.11\pm149.42\text{ }\mu\text{V}$) ($p=0.021$) when the low torque was applied (Figure 9a).

- High torque

No significant differences were found when comparing the stretch reflex amplitude values in the non impaired population with those in the for post stroke population when the high torque was applied (Figure 9a).

The amplitudes found at the lower torque are consistent with the ones found at the extended position. The lack of differences at the high torque resulting from a decrease in the EMG activity in the non-impaired subjects was unexpected and will need to be explored further.

6.3.4.2.2 Stretch reflex latency

No significant differences were found when comparing the stretch reflex latencies in the non-impaired population with those in the post-stroke population when either the low or the high torque were applied (Figure 9b).

These results suggest that the latency values are remained unchanged when the muscle is under less initial tension, this again can be attributed to the Golgi tendon organ contribution.

6.3.4.2.3 Stretch reflex rise time

No significant differences were found when comparing the stretch reflex rise time values in the non-impaired population with those in the for post-stroke population when either the low or the high torque were applied (Figure 9c).

This results support the findings from the extended position trials.

6.3.4.2.4 Stretch reflex duration

- Low torque

Stretch reflex duration values in the post-stroke population (131.57 ± 77.05 ms) were significantly higher when compared with those in the non-impaired population (88.59 ± 25.32) ($p=0.046$) (Figure 9d).

- High torque

No significant differences were found when comparing the stretch reflex duration in the non-impaired population with those in the post-stroke population (Figure 9d).

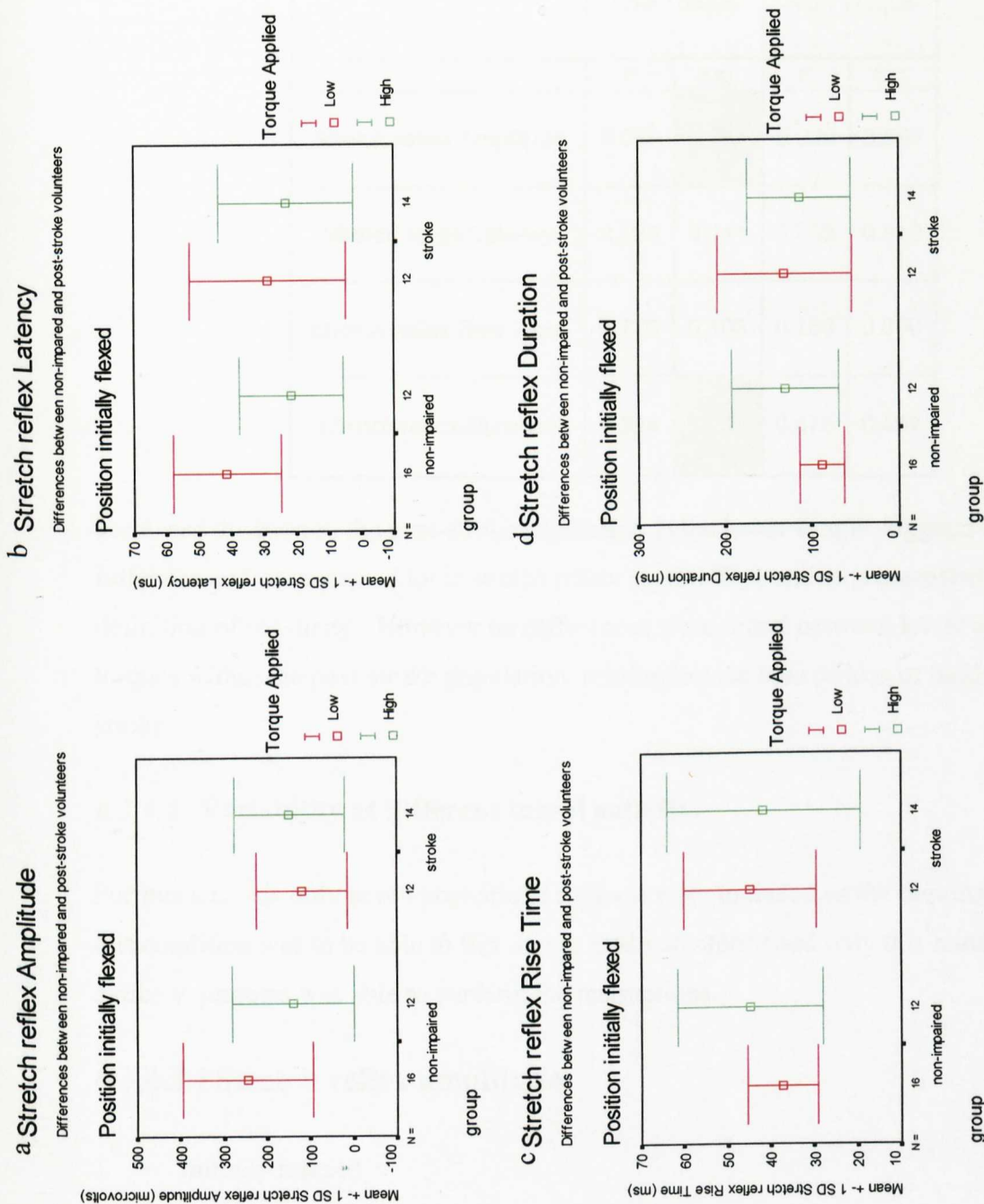


Figure 9: Mean and standard deviation plot of the stretch reflex parameters showing the differences between the post-stroke and non-impaired populations at the high and low torque when the initial position is flexed. A) Amplitude, b) latency, c) rise time and d) duration

Table 16: Statistical analysis determining the differences between the post-stroke and non-impaired populations at the flexed position with high and low torque. The red cells represent the significantly different values obtained with ANOVA with 95% confidence.

	Low Torque		High Torque	
	F	Sig.	F	Sig.
Stretch reflex Amplitude	6.065	0.021	0.020	0.889
Stretch reflex Latency	2.725	0.111	0.053	0.819
Stretch reflex Rise Time	2.779	0.108	0.163	0.690
Stretch reflex Duration	4.394	0.046	0.475	0.497

Increased duration in the post-stroke population at the lower torque suggests cortical influence and/or increased tonic stretch reflex excitability, which is consistent with the definition of spasticity. However no differences were found between lower and higher torques within the post-stroke population, reinforcing the idea of loss of modulation after stroke.

6.3.4.3 Variability at different initial activity.

For this analysis only seven post-stroke subjects were included as the requirement for this condition was to be able to flex and extend voluntarily and only this number of post-stroke volunteers was able to perform the instructions.

6.3.4.3.1 Stretch reflex amplitude

- Initially relaxed

No significant differences were found in the stretch reflex amplitudes between the non-impaired population and the post-stroke population for combination of initial position (extended (Figure 10a) or flexed (Figure 11a)) and initial applied torque (low or high) when the voluntary activity previous to de perturbation is relaxed (Table 17).

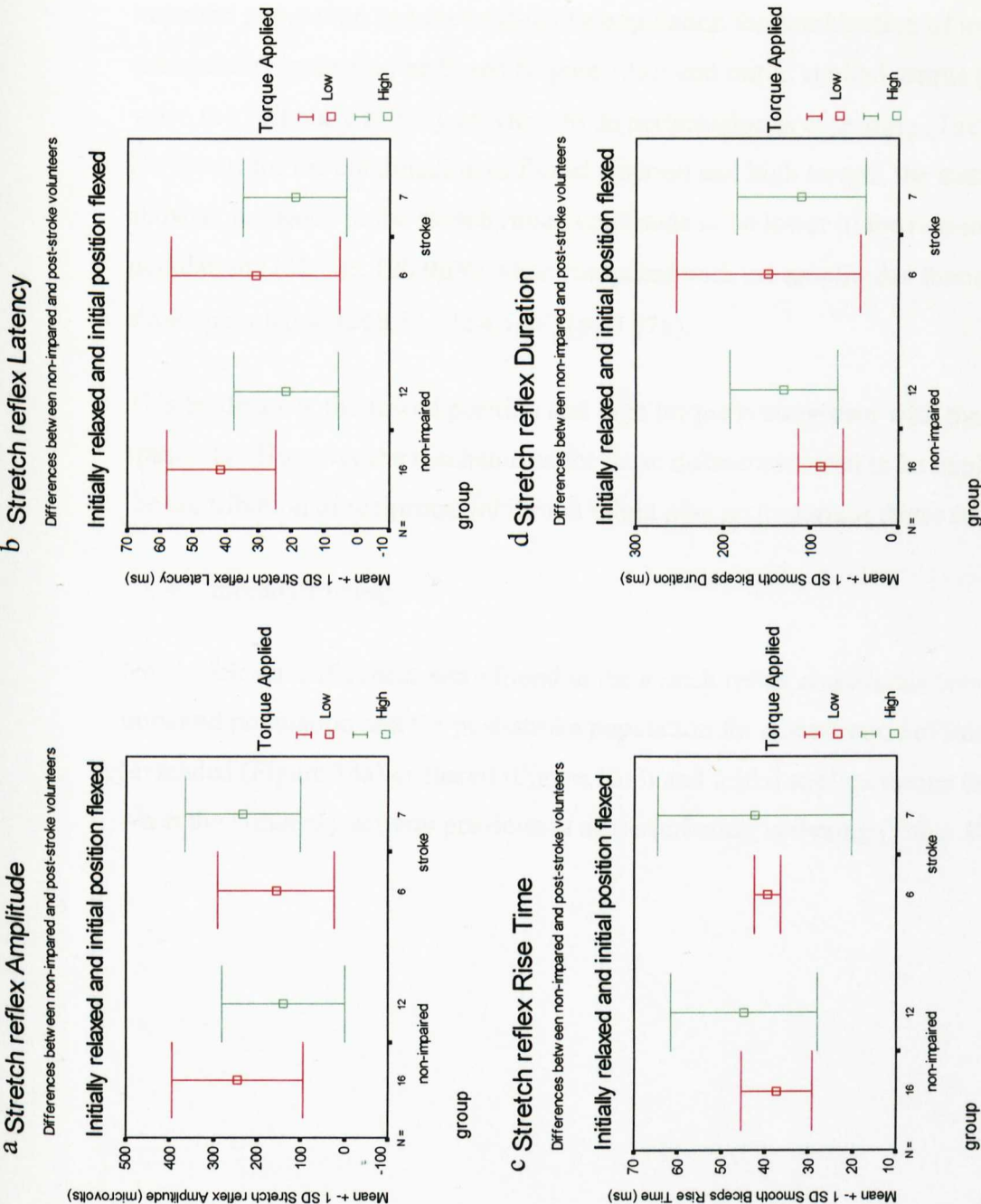


Figure 11: Mean and standard deviation plot of the stretch reflex parameters showing the differences between the post-stroke and non-impaired populations at the high and low torque when the initial position is flexed and the initial voluntary activity is relaxed. A) Amplitude, b) latency, c) rise time and d) duration

- Initially extending

No significant differences were found in the stretch reflex amplitudes between the non-impaired population and the post-stroke population for combination of initial position (extended (Figure 12a) or flexed (Figure 13a)) and initial applied torque (low or high) when the voluntary activity previous to de perturbation is extending (Table 18).

However, for the combination of flexed position and high torque, the statistical analysis showed tendency in the stretch reflex amplitude to be lower in the non-impaired population ($132.74 \pm 120.9 \mu\text{V}$) when compared with the amplitudes found in the post-stroke population ($268.38 \pm 154.5 \mu\text{V}$) ($p=0.076$).

This tendency at the flexed position and high torque is congruent with the definition of spasticity. However the mechanisms for these differences need to be explored further as the contribution of reciprocal inhibition might play an important factor in these findings.

- Initially flexing

No significant differences were found in the stretch reflex amplitudes between the non-impaired population and the post-stroke population for combination of initial position (extended (Figure 14a) or flexed (Figure 15a)) and initial applied torque (low or high) when the voluntary activity previous to de perturbation is flexing (Table 19).

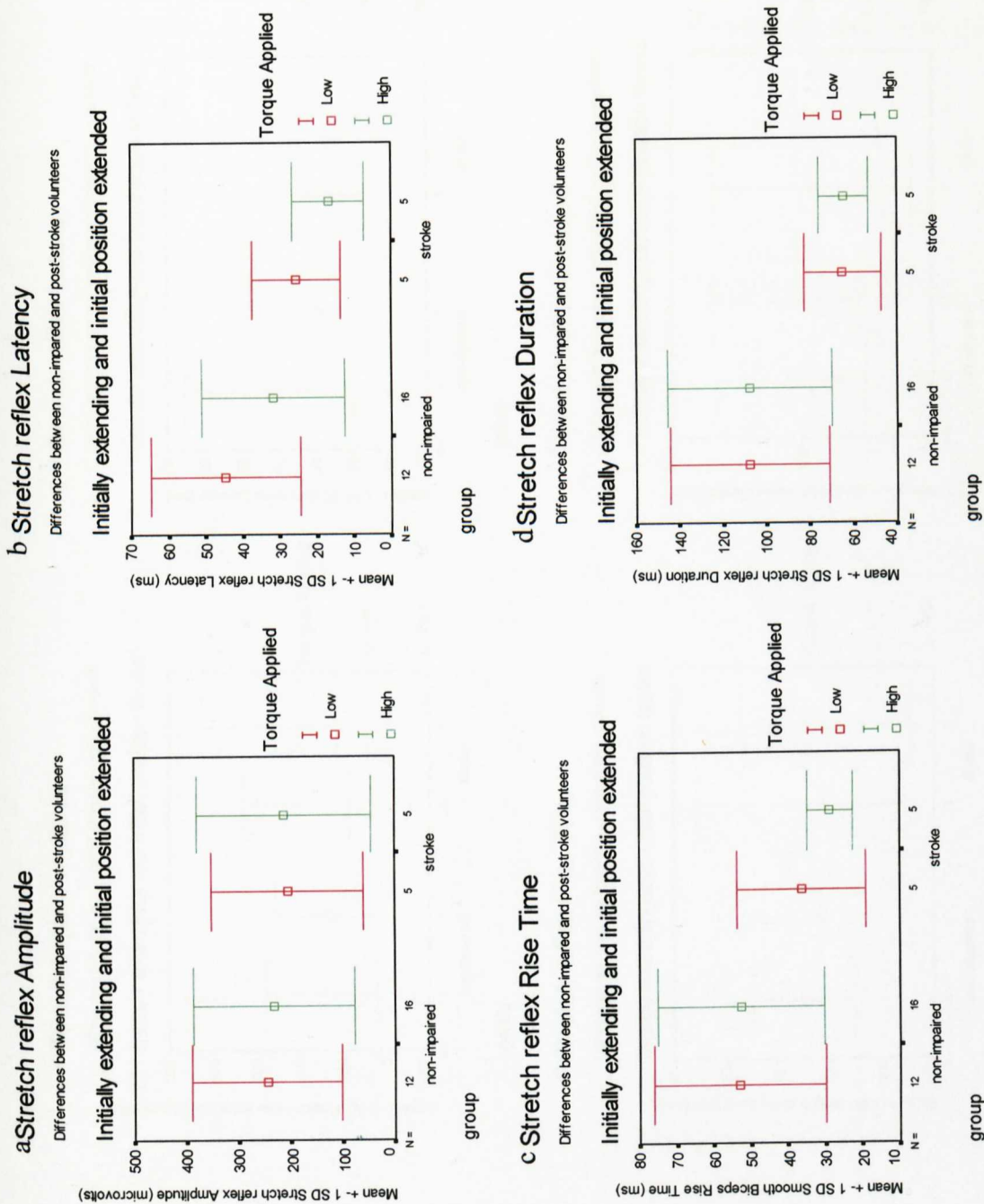


Figure 12: Mean and standard deviation plot of the stretch reflex parameters showing the differences between the post-stroke and non-impaired populations at the high and low torque when the initial position is extended and the initial voluntary activity is extending. A) Amplitude, b) latency, c) rise time and d) duration

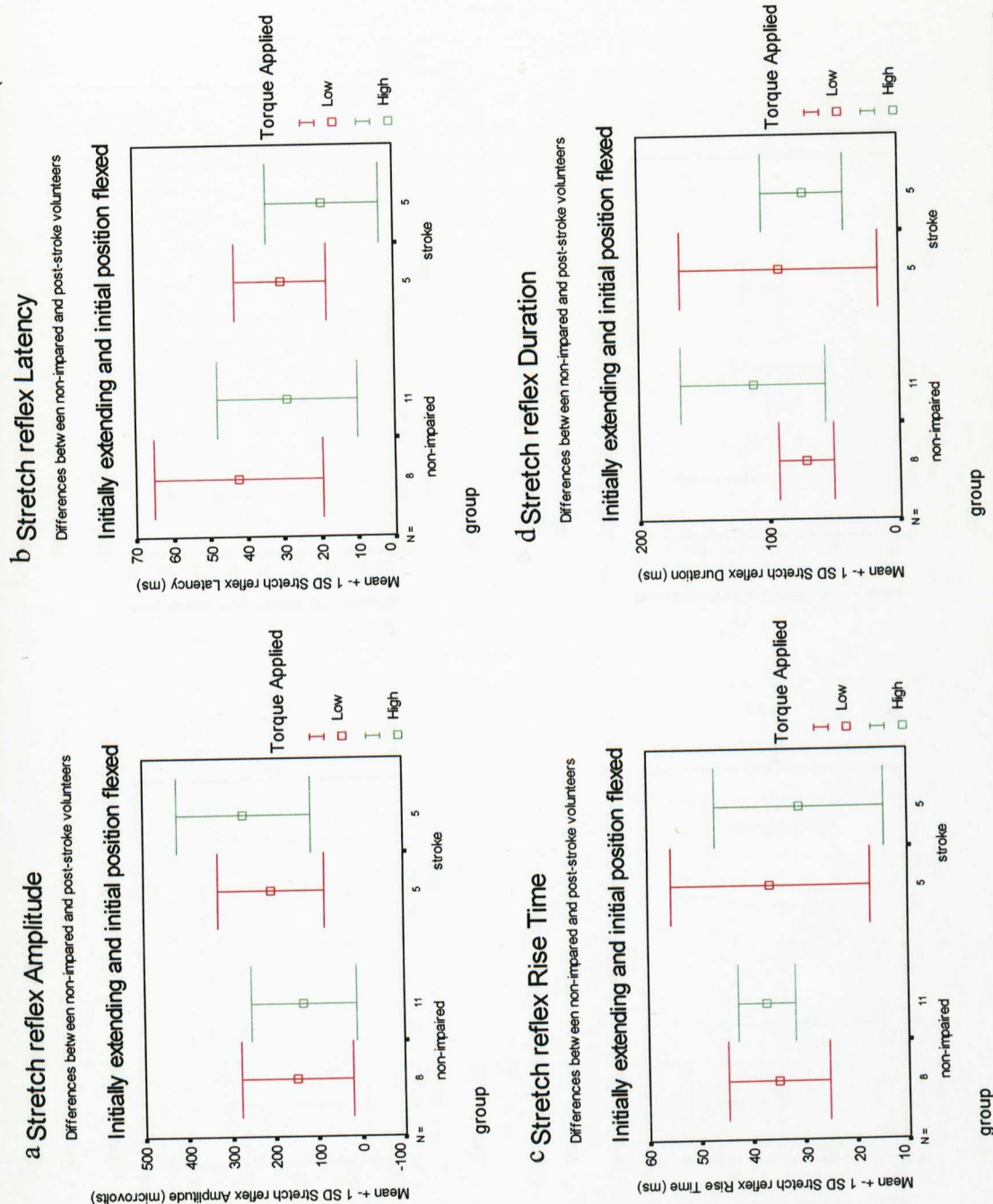


Figure 13: Mean and standard deviation plot of the stretch reflex parameters showing the differences between the post-stroke and non-impaired populations at the high and low torque when the initial position is flexed and the initial voluntary activity is extending. A) Amplitude, b) latency, c) rise time and d) duration

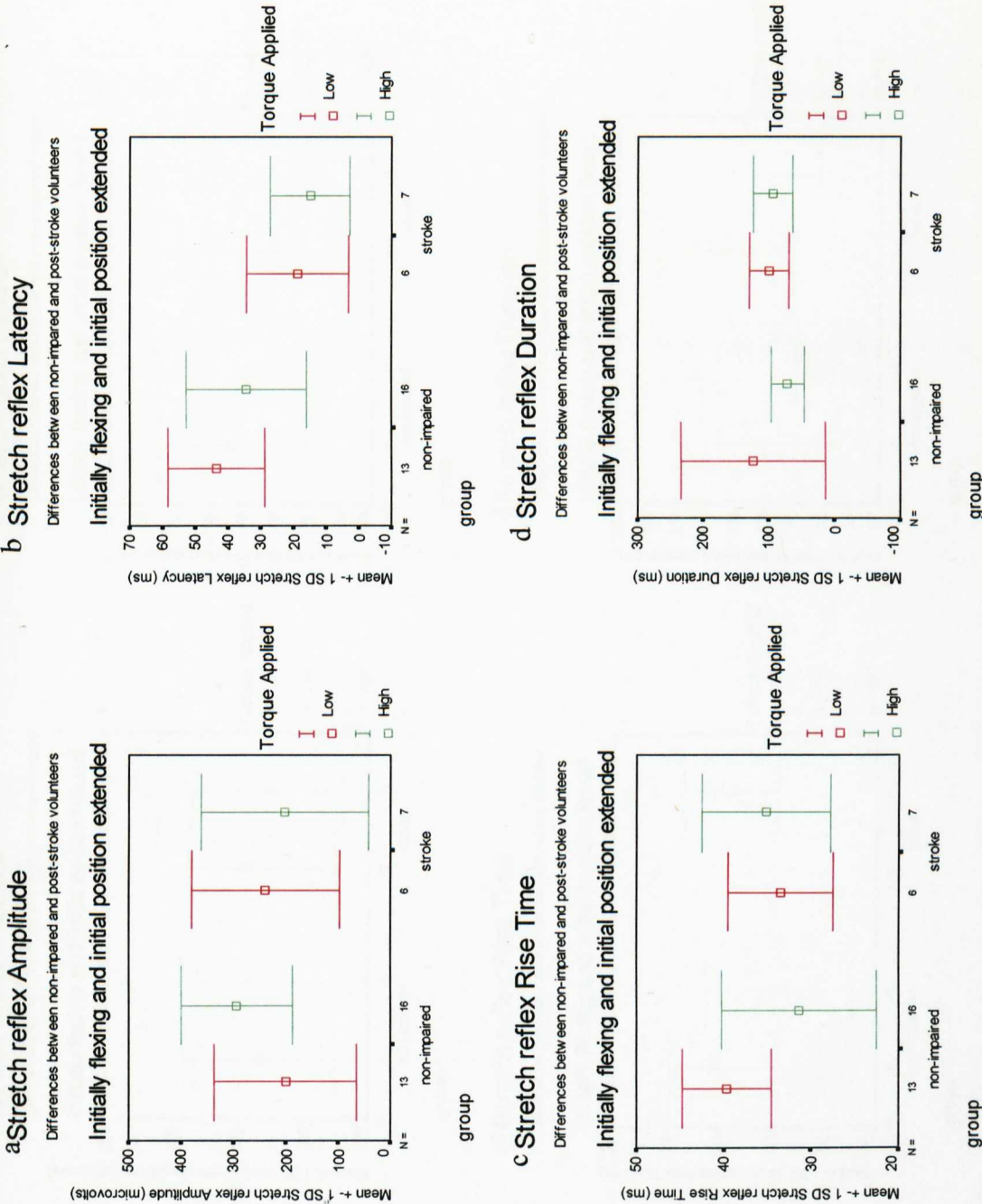


Figure 14: Mean and standard deviation plot of the stretch reflex parameters showing the differences between the post-stroke and non-impaired populations at the high and low torque when the initial position is extended and the initial voluntary activity is flexing. A) Amplitude, b) latency, c) rise time and d) duration

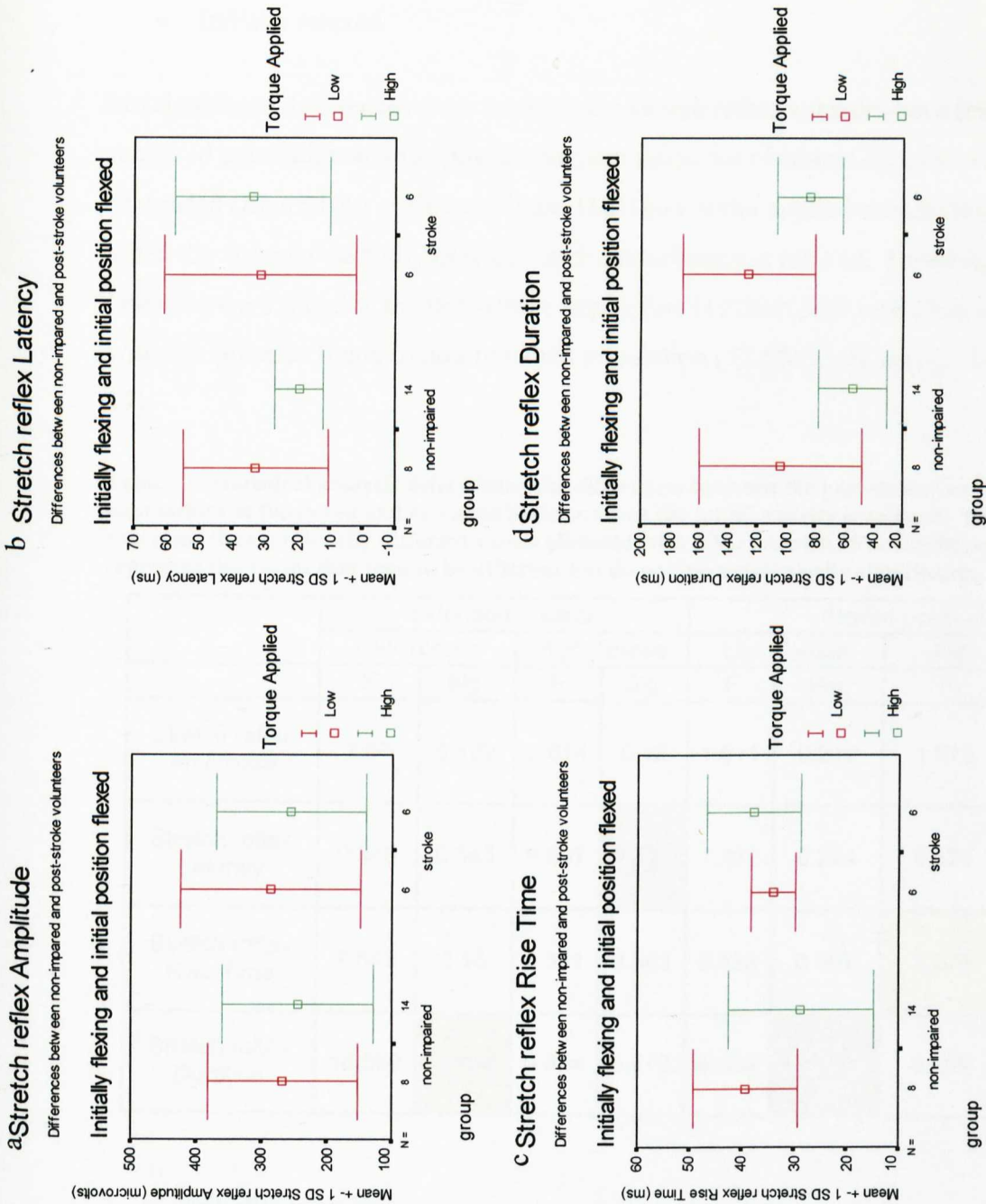


Figure 15: Mean and standard deviation plot of the stretch reflex parameters showing the differences between the post-stroke and non-impaired populations at the high and low torque when the initial position is flexed and the initial voluntary activity is flexing. A) Amplitude, b) latency, c) rise time and d) duration

6.3.4.3.2 Stretch reflex latency

- Initially relaxed

No significant differences were found in the stretch reflex latencies between the non-impaired population and the post-stroke population for combination of initial position (extended (figure 10b) or flexed (figure 11b)) and initial applied torque (low or high) when the voluntary activity previous to de perturbation is relaxed. However, shorter latencies were found in the post-stroke population (19.39 ± 15.37 ms) when compared with the latencies found in non-impaired population (37.58 ± 15.91 ms) ($p=0.018$) (Table 17).

Table 17: Statistical analysis determining the differences between the post-stroke and non-impaired populations at the flexed and extended position when the initial activity is relaxed. The red cells represent the significantly different values obtained with ANOVA with 95% confidence. Yellow cells represent the values that tend to be different but do not have statistically significance.

	Extended position				Flexed position			
	Low Torque		High Torque		Low Torque		High Torque	
	F	Sig.	F	Sig.	F	Sig.	F	Sig.
Stretch reflex Amplitude	2.67	0.122	2.614	0.12	1.611	0.219	1.978	0.178
Stretch reflex Latency	0.946	0.345	6.597	0.018	1.322	0.264	0.126	0.727
Stretch reflex Rise Time	2.542	0.13	0.381	0.543	0.339	0.567	0.076	0.787
Stretch reflex Duration	10.059	0.006	0.024	0.878	4.833	0.04	0.409	0.531

- Initially extending

Statistical analysis showed a tendency in the stretch reflex latencies in the post-stroke population (25.53 ± 11.83 ms) to be lower with the ones found in the non-impaired population (44.75 ± 20.13 ms) ($p=0.067$) at the extended position with low torque (Figure 12b). All other combinations of initial position and applied torque showed no significant differences (Table 18).

- Initially flexing (Table 19)

Significantly shorter latencies were found in the post-stroke population when compared with the ones found in the non-impaired population when the initial position is extended and both the low (post-stroke: 18.71 ± 15.5 ms; non-impaired: 43.58 ± 14.89 ms ($p=0.004$)) and high (post-stroke: 14.96 ± 12.04 ms; non-impaired: 34.33 ± 18.46 ms ($p=0.019$)) torques are applied (Figure 14b).

These findings are consistent with the suggestion of the lack of modulation and lower latencies for the post-stroke population.

No significant differences were found when comparing the stretch reflex latencies in the non-impaired population with those in the post-stroke population when the initial position is flexed and the low torque is applied.

A strong tendency for lower stretch reflex latencies in the non-impaired subjects (19.37 ± 7.46 ms) when compared with those in the post-stroke population (33.73 ± 23.73 ms) ($p=0.05$) when the initial position is flexed and the high torque is applied (Figure 15b).

These findings are opposite to what has been observed previously. This difference may be attributed to the interaction between the spindle sensitivity and the tendency of the non-impaired subjects to maintain a certain level of muscle activity that would be enhanced with the feed forward mechanisms trying to maintain a certain position against a higher torque.

6.3.4.3.3 Stretch reflex rise time

- Initially relaxed

No significant differences were found in the stretch reflex rise times between the non-impaired population and the post-stroke population for combination of initial position (extended (Figure 10c) or flexed Figure 11c) and initial applied torque (low or high) when the voluntary activity previous to de perturbation is relaxed (Table 17).

- Initially extending (Table 18)

No significant differences were found between the stretch reflex rise times in the non-impaired population and those in the post-stroke population when the initial position is extended and the low torque is applied.

Significantly lower stretch reflex rise times were found in the post-stroke population (28.75 ± 6.12 ms) when compared with those in the non-impaired population (52.59 ± 22.38 ms) ($p > 0.1$, = .032) when the initial position is extended and the high torque is applied (Figure 12c).

No significant differences were found between the stretch reflex rise time values in the non-impaired population when compared with those in the post-stroke population when the initial position is flexed and any of the initially torques (high and low) is applied (Figure 13c).

Table 18: Statistical analysis determining the differences between the post-stroke and non-impaired populations at the flexed and extended position when the initial activity is extending. The red cells represent the significantly different values obtained with ANOVA with 95% confidence. Yellow cells represent the values that tend to be different but do not have statistically significance.

	Extended position				Flexed position			
	Low Torque		High Torque		Low Torque		High Torque	
	F	Sig.	F	Sig.	F	Sig.	F	Sig.
Stretch reflex Amplitude	0.271	0.61	0.062	0.806	0.62	0.448	3.664	0.076
Stretch reflex Latency	3.897	0.067	2.687	0.118	1.098	0.317	1.045	0.324
Stretch reflex Rise Time	2.053	0.172	5.363	0.032	0.028	0.87	1.499	0.241
Stretch reflex Duration	6.021	0.027	6.112	0.023	0.514	0.488	2.103	0.169

The increased rise times in the non-impaired subjects under the initially extended position and the high torque combination is explored in previous sections, the results observed in the post-stroke subjects support the idea of lack of modulation.

- Initially flexing (Table 19)

Significantly lower stretch reflex rise times were found in the post-stroke population (33.52 ± 6.03 ms) when compared with those in the non-impaired population (39.64 ± 5.13 ms) ($p=0.035$) when the initial position is extended and the low torque is applied (Figure 14c).

No significant differences were found between the stretch reflex rise times in the non-impaired population and those in the post-stroke population when the initial position is extended and the high torque is applied.

Although the statistical analysis shows significant differences in the extended position at the lower torque, these values are only 6 ms apart from each other suggesting that there could be an artefact in the analysis. It could also be possible that this difference is a result of the post-stroke subjects reaching the maximum value a lot faster than the non-impaired subjects.

No significant differences were found between the stretch reflex rise times in the non-impaired population and those in the post-stroke population at any of the initially applied torques (high and low) when the initial position is flexed (Figure 15c).

6.3.4.3.4 Stretch reflex duration

- Initially relaxed (Table 17)

Significant longer stretch reflex durations were found in the post-stroke population when compared with those in the non-impaired population when the initial applied torque is low at both the extended (post-stroke: 102.47 ± 30.87 ms; non-impaired: 65.51 ± 17.32 ms ($p=0.006$)) (Figure 10d) and flexed position (post-stroke: 148.31 ± 104.68 ms; non-impaired: 88.59 ± 25.32 ms (<0.05 , $=0.04$)) (Figure 11d).

No significant differences were found between stretch reflex durations values in the non-impaired population and those in the post-stroke population when the initial applied torque is high at any of the initial positions (extended or flexed).

Table 19: Statistical analysis determining the differences between the post-stroke and non-impaired populations at the flexed and extended position when the initial activity is flexing. The red cells represent the significantly different values obtained with ANOVA with 95% confidence. Yellow cells represent the values that tend to be different but do not have statistical significance.

	Extended position				Flexed position			
	Low Torque		High Torque		Low Torque		High Torque	
	F	Sig.	F	Sig.	F	Sig.	F	Sig.
Stretch reflex Amplitude	0.33	0.573	2.651	0.118	0.082	0.78	0.041	0.841
Stretch reflex Latency	11.173	0.004	6.41	0.019	0.01	0.922	4.403	0.05
Stretch reflex Rise Time	5.236	0.035	0.951	0.341	1.452	0.251	2.21	0.154
Stretch reflex Duration	0.276	0.606	3.637	0.07	0.653	0.435	6.955	0.017

Longer durations in the post-stroke volunteers suggest an increased tonic stretch reflex component.

- Initially extending (Table 18)

Significantly shorter stretch reflex durations were found in the post-stroke population when compared with those in the non-impaired population when the initial position is extended (Figure 12d) and the applied torque is both low (post-stroke: 64.94 ± 17.5 ms; non-impaired: 107.54 ± 36.59 ms ($p = 0.027$)) and high (post-stroke: 64.64 ± 11.5 ms; non-impaired: 107.60 ± 37.70 ms ($p = 0.023$)).

Shorter durations in the post-stroke subjects when voluntarily extending can be a result of the reciprocal inhibition and/or some other different neural pathways that are not modulating the duration in the same way of the non-impaired population.

No significant differences were found between the stretch reflex durations in the non-impaired population and those in the post-stroke population when the starting position is flexed and both the low and high torques are applied (Figure 13d).

- Initially flexing (Table 19)

No significant differences were found between the stretch reflex durations in the non-impaired population and those in the post-stroke population at the extended position and both the low and high torque (Figure 14d). However there is a tendency for these values to be lower in the non-impaired population (71.35 ± 25.13 ms) when compared with those in the post-stroke population (94.34 ± 29.96 ms) ($p > 0.05$, $= 0.070$) at this extended position with the high torque.

No significant differences were found when comparing the stretch reflex durations in the non-impaired population with the those in the post-stroke population at the flexed position when the low torque is applied (Figure 15d).

Significantly shorter stretch reflex durations were found in the non-impaired population (54.28 ± 23.25 ms) when compared with the ones in the post-stroke population (83.82 ± 22.13 ms) ($p = 0.017$) at the flexed position when the higher torque is applied (Figure 15d).

Longer durations in the post-stroke population suggest increased tonic stretch reflex components.

6.3.4.4 Variability due to different head position

6.3.4.4.1 Stretch reflex amplitude

- Head straight

Stretch reflex amplitudes were significantly lower in the post-stroke population (121.52 ± 83.150 μ V) when compared with those in the non-impaired population (264.79 ± 158.64 μ V) ($p = 0.005$) (Table 20).

- Head left

Stretch reflex amplitudes were significantly lower in the post-stroke population (136.26 ± 105.79 μ V) when compared with those in the non-impaired population (303.74 ± 122.99 μ V) ($p = 0.001$) (Table 21).

- Head right

Stretch reflex amplitudes were significantly lower in the post-stroke population ($124.37 \pm 101.05 \mu\text{V}$) when compared with those in the non-impaired population ($314.42 \pm 98.94 \mu\text{V}$) ($p = 0.00$) (Table 22).

Figure 16a shows the mean and standard deviation plot of the differences in the stretch reflex amplitude between non-impaired and post-stroke subjects.

6.3.4.4.2 Stretch reflex latency

- Head straight

Stretch reflex latencies were significantly shorter in the post-stroke population ($18.06 \pm 11.72 \mu\sigma$) when compared with those in the non-impaired population ($37.58 \pm 15.91 \text{ ms}$) ($p = 0.001$) (Table 20).

- Head left

Stretch reflex latencies were significantly shorter in the post-stroke population ($15.51 \pm 6.97 \text{ ms}$) when compared with those in the non-impaired population ($27.91 \pm 16.8 \text{ ms}$) ($p = 0.029$) (Table 21).

- Head right

Stretch reflex latencies were significantly shorter in the post-stroke population ($16.37 \pm 9.77 \text{ ms}$) when compared with those in the non-impaired population ($39.78 \pm 13.47 \text{ ms}$) ($p = 0.000$) (Table 22).

Figure 16b shows the mean and standard deviation plot of the differences in the stretch reflex latencies between non-impaired and post-stroke subjects.

6.3.4.4.3 Stretch reflex rise time

No significant differences were found when comparing the stretch reflex rise time values in the non-impaired population with the those in the post-stroke population where subjects are facing straight (Table 20) or to the left (Table 21) .

Stretch reflex rise time values were significantly higher in the non-impaired population (49.27 ± 20.63 ms) when compared with those in the post-stroke population (32.32 ± 8.47 ms). ($p < 0.05$, $= 0.01$) where subjects were facing to the right (Table 22).

Figure 16c shows the mean and standard deviation plot of the differences in the stretch reflex rise time between non-impaired and post-stroke subjects.

6.3.4.4.4 Stretch reflex duration

No significant differences were found when comparing the durations of the stretch reflex in the non-impaired population with those in the post-stroke population where subjects were facing straight (Table 20), to the left (Table 21) or to the right (Table 22).

However, this parameter showed a tendency to be lower in the non-impaired population (67.74 ± 23.19 ms) when compared with the duration of the reflex in the post-stroke population where subjects were facing to the left (Table 21) (94.10 ± 67.74 ms) ($p > 0.05$, $= 0.078$).

Figure 16a shows the mean and standard deviation plot of the differences in the stretch reflex duration between non-impaired and post-stroke subjects.

The changes in the parameters due to the direction where the head is turned could be a result of higher centre contribution to the modulation of the stretch reflex.

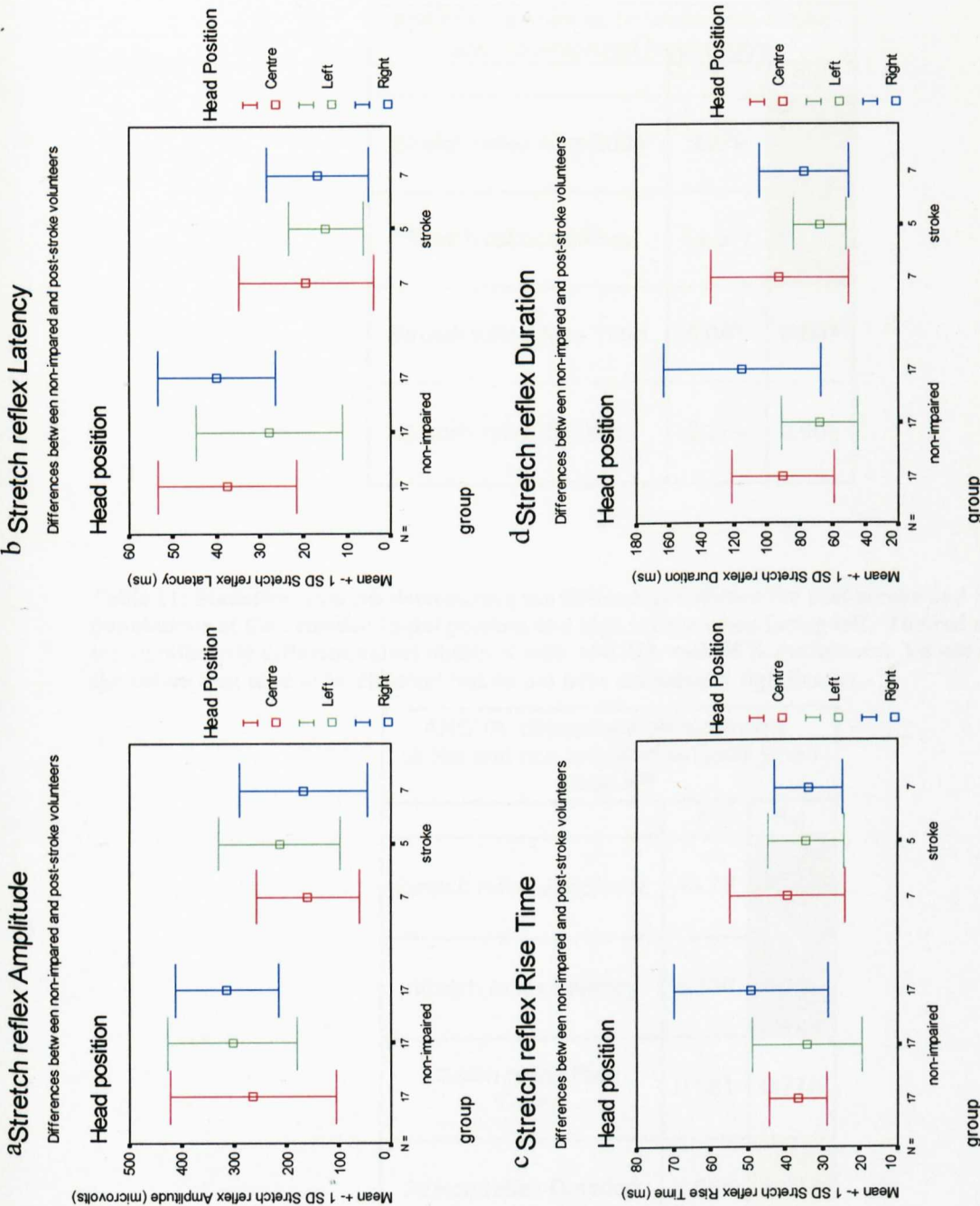


Figure 16: Mean and standard deviation plot of the stretch reflex amplitude showing the differences between the post-stroke and non-impaired populations at the different head positions during the perturbation (centre, left or right).

Table 20: Statistical analysis determining the differences between the post-stroke and non-impaired populations at the extended initial position at a high torque when facing straight. The red cells represent the significantly different values obtained with ANOVA with 95% confidence.

ANOVA differences between post-stroke and non-impaired head centre		
	F	Sig.
Stretch reflex Amplitude	9.279	0.005
Stretch reflex Latency	14.517	0.001
Stretch reflex Rise Time	0.061	0.807
Stretch reflex Duration	0.274	0.605

Table 21: Statistical analysis determining the differences between the post-stroke and non-impaired populations at the extended initial position at a high torque when facing left. The red cells represent the significantly different values obtained with ANOVA with 95% confidence. Yellow cells represent the values that tend to be different but do not have statistical significance.

ANOVA differences between post-stroke and non-impaired subjects when head left		
	F	Sig.
Stretch reflex Amplitude	13.76	0.001
Stretch reflex Latency	5.336	0.029
Stretch reflex Rise Time	0.081	0.778
Stretch reflex Duration	3.366	0.078

Table 22: Statistical analysis determining the differences between the post-stroke and non-impaired populations at the extended initial position at a high torque when facing right. The red cells represent the significantly different values obtained with ANOVA with 95% confidence.

ANOVA differences between post-stroke and non-impaired subjects when head right		
	F	Sig.
Stretch reflex Amplitude	26.684	0.000
Stretch reflex Latency	27.889	0.000
Stretch reflex Rise Time	7.727	0.010
Stretch reflex Duration	2.367	0.135

6.3.5 General Linear Model

The GLM Multivariate procedure provides regression analysis and analysis of variance for multiple dependent variables by one or more factor variables or covariates. The factor variables divide the population into groups. It is possible to test null hypotheses about the effects of factor variables on the means of various groupings of a joint distribution of dependent variables using this general linear model procedure. Also interactions between factors as well as the effects of individual factors can be investigated.

Two multivariate general linear model analyses were computed in SPSS, one for the non-impaired subjects and one with the post-stroke volunteers, with the aim of looking at the effect of the different conditions and their interaction on the stretch reflex parameters previously described (amplitude, latency, rise time and duration).

6.3.5.1 Non-impaired subjects

6.3.5.1.1 Stretch reflex amplitude

It was observed that the stretch reflex amplitude was considerably influenced by the initial position ($p=0.007$) and to a lesser extent by the initial activity ($p=0.069$) (Table 24), particularly where subjects are extending voluntarily ($p=0.013$) when compared with the initially relaxed condition (Table 25) (Figure 17).

6.3.5.1.2 Stretch reflex latency

It was observed that the stretch reflex latency was considerably influenced by the applied torque ($p=0.001$), the interaction between applied torque and starting angular position ($p=0.021$), and the interaction between initial angle, applied torque and initial activity ($p=0.020$) (Table 24) (Figure 18).

6.3.5.1.3 Stretch reflex rise time

It was observed that the stretch reflex rise time is mostly influenced by the initial activity ($p=0.004$) (Table 24), particular differences were found when comparing initially relaxed with initially extending ($p=0.020$) and initially flexing with initially extending ($p=0.000$) (Table 25). This parameter is also influence by the head position ($p=0.004$), the differences found were when comparing the rise time when facing right with facing straight ($p=0.015$) and when facing left with facing right ($p=0.006$) (Table 26). The interaction between the initial activity and the starting angular position ($p=0.000$) and the interaction between initial activity and applied torque ($p=0.026$) (Figure 19).

6.3.5.1.4 Stretch reflex duration

The stretch reflex duration is mostly influenced by the head position ($p=0.011$). The main differences were found when comparing between facing left with facing right ($p=0.009$) (Table 26). This parameter is also influenced by the interaction between the initial activity and the starting angle ($p=0.006$) and the torque applied and initial activity ($p=0.000$) (Table 24) (Figure 20).

Table 23: non-impaired population Pillai’s trace multivariate test determining the effects of each of the factors towards the GLM analysis. Pillai’s trace is considered to be the most robust and powerful criterion. The red cells represent the significantly effects in the model with 95% confidence.

Pillai's Trace Multivariate Test	
Effect	Sig.
Intercept	0
Initial position	0.035
Initially applied torque	0.015
Voluntary activity	0
Head position	0.002
Initial position * Initially applied torque	0.143
Initial position * Voluntary activity	0
Initially applied torque * Voluntary activity	0
Initial position * Initially applied torque * Voluntary activity	0.173

The differences due to the interaction between the conditions strongly suggest that the stretch reflex parameters values depend on physiological variables interacting with each other such as the muscle spindle and Golgi tendon organ sensitivity and gain. These variables along with feed forward mechanisms and presynaptic, reciprocal and autogenic inhibition increase or decrease the overall stretch reflex excitability.

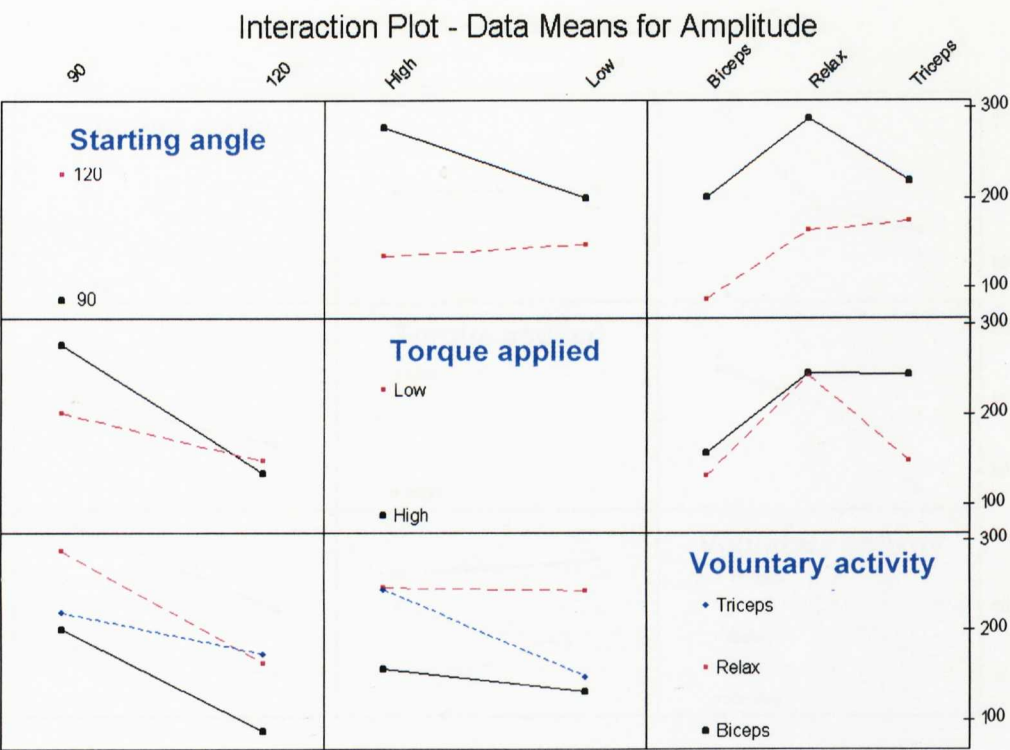


Figure 17: Interaction plot of the different conditions (starting angle, torque applied and voluntary activity) influencing the stretch reflex amplitude (μV) within the non-impaired population.

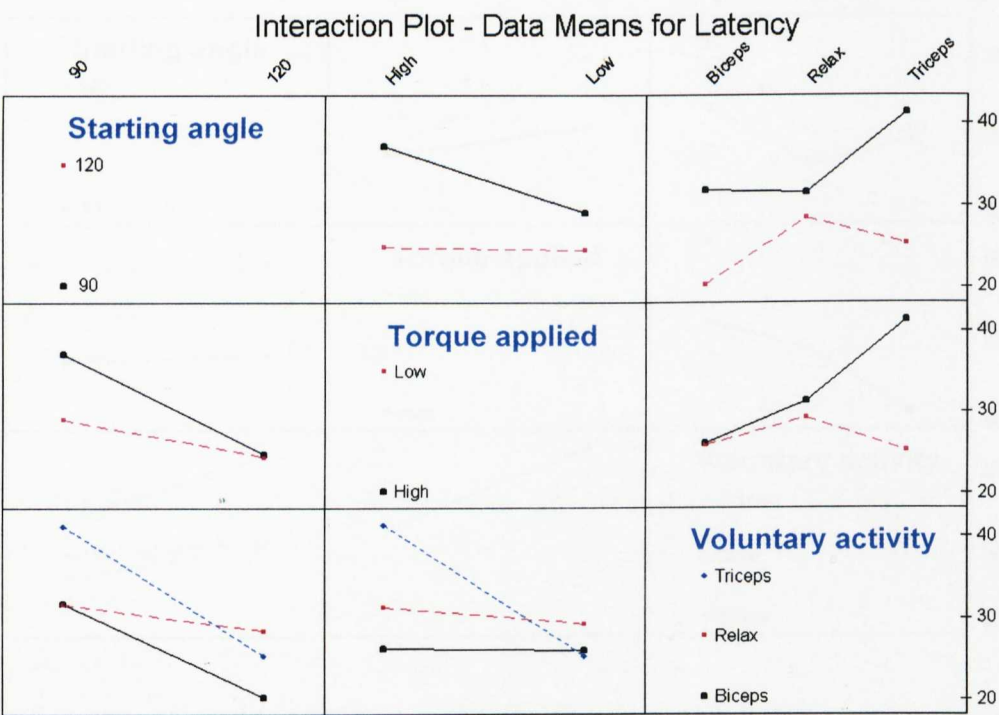


Figure 18: Interaction plot of the different conditions (starting angle, torque applied and voluntary activity) influencing stretch reflex latency (ms) within the non-impaired population.

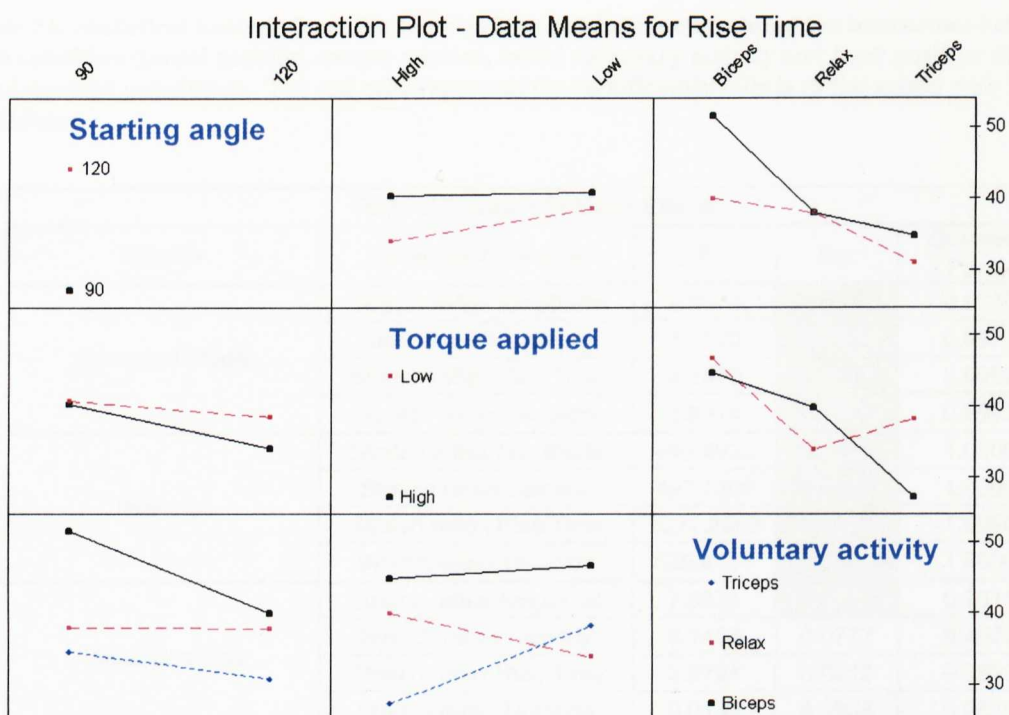


Figure 19: Interaction plot of the different conditions (starting angle, torque applied and voluntary activity) influencing the stretch reflex rise time (ms) within the non-impaired population.

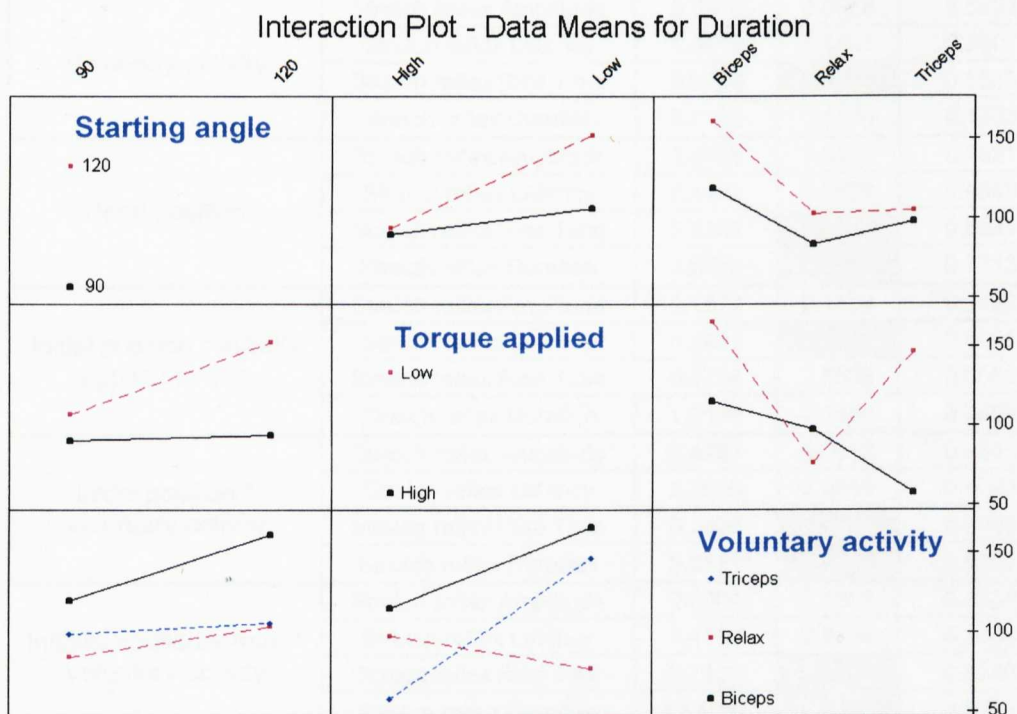


Figure 20: Interaction plot of the different conditions (starting angle, torque applied and voluntary activity) influencing the stretch reflex duration (ms) within the non-impaired population.

Table 24: Statistical analysis from the General Linear Model determining the interaction between each condition (initial position, torque applied, initial voluntary activity and head position for the non-impaired population. The red cells represent the significantly effects in the model with 95% confidence.

Tests of Between-Subjects Effects				
Source	Dependent Variable	F	Sig.	Observed Power
Corrected Model	Stretch reflex Amplitude	2.6674	0.0019	0.9838
	Stretch reflex Latency	3.3620	0.0001	0.9972
	Stretch reflex Rise Time	4.2370	0.0000	0.9998
	Stretch reflex Duration	3.8214	0.0000	0.9992
Intercept	Stretch reflex Amplitude	445.4933	0.0000	1.0000
	Stretch reflex Latency	497.1306	0.0000	1.0000
	Stretch reflex Rise Time	1021.2863	0.0000	1.0000
	Stretch reflex Duration	535.4117	0.0000	1.0000
Initial position	Stretch reflex Amplitude	7.5674	0.0066	0.7811
	Stretch reflex Latency	3.1493	0.0777	0.4228
	Stretch reflex Rise Time	2.8721	0.0919	0.3920
	Stretch reflex Duration	0.0132	0.9088	0.0515
Initially applied torque	Stretch reflex Amplitude	0.5301	0.4675	0.1119
	Stretch reflex Latency	10.9973	0.0011	0.9096
	Stretch reflex Rise Time	0.1776	0.6739	0.0704
	Stretch reflex Duration	0.0156	0.9006	0.0518
Voluntary activity	Stretch reflex Amplitude	2.7180	0.0688	0.5323
	Stretch reflex Latency	1.6814	0.1891	0.3507
	Stretch reflex Rise Time	5.5768	0.0045	0.8515
	Stretch reflex Duration	0.7186	0.4889	0.1703
Head position	Stretch reflex Amplitude	0.6735	0.5112	0.1621
	Stretch reflex Latency	2.4422	0.0899	0.4868
	Stretch reflex Rise Time	5.5858	0.0044	0.8521
	Stretch reflex Duration	4.5832	0.0115	0.7713
Initial position * Initially applied torque	Stretch reflex Amplitude	2.1877	0.1409	0.3128
	Stretch reflex Latency	5.4405	0.0208	0.6405
	Stretch reflex Rise Time	0.0754	0.7839	0.0586
	Stretch reflex Duration	1.9139	0.1683	0.2800
Initial position * Voluntary activity	Stretch reflex Amplitude	2.4257	0.0913	0.4840
	Stretch reflex Latency	2.3906	0.0945	0.4780
	Stretch reflex Rise Time	8.2808	0.0004	0.9598
	Stretch reflex Duration	5.2171	0.0063	0.8257
Initially applied torque * Voluntary activity	Stretch reflex Amplitude	2.0004	0.1383	0.4093
	Stretch reflex Latency	1.4160	0.2454	0.3009
	Stretch reflex Rise Time	3.7109	0.0264	0.6749
	Stretch reflex Duration	12.4207	0.0000	0.9958
Initial position * Initially applied torque * Voluntary activity	Stretch reflex Amplitude	0.5469	0.5797	0.1395
	Stretch reflex Latency	4.0215	0.0196	0.7122
	Stretch reflex Rise Time	0.1599	0.8524	0.0744
	Stretch reflex Duration	0.4681	0.6270	0.1257

Table 25: Bonferroni post-hoc analysis to determine the influence of the initial voluntary activity from the general linear model for the non-impaired population. The red cells represent the significantly effects in the model with 95% confidence.

Multiple Comparisons					
Bonferroni					
Dependent Variable	(I) Voluntary Activity	(J) Voluntary Activity	Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Relax	Triceps	67.760	0.2349	0.0132
		Biceps	14.090	0.2287	1.0000
	Triceps	Relax	-67.760	0.2349	0.0132
		Biceps	-53.670	0.2654	0.1339
	Biceps	Relax	-14.090	0.2287	1.0000
		Triceps	53.670	0.2654	0.1339
Stretch reflex Latency	Relax	Triceps	-3.5739	2.9798	0.6960
		Biceps	0.3873	2.9012	1.0000
	Triceps	Relax	3.5739	2.9798	0.6960
		Biceps	3.9611	3.3666	0.7228
	Biceps	Relax	-0.3873	2.9012	1.0000
		Triceps	-3.9611	3.3666	0.7228
Stretch reflex Rise Time	Relax	Triceps	-6.9781	2.5384	0.0198
		Biceps	5.1442	2.4714	0.1165
	Triceps	Relax	6.9781	2.5384	0.0198
		Biceps	12.1224	2.8679	0.0001
	Biceps	Relax	-5.1442	2.4714	0.1165
		Triceps	-12.1224	2.8679	0.0001
Stretch reflex Duration	Relax	Triceps	-10.6447	8.1664	0.5823
		Biceps	6.7113	7.9508	1.0000
	Triceps	Relax	10.6447	8.1664	0.5823
		Biceps	17.3560	9.2264	0.1848
	Biceps	Relax	-6.7113	7.9508	1.0000
		Triceps	-17.3560	9.2264	0.1848

Table 26: Bonferroni post-hoc analysis to determine the influence of the head position from the general linear model for the non-impaired population. The red cells represent the significantly effects in the model with 95% confidence.

Multiple Comparisons					
Bonferroni					
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Centre	Left	-0.717	0.3351	0.1009
		Right	-0.824	0.3351	0.0446
	Left	Centre	0.717	0.3351	0.1009
		Right	-0.107	0.4501	1.0000
	Right	Centre	0.824	0.3351	0.0446
		Left	0.107	0.4501	1.0000
Stretch reflex Latency	Centre	Left	5.4886	4.2513	0.5951
		Right	-6.3737	4.2513	0.4068
	Left	Centre	-5.4886	4.2513	0.5951
		Right	-11.8624	5.7110	0.1177
	Right	Centre	6.3737	4.2513	0.4068
		Left	11.8624	5.7110	0.1177
Stretch reflex Rise Time	Centre	Left	4.8928	3.6215	0.5352
		Right	-10.2870	3.6215	0.0151
	Left	Centre	-4.8928	3.6215	0.5352
		Right	-15.1798	4.8649	0.0063
	Right	Centre	10.2870	3.6215	0.0151
		Left	15.1798	4.8649	0.0063
Stretch reflex Duration	Centre	Left	25.3053	11.6508	0.0936
		Right	-22.0580	11.6508	0.1799
	Left	Centre	-25.3053	11.6508	0.0936
		Right	-47.3633	15.6511	0.0085
	Right	Centre	22.0580	11.6508	0.1799
		Left	47.3633	15.6511	0.0085

6.3.5.2 Post-stroke subjects model

According to the general linear model, only the angular position has a tendency to influence the model (p=0.065).

The individual effects on each parameter are explained below:

6.3.5.2.1 Stretch reflex amplitude

It was observed that the stretch reflex amplitude was not influenced by any of the factors (Table 28). The interaction plot for this parameter between the conditions is shown in figure 21.

6.3.5.2.2 Stretch reflex latency

It was observed that the stretch reflex latency was influenced by the initial angular position ($p=0.043$) (Table 28). The interaction plot for this parameter between the conditions is shown in figure 22. The influence of the angular position on the latency of the stretch reflex can be due to the tension of the Golgi tendon organ and/or the sensitivity of the muscle spindle due to the initial length of the intrafusal muscle fibres.

6.3.5.2.3 Stretch reflex rise time

It was observed that the stretch reflex rise time was not influenced by any of the factors (Table 28). The interaction plot for this parameter between the conditions is shown in figure 23.

6.3.5.2.4 Stretch reflex duration

According to the model, the stretch reflex duration was mostly influenced by the initial activity ($p=0.029$) (Table 28). However the post-hoc tests did not show any significant variability due to this factor on the stretch reflex duration (Table 29). The interaction plot for this parameter between the conditions is shown in figure 24.

The lack of variation of the stretch reflex parameters strongly suggests that there is a lack of modulation on the stretch reflex excitability after the stroke. This lack of modulation can be responsible for the clinical signs that influence the diagnosis of spasticity which could also be masked by the altered biomechanical properties of the muscle which are also found along with spasticity.

Table 27: Post-stroke population Pillai’s trace multivariate test determining the effects of each of the factors towards the GLM analysis. Pillai’s trace is considered to be the most robust and powerful criterion among the others. The red cells represent the significantly effects in the model with 95% confidence.

Pillai's Trace Multivariate Test	
Effect	Sig.
Intercept	0
Initial position	0.065
Initially applied torque	0.202
Voluntary activity	0.117
Head position	0.95
Initial position * Initially applied torque	0.41
Initial position * Voluntary activity	0.972
Initially applied torque * Voluntary activity	0.801
Initial position * Initially applied torque * Voluntary activity	0.98

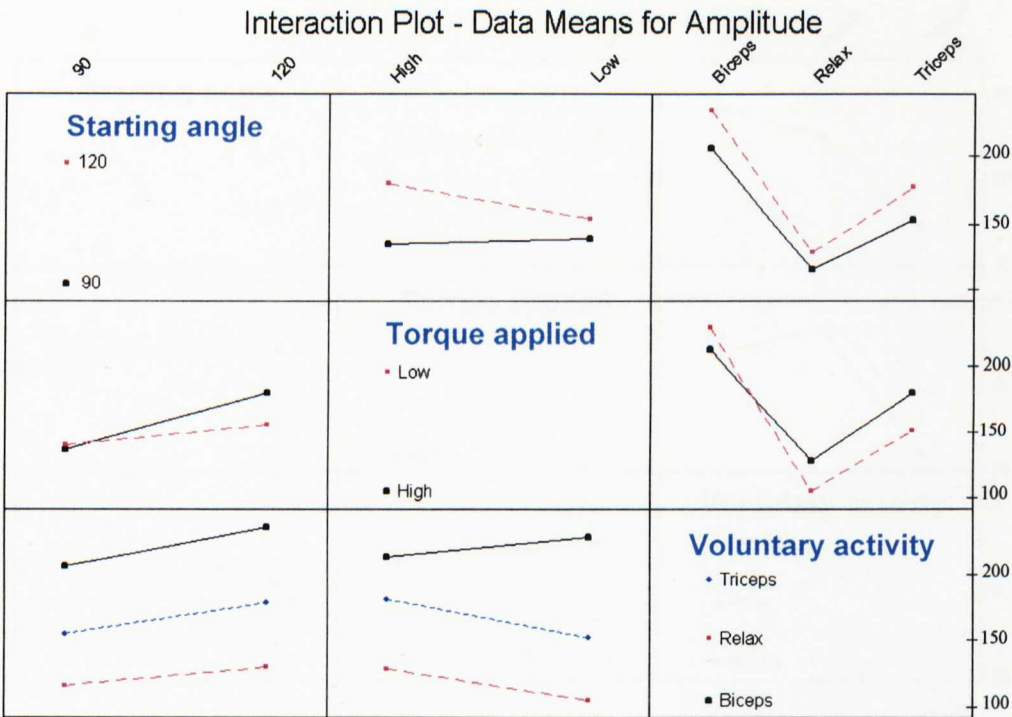


Figure 21: Interaction plot of the different conditions (starting angle, torque applied and voluntary activity) influencing the stretch reflex amplitude (μV) within the post-stroke population.

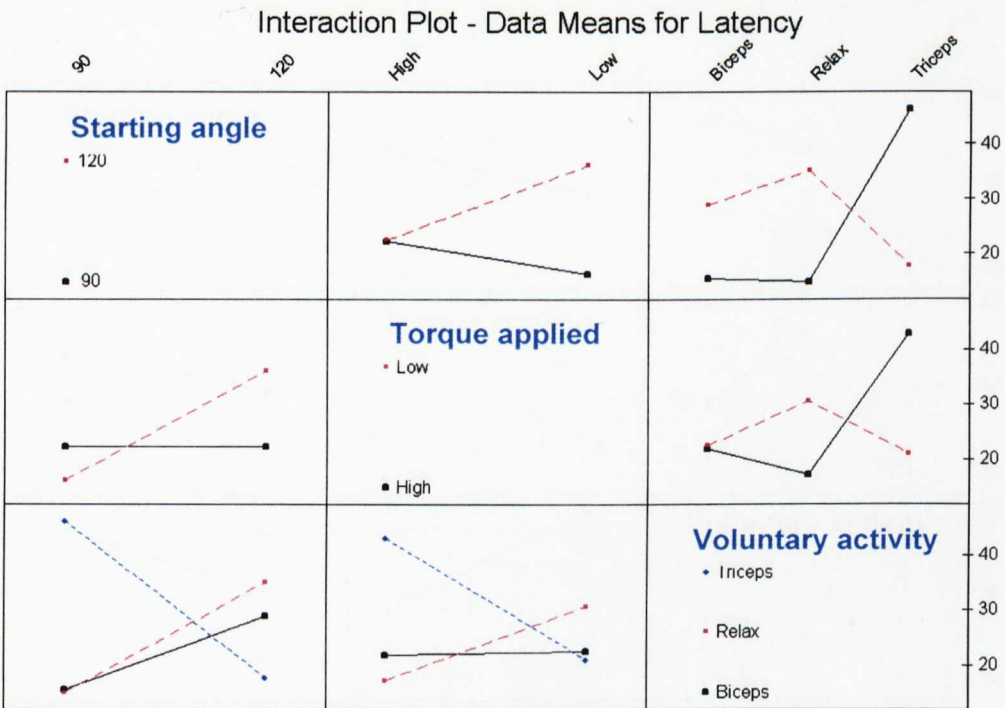


Figure 22: Interaction plot of the different conditions (starting angle, torque applied and voluntary activity) influencing the stretch reflex latency (ms) within the post-stroke population.

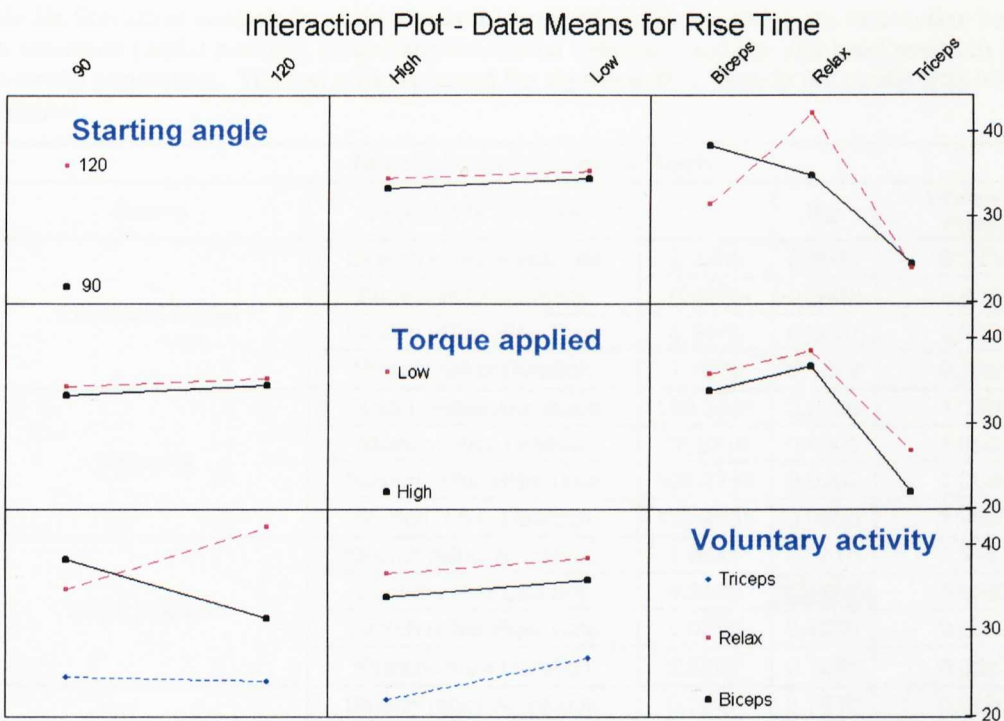


Figure 23: Interaction plot of the different conditions (starting angle, torque applied and voluntary activity) influencing the stretch reflex rise time (ms) within the post-stroke population.

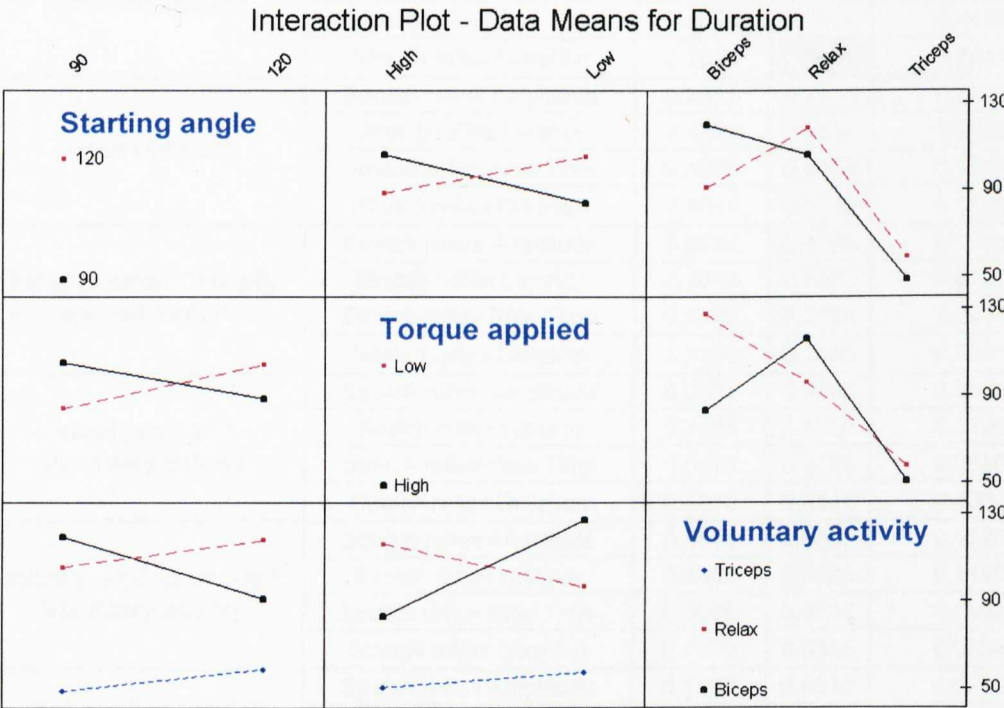


Figure 24: Interaction plot of the different conditions (starting angle, torque applied and voluntary activity) influencing the stretch reflex duration (ms) within the post-stroke population.

Table 28: Statistical analysis from the General Linear Model determining the interaction between each condition (initial position, torque applied, initial voluntary activity and head position) for the post-stroke population. The red cells represent the significant effects in the model with 95% confidence.

Tests of Between-Subjects Effects				
Source	Dependent Variable	F	Sig.	Observed Power
Corrected Model	Stretch reflex Amplitude	0.5205	0.9041	0.2755
	Stretch reflex Latency	0.8929	0.5642	0.4850
	Stretch reflex Rise Time	0.5475	0.8857	0.2904
	Stretch reflex Duration	1.4056	0.1802	0.7296
Intercept	Stretch reflex Amplitude	130.3867	0.0000	1.0000
	Stretch reflex Latency	77.5216	0.0000	1.0000
	Stretch reflex Rise Time	401.3746	0.0000	1.0000
	Stretch reflex Duration	173.4837	0.0000	1.0000
Initial position	Stretch reflex Amplitude	1.0841	0.3015	0.1767
	Stretch reflex Latency	4.2480	0.0432	0.5285
	Stretch reflex Rise Time	0.0865	0.7696	0.0597
	Stretch reflex Duration	2.6267	0.1098	0.3587
Initially applied torque	Stretch reflex Amplitude	0.0947	0.7593	0.0606
	Stretch reflex Latency	1.4294	0.2361	0.2181
	Stretch reflex Rise Time	0.1613	0.6892	0.0681
	Stretch reflex Duration	2.5585	0.1144	0.3509
Voluntary activity	Stretch reflex Amplitude	1.3605	0.2635	0.2833
	Stretch reflex Latency	0.2072	0.8134	0.0811
	Stretch reflex Rise Time	2.2653	0.1117	0.4455
	Stretch reflex Duration	3.7293	0.0291	0.6641
Head position	Stretch reflex Amplitude	0.2477	0.7813	0.0874
	Stretch reflex Latency	0.1020	0.9032	0.0650
	Stretch reflex Rise Time	0.4208	0.6583	0.1155
	Stretch reflex Duration	0.4014	0.6710	0.1123
Initial position * Initially applied torque	Stretch reflex Amplitude	0.6072	0.4386	0.1198
	Stretch reflex Latency	0.2013	0.6552	0.0727
	Stretch reflex Rise Time	0.2770	0.6004	0.0814
	Stretch reflex Duration	1.3208	0.2545	0.2051
Initial position * Voluntary activity	Stretch reflex Amplitude	0.0551	0.9465	0.0580
	Stretch reflex Latency	0.7415	0.4802	0.1708
	Stretch reflex Rise Time	0.0210	0.9792	0.0530
	Stretch reflex Duration	0.3810	0.6847	0.1089
Initially applied torque * Voluntary activity	Stretch reflex Amplitude	0.4641	0.6307	0.1228
	Stretch reflex Latency	0.4052	0.6685	0.1129
	Stretch reflex Rise Time	0.8005	0.4534	0.1812
	Stretch reflex Duration	0.1576	0.8545	0.0734
Initial position * Initially applied torque * Voluntary activity	Stretch reflex Amplitude	0.1617	0.8510	0.0740
	Stretch reflex Latency	0.5409	0.5848	0.1358
	Stretch reflex Rise Time	0.0360	0.9647	0.0552
	Stretch reflex Duration	0.0499	0.9514	0.0572

Table 29: Bonferroni post-hoc analysis to determine the influence of the initial voluntary activity from the general linear model for the post-stroke population.

Multiple Comparisons					
Bonferroni					
Dependent Variable	(I) Voluntary Activity	(J) Voluntary Activity	Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude (mV)	Relax	Triceps	-0.389	0.3804	0.9298
		Biceps	-0.601	0.3551	0.2863
	Triceps	Relax	0.389	0.3804	0.9298
		Biceps	-0.211	0.4092	1.0000
	Biceps	Relax	0.601	0.3551	0.2863
		Triceps	0.211	0.4092	1.0000
Stretch reflex Latency (ms)	Relax	Triceps	-3.2666	4.8415	1.0000
		Biceps	-4.5183	4.5195	0.9631
	Triceps	Relax	3.2666	4.8415	1.0000
		Biceps	-1.2518	5.2080	1.0000
	Biceps	Relax	4.5183	4.5195	0.9631
		Triceps	1.2518	5.2080	1.0000
Stretch reflex Rise Time (ms)	Relax	Triceps	5.4650	3.5038	0.3706
		Biceps	3.4064	3.2708	0.9042
	Triceps	Relax	-5.4650	3.5038	0.3706
		Biceps	-2.0586	3.7691	1.0000
	Biceps	Relax	-3.4064	3.2708	0.9042
		Triceps	2.0586	3.7691	1.0000
Stretch reflex Duration (ms)	Relax	Triceps	26.5036	13.5330	0.1630
		Biceps	-0.6152	12.6331	1.0000
	Triceps	Relax	-26.5036	13.5330	0.1630
		Biceps	-27.1188	14.5575	0.2006
	Biceps	Relax	0.6152	12.6331	1.0000
		Triceps	27.1188	14.5575	0.2006

Table 30: Bonferroni post-hoc analysis to determine the influence of the head position from the general linear model for the post-stroke population The red cells represent the significantly effects in the model with 95% confidence.

Multiple Comparisons					
Bonferroni					
Dependent Variable			Mean Difference (I-J)	Std. Error	Sig.
Stretch reflex Amplitude	Centre	Left	0.037	0.6317	1.0000
		Right	0.490	0.5410	1.0000
	Left	Centre	-0.037	0.6317	1.0000
		Right	0.453	0.7986	1.0000
	Right	Centre	-0.490	0.5410	1.0000
		Left	-0.453	0.7986	1.0000
Stretch reflex Latency	Centre	Left	8.1166	8.0400	0.9491
		Right	6.2329	6.8862	1.0000
	Left	Centre	-8.1166	8.0400	0.9491
		Right	-1.8837	10.1650	1.0000
	Right	Centre	-6.2329	6.8862	1.0000
		Left	1.8837	10.1650	1.0000
Stretch reflex Rise Time	Centre	Left	1.9207	5.8186	1.0000
		Right	2.7640	4.9837	1.0000
	Left	Centre	-1.9207	5.8186	1.0000
		Right	0.8433	7.3565	1.0000
	Right	Centre	-2.7640	4.9837	1.0000
		Left	-0.8433	7.3565	1.0000
Stretch reflex Duration	Centre	Left	29.6003	22.4736	0.5769
		Right	19.8418	19.2486	0.9190
	Left	Centre	-29.6003	22.4736	0.5769
		Right	-9.7586	28.4133	1.0000
	Right	Centre	-19.8418	19.2486	0.9190
		Left	9.7586	28.4133	1.0000

6.3.6 Multiple initially applied torques

The variation in the protocol for the post-stroke subjects who were not able to flex or extend voluntarily consisted in introducing additional torque values. Thus, the torque values used in this protocol are:

- 1) 2.28 N.m
- 2) 3.42 N.m
- 3) 4.56 N.m
- 4) 5.70 N.m
- 5) 6.84 N.m

An analysis of variance was used to determine the differences in the stretch reflex parameter as well as in the range of movement and average angular velocity between the different initial torque values. Also the relationship between these variables and the initially applied torque values was determined using a regression analysis.

6.3.6.1 Kinematics

6.3.6.1.1 Range of movement

- Initially flexed

The analysis of variance showed that, when the whole post-stroke population is considered, there is no significant differences in the range of movement between the first and the second values of applied torque ($p=0.363$); between the second and third ($p=0.307$) and between the third and fourth ($p=0.154$) (Table 31). However a clear linear behaviour is observed for each individual. Figure 25 shows the regression plot and the correlation for each individual and the whole population.

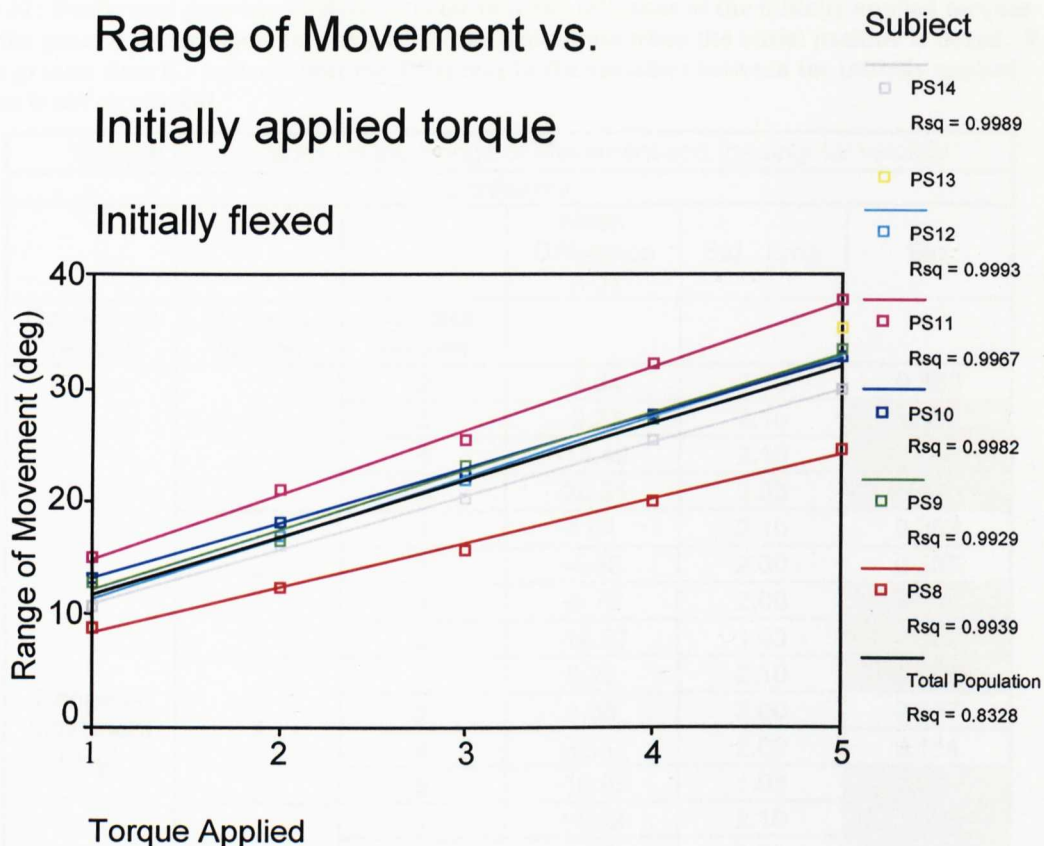


Figure 25: Regression plots of the range of movement and the initially applied torque values for each post-stroke subject and for the total population from the multiple torques protocol when the initial position is flexed.

Table 31: Bonferroni post-hoc analysis to determine the influence of the initially applied torques from the general linear model for the post-stroke population when the initial position is flexed. P values greater than 0.1 indicate that the difference in the variables between the initially applied torques is not significant.

Multiple Comparisons for the Range of Movement and the angular velocity					
Bonferroni					
Dependent Variable	(I) Force Applied	(J) Force Applied	Mean Difference (I-J)	Std. Error	Sig.
Range of Movement (deg)	1	2	-4.64	2.10	0.363
		3	-9.22	2.10	0.002
		4	-14.42	2.10	0.000
		5	-20.21	2.03	0.000
	2	1	4.64	2.10	0.363
		3	-4.58	2.00	0.307
		4	-9.78	2.00	0.000
		5	-15.57	1.93	0.000
	3	1	9.22	2.10	0.002
		2	4.58	2.00	0.307
		4	-5.20	2.00	0.154
		5	-10.98	1.93	0.000
	4	1	14.42	2.10	0.000
		2	9.78	2.00	0.000
		3	5.20	2.00	0.154
		5	-5.79	1.93	0.060
	5	1	20.21	2.03	0.000
		2	15.57	1.93	0.000
		3	10.98	1.93	0.000
		4	5.79	1.93	0.060
Angular Velocity (deg/s)	1	2	-44.66	23.09	0.645
		3	-87.46	23.09	0.009
		4	-129.79	23.09	0.000
		5	-167.61	22.33	0.000
	2	1	44.66	23.09	0.645
		3	-42.80	22.02	0.633
		4	-85.13	22.02	0.007
		5	-122.95	21.22	0.000
	3	1	87.46	23.09	0.009
		2	42.80	22.02	0.633
		4	-42.33	22.02	0.660
		5	-80.15	21.22	0.009
	4	1	129.79	23.09	0.000
		2	85.13	22.02	0.007
		3	42.33	22.02	0.660
		5	-37.82	21.22	0.868
	5	1	167.61	22.33	0.000
		2	122.95	21.22	0.000
		3	80.15	21.22	0.009
		4	37.82	21.22	0.868

- Initially extended

The analysis of variance in this condition showed a significant difference between all the applied torques (Table 32). Figure 26 shows the regression plot and the correlation for each individual and the whole population.

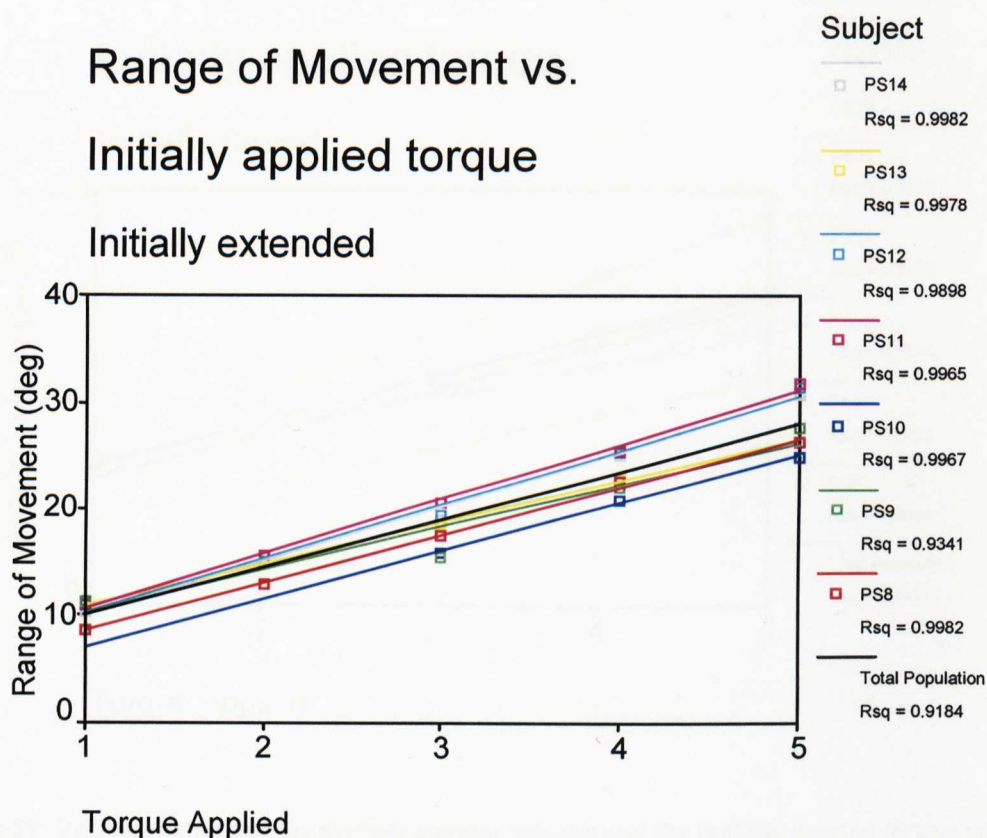


Figure 26: Regression plots of the range of movement and the initially applied torque values for each post-stroke subject and for the total population from the multiple torques protocol when the initial position is extended.

The overlap in the initially flexed position may be a result of the differences in the biomechanical characteristics of each volunteer.

6.3.6.1.2 Average angular velocity

- Initially flexed

The analysis of variance did not show significant differences in the average angular velocity between the first and the second values of applied torque ($p=0.645$); between the second and third ($p=0.633$); between the third and fourth ($p=0.660$) and between the

fourth and fifth ($p=0.868$) when the whole post-stroke population is considered (Table 31) Figure 27 shows the regression plot and the correlation for each individual and the whole population.

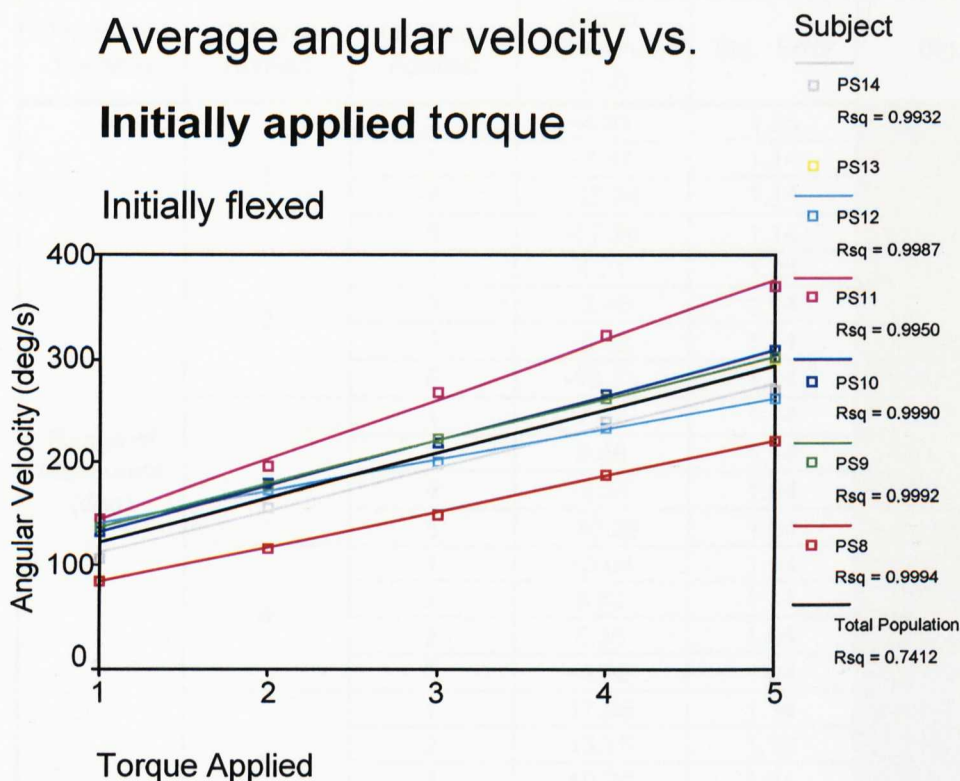


Figure 27: Regression plots of the average angular velocity and the initially applied torque values for each post-stroke subject and for the total population from the multiple torques protocol when the initial position is flexed.

- Initially extended

There was not significant differences in the average angular velocity between the second and third values of applied torque ($p=0.504$) (Table 32). Figure 28 shows the regression plot and the correlation for each individual and the whole population.

Angular velocities showed to be linear for every subject ($R^2>0.99$) however when the whole population is analyse it showed a lower correlation at the flexed position (slope = 36.749; intercept = 40.40; $R^2=0.7412$) than at the extended position (slope= 36.279; intercept= 25.584; $R^2=0.862$). These differences were assumed to be resultant of the anthropometric differences between the subjects.

Table 32: Bonferroni post-hoc analysis to determine the influence of the initially applied torques from the general linear model for the post-stroke population when the initial position is extended. P values greater than 0.1 indicate that the difference in the variables between the initially applied torques is not significant.

Multiple Comparisons for the Range of Movement and the angular velocity					
Bonferroni					
Dependent Variable	(I) Force Applied	(J) Force Applied	Mean Difference (I-J)	Std. Error	Sig.
Range of Movement (deg)	1	2	-4.21	1.23	0.021
		3	-7.67	1.14	0.000
		4	-13.04	1.14	0.000
		5	-17.96	1.14	0.000
	2	1	4.21	1.23	0.021
		3	-3.46	1.14	0.054
		4	-8.82	1.14	0.000
		5	-13.75	1.14	0.000
	3	1	7.67	1.14	0.000
		2	3.46	1.14	0.054
		4	-5.36	1.04	0.000
		5	-10.29	1.04	0.000
	4	1	13.04	1.14	0.000
		2	8.82	1.14	0.000
		3	5.36	1.04	0.000
		5	-4.92	1.04	0.001
	5	1	17.96	1.14	0.000
		2	13.75	1.14	0.000
		3	10.29	1.04	0.000
		4	4.92	1.04	0.001
Angular Velocity (deg/s)	1	2	-46.58	15.46	0.057
		3	-75.95	14.31	0.000
		4	-124.25	14.31	0.000
		5	-167.10	14.31	0.000
	2	1	46.58	15.46	0.057
		3	-29.36	14.31	0.504
		4	-77.67	14.31	0.000
		5	-120.51	14.31	0.000
	3	1	75.95	14.31	0.000
		2	29.36	14.31	0.504
		4	-48.30	13.07	0.010
		5	-91.15	13.07	0.000
	4	1	124.25	14.31	0.000
		2	77.67	14.31	0.000
		3	48.30	13.07	0.010
		5	-42.85	13.07	0.030
	5	1	167.10	14.31	0.000
		2	120.51	14.31	0.000
		3	91.15	13.07	0.000
		4	42.85	13.07	0.030

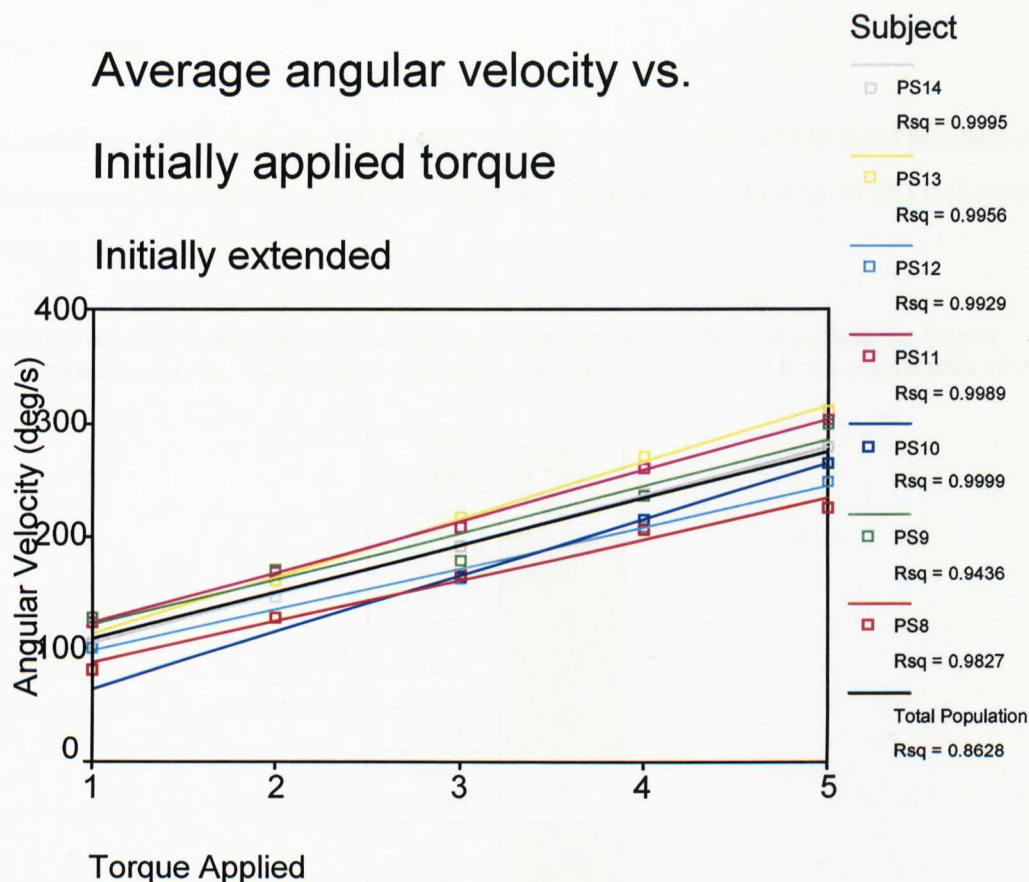


Figure 28: Regression plots of the average angular velocity and the initially applied torque values for each post-stroke subject and for the total population from the multiple torques protocol when the initial position is extended.

6.3.6.2 Stretch reflex parameters

An analysis of variance was used to determine the differences of the stretch reflex parameters between the five initially applied torque values at the extended and flexed initial positions. No significant differences were found in any of the parameters between any of the initially applied torques at either the extended or the flexed initial positions ($p>0.1$). These results support the previous post-stroke results from the original protocol suggesting lack of modulation of the stretch reflex parameters.

6.3.6.3 General linear model

In order to determine whether the initial conditions or their interactions (initially applied torque, initial position, and angular velocity and head position) had any effect on the

stretch reflex parameters throughout the whole protocol, a general linear model analysis was implemented.

This model determined that all the conditions with the exception of the head position and the interaction of the initially applied torque with the initial position ($p>0.05$) influence the model (Table 33).

Table 33: post-stroke population multivariate test determining the effects of each of the factors towards the GLM analysis. The red cells represent the significantly effects in the model with 95% confidence.

Pillai's Trace Multivariate Test	
Effect	Sig.
Intercept	0
Angular velocity	0
Initially applied torque	0.002
Head position	0.933
Initial position	0.001
Initially applied torque * Initial position	0.846

The individual effects of each condition and their interaction on each parameter are summarised below (Table 34).

6.3.6.3.1 Stretch reflex amplitude

It was observed that none of the conditions or their interactions affected the stretch reflex amplitude (Figure 29a). Furthermore, no specific pattern is found when plotting the marginal means obtained from the GLM analysis (Figure 30a).

6.3.6.3.2 Stretch reflex latency

The stretch reflex latency was influenced by the angular velocity ($p=0$), and the initially applied torque ($p=0$) (Figure 29b). This parameter shows a clear tendency to decrease when the marginal means obtained from the GLM analysis are plotted suggesting increased excitability at higher torques, therefore at higher velocities of muscle stretch (Figure 30b).

6.3.6.3.3 Stretch reflex rise time

The stretch reflex rise time was only affected by the initial angular position ($p= 0.009$) (Figure 29c). Opposite patterns are found in this parameter for the two initial positions when plotting the marginal means obtained from the GLM analysis (Figure 30c).

6.3.6.3.4 Stretch reflex duration

The stretch reflex duration was influenced by the angular velocity, the applied torque and the initial position ($p=0$) (Figure 29d). This parameter shows a tendency to increase when the marginal means obtained from the GLM analysis are plotted, suggesting increased tonic stretch reflex components at increased torques and velocities. It is also evident that the durations are longer when the initial position is initially flexed, suggesting that the initial muscle length and/ or the static tension affect (Golgi tendon organ contribution) the tonic stretch reflex excitability (Figure 30d).

Table 34: Statistical analysis from the General Linear Model determining the interaction between each condition (initial position, torque applied and head position for the post-stroke population in the multiple applied torques protocol. The red cells represent the significantly effects in the model with 95% confidence.

Tests of Between-Subjects Effects			
Source	Dependent Variable	F	Sig.
Corrected Model	Raw Biceps Amplitude (a)	0.588	0.843
	Smooth Biceps Latency (a)	4.787	0
	Smooth Biceps Rise Time (a)	1.306	0.239
	Smooth Biceps Duration (a)	2.482	0.01
Intercept	Raw Biceps Amplitude (a)	6.988	0.01
	Smooth Biceps Latency (a)	23.199	0
	Smooth Biceps Rise Time (a)	2.391	0.127
	Smooth Biceps Duration (a)	29.705	0
Angular velocity	Raw Biceps Amplitude (a)	0.104	0.748
	Smooth Biceps Latency (a)	46.979	0
	Smooth Biceps Rise Time (a)	1.097	0.299
	Smooth Biceps Duration (a)	6.344	0.014
Initially applied torque	Raw Biceps Amplitude (a)	0.336	0.853
	Smooth Biceps Latency (a)	11.297	0
	Smooth Biceps Rise Time (a)	0.369	0.83
	Smooth Biceps Duration (a)	2.692	0.039
Head position	Raw Biceps Amplitude (a)	0.223	0.801
	Smooth Biceps Latency (a)	0.042	0.959
	Smooth Biceps Rise Time (a)	0.009	0.991
	Smooth Biceps Duration (a)	0.338	0.714
Initial position	Raw Biceps Amplitude (a)	0.036	0.851
	Smooth Biceps Latency (a)	0.149	0.701
	Smooth Biceps Rise Time (a)	7.299	0.009
	Smooth Biceps Duration (a)	18.347	0
Initially applied torque * Initial position	Raw Biceps Amplitude (a)	1.353	0.261
	Smooth Biceps Latency (a)	0.34	0.85
	Smooth Biceps Rise Time (a)	0.531	0.713
	Smooth Biceps Duration (a)	0.589	0.672

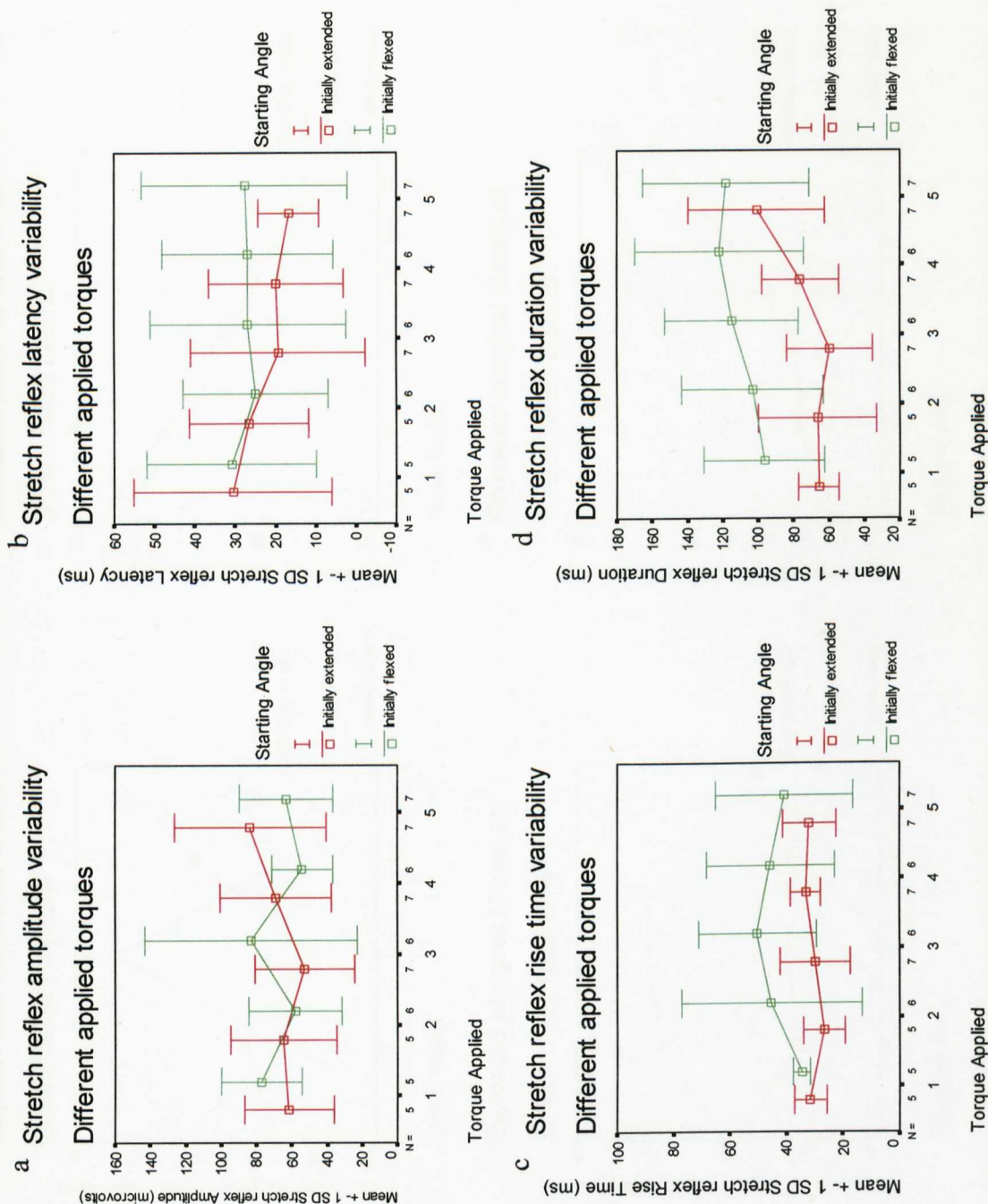


Figure 29: mean and standard deviation plots of the stretch reflex parameters, a) amplitude, b) latency, c) rise time and d) duration, comparing their differences between five applied torque values.

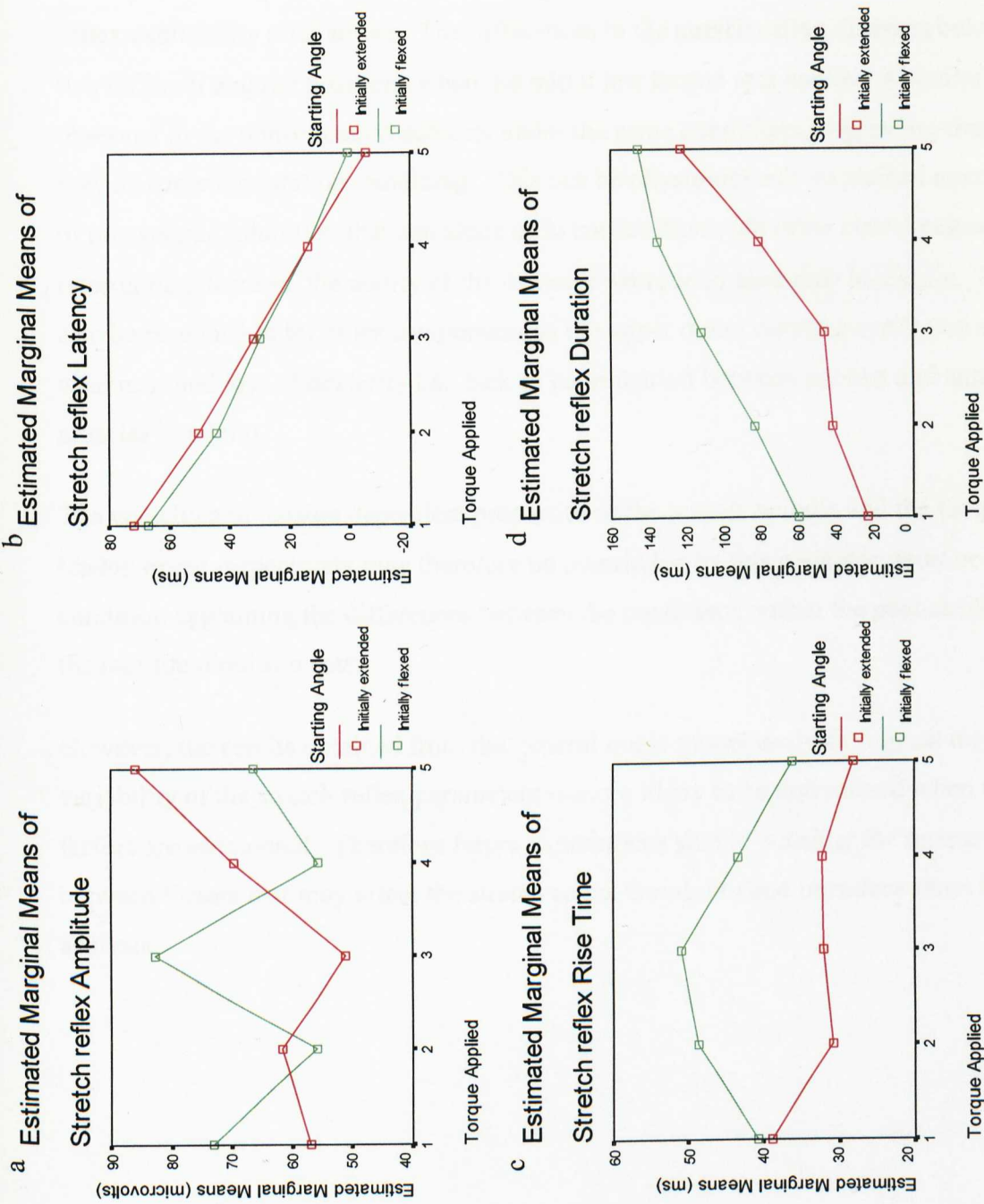


Figure 30: Estimated marginal means of the stretch reflex parameters, a) amplitude b) latency c) rise time and d) duration, obtained from the general linear model analysis

6.3.7 Discussion

The lack of significant differences in the majority of the parameters of the stretch reflex in the post-stroke population suggests that there is a lack of modulation of the stretch reflex excitability after stroke. The differences in the stretch reflex duration between the two different angular positions when the initial low torque was applied is similar to that observed in the non-impaired subjects under the same conditions, suggesting that there may be some modulation remaining. This can be physiologically explained as reduction of presynaptic inhibition that can alone or in conjunction with other neural pathways interruption decrease the ability of the α -motor neurone to modulate its output. This can also be responsible for other components of the upper motor neurone syndrome such as weakness and loss of dexterity i.e. lack of coordination between agonist and antagonist muscles in a joint.

The velocity and tension dependent properties of the muscle spindle and the Golgi tendon organ respectively may therefore be overridden by this α -motor neurone condition explaining the differences between the conditions within the post-stroke and the non-impaired subjects.

However, the results obtained from the general linear model analysis suggest that the variability of the stretch reflex parameters is more likely to be determined when more factors are considered. Therefore future experiments should consider the interaction between factors that may affect the stretch reflex variability and introduce them in the analysis.

7 Conclusions

7.1 Introduction

Spasticity has been defined as a velocity dependent increase in the stretch reflex excitability as a result of a lesion to the CNS. This type of lesion generally results in a group of symptoms that are clinically referred to as the Upper Motor Neurone Syndrome of which spasticity is only one component along with weakness and loss of dexterity. Nevertheless, the definition of spasticity is an adapted version of the description of the functionality of the muscle spindle, where the stretch reflex is velocity dependent itself (chapter 2).

There are many references in the literature about how it is assumed that spasticity increases the resistance to passive movement when a muscle is being stretched. This increased resistance has long been attributed to the hyperexcitability of the stretch reflex response but few make reference to the biomechanical changes that occur to the muscle and surrounding soft-tissue often seen accompanying neural lesion and potentially affect such resistance to passive movement.

Many authors have tried to analyse the velocity dependency of spasticity however they neglect the contribution of the biomechanical changes by stretching the joints at very slow velocities to establish a "passive" resistance and then increase gradually the velocities, in some cases verifying the presence of a stretch reflex, some others not, and attribute the velocity dependent increased resistance to the stretch reflex properties.

However it has been reported that the resistance due to the biomechanical properties of the muscles and surrounding tissue are also velocity dependent (Singer et al, 2003), thus increasing with the stretch velocity altering the pre-established base line, making more difficult to dissociate the neural from the non-neural components by simply assessing or even measuring the resistance to passive motion. Nevertheless, this has been only reported experimentally regarding the ankle joint and it may be different from other joints such as knee and elbow as the arrangement of muscles and tendons surrounding the joints are different.

The purpose of this work was to study the stretch reflex responses independently of the resistance to passive movement from the elbow joint flexors in a group of non-impaired volunteers and a group of post-stroke volunteers. Such stretch reflex responses were obtained from electromyographic recordings in response to standardised perturbations using known initial applied torques and a combination of different initial conditions including initial elbow angle, initial muscle activity and the head position during the tests.

Comparisons within and between groups under the different conditions were statistically analyzed using ANOVA and a General Linear Model analysis. These analyses showed significant differences within the non-impaired populations that depend on the different initial conditions and their interactions. Such differences were not found in the post-stroke population.

Although the differences found in the non-impaired group are subject to interpretation, it is the lack of modulation in the post stroke population that needs to be addressed to understand better the concept of spasticity and to determine the therapeutic approach towards it.

7.2 Measurement of spasticity (state of the art)

The lack of a proper concept and therefore definition of spasticity has affected the diagnosis for this phenomenon. It was found that the most common assumption is that the stretch reflex hyperexcitability increases the resistance of a joint to be passively stretched throughout its range of motion. Nevertheless some of the papers reviewed have mentioned that, in the presence of the upper motor neurone syndrome, spasticity may or may not be clinically evident and yet hyperreflexia is present (Sherman, Koshland, & Laguna 2000) and that patients with clinically diagnosed spasticity do not have hyperreflexia (O'Dwyer, Ada, & Neilson 1996). However these are isolated cases and the methodology used may be flawed in principle and lack of concept. Therefore such studies should be interpreted with caution and regard them as exceptional case studies.

Current available techniques for the measurement of spasticity were categorised in three main groups:

- Clinical
- Neurophysiological
- Biomechanical

Biomechanical techniques alone are mainly based on the clinical ones adding instrumentation to control the perturbation and register the resistance to passive movement by means of torque and angular displacement sensors. Unfortunately this adds complexity to the techniques since the instrumentation required may be time consuming and difficult to implement, particularly in patients with higher levels of post-stroke sequels such as contractures, associated reactions and abnormal posture. Furthermore, complex instrumentation where motors, computers and/or motion analysis cameras are required, are difficult to store and maintain and also require a technical level of expertise which may not always be available in stroke units.

Neurophysiological techniques, on the other hand, provide with a better insight of the neurophysiological pathways although they bypass physiological sensors (i.e. muscle spindle and Golgi tendon organ) and may affect others (i.e. cutaneous and nociceptive). They are sometimes invasive and uncomfortable for the patient and also require technical expertise.

As it can be seen, each one of these categories has advantages and disadvantages that are inherent to them and cannot be avoided. However, these techniques can be combined and complement each other.

For instance, if biomechanical techniques are used alone they, as well as the clinical ones, are limited in dissociating the neural from the non-neural mechanisms. However, EMG activity resulting from the muscle stretch can be registered during the perturbation increasing the capabilities of the technique to determine neurological activity. Nevertheless, sometimes the instrumentation used may be still be limited in differentiating the resistive torque due to biomechanical changes from those of neural origin. Some authors have developed models

that include the estimation of the effects of the recorded EMG measurements towards the resistive torque. Nevertheless, these models are obtained from the filtered EMG data and may not always be proportional to the registered torque (Schmit et al. 1999a; Schmit et al. 1999b; Schmit & Rymer 2001; Schmit, Dewald, & Rymer 2000).

7.3 Instrumentation and Methodology

Spasticity can affect different joints depending on the site and the severity of the lesion. The elbow joint was chosen for this work because it is experimentally easier to access and more straight forward to place electrodes and instrumentation on it than other joints such as wrists, fingers and lower limb joints.

The literature review showed that existing devices and protocols were not likely to be used in a wide range of patients due to positioning and patient-device interaction. Therefore, the custom made device was designed to be able to fit most of the volunteers (impaired and non-impaired).

The applied torque (chapter 5) acted as a step input and the behaviour of the device was considered to be a second order system. The first oscillation of the angular displacement was of particular interest for this work since it elicits the stretch reflex response and EMG activity due to further oscillations cannot always be considered to be of reflex origin. A second order model was used to describe the behaviour of the device, which proved to be accurate for the first oscillation for every initially applied torque value.

The measurement protocol consisted of a battery of tests where the following conditions were thought to affect the stretch reflex excitability:

- Initially applied torque
- Voluntary activity
- Initial angular position,
- Head position

These conditions influence the behaviour of the stretch reflex. With this in mind, they were analysed independently within and between populations using ANOVA and their interaction effects were analysed using a General Linear Model analysis.

Each test session lasted for at least fifty minutes considering the time to set the patient ready for the test. Unfortunately, the stretch reflex activity has shown to be affected by several repetitions of stretches changing the results from trial to trial (Schmit, Dewald, & Rymer 2000). This drawback could not be controlled and was tried to be minimised by applying the perturbations randomly. Therefore future protocols should involve less conditions and more repeated measurements to observe the variability within each combination of conditions.

The following sections of this chapter will conclude on the results obtained and hypothesise a physiological explanation for the changes in the behaviour of the stretch reflex parameters.

7.4 Differences within the non-impaired and post-stroke populations

7.4.1 Non-impaired population

7.4.1.1 Relaxed conditions

The stretch reflex parameters obtained from the non-impaired subjects yielded somewhat unexpected results. The amplitude was found to be dependent on the initial angular position. Higher stretch reflex amplitudes were found when the elbow joint was positioned in the extended position, suggesting that the muscle spindle excitability may be increased. This observation is supported by the literature (chapter 2). The latency results also support this observation as it was found to be shorter at the extended position when the low torque was applied. However the latency values trend was found to be opposite when the high torque was applied. These latter results may be explained by means of the Golgi tendon organ autogenic inhibition (Ib) in addition to that of the muscle spindle and/or the behaviour of the intrafusal muscle fibres at different lengths.

The background hypothesis for the contribution of the Golgi tendon organ towards these measurements can be found in chapter 2. Two types of Golgi tendon have been identified.

One type responds only to changes in tension (dynamic) and the other type to absolute tension and the changes in tension (dynamic and static). Increased muscle length, even at rest may be responsible for some of the Golgi tendon organs to fire inhibiting the stretch reflex activity. Such inhibition would be reflected in prolonged latencies particularly when higher torques are applied. Therefore it can be suggested that the stretch reflex latencies are affected by a combination of muscle spindle, muscle fibres length tension properties and Golgi tendon organ activity that facilitates and inhibits the stretch reflex activity respectively. Such activity is determined by the initial muscle length and the applied torque used to stretch the muscle.

It is also possible that the latency and the amplitude of the stretch reflex may be interdependent. Lin and Rymer experiments (1999) have shown that the stretch reflex may not regulate length but stiffness of the muscle during stretch. Based on this premise, if for any reason the latency of the stretch reflex is long then a higher magnitude of the muscle activity would be expected to increase the stiffness of a muscle closer to the yielding point whereas if there is a shorter latency the magnitude would not be required to be high to maintain a particular muscle stiffness.

The stretch reflex rise time did not show any significant variability at the different initial position or the initially applied torque. This suggests that the synaptic behaviour is not influenced by these conditions. This was expected as the synaptic activity would be influenced by feed-forward mechanisms which would be unaffected in relaxed conditions.

The stretch reflex duration was considered to be an indicator for the stretch reflex tonic response. Longer durations imply prolonged reflex activity. Increased reflex durations were observed when the higher torque was applied (i.e. faster velocities and wider range of movement). This suggests that the tonic component of the stretch reflex is elicited either by increased stretch velocity or increased range of movement. Also shorter durations were found at the initially extended position, suggesting increased Golgi tendon organ contribution and/or dependency on the muscle stiffness associated with the initial muscle length.

7.4.1.2 Voluntary activity conditions

The amplitude of the reflex showed no differences between the initial voluntary activity conditions (relaxed, flexing and extending) implying that the stretch reflex amplitude remained the same relative to the muscle background activity.

The differences found in the latency, rise time and duration values of the stretch reflex did not always show specific pattern between the previous voluntary activities for all the initial conditions.

Differences in the stretch reflex latencies were found only at the extended position when the lower torque was applied. In this case the latencies were shorter when the subjects were relaxed when compared to voluntarily flexing and extending. The possible explanations for these findings are the reciprocal inhibition when the subjects were attempting to extend the elbow and the autogenic inhibition mechanisms when the subjects were attempting to flex the elbow and more importantly the possibility of a slight displacement towards flexion during the initial voluntary flexion. Reciprocal inhibition from the triceps activity may delay the stretch reflex response. Golgi tendon organ activity due to increased biceps activity and the initially applied torque may increase the tension at the tendon causing inhibition of the biceps activity. However there cannot be a definite conclusion from these observations since no further differences were found at the flexed position using the lower torque or at any of the initial positions at the higher torque. Further investigation is required for the understanding the complexity of such findings.

The differences in the stretch reflex rise time values were consistent at the extended position for both applied torques when comparing the initially relaxed with the initially extending conditions. Shorter values when the subjects were initially relaxed can be explained also by reciprocal inhibition mechanisms. These mechanisms would be triggered when the perturbation is applied in the opposite direction of the voluntary activity.

The differences observed in the rise time values between the voluntarily flexing and voluntarily extending conditions (i.e. shorter rise times when voluntarily flexing) when the initial position is extended and the high torque is applied can be explained by the fact that

the elbow flexors are already active facilitating the stretch reflex activation thus reaching the maximum stretch reflex amplitude faster.

This explanation can also be applied to the results observed at the high torque and flexed position when the stretch reflex rise time values are lower when compared with the ones when the subjects are flexing voluntarily.

The difference found in the stretch reflex duration values when comparing the different initial voluntary activity are consistent with the mechanisms previously described. A general pattern of decreased duration was found when the subjects are voluntarily flexing when comparing to voluntarily extending (high torque at both initial positions) and/or relaxed (high torque and flexed position) prior the stretch. These differences may be attributed to the autogenic inhibition mechanisms of the Golgi tendon organ inhibiting the flexor activity due to the increased tension from applying a torque to the flexors when they are active.

The differences in the stretch reflex parameters due to the position of the head were aimed to assess the influence of the asymmetric tonic neck reflex. It was found that this variable does not influence the stretch reflex amplitude or latency but it affects the rise time and duration.

Lower rise time values were observed in the trials where the subjects were facing towards the same side of the limb being tested when compared to the trials where the subjects were facing the opposite side. The stretch reflexes lasted longer when the subjects were looking at the opposite side of the limb being tested when compared with the trials when they were facing the same side. These results suggest that the tonic component of the stretch reflex is increased when facing the opposite side. These can be explained by assuming increased facilitation or reduced presynaptic inhibition when the head is turning to the opposite side of the arm test.

Additional information regarding the asymmetric neck tonic reflexes can be seen in a study by Le Pellec and Maton (1996) where the authors observed the reflexes in the extensor muscles of the elbow joint when turning the head left, right or straight. In their results the elbow extensor muscles of the opposite side presented higher tonic stretch reflexes when the

head was facing the side of the limb being tested (Le Pellec & Maton 1996). Results from this study complement the results observed in this work which can be explained by reciprocal inhibition mechanisms. While tonic stretch reflexes from elbow extensors are increased when looking at the ipsilateral side of the arm being tested, stretch reflexes from the flexors of the contralateral arm are also increased.

7.4.2 Post-stroke subjects

The results presented in this work suggest that there is a lack of modulation in the stretch reflex excitability after the occurrence of a stroke as no statistical differences were found between the different conditions within the post-stroke population. These results are supported by a study by Leonard and colleagues (1998) and Morita and colleagues (2001) where they studied soleus H-reflexes preceding and during voluntary tibialis anterior muscle contraction of standing subjects and during balance platform induced postural perturbations and H-reflex modulation during voluntary movement in healthy and spastic groups respectively. In both studies it was found that regardless of the level of tibialis anterior activation, soleus H-reflexes of subjects with UMN lesions did not demonstrate inhibition in any of the cases unlike the non-disabled volunteers (Leonard et al. 1998) and H-reflexes varied more in the healthy subjects group than in the spastic group suggesting an abnormal modulation of stretch reflexes and attributed this to an abnormal regulation of reciprocal inhibition and presynaptic inhibition in patients with spasticity and explained that these abnormalities may be responsible for the tendency to elicitation of unwanted stretch reflex activity seen in spasticity (Morita et al. 2001).

These results are congruent with those of Kasai and Komiyama (1996) where it was observed that voluntary arm movements caused H-reflex depression in the soleus muscle of non-impaired subjects (Kasai & Komiyama 1996).

The stretch reflex duration was the only parameter that showed differences between the initial positions when the lower torque was applied. Longer durations were observed when the elbow joint is initially flexed when compared with the ones when the elbow joint is initially extended. These differences can be attributed to either the Golgi tendon activity inhibiting the reflex due to increased tension at the extended position or to the difference in

intrafusal (muscle spindle) and extrafusal muscle fibres stiffness between the initial positions. Assuming lower muscle stiffness at the flexed position, the stretch reflex would require being active for a longer period of time to increase the stiffness throughout the range of motion.

7.4.3 Differences between populations

The lower stretch reflex amplitudes found in the post-stroke subjects when compared with the non-impaired volunteers were unexpected. These results suggest two possible explanations. First, the amplitudes are not representative of the stretch reflex excitability and the effects of the biomechanical changes may also affect this value. Second, the ongoing therapy and botulinum toxin treatment may have been responsible for these findings even when tests were carried out within the theoretical window where the effects of botulinum toxin were not effective (chapter 5).

Shorter latencies found in the post-stroke population in most of the conditions imply higher excitability of the stretch reflex suggesting reduced presynaptic inhibition and/or reduced stretch reflex threshold.

Shorter rise time values found in the post-stroke subjects when compared with the non-impaired population suggest higher excitability of the motor-neurone and synaptic interneurons due to reduced presynaptic inhibition.

Longer stretch reflex durations in the post-stroke subject suggest increased tonic stretch reflex activity. These findings are consistent with the definition of spasticity.

These results suggest that the latency, rise time and duration are the most useful to differentiate between non-impaired subjects and people with diagnosed post-stroke spasticity. EMG amplitudes are difficult to compare between subjects as they may be dependent on skin conductance (Leao & Burne 2004) and physiological cross-sectional area (Klein, Rice, & Marsh 2001).

7.5 General linear model

A multivariate general linear model analysis was used to study the effects of the interactions between the different conditions on the stretch reflex parameters in the post-stroke and non-impaired populations.

7.5.1 Non-impaired subjects

In the analysis of non-impaired subjects it was found that the stretch reflex parameters were influenced by the initial position, voluntary activity, applied torque and head position and their interactions. The interactions between the applied torque and the starting angle and initial activity had no influence on any of the parameters.

The amplitude was strongly influenced by the initial position and the initial activity. The influence of the initial position in the amplitude suggests the contribution of the Golgi tendon organ activity and/or the initial tension of the intrafusal muscle fibres affecting the sensitivity of the muscle spindle. The influence of the initial activity suggests changes in the threshold of the α -motor neuron and/or reciprocal and autogenic inhibition for extending and flexing respectively.

The latencies were influenced by the applied torque, initial angular position and voluntary activity and their interaction. This suggests a combination of effects in the muscle spindle sensitivity, Golgi tendon organ activity and reciprocal and autogenic inhibition.

The rise time and duration were influenced by the initial voluntary activity and its interaction with the starting angular position and with the initial applied torque. This variability can be explained by the same mechanisms affecting the latencies. Changes due to head position can be explained by the influence of the asymmetric tonic neck reflexes influencing the excitability of the elbow flexors when the head is turned to the opposite side of the arm being tested.

7.5.2 Post-stroke subjects

There was no particular factor or influencing the stretch reflex parameters in the post-stroke group. However, the stretch reflex latency was influenced by the initial angular position. This can be explained also by the changes in tension of the Golgi tendon organ and/or the sensitivity of the muscle spindle dependent on the initial length of the muscle fibres.

The lack of variability in most of the stretch reflex parameters strongly suggests a lack of modulation. These findings are consistent with the results observed when each of the conditions was analysed separately.

The difference in the results from the General Linear Model with those from the independent analysis of each factor imply that the experimental design for the measurement of physiological data requires to consider as many factors as possible to obtain a better estimation of the variability of the data. However the analysis of each factor under a particular set of conditions is still required to explain the behaviour of the parameters for that particular set of conditions and provide the background for further analyses.

7.6 Multiple applied torques

This later protocol was applied only to post-stroke volunteers that were not able to voluntarily flex or extend the elbow joint. The ANOVA tests for each independent condition showed that the stretch reflex parameters did not depend on the initially applied torque supporting the hypothesis of lack of modulation of the stretch reflex excitability. However when the General Linear Model analysis was used to observe the effects of the conditions it was found that most of the stretch reflex parameters were influenced by all the conditions except the head position.

None of the initial conditions and their interactions affect the stretch reflex amplitude suggesting that the gain of the stretch reflex is not dependent on the applied torque, initial position and/or the velocity of the stretch.

The stretch reflex latencies were very much influenced by the angular velocity and the initially applied torque. It was found that the latencies were reduced when the angular

velocity increased supporting the view that the stretch reflex threshold decreases when the velocity of the stretch increases. However the neurophysiological background suggests that this threshold may not be solely dependent on the changes of length (position) but also result from the muscle spindle response to the increment in speed of stretch (velocity).

The stretch reflex rise time was dependent on the starting angular position. Generally shorter rise times observed when the elbow joint is initially extended suggest increased firing of the muscle spindle exciting the α -motor neurone pool.

Stretch reflex durations were affected by the angular velocity, the applied torque and the initial position. Shorter durations when the elbow joint is initially extended suggest Golgi tendon organ activity. Increased durations for both initial positions when the applied torque and the velocity of the stretch increased suggest increased tonic stretch reflexes which are consistent with the definition of spasticity.

7.7 Final thought

The stretch reflex parameters suggested in this work as measure of the excitability of the stretch reflex have shown differences between post-stroke and non-impaired volunteers. The stretch reflex excitability was more variable and more dependent on the initial conditions in the non-impaired population but it was observed more often in the post-stroke population.

The parameters found to be more representative of the stretch reflex excitability were latency and the duration. These parameters showed more dependency on the velocity of the stretch and/or the applied torque, especially when the second protocol was applied in the post-stroke population. Also when comparing the latencies and durations between the populations, shorter latencies and longer durations were generally found in the post-stroke group, supporting the theory of reduced thresholds and increased tonic stretch reflexes respectively.

The variability in the stretch reflex parameters however cannot be explained by the velocity of the stretch and/or the muscle spindle sensitivity alone. Intrinsic muscle fibres properties as well as other physiological sensors activity such as the Golgi tendon organ cannot be

neglected and should be considered in further experimental designs. The results from this work suggest that the excitability of the stretch reflex depends on many factors that cannot be controlled but can be estimated and therefore analysed.

However, from a pragmatic point of view and based on the findings of this work it is possible to say that the measurement of spasticity based on the quantification of the stretch reflex excitability alone is far from being definitive. Therefore, it is necessary to promote interdisciplinary research groups in order to determine the clinical importance of the stretch reflex quantification in combination with other scientifically accepted techniques in order to develop protocols aimed to increase the quality of life of people who have had a neurological lesion.

8 Recommendations for future work

8.1 Introduction

Quantifying and making sense of physiological data is always difficult due to the intrinsic non-linearities and differences between and within subjects. As was seen in chapter 2, the modulation of stretch reflex excitability depends on feedback and feed-forward mechanisms which are regulated by many other structures in the reflex pathways (interneurones, muscle spindles, Golgi tendon organs, Renshaw cells, etc.) whose response and behaviour are also variable in themselves and depend on many factors (i.e. physiological, biomechanical, neurological, diurnal, psychological, etc.). All these factors need to be explored to estimate their effect upon the excitability of the stretch reflex and their changes due to neurological damage. Unfortunately, experimental protocols are unlikely to be able to consider all the influences and interactions between different factors at once as it will be time consuming (also affecting the outcome measurements). Nevertheless, even when some variables cannot be controlled, they can be measured and introduced to the statistical analysis as factors.

8.2 Instrumentation

The technique developed proved to be reliable to elicit, record and analyse the stretch reflex response from the elbow flexors due to a fast angular perturbation. However its use was limited to the left elbow joint and for people with seating balance and able to follow instructions. Also the range of movement and the velocity of the stretch were dependent on the torque applied making it difficult to determine whether some parameters were dependent on velocity or position. Furthermore, when the possibility of its clinical use was discussed with physiotherapists and clinicians, their comments were that it was too big for storage and that it required specialised knowledge to be operated. Therefore, modifications to the existing device or even recommendations for future designs should include:

- Linkages or additional mechanisms to test the stretch reflex on both sides and on different joints.

- Modifications to maintain a constant displacement regardless of the applied torque as the rise time and duration may be dependent on either range of movement or speed of movement.
- The ability to adapt the device to be used next to a bed as some patients do not have seating stability.
- With the evolution of technology it may be expected at some point to replace the springs with another type of actuators.
- Include a report generator for single or multiple tests for easy interpretation of the results in a clinical environment.

8.3 Measurement protocols

8.3.1 Factors and variables affecting the stretch reflex excitability- Strategies towards neuromuscular modelling

The protocols described and used in this work were necessary to estimate the variability of the stretch reflex response to some physiological factors known to affect it. However there are other factors that may also have effect on the excitability that are not possible to control but that can be measured. Taking into account these factors and variables it may be possible to define a model that can describe the parameters proposed in this work, thus the stretch reflex excitability in a more general picture. Furthermore it may be possible to determine the resistive torque associated with the onset and duration of the muscle activation due to the stretch reflex response. This resistive torque may be expressed as a function of the changes in the biomechanical properties of the muscle due to the electrical activity associated with the muscle activation. The torque produced may also be found to be a function of the physiological cross-sectional area of the muscle(s) and the joint angle (muscle moment arms). Other factors that should be taken into account are the estimation of the effects of adjacent joints on the excitability of the reflex, particularly in bi-articular muscles as the relative length of the muscle is dependent on these.

If the models for these studies are to be individualised for each volunteer, additional information would be necessary for the determination of muscle lengths at different joint angles (and subsequent calculation of their moment arms) which may be possible to gather using ultrasound or MRI imaging.

8.3.2 Stretch reflex analysis

This work has focused on the parameterisation of the stretch reflex signal as a result of the stretch reflex to a sudden elbow extension perturbation. The analysis technique used involved the detection of maximum amplitudes and ranges of EMG activation with thresholds determined by previous voluntary activity. This technique was shown to provide a good approximation of visually detectable thresholds and amplitudes.

However recent techniques for signal processing such as wavelets are recommended for future analysis for two main reasons:

- They provide a more sensitive detection of changes within a signal and time-frequency analysis for improved analysis of signal variation.
- Signals can be reconstructed after analysis with minimum loss of the original signal improving modelling capabilities.

8.3.3 Characteristics of the volunteers

This work was mainly focused on observing the differences in the stretch reflex parameters between non-impaired volunteers and post-stroke patients with diagnosed spasticity with more than six months after the onset of the stroke. However, it is acknowledged that most of the neurophysiological and biomechanical changes occur gradually after the onset of the lesion. Therefore it is also important to design experimental protocols to include the evolution of the stretch reflex excitability and the changes in the biomechanical properties from a few days after the onset of the lesion with repeated measurements in regular intervals.

The technique developed in this work can also be used in determining the clinical effectiveness of treatments to reduce spasticity (stretch reflex excitability according to the definition) such as Botulinum Toxin injections, electrical stimulation and physical therapy among others.

8.3.4 Interdisciplinary projects

The main objectives of this work were to analyse and differentiate the stretch reflex excitability between people with no neural impairment and people who have suffered a stroke and have consequently developed spasticity.

For this technique to be useful from the patients' perspective, it is necessary to develop interdisciplinary projects with a holistic approach to determine clinical effectiveness of treatments to reduce spasticity and/or improve quality of life. These projects should prove to be effective for the patients and their relatives, both at the individual and scientific levels, then these should be followed with the proper validation and with help of technological advances.

References

- Abbruzzese, G., Abbruzzese, M., Ratto, S., & Favale, E. 1982, "Contribution of tonic vibration reflex to the evaluation and diagnosis of cerebellar disorders", *J.Neurol.Neurosurg.Psychiatry*, vol. 45, no. 6, pp. 526-530.
- Ada, L., Vattanasilp, W., O'Dwyer, N. J., & Crosbie, J. 1998, "Does spasticity contribute to walking dysfunction after stroke?", *J.Neurol.Neurosurg.Psychiatry*, vol. 64, no. 5, pp. 628-635.
- Ageranioti, S. A. & Hayes, K. C. 1990, "Effects of vibration on hypertonia and hyperreflexia in the wrist joint of patients with spastic hemiparesis", *Physiotherapy Canada*, vol. 42, no. 1, pp. 24-33.
- Ashworth, N. L., Satkunam, L. E., & Deforge, D. 2004, "Treatment for spasticity in amyotrophic lateral sclerosis/motor neuron disease", *Cochrane.Database.Syst.Rev.* no. 1, p. CD004156.
- Aymard, C., Katz, R., Lafitte, C., Lo, E., Penicaud, A., Pradat-Diehl, P., & Raoul, S. 2000, "Presynaptic inhibition and homosynaptic depression: a comparison between lower and upper limbs in normal human subjects and patients with hemiplegia", *Brain*, vol. 123 (Pt 8), pp. 1688-1702.
- Bajd T & Vodovnik L 1984, "Pendulum testing of spasticity", *Journal of Biomedical Engineering*, vol. 6, pp. 9-16.
- Barnes, M. P. 2001, "Medical management of spasticity in stroke", *Age Ageing*, vol. 30 Suppl 1, pp. 13-16.
- Basmajian, J. V. & DeLuca, C. J. 1985, *Muscles Alive: Their functions revealed by electromyography*, 5th edn, Baltimore.
- Benson, L. J., Harris, G. F., Light, T. R., & Acharaya, K. R. 1990, "Biomechanical assessment of the wrist in spastic cerebral palsy with isometric torque recording (abstract)", *Developmental Medicine and Child Neurology*, vol. 32, p. 38.
- Bhakta, B. B., Cozens, J. A., Bamford, J. M., & Chamberlain, M. A. 1996, "Use of botulinum toxin in stroke patients with severe upper limb spasticity", *J.Neurol.Neurosurg.Psychiatry*, vol. 61, no. 1, pp. 30-35.
- Bischoff, C., Schoenle, P. W., & Conrad, B. 1992, "Increased F-wave duration in patients with spasticity", *Electromyogr.Clin.Neurophysiol.*, vol. 32, no. 9, pp. 449-453.
- Black-Schaffer, R. M., Kirsteins, A. E., & Harvey, R. L. 1999, "Stroke rehabilitation. 2. Co-morbidities and complications", *Arch.Phys.Med.Rehabil.*, vol. 80, no. 5 Suppl 1, pp. S8-16.

- Bobath, B. 1978, *Adult hemiplegia, evaluation and treatment*, 2nd edn, Champaign IL: University of Illinois Press.
- Bobath, B. 1990, *Hemiplegia*, 3rd edn, Harcourt Publishers Ltd.
- Bodofsky, E. B. 1999, "Contraction-induced upper extremity H reflexes: normative values", *Arch.Phys.Med.Rehabil.*, vol. 80, no. 5, pp. 562-565.
- Bohannon, R. W. & Smith, M. B. 1987, "Interrater reliability of a modified Ashworth scale of muscle spasticity", *Phys.Ther.*, vol. 67, no. 2, pp. 206-207.
- Botte, M. J., Nickel, V. L., & Akeson, W. H. 1988, "Spasticity and contracture. Physiologic aspects of formation", *Clin.Orthop.* no. 233, pp. 7-18.
- Botte, M. J., Waters, R. L., Keenan, M. A., Jordan, C., & Garland, D. E. 1988, "Approaches to senior care #2. Orthopaedic management of the stroke patient. Part I. Pathophysiology, limb deformity, and patient evaluation", *Orthop.Rev.*, vol. 17, no. 6, pp. 637-647.
- Braddom, R. L. & Johnson, E. W. 1974, "H reflex: review and classification with suggested clinical uses", *Arch.Phys.Med.Rehabil.*, vol. 55, no. 9, pp. 412-417.
- Brashear, A., McAfee, A. L., Kuhn, E. R., & Fyffe, J. 2004, "Botulinum toxin type B in upper-limb poststroke spasticity: a double-blind, placebo-controlled trial", *Arch.Phys.Med.Rehabil.*, vol. 85, no. 5, pp. 705-709.
- Brooke JD, Peritore G, Staines WR, McIlroy WE, & Aimee N 2000, "**Upper limb H reflexes and somatosensory evoked potentials modulated by movement**", *Journal of Electromyography and Kinesiology*, vol. 10, no. 3, pp. 211-215.
- Brooke, J. D., Cheng, J., Misiaszek, J. E., & Lafferty, K. 1995a, "Amplitude modulation of the soleus H reflex in the human during active and passive stepping movements", *J.Neuropsychiol.*, vol. 73, no. 1, pp. 102-111.
- Brooke, J. D., McIlroy, W. E., Collins, D. F., & Misiaszek, J. E. 1995b, "Mechanisms within the human spinal cord suppress fast reflexes to control the movement of the legs", *Brain Res.*, vol. 679, no. 2, pp. 255-260.
- Brown, J. K., van, R. F., Walsh, G., Lakie, M., & Wright, G. W. 1987, "A neurological study of hand function of hemiplegic children", *Dev.Med.Child Neurol.*, vol. 29, no. 3, pp. 287-304.
- Burke, D. 1983, "Critical examination of the case for or against fusimotor involvement in disorders of muscle tone", *Adv.Neurol.*, vol. 39, pp. 133-150.
- Burke, D. 1988, "Spasticity as an adaptation to pyramidal tract injury", *Adv.Neurol.*, vol. 47, pp. 401-423.

- Byrne, P. C., Webb, S., McSweeney, F., Burke, T., Hutchinson, M., & Parfrey, N. A. 1998, "Linkage of AD HSP and cognitive impairment to chromosome 2p: haplotype and phenotype analysis indicates variable expression and low or delayed penetrance", *Eur.J.Hum.Genet.*, vol. 6, no. 3, pp. 275-282.
- Calderon-Gonzalez, R. & Calderon-Sepulveda, R. F. 2002, "[Treatment of spasticity in cerebral palsy with botulinum toxin]", *Rev.Neurol.*, vol. 34, no. 1, pp. 52-59.
- Carr, J. H., Shepherd, R. B., & Ada, L. 1995, "Spasticity: research findings and implications for intervention", *Physiotherapy*, vol. 81, pp. 421-429.
- Cavanagh, P. R. & Komi, P. V. 1979, "Electromechanical delay in human skeletal muscle under concentric and eccentric contractions", *Eur.J.Appl.Physiol Occup.Physiol*, vol. 42, no. 3, pp. 159-163.
- Chapman, C. E. & Wiesendanger, M. 1982, "The physiological and anatomical basis of spasticity: a review", *Physiotherapy Canada*, vol. 34, no. 3, pp. 125-136.
- Childers, M. K., Brashear, A., Jozefczyk, P., Reding, M., Alexander, D., Good, D., Walcott, J. M., Jenkins, S. W., Turkel, C., & Molloy, P. T. 2004, "Dose-dependent response to intramuscular botulinum toxin type A for upper-limb spasticity in patients after a stroke", *Arch.Phys.Med.Rehabil.*, vol. 85, no. 7, pp. 1063-1069.
- Crago, P. E., Houk, J. C., & Hasan, Z. 1976, "Regulatory actions of human stretch reflex", *J.Neurophysiol.*, vol. 39, no. 5, pp. 925-935.
- Damiano, D. L., Quinlivan, J. M., Owen, B. F., Payne, P., Nelson, K. C., & Abel, M. F. 2002, "What does the Ashworth scale really measure and are instrumented measures more valid and precise?", *Dev.Med.Child Neurol.*, vol. 44, no. 2, pp. 112-118.
- Davies, P., Petit, J., & Scott, J. J. 1995, "The dynamic response of Golgi tendon organs to tetanic contraction of in-series motor units", *Brain Res.*, vol. 690, no. 1, pp. 82-91.
- de Paiva, A., Meunier, F. A., Molgo, J., Aoki, K. R., & Dolly, J. O. 1999, "Functional repair of motor endplates after botulinum neurotoxin type A poisoning: biphasic switch of synaptic activity between nerve sprouts and their parent terminals", *Proc.Natl.Acad.Sci.U.S.A*, vol. 96, no. 6, pp. 3200-3205.
- De, E. G., Brooker, G., Costa, E., & Wojcik, W. J. 1992, "Stimulation of high affinity gamma-aminobutyric acidB receptors potentiates the depolarization-induced increase of intraneuronal ionized calcium content in cerebellar granule neurons", *Mol.Pharmacol.*, vol. 42, no. 3, pp. 407-414.
- Deltombe, T., De Wispelaere, J. F., Gustin, T., Jamart, J., & Hanson, P. 2004, "Selective blocks of the motor nerve branches to the soleus and tibialis posterior muscles in the management of the spastic equinovarus foot", *Arch.Phys.Med.Rehabil.*, vol. 85, no. 1, pp. 54-58.

- Delwaide, P. J. 1989, "[Pyramidal syndrome: its physiopathology and treatment]", *Recenti Prog.Med.*, vol. 80, no. 12, pp. 681-685.
- Delwaide, P. J., Pepin, J. L., & Maertens de, N. A. 1993, "The audiospinal reaction in parkinsonian patients reflects functional changes in reticular nuclei", *Ann.Neurol.*, vol. 33, no. 1, pp. 63-69.
- Dewald, J. P. & Given JD 1994, "Electrical Stimulation and Spasticity Reduction: Fact or Fiction?", *Physical Medicine and Rehabilitation:state of the art reviews*, vol. 8, no. 3, pp. 507-522.
- Dewald, J. P., Given, J. D., & Rymer, W. Z. 1996, "Long-lasting reductions of spasticity induced by skin electrical stimulation", *IEEE Trans.Rehabil.Eng*, vol. 4, no. 4, pp. 231-242.
- Dietz, V. 2003, "Spinal cord pattern generators for locomotion", *Clin.Neurophysiol.*, vol. 114, no. 8, pp. 1379-1389.
- Dressnandt, J., Auer, C., & Conrad, B. 1995, "Influence of baclofen upon the alpha-motoneuron in spasticity by means of F-wave analysis", *Muscle Nerve*, vol. 18, no. 1, pp. 103-107.
- Duckworth, D. & Toth, H. H. Two cases of general spastic rigidity. 49[ii]. 18. Medical tracts.
- Dvir, Z. & Panturin, E. 1993, "Measurement of spasticity and associated reactions in stroke patients before and after physiotherapeutic intervention", *Clinical Rehabilitation*, vol. 7, pp. 15-21.
- Evarts, E. V. & GRANIT, R. 1976, "Relations of reflexes and intended movements", *Prog.Brain Res.*, vol. 44, pp. 1-14.
- Faist, M., Ertel, M., Berger, W., & Dietz, V. 1999, "Impaired modulation of quadriceps tendon jerk reflex during spastic gait: differences between spinal and cerebral lesions", *Brain*, vol. 122 (Pt 3), pp. 567-579.
- Fee, J. W., Jr. & Foulds, R. A. 2004, "Neuromuscular modeling of spasticity in cerebral palsy", *IEEE Trans.Neural Syst.Rehabil.Eng*, vol. 12, no. 1, pp. 55-64.
- Fellows, S. J., Ross, H. F., & Thilmann, A. F. 1993, "The limitations of the tendon jerk as a marker of pathological stretch reflex activity in human spasticity", *J.Neurol.Neurosurg.Psychiatry*, vol. 56, no. 5, pp. 531-537.
- Firoozbakhsh, K. K., Kunkel, C. F., Scremin, A. M., & Moneim, M. S. 1993, "Isokinetic dynamometric technique for spasticity assessment", *Am.J.Phys.Med.Rehabil.*, vol. 72, no. 6, pp. 379-385.

- Fisher, M. A. **Whither F waves – clinical and research perspectives** . Electroencephalography and Clinical Neurophysiology/Electromyography and Motor Control 97[4], s62-s63. 1995.
- Fowler, E. G., Nwigwe, A. I., & Ho, T. W. 2000, "Sensitivity of the pendulum test for assessing spasticity in persons with cerebral palsy", *Dev.Med.Child Neurol.*, vol. 42, no. 3, pp. 182-189.
- Gardner, M. J., Ong, B. C., Liporace, F., & Koval, K. J. 2002, "Orthopedic issues after cerebrovascular accident", *Am.J.Orthop.*, vol. 31, no. 10, pp. 559-568.
- Ghez, C. & Krakauer, J. 2000, "The Organization of Movement," in *Principles of neural science*, fourth edn, E. R. Kandel, J. H. Schwartz, & T. M. Jessel, eds., McGraw Hill, pp. 653-673.
- Ghosh, B. & Das, S. K. 2002, "Botulinum toxin: a dreaded toxin for use in human being", *J.Indian Med.Assoc.*, vol. 100, no. 10, pp. 607-2, 614.
- Given JD et al 1995, "Joint dependent passive stiffness in paretic and contralateral limbs of spastic patients with hemiparetic stroke", *Journal of Neurology, Neurosurgery, and Psychiatry*, vol. 59, pp. 271-279.
- Gracies, J. M., Marosszeky, J. E., Renton, R., Sandanam, J., Gandevia, S. C., & Burke, D. 2000, "Short-term effects of dynamic lycra splints on upper limb in hemiplegic patients", *Arch.Phys.Med.Rehabil.*, vol. 81, no. 12, pp. 1547-1555.
- Gregory, J. E., Brockett, C. L., Morgan, D. L., Whitehead, N. P., & Proske, U. 2002, "Effect of eccentric muscle contractions on Golgi tendon organ responses to passive and active tension in the cat", *J.Physiol*, vol. 538, no. Pt 1, pp. 209-218.
- Grill, S. E. & Rymer, W. Z. 1985, "Reflex actions of muscle afferents on fusimotor innervation in decerebrated cats: an assessment of beta contributions", *Exp.Brain Res.*, vol. 59, no. 2, pp. 282-295.
- Harburn, K. L. & Potter, P. J. 1993, "Spasticity and contractures", *Physical Medicine and Rehabilitation*, vol. 7, no. 1, pp. 113-132.
- Harburn, K. L., Vandervoort, A. A., Helewa, A., Goldsmith, C. H., Kertesz, A., Teasell, R. W., & Hill, K. M. 1995, "A reflex technique to measure presynaptic inhibition in cerebral stroke", *Electromyogr.Clin.Neurophysiol.*, vol. 35, no. 3, pp. 149-163.
- Hayashi, R., Hashimoto, T., Tada, T., & Ikeda, S. 2001, "Relation between changes in long-latency stretch reflexes and muscle stiffness in Parkinson's disease - comparison before and after unilateral pallidotomy", *Clinical Neurophysiology*, vol. 112, no. 10, pp. 1814-1821.
- He, J. 1998, "Stretch reflex sensitivity: effects of postural and muscle length changes", *IEEE Trans.Rehabil.Eng*, vol. 6, no. 2, pp. 182-189.

- Heald, A., Bates, D., Cartlidge, N. E., French, J. M., & Miller, S. 1993a, "Longitudinal study of central motor conduction time following stroke. 1. Natural history of central motor conduction", *Brain*, vol. 116 (Pt 6), pp. 1355-1370.
- Heald, A., Bates, D., Cartlidge, N. E., French, J. M., & Miller, S. 1993b, "Longitudinal study of central motor conduction time following stroke. 2. Central motor conduction measured within 72 h after stroke as a predictor of functional outcome at 12 months", *Brain*, vol. 116 (Pt 6), pp. 1371-1385.
- Higashi, T., Funase, K., Kusano, K., Tabira, T., Harada, N., Sakakibara, A., & Yoshimura, T. 2001, "Motoneuron pool excitability of hemiplegic patients: assessing recovery stages by using H-reflex and M response", *Arch.Phys.Med.Rehabil.*, vol. 82, no. 11, pp. 1604-1610.
- Homma, S. 1976, "Frequency characteristics of the impulse decoding ratio between the spinal afferents and efferents in the stretch reflex", *Prog.Brain Res.*, vol. 44, pp. 15-30.
- Hsueh, I. P., Lee, M. M., & Hsieh, C. L. 2002, "The Action Research Arm Test: is it necessary for patients being tested to sit at a standardized table?", *Clinical Rehabilitation*, vol. 16, pp. 382-388.
- Hughes, A. J. 1994, "Botulinum toxin in clinical practice", *Drugs*, vol. 48, no. 6, pp. 888-893.
- Iloeje, S. O. 1994, "Measurement of muscle tone in children with cerebellar ataxia", *East Afr.Med J.*, vol. 71, no. 4, pp. 256-260.
- Jankus, W. R., Robinson, L. R., & Little, J. W. 1994, "Normal limits of side-to-side H-reflex amplitude variability", *Arch.Phys.Med.Rehabil.*, vol. 75, no. 1, pp. 3-7.
- Johnson, G. R. 2002, "Outcome measures of spasticity", *Eur.J.Neurol.*, vol. 9 Suppl 1, pp. 10-16.
- Joodaki, M. R., Olyaei, G. R., & Bagheri, H. 2001, "The effects of electrical nerve stimulation of the lower extremity on H-reflex and F-wave parameters", *Electromyogr.Clin.Neuropsychiol.*, vol. 41, no. 1, pp. 23-28.
- Ju, M. S., Chen, J. J., Lee, H. M., Lin, T. S., Lin, C. C., & Huang, Y. Z. 2000, "Time-course analysis of stretch reflexes in hemiparetic subjects using an on-line spasticity measurement system", *J.Electromyogr.Kinesiol.*, vol. 10, no. 1, pp. 1-14.
- Kakuda, N. & Nagaoka, M. 1998, "Dynamic response of human muscle spindle afferents to stretch during voluntary contraction", *J.Physiol*, vol. 513 (Pt 2), pp. 621-628.
- Kakuda, N., Miwa, T., & Nagaoka, M. 1998, "Coupling between single muscle spindle afferent and EMG in human wrist extensor muscles: physiological evidence of skeletofusimotor (beta) innervation", *Electroencephalogr.Clin.Neuropsychiol.*, vol. 109, no. 4, pp. 360-363.

- Kamper, D. G. & Rymer, W. Z. 2000, "Quantitative features of the stretch response of extrinsic finger muscles in hemiparetic stroke", *Muscle Nerve*, vol. 23, no. 6, pp. 954-961.
- Kamper, D. G., Schmit, B. D., & Rymer, W. Z. 2001, "Effect of muscle biomechanics on the quantification of spasticity", *Ann.Biomed.Eng.*, vol. 29, no. 12, pp. 1122-1134.
- Kasai, T. & Komiyama, T. 1996, "Soleus H-reflex depression induced by ballistic voluntary arm movement in human", *Brain Res.*, vol. 714, no. 1-2, pp. 125-134.
- Katz RT 1994, "Electrophysiologic Assessment of Spastic Hypertonia", *Physical Medicine and Rehabilitation*, vol. 8, no. 3, pp. 465-471.
- Klein, C. S., Rice, C. L., & Marsh, G. D. 2001, "Normalized force, activation, and coactivation in the arm muscles of young and old men", *J.Appl.Physiol*, vol. 91, pp. 1341-1349.
- Kong, K. H. & Chua, K. S. 2002, "Intramuscular neurolysis with alcohol to treat post-stroke finger flexor spasticity", *Clin.Rehabil.*, vol. 16, no. 4, pp. 378-381.
- Lai, C. H., Gau, J. L., Ju, M. S., & Chen, J. J. 1996, "Development of Spasticity Measurement System for Quantitative Analysis of Stretch Reflexes in Spastic Subjects", *Chinese J.Med.Biol.Eng.*, vol. 16, no. 1, pp. 16-30.
- Lamontagne, A., Malouin, F., & Richards, C. L. 2001, "Locomotor-specific measure of spasticity of plantarflexor muscles after stroke", *Arch.Phys.Med.Rehabil.*, vol. 82, no. 12, pp. 1696-1704.
- Lance JW 1980, *Symposium Synopsis, in Spasticity: Disordered Motor control*, Eds. Symposia Specialist. Miami.
- Lance, J. W. 1980, "The control of muscle tone, reflexes, and movement: Robert Wartenberg Lecture", *Neurology*, vol. 30, no. 12, pp. 1303-1313.
- Lance, J. W. 1990, "What is spasticity?", *Lancet*, vol. 335, no. 8689, p. 606.
- Laporte, Y. & Emonet-Denand, F. 1976, "The skeleto-fusimotor innervation of cat muscle spindle", *Prog.Brain Res.*, vol. 44, pp. 99-109.
- Le Pellec, A. & Maton, B. 1996, "Influence of Tonic Neck Reflexes on the Upper Limb Stretch Reflex in Man", *Journal of Electromyography & Kinesiology*, vol. 6, no. 2, pp. 73-82.
- Le, C. P., Hernot, X., Bartier, O., Carrault, G., Chagneau, F., Gallien, P., Allain, H., & Rochcongar, P. 2002, "[Evaluation of pendulum testing of spasticity]", *Ann.Readapt.Med.Phys.*, vol. 45, no. 9, pp. 510-516.

- Le, C. P., Poudens, S. A., Chagneau, F., Carrault, G., Allain, H., & Rochcongar, P. 2001, "A comprehensive model of spastic hypertonia derived from the pendulum test of the leg", *Muscle Nerve*, vol. 24, no. 12, pp. 1612-1621.
- Leao, R. N. & Burne, J. A. 2004, "**Continuous wavelet transform in the evaluation of stretch reflex responses from surface EMG**", *Journal of Neuroscience Methods*, vol. 133, no. 1-2, pp. 115-125.
- Lee, H. M., Chen, J. J., Ju, M. S., Lin, C. C., & Poon, P. P. 2004, "Validation of portable muscle tone measurement device for quantifying velocity-dependent properties in elbow spasticity", *J.Electromyogr.Kinesiol.*, vol. 14, no. 5, pp. 577-589.
- Lee, H. M., Huang, Y. Z., Chen, J. J., & Hwang, I. S. 2002, "Quantitative analysis of the velocity related pathophysiology of spasticity and rigidity in the elbow flexors", *J.Neurol.Neurosurg.Psychiatry*, vol. 72, no. 5, pp. 621-629.
- Lehmann, J. F., Price, R., deLateur, B. J., Hinderer, S., & Traynor, C. 1989, "Spasticity: quantitative measurements as a basis for assessing effectiveness of therapeutic intervention", *Arch.Phys.Med.Rehabil.*, vol. 70, no. 1, pp. 6-15.
- Leonard, C. T., Diedrich, P. M., Matsumoto, T., Moritani, T., & McMillan, J. A. 1998, "H-reflex modulations during voluntary and automatic movements following upper motor neuron damage", *Electroencephalogr.Clin.Neurophysiol.*, vol. 109, no. 6, pp. 475-483.
- Lieberson WT, Holmquest HJ, & Scot D, D. M. Functional electrotherapy: stimulation of the peroneal nerve synchronized with the swing phase of gait of hemiplegic patients. *Arch Phys Med Rehabil* 42, 101-105. 1961.
- Lin, C. C., Ju, M. S., & Lin, C. W. 2003, "The pendulum test for evaluating spasticity of the elbow joint", *Arch.Phys.Med.Rehabil.*, vol. 84, no. 1, pp. 69-74.
- Lin, C. C., Lin, C. W., Ke, T. Y., & Ju, M. S. 2001, "Biomechanical Models for Modified Pendulum Test of the Upper Limb", *Journal of Medical and Biological Engineering*, vol. 21, no. 4, pp. 257-263.
- Lin, D. C. & Rymer, W. Z. 1991, "A quantitative analysis of pendular motion of the lower leg in spastic human subjects", *IEEE Trans.Biomed.Eng*, vol. 38, no. 9, pp. 906-918.
- Lin, D. C. & Rymer, W. Z. 1998, "Damping in reflexively active and areflexive lengthening muscle evaluated with inertial loads", *J.Neurophysiol.*, vol. 80, no. 6, pp. 3369-3372.
- Lin, D. C. & Rymer, W. Z. 2001, "Damping actions of the neuromuscular system with inertial loads: human flexor pollicis longus muscle", *J.Neurophysiol.*, vol. 85, no. 3, pp. 1059-1066.

- Lin, F. M. & Sabbahi, M. 1999, "Correlation of spasticity with hyperactive stretch reflexes and motor dysfunction in hemiplegia", *Arch.Phys.Med.Rehabil.*, vol. 80, no. 5, pp. 526-530.
- Magladery JW & McDougal DB 1950, "Electrophysiologic studies of nerve and reflex activities in normal man & identification of certain reflexes in the electromyogram and conduction velocity of peripheral nerve fibers.", *Bull Johns Hopkins Hosp*, vol. 86: pp. 265-290.
- Marchese, S. S., Di, B. P., Sessa, E., Donato, S., & Bramanti, P. 2001, "The spasticity evaluation test (SeT): a pilot study", *J.Rehabil.Res.Dev.*, vol. 38, no. 1, pp. 93-100.
- Marque, P., Simonetta-Moreau, M., Maupas, E., & Roques, C. F. 2001, "Facilitation of transmission in heteronymous group II pathways in spastic hemiplegic patients", *J.Neurol.Neurosurg.Psychiatry*, vol. 70, no. 1, pp. 36-42.
- Matthews PBC 1972a, "Tone and The Stretch Reflex," in *Mammalian Muscle Receptors and their Central Actions*, Edward Arnold Ltd, London, pp. 414-418.
- Matthews PBC 1972b, "The Functional Distinctiveness of the Primary and Secondary Spindle Endings," in *Mammalian Muscle Receptors and their Central Actions*, Edward Arnold Ltd., London, p. 157.
- Matthews, W. B. 1966, "Ratio of maximum H reflex to maximum M response as a measure of spasticity", *J.Neurol.Neurosurg.Psychiatry*, vol. 29, no. 3, pp. 201-204.
- Miscio, G., Del, C. C., Pianca, D., Colombo, R., Panizza, M., Schieppati, M., & Pisano, F. 2004, "Botulinum toxin in post-stroke patients: stiffness modifications and clinical implications", *J.Neurol.*, vol. 251, no. 2, pp. 189-196.
- Misiaszek, J. E., de Serres, S. J., Stein, R. B., Jiang, W., & Pearson, K. G. 2000, "Stretch and H reflexes in triceps surae are similar during tonic and rhythmic contractions in high decerebrate cats", *J.Neurophysiol.*, vol. 83, no. 4, pp. 1941-1950.
- Morita, H., Crone, C., Christenhuis, D., Petersen, N. T., & Nielsen, J. B. 2001, "Modulation of presynaptic inhibition and disynaptic reciprocal Ia inhibition during voluntary movement in spasticity", *Brain*, vol. 124, no. Pt 4, pp. 826-837.
- Neilson, P. D. & McCaughey, J. 1981, "Effect of contraction level and magnitude of stretch on tonic stretch reflex transmission characteristics", *J.Neurol.Neurosurg.Psychiatry*, vol. 44, no. 11, pp. 1007-1012.
- O'Brien, C. F., Seeberger, L. C., & Smith, D. B. 1996, "Spasticity after stroke. Epidemiology and optimal treatment", *Drugs Aging*, vol. 9, no. 5, pp. 332-340.
- Oddsson, L. I. 1990, "Control of voluntary trunk movements in man. Mechanisms for postural equilibrium during standing", *Acta Physiol Scand.Suppl*, vol. 595, pp. 1-60.

- O'Dwyer, N. J. & Ada, L. 1996, "Reflex hyperexcitability and muscle contracture in relation to spastic hypertonia", *Curr.Opin.Neurol.*, vol. 9, no. 6, pp. 451-455.
- O'Dwyer, N. J., Ada, L., & Neilson, P. D. 1996, "Spasticity and muscle contracture following stroke", *Brain*, vol. 119 (Pt 5), pp. 1737-1749.
- Ogata, K. 1998, "Linear Systems Analyses in the Time Domain," 3rd edn, Prentice-Hall, New Jersey, pp. 236-291.
- Okuma, Y., Mizuno, Y., & Lee, R. G. 2002, "Reciprocal Ia inhibition in patients with asymmetric spinal spasticity", *Clin.Neurophysiol.*, vol. 113, no. 2, pp. 292-297.
- Pagliaro, P. & Zamparo, P. 1999, "Quantitative evaluation of the stretch reflex before and after hydro kinesy therapy in patients affected by spastic paresis", *J.Electromyogr.Kinesiol.*, vol. 9, no. 2, pp. 141-148.
- Pandyan, A. D., Price, C. I., Barnes, M. P., & Johnson, G. R. 2003, "A biomechanical investigation into the validity of the modified Ashworth Scale as a measure of elbow spasticity", *Clin.Rehabil.*, vol. 17, no. 3, pp. 290-293.
- Pandyan, A. D., Price, C. I., Rodgers, H., Barnes, M. P., & Johnson, G. R. 2001, "Biomechanical examination of a commonly used measure of spasticity", *Clin.Biomech.(Bristol., Avon.)*, vol. 16, no. 10, pp. 859-865.
- Panizza, M., Balbi, P., Russo, G., & Nilsson, J. 1995, "H-reflex recovery curve and reciprocal inhibition of H-reflex of the upper limbs in patients with spasticity secondary to stroke", *Am.J.Phys.Med.Rehabil.*, vol. 74, no. 5, pp. 357-363.
- Pearson, K. & Gordon, J. 2000, "Spinal reflexes," in *Principles of neural science*, fourth edn, E. R. Kandel, J. H. Schwartz, & T. M. Jessel, eds., McGraw Hill, pp. 713-735.
- Pike, M. G., Applegarth, D. A., Dunn, H. G., Bamforth, S. J., Tingle, A. J., Wood, B. J., Dimmick, J. E., Harris, H., Chantler, J. K., & Hall, J. G. 1990, "Congenital rubella syndrome associated with calcific epiphyseal stippling and peroxisomal dysfunction", *J.Pediatr.*, vol. 116, no. 1, pp. 88-94.
- Pisano, F., Miscio, G., Del Conte, C., Pianca, D., Candeloro, E., & Colombo, R. 2000, "Quantitative measures of spasticity in post-stroke patients", *Clin.Neurophysiol.*, vol. 111, no. 6, pp. 1015-1022.
- Price, R. 1990, "Mechanical Spasticity Evaluation Techniques", *Critical Reviews in Physical and rehabilitation Medicine*, vol. 2, no. 2, pp. 65-73.
- Rosche, J., Rub, K., Niemann-Delius, B., Mauch, E., & Kornhuber, H. H. 1996, "Effects of physiotherapy on F-wave-amplitudes in spasticity", *Electromyogr.Clin.Neurophysiol.*, vol. 36, no. 8, pp. 509-511.
- Rothwell, J. 1994, "Mechanical properties of muscles," in *Control of Human Voluntary Movement*, Second edn, Chapman & Hall, London, pp. 6-9.

- Rymer WZ & Katz RT 1994, "Mechanical Quantification of spastic hypertonia", *Phys.Med.Rehabil*, vol. 8, pp. 455-463.
- Schafer, S. S., Berkelmann, B., & Schuppan, K. 1999, "Two groups of Golgi tendon organs in cat tibial anterior muscle identified from the discharge frequency recorded under a ramp-and-hold stretch", *Brain Res.*, vol. 846, no. 2, pp. 210-218.
- Schafer, S. S., Schuppan, O., & Dadfar, F. 1999, "The response of muscle spindle primary afferents to simultaneously presented sinusoidal and ramp-and-hold stretches", *Brain Res.*, vol. 819, no. 1-2, pp. 89-107.
- Schmit, B. D. & Rymer, W. Z. 2001, "Identification of static and dynamic components of reflex sensitivity in spastic elbow flexors using a muscle activation model", *Ann.Biomed.Eng*, vol. 29, no. 4, pp. 330-339.
- Schmit, B. D. 2001, "Mechanical measures of spasticity in stroke", *Top.Stroke Rehabil.*, vol. 8, no. 1, pp. 13-26.
- Schmit, B. D., Dewald, J. P., & Rymer, W. Z. 2000, "Stretch reflex adaptation in elbow flexors during repeated passive movements in unilateral brain-injured patients", *Arch.Phys.Med.Rehabil.*, vol. 81, no. 3, pp. 269-278.
- Schmit, B. D., Dhaher, Y., Dewald, J. P., & Rymer, W. Z. 1999, "Reflex torque response to movement of the spastic elbow: theoretical analyses and implications for quantification of spasticity", *Ann.Biomed.Eng*, vol. 27, no. 6, pp. 815-829.
- Shapiro, R. T. 2001, "Management of Spasticity, Pain, and Paroxysmal Phenomena in Multiple Sclerosis", *Current Neurology and Neuroscience reports*, vol. 1, pp. 299-302.
- Sheean, G. 1998, "Pathophysiology of spasticity," in *Spasticity rehabilitation*, G. Sheean, ed., Churchill communications Europe, London.
- Sheean, G. 2002, "The pathophysiology of spasticity", *Eur.J.Neurol.*, vol. 9 Suppl 1, pp. 3-9.
- Sherman, S. J., Koshland, G. F., & Laguna, J. F. 2000, "Hyper-reflexia without spasticity after unilateral infarct of the medullary pyramid", *J.Neurol.Sci.*, vol. 175, no. 2, pp. 145-155.
- Sherrington, C. S. & Denny-Brown, D. 1979, *Selected writings of Sir Charles Sherrington: a testimonial presented by the neurologists forming the guarantors of the journal Brain* Oxford University Press, Oxford.
- Sherrington, C. S. 1898, "Decerebrate rigidity and reflex coordination of movements", *J Physiol (Lond)*, vol. 22, pp. 319-332.
- Singer, B. J., Dunne, J. W., Singer, K. P., & Allison, G. T. 2003, "Velocity dependent passive plantarflexor resistive torque in patients with acquired brain injury", *Clin.Biomech.(Bristol, Avon.)*, vol. 18, no. 2, pp. 157-165.

- Sommerfeld, D. K., Eek, E. U., Svensson, A. K., Holmqvist, L. W., & von Arbin, M. H. 2004, "Spasticity after stroke: its occurrence and association with motor impairments and activity limitations", *Stroke*, vol. 35, no. 1, pp. 134-139.
- Stampacchia, G., Bradaschia, E., & Rossi, B. 2004, "Change of stretch reflex threshold in spasticity: effect of botulinum toxin injections", *Arch.Ital.Biol.*, vol. 142, no. 3, pp. 265-273.
- Stefanovska, A., Gros, N., Vodovnik, L., Rebersek, S., & cimovic-Janezic, R. 1988, "Chronic electrical stimulation for the modification of spasticity in hemiplegic patients", *Scand.J.Rehabil.Med.Suppl.*, vol. 17, pp. 115-121.
- Stefanovska, A., Rebersek S, Bajd T, & Vodovnik L 1991, "Effects of Electrical Stimulation on Spasticity", *Critical Reviews in Physical and rehabilitation Medicine*, vol. 3, no. 1, pp. 59-99.
- Stevens, S. On the Theory of Scales of Measurement. *Science Magazine* 103[2684], 677-680. 1946.
- Stokes, I. A. F. 2004, "Relationships of EMG to effort in the trunk under isometric conditions: force-increasing and decreasing effects and temporal delays", *Clinical Biomechanics*. *In press*
- Takata, Y., Nakajima, T., & Yamada, Y. 1996, "Quantitative evaluation of the tonic vibration reflex (TVR) in the masseter muscle", *J Oral Maxillofac.Surg.*, vol. 54, no. 11, pp. 1307-1313.
- [Taylor, A. & Durbaba, R. Muscle spindle basics. McGarrick, J. www.kcl.ac.uk/teares/gktvc/vc/lt/mspindle/spin1.htm . 16-1-2003. King's College London.](http://www.kcl.ac.uk/teares/gktvc/vc/lt/mspindle/spin1.htm)
- Tsai, K. H., Yeh, C. Y., Chang, H. Y., & Chen, J. J. 2001, "Effects of a single session of prolonged muscle stretch on spastic muscle of stroke patients", *Proc.Natl.Sci.Counc.Repub.China B*, vol. 25, no. 2, pp. 76-81.
- Turner, M. S. 2003, "Early use of intrathecal baclofen in brain injury in pediatric patients", *Acta Neurochir.Suppl.*, vol. 87, pp. 81-83.
- Vattanasilp, W. & Ada, L. 1999, "The relationship between clinical and laboratory measures of spasticity", *Aust.J.Physiother.*, vol. 45, no. 2, pp. 135-139.
- Vodovnik, L., Bowman, B. R., & Bajd, T. 1984, "Dynamics of spastic knee joint", *Med Biol.Eng Comput.*, vol. 22, no. 1, pp. 63-69.
- Vodovnik, L., Bowman, B. R., & Hufford, P. 1984, "Effects of electrical stimulation on spinal spasticity", *Scand.J.Rehabil.Med.*, vol. 16, no. 1, pp. 29-34.
- Watkins, C. L., Leathley, M. J., Gregson, J. M., Moore, A. P., Smith, T. L., & Sharma, A. K. 2002, "Prevalence of spasticity post stroke", *Clin.Rehabil.*, vol. 16, no. 5, pp. 515-522.

- Wilson, L. R., Gandevia, S. C., Inglis, J. T., Gracies, J., & Burke, D. 1999, "Muscle spindle activity in the affected upper limb after a unilateral stroke", *Brain*, vol. 122 (Pt 11), pp. 2079-2088.
- Windhorst, U. 1996, "ON THE ROLE OF RECURRENT INHIBITORY FEEDBACK IN MOTOR CONTROL", *Prog.Neurobiol.*, vol. 49, no. 6, pp. 517-587.
- Winter, D. A. 1990, *Biomechanics and Motor Control of Human Movement* John Wiley & Sons..
- Wolf, S. L., Catlin, P. A., Ellis, M., Archer, A. L., Morgan, B., & Piacentino, A. 2001, "Assessing Wolf motor function test as outcome measure for research in patients after stroke", *Stroke*, vol. 32, no. 7, pp. 1635-1639.
- Wolf, S. L., Segal, R. L., Catlin, P. A., Tschorn, J., Raleigh, T., Kontos, H., & Pate, P. 1996, "Determining consistency of elbow joint threshold angle in elbow flexor muscles with spastic hypertonia", *Phys.Ther.*, vol. 76, no. 6, pp. 586-600.
- Yamamoto, S., Nakazawa, K., Yano, H., & Ohtsuki, T. 2000, "Differential angle-dependent modulation of the long-latency stretch reflex responses in elbow flexion synergists", *J.Electromyogr.Kinesiol.*, vol. 10, no. 2, pp. 135-142.
- Yang, T. F., Fu, C. P., Kao, N. T., Chan, R. C., & Chen, S. J. 2003, "Effect of botulinum toxin type A on cerebral palsy with upper limb spasticity", *Am.J.Phys.Med.Rehabil.*, vol. 82, no. 4, pp. 284-289.
- Young RR 1989, "Treatment of spastic paresis", *N.Engl.J.Med.*, vol. 320, pp. 1553-1555.
- Young, R. R. 1994, "Spasticity: a review", *Neurology*, vol. 44, no. 11 Suppl 9, p. S12-S20.
- Zamparo, P., Capelli, C., Pagliaro, P., De, L. G., Pertoldi, S., Saccavini, M., & Di Prampero, P. E. 1997, "Quantitative evaluation of the Myotatic Reflex in Hemiplegic and Paraplegic patients", *Adv.Clin.Path.*, vol. 1, no. 1, pp. 49-57.
- Zhang, L. Q., Wang, G., Nishida, T., Xu, D., Sliwa, J. A., & Rymer, W. Z. 2000, "Hyperactive tendon reflexes in spastic multiple sclerosis: measures and mechanisms of action", *Arch Phys Med Rehabil*, vol. 81, no. 7, pp. 901-909.

Appendix A

**Non-impaired (NI) and Post-stroke (PS)
subjects individual measurements**

Subject

NI1

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	145.72	52.00	41.75	96.68	21.62	341.91
Flexed	6.84	Flexing	Centre	276.64	20.51	25.39	49.80	11.62	143.36
Flexed	4.56	Relax	Centre	363.19	34.67	37.11	70.31	30.66	465.18
Flexed	4.56	Flexing	Centre	236.65	34.91	36.87	217.04	45.82	553.64
Flexed	6.84	Relax	Centre	158.65	27.59	45.17	280.52	45.81	553.52
Flexed	6.84	Extending	Centre	209.18	32.23	50.29	235.60	45.92	543.59
Extended	4.56	Relax	Centre	224.46	20.26	35.64	66.89	23.00	354.17
Extended	6.84	Extending	Centre	281.61	41.50	83.50	155.27	45.99	656.34
Extended	4.56	Flexing	Centre	249.42	45.41	48.10	95.70	28.61	290.77
Extended	6.84	Flexing	Centre	256.71	33.94	36.38	75.93	14.78	184.60
Extended	6.84	Relax	Centre	242.83	26.86	43.21	96.68	36.44	391.76
Extended	4.56	Extending	Centre	345.82	35.16	68.85	167.72	46.10	590.06
Extended	6.84	Relax	Left	371.52	31.49	35.89	69.58	33.82	398.10
Extended	6.84	Relax	Right	339.99	27.34	60.55	259.52	46.21	698.45

Subject

NI2

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	410.57	74.46	33.69	69.34	24.96	229.70
Flexed	6.84	Flexing	Centre	459.90	21.97	64.45	108.89	17.84	179.10
Flexed	4.56	Relax	Centre	481.14	44.68	35.16	75.20	31.37	298.81
Flexed	4.56	Flexing	Centre	307.72	41.26	35.16	73.24	46.11	453.99
Flexed	6.84	Relax	Centre	382.65	31.01	66.89	113.53	46.13	445.64
Flexed	6.84	Extending	Centre	21.48	25.39	33.94	129.39	46.11	447.56
Extended	4.56	Relax	Centre	447.03	28.32	37.11	70.31	36.54	340.94
Extended	6.84	Extending	Centre	475.78	36.13	43.46	93.51	46.12	485.60
Extended	4.56	Flexing	Centre	437.53	48.10	41.26	309.57	24.38	222.43
Extended	6.84	Flexing	Centre	422.61	34.18	47.85	91.31	10.28	116.26
Extended	6.84	Relax	Centre	413.74	35.40	34.91	73.00	29.72	299.86
Extended	4.56	Extending	Centre	419.95	44.92	56.88	108.89	46.21	458.30
Extended	6.84	Relax	Left	448.57	29.54	34.91	65.92	35.31	352.80
Extended	6.84	Relax	Right	425.89	38.09	47.85	138.67	46.15	488.47

Subject

NI3

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	12.72	0.00	40.28	134.52	23.86	228.92
Flexed	6.84	Flexing	Centre	16.16	0.00	0.00	0.00	12.75	129.89
Flexed	4.56	Relax	Centre	249.42	72.75	38.33	79.35	33.21	320.87
Flexed	4.56	Flexing	Centre	9.42	0.00	13.67	26.86	46.19	463.70
Flexed	6.84	Relax	Centre	20.77	41.99	37.35	126.95	46.21	457.16
Flexed	6.84	Extending	Centre	23.28	0.00	22.71	178.22	46.18	454.68
Extended	4.56	Relax	Centre	267.50	12.45	40.77	82.76	34.28	331.12
Extended	6.84	Extending	Centre	245.36	57.86	43.70	90.58	46.29	562.64
Extended	4.56	Flexing	Centre	210.08	74.22	34.91	73.49	22.53	222.36
Extended	6.84	Flexing	Centre	292.54	51.76	28.08	51.03	20.88	205.63
Extended	6.84	Relax	Centre	406.27	57.37	38.82	82.28	31.87	303.58
Extended	4.56	Extending	Centre	124.18	46.88	37.11	72.51	46.04	458.83
Extended	6.84	Relax	Left	263.88	5.62	69.82	103.27	37.44	354.15
Extended	6.84	Relax	Right	349.68	50.78	41.75	113.77	46.22	520.10

Subject

NI4

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	69.97	22.71	37.84	74.22	16.71	167.79
Flexed	6.84	Flexing	Centre	64.51	308.11	10.74	21.00	4.08	40.58
Flexed	4.56	Relax	Centre	110.62	7.81	34.67	133.79	30.17	264.62
Flexed	4.56	Flexing	Centre	94.61	0.00	95.21	196.53	42.11	379.11
Flexed	6.84	Relax	Centre	7.21	7.81	9.77	18.80	43.17	377.83
Flexed	6.84	Extending	Centre	165.66	60.30	37.35	85.45	43.56	367.86
Extended	4.56	Relax	Centre	318.02	36.38	39.55	70.80	11.44	114.81
Extended	6.84	Extending	Centre	369.14	54.44	44.68	82.76	45.44	440.02
Extended	4.56	Flexing	Centre	7.55	0.00	9.52	18.31	23.31	192.85
Extended	6.84	Flexing	Centre	91.33	275.88	10.01	19.78	3.42	36.48
Extended	6.84	Relax	Centre	62.13	18.80	51.51	102.78	39.28	351.32
Extended	4.56	Extending	Centre	64.60	21.97	82.03	148.44	44.56	317.32
Extended	6.84	Relax	Left	264.92	75.68	20.02	37.11	5.30	54.93
Extended	6.84	Relax	Right	133.86	8.54	93.75	150.39	45.69	488.64

Subject

NI5

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	23.50	0.00	84.23	124.51	20.28	193.20
Flexed	6.84	Flexing	Centre	338.13	16.36	29.05	56.40	14.44	158.18
Flexed	4.56	Relax	Centre	397.23	26.37	32.47	67.14	23.76	226.34
Flexed	4.56	Flexing	Centre	161.40	1.71	30.76	62.74	42.50	371.21
Flexed	6.84	Relax	Centre	149.07	11.47	31.01	63.48	43.55	392.00
Flexed	6.84	Extending	Centre	149.07	11.47	31.01	63.48	43.55	392.00
Extended	4.56	Relax	Centre	450.15	16.11	45.17	84.23	29.98	311.68
Extended	6.84	Extending	Centre	54.18	3.91	34.91	72.02	45.49	416.86
Extended	4.56	Flexing	Centre	35.07	23.44	41.02	82.52	23.41	221.44
Extended	6.84	Flexing	Centre	288.42	26.37	31.74	59.08	8.15	98.73
Extended	6.84	Relax	Centre	343.57	22.95	31.25	64.70	26.64	254.96
Extended	4.56	Extending	Centre	61.39	15.87	49.32	104.98	45.84	448.11
Extended	6.84	Relax	Left	410.66	25.39	31.74	63.23	28.52	304.16
Extended	6.84	Relax	Right	365.95	42.48	41.99	152.83	45.99	434.07

Subject

N16

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	13.78	0.00	36.87	486.82	16.60	243.68
Flexed	6.84	Flexing	Centre	285.99	33.69	26.61	53.22	7.91	96.66
Flexed	4.56	Relax	Centre	24.84	49.32	41.02	86.43	17.08	290.29
Flexed	4.56	Flexing	Centre	11.11	0.00	7.81	14.89	38.32	344.20
Flexed	6.84	Relax	Centre	6.79	47.12	32.96	65.92	37.97	318.72
Flexed	6.84	Extending	Centre	98.90	18.31	31.98	65.92	36.07	361.27
Extended	4.56	Relax	Centre	155.24	34.67	21.73	41.50	18.20	292.30
Extended	6.84	Extending	Centre	18.94	31.25	31.25	131.84	41.29	431.40
Extended	4.56	Flexing	Centre	19.13	0.00	120.12	505.13	21.47	235.16
Extended	6.84	Flexing	Centre	209.56	51.51	24.90	49.56	13.67	163.20
Extended	6.84	Relax	Centre	139.29	41.50	35.16	85.21	28.29	273.34
Extended	4.56	Extending	Centre	157.85	71.78	43.70	87.89	43.91	422.23
Extended	6.84	Relax	Left	250.38	39.06	33.45	67.63	25.33	285.79
Extended	6.84	Relax	Right	184.66	48.58	35.64	78.37	45.81	497.69

Subject

NI7

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	14.55	0.00	38.82	477.54	22.26	221.84
Flexed	6.84	Flexing	Centre	101.90	8.79	33.69	69.82	16.37	164.36
Flexed	4.56	Relax	Centre	67.43	40.04	24.90	126.46	27.04	254.65
Flexed	4.56	Flexing	Centre	16.53	0.00	53.47	479.49	43.69	402.11
Flexed	6.84	Relax	Centre	38.88	2.93	32.47	116.94	44.91	393.90
Flexed	6.84	Extending	Centre	16.09	9.77	37.11	150.39	45.56	410.18
Extended	4.56	Relax	Centre	192.80	0.00	52.98	93.75	33.49	315.32
Extended	6.84	Extending	Centre	120.00	66.41	33.20	68.85	45.43	451.66
Extended	4.56	Flexing	Centre	9.95	10.50	7.81	15.14	23.37	240.51
Extended	6.84	Flexing	Centre	389.58	75.44	40.04	75.44	17.51	191.81
Extended	6.84	Relax	Centre	389.43	73.97	36.62	76.90	28.64	260.09
Extended	4.56	Extending	Centre	80.76	14.40	109.62	168.21	45.60	471.69
Extended	6.84	Relax	Left	113.64	6.10	39.79	86.67	37.93	353.14
Extended	6.84	Relax	Right	402.54	64.70	36.62	81.79	45.48	404.07

Subject

NI8

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	259.72	57.37	44.68	86.91	18.74	175.20
Flexed	6.84	Flexing	Centre	261.83	26.37	37.35	68.60	12.80	113.99
Flexed	4.56	Relax	Centre	409.27	51.76	55.42	96.19	28.44	237.76
Flexed	4.56	Flexing	Centre	437.09	48.34	37.35	80.81	41.97	335.74
Flexed	6.84	Relax	Centre	441.43	49.56	43.21	97.41	41.83	344.71
Flexed	6.84	Extending	Centre	331.50	31.74	37.84	112.30	42.18	342.16
Extended	4.56	Relax	Centre	118.04	18.31	5.86	11.96	33.99	284.68
Extended	6.84	Extending	Centre	96.99	9.28	90.09	140.87	43.80	351.05
Extended	4.56	Flexing	Centre	248.30	54.93	39.31	79.10	21.52	164.18
Extended	6.84	Flexing	Centre	231.16	50.78	29.79	56.40	14.32	149.22
Extended	6.84	Relax	Centre	85.46	42.97	40.77	95.95	30.14	224.47
Extended	4.56	Extending	Centre	386.32	70.80	40.28	80.32	42.88	340.36
Extended	6.84	Relax	Left	405.52	37.35	36.13	70.31	24.85	215.18
Extended	6.84	Relax	Right	462.88	60.55	40.28	76.66	43.72	380.23

Subject

NI9

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	118.19	40.04	33.69	67.87	21.49	211.58
Flexed	6.84	Flexing	Centre	14.64	0.00	0.00	0.00	5.93	61.47
Flexed	4.56	Relax	Centre	389.14	52.00	36.13	77.64	28.46	278.19
Flexed	4.56	Flexing	Centre	74.35	0.73	62.74	162.84	41.92	369.25
Flexed	6.84	Relax	Centre	123.29	34.67	33.69	125.49	41.96	373.66
Flexed	6.84	Extending	Centre	49.74	40.28	36.13	125.24	41.13	370.22
Extended	4.56	Relax	Centre	195.57	47.12	31.49	56.88	19.21	202.77
Extended	6.84	Extending	Centre	178.88	27.59	59.33	125.00	44.25	412.87
Extended	4.56	Flexing	Centre	252.04	51.76	35.40	73.49	22.36	208.19
Extended	6.84	Flexing	Centre	361.13	40.28	36.13	77.39	9.53	98.58
Extended	6.84	Relax	Centre	426.81	38.09	33.69	67.14	27.56	270.71
Extended	4.56	Extending	Centre	10.78	4.15	7.57	15.87	45.44	423.05
Extended	6.84	Relax	Left	203.51	24.90	25.39	50.54	27.57	259.02
Extended	6.84	Relax	Right	256.63	37.11	38.57	76.42	45.53	435.77

Subject

NI10

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	24.87	4.88	12.94	25.39	21.78	193.50
Flexed	6.84	Flexing	Centre	231.88	22.46	31.49	63.72	5.07	55.50
Flexed	4.56	Relax	Centre	258.66	65.67	46.39	133.30	33.40	317.40
Flexed	4.56	Flexing	Centre	385.35	60.30	39.79	82.28	44.13	381.33
Flexed	6.84	Relax	Centre	206.33	28.08	74.95	162.35	42.03	370.18
Flexed	6.84	Extending	Centre	348.36	63.23	39.06	78.86	40.33	363.86
Extended	4.56	Relax	Centre	375.86	33.94	30.03	60.30	15.77	174.58
Extended	6.84	Extending	Centre	415.89	36.62	98.14	182.37	44.57	476.70
Extended	4.56	Flexing	Centre	99.22	28.56	50.29	106.69	23.14	218.36
Extended	6.84	Flexing	Centre	147.74	6.84	28.32	138.43	5.63	64.75
Extended	6.84	Relax	Centre	244.58	22.46	21.73	136.72	35.37	340.84
Extended	4.56	Extending	Centre	381.71	48.83	37.84	74.71	44.55	381.72
Extended	6.84	Relax	Left	357.51	26.12	45.41	87.65	19.82	219.42
Extended	6.84	Relax	Right	277.70	31.49	98.88	157.23	45.26	548.50

Subject

NI11

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	17.15	0.00	40.77	478.76	23.26	215.08
Flexed	6.84	Flexing	Centre	110.64	10.50	7.57	14.65	19.35	195.22
Flexed	4.56	Relax	Centre	356.21	26.61	33.69	69.09	30.80	302.50
Flexed	4.56	Flexing	Centre	12.28	0.00	33.45	484.86	45.89	416.78
Flexed	6.84	Relax	Centre	28.08	1.71	31.49	63.23	44.87	384.51
Flexed	6.84	Extending	Centre	9.50	10.25	45.17	87.40	45.69	405.09
Extended	4.56	Relax	Centre	259.68	31.98	38.09	70.80	35.26	330.45
Extended	6.84	Extending	Centre	399.70	34.67	34.42	73.49	45.98	485.45
Extended	4.56	Flexing	Centre	335.25	60.79	32.96	68.12	24.34	222.06
Extended	6.84	Flexing	Centre	409.85	39.06	32.96	69.82	16.36	168.36
Extended	6.84	Relax	Centre	400.65	37.84	32.71	69.34	30.02	288.62
Extended	4.56	Extending	Centre	432.25	66.89	34.42	71.78	46.10	494.28
Extended	6.84	Relax	Left	406.06	41.26	33.94	71.04	30.66	322.87
Extended	6.84	Relax	Right	348.78	35.16	40.77	89.60	46.24	526.08

Subject

NI12

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	12.00	0.00	39.31	477.54	20.61	206.87
Flexed	6.84	Flexing	Centre	251.58	11.72	42.97	75.68	10.54	114.82
Flexed	4.56	Relax	Centre	9.63	0.00	12.45	26.12	23.37	220.04
Flexed	4.56	Flexing	Centre	16.87	0.00	46.14	478.27	40.76	381.16
Flexed	6.84	Relax	Centre	32.16	0.00	33.20	194.34	40.54	381.75
Flexed	6.84	Extending	Centre	26.19	6.84	42.48	188.23	41.30	396.15
Extended	4.56	Relax	Centre	360.29	19.78	41.02	78.61	25.16	253.18
Extended	6.84	Extending	Centre	87.21	31.01	35.16	73.97	40.30	377.74
Extended	4.56	Flexing	Centre	156.94	41.99	35.16	70.56	23.50	249.38
Extended	6.84	Flexing	Centre	279.06	36.87	31.74	60.79	16.44	206.56
Extended	6.84	Relax	Centre	46.21	40.28	46.88	169.68	27.97	290.73
Extended	4.56	Extending	Centre	26.99	0.00	53.47	232.91	45.79	444.44
Extended	6.84	Relax	Left	345.81	17.58	56.88	97.41	33.37	366.44
Extended	6.84	Relax	Right	178.61	55.18	31.74	135.01	46.01	490.83

Subject

NI13

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	37.04	27.59	39.79	75.68	22.93	243.31
Flexed	6.84	Flexing	Centre	197.67	31.01	18.07	35.64	16.13	181.54
Flexed	4.56	Relax	Centre	146.37	28.08	37.11	83.25	25.25	279.53
Flexed	4.56	Flexing	Centre	270.51	24.41	32.96	66.65	45.31	467.45
Flexed	6.84	Relax	Centre	57.22	9.28	34.91	71.78	43.13	442.77
Flexed	6.84	Extending	Centre	22.34	14.40	33.69	70.56	45.75	502.42
Extended	4.56	Relax	Centre	301.58	16.60	29.05	64.70	32.27	433.37
Extended	6.84	Extending	Centre	398.83	24.41	80.81	118.16	38.67	448.66
Extended	4.56	Flexing	Centre	77.05	22.46	36.87	74.71	26.46	277.85
Extended	6.84	Flexing	Centre	370.88	22.22	44.68	83.01	13.20	159.91
Extended	6.84	Relax	Centre	409.04	29.54	35.89	75.68	30.28	327.23
Extended	4.56	Extending	Centre	13.57	0.00	16.36	39.55	46.11	597.62
Extended	6.84	Relax	Left	343.57	13.92	36.38	73.73	36.08	392.99
Extended	6.84	Relax	Right	420.72	38.09	40.53	105.71	46.06	618.50

Subject

NI14

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	48.89	0.00	21.24	132.08	24.22	229.66
Flexed	6.84	Flexing	Centre	45.55	17.58	13.92	25.39	12.27	124.40
Flexed	4.56	Relax	Centre	15.87	0.00	25.88	136.23	23.58	223.08
Flexed	4.56	Flexing	Centre	11.08	12.94	27.83	118.65	43.70	446.40
Flexed	6.84	Relax	Centre	8.51	0.00	20.26	39.31	44.04	454.39
Flexed	6.84	Extending	Centre	19.15	9.77	35.64	137.21	44.34	454.04
Extended	4.56	Relax	Centre	54.48	14.89	21.24	62.01	27.47	267.28
Extended	6.84	Extending	Centre	20.86	1.95	46.88	167.48	43.31	428.46
Extended	4.56	Flexing	Centre	12.93	0.00	10.50	22.95	23.17	218.17
Extended	6.84	Flexing	Centre	84.22	11.47	15.14	31.01	7.87	88.86
Extended	6.84	Relax	Centre	41.12	52.73	21.48	47.85	28.74	273.11
Extended	4.56	Extending	Centre	28.89	0.00	62.01	472.41	46.19	470.67
Extended	6.84	Relax	Left	107.21	10.01	20.75	87.40	19.21	207.05
Extended	6.84	Relax	Right	202.18	35.64	75.68	114.26	46.13	467.66

Subject

NI15

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	125.20	59.81	36.38	77.64	26.27	253.78
Flexed	6.84	Flexing	Centre	146.64	18.80	23.93	48.34	8.50	96.12
Flexed	4.56	Relax	Centre	335.57	48.34	41.99	94.48	31.46	322.91
Flexed	4.56	Flexing	Centre	13.97	5.86	9.03	17.58	46.13	499.85
Flexed	6.84	Relax	Centre	57.26	7.08	72.75	174.07	46.05	488.64
Flexed	6.84	Extending	Centre	26.44	0.00	64.94	185.30	46.07	497.90
Extended	4.56	Relax	Centre	364.10	18.80	32.71	67.87	28.77	299.86
Extended	6.84	Extending	Centre	374.51	51.27	37.60	76.42	46.08	545.47
Extended	4.56	Flexing	Centre	402.09	41.99	39.31	84.72	26.02	252.51
Extended	6.84	Flexing	Centre	162.02	10.01	15.14	103.27	11.21	124.08
Extended	6.84	Relax	Centre	415.06	45.90	38.82	79.35	32.48	342.92
Extended	4.56	Extending	Centre	250.73	41.99	36.62	80.32	46.20	519.83
Extended	6.84	Relax	Left	381.31	23.68	28.56	55.91	25.03	277.86
Extended	6.84	Relax	Right	357.22	40.77	41.50	81.54	46.20	596.96

Subject

NI16

Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)
Flexed	4.56	Extending	Centre	18.70	0.00	20.02	39.79	22.41	209.12
Flexed	6.84	Flexing	Centre	318.59	11.96	26.61	50.78	6.20	65.93
Flexed	4.56	Relax	Centre	123.44	27.10	36.13	81.05	29.28	274.40
Flexed	4.56	Flexing	Centre	264.44	49.56	38.09	81.79	40.85	440.34
Flexed	6.84	Relax	Centre	25.55	14.40	34.18	177.73	41.02	439.84
Flexed	6.84	Extending	Centre	37.74	15.14	35.64	80.08	41.30	442.80
Extended	4.56	Relax	Centre	411.07	14.40	33.69	69.09	21.12	219.56
Extended	6.84	Extending	Centre	158.80	17.82	38.57	124.02	40.60	447.02
Extended	4.56	Flexing	Centre	52.27	32.96	37.84	78.61	23.78	220.36
Extended	6.84	Flexing	Centre	399.76	15.87	31.98	65.43	3.65	50.39
Extended	6.84	Relax	Centre	388.65	45.41	35.40	72.75	33.59	341.43
Extended	4.56	Extending	Centre	245.68	57.62	38.33	124.76	39.98	458.74
Extended	6.84	Relax	Left	437.43	25.15	27.34	57.86	16.28	175.46
Extended	6.84	Relax	Right	390.75	30.52	33.20	67.87	34.58	358.60

Subject	NI17									
Initial Angle	Applied Torque	Voluntary activity	Head position	Amplitude (μV)	Latency (ms)	Rise Time (ms)	Duration (ms)	Range of Movement (deg)	Angular velocity (deg/s)	
Flexed	4.56	Extending	Centre	16.01	0.00	105.47	511.47	19.62	187.78	
Flexed	6.84	Flexing	Centre	386.10	19.53	20.02	39.06	10.43	115.42	
Flexed	4.56	Relax	Centre	37.93	53.22	46.63	102.29	31.28	280.35	
Flexed	4.56	Flexing	Centre	43.03	0.00	34.67	147.46	38.59	308.71	
Flexed	6.84	Relax	Centre	11.59	22.22	35.64	70.56	38.13	308.09	
Flexed	6.84	Extending	Centre	20.80	0.00	33.20	68.60	31.37	264.40	
Extended	4.56	Relax	Centre	0.72	0.00	0.00	0.00	32.47	286.64	
Extended	6.84	Extending	Centre	44.18	15.14	37.11	76.90	46.20	452.75	
Extended	4.56	Flexing	Centre	49.18	40.04	42.97	419.92	19.85	213.93	
Extended	6.84	Flexing	Centre	395.05	42.72	27.83	53.71	11.32	139.21	
Extended	6.84	Relax	Centre							

Torque (N.m.) Starting angle	2.28				3.42				4.56				5.7				6.84			
	extended		flexed		extended		flexed		extended		flexed		extended		flexed		extended		flexed	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Parameters																				
Stretch reflex Amplitude (μ V)	61.46	25.33	77.13	22.51	64.32	30.11	57.94	26.37	52.58	28.43	82.94	60.17	69.00	31.38	54.27	17.21	83.39	42.68	63.27	26.62
Stretch reflex Latency (ms)	30.52	24.56	30.91	20.93	26.66	14.87	24.98	17.88	19.36	21.55	26.81	24.26	19.85	16.68	27.06	21.17	16.74	7.58	27.73	25.48
Stretch reflex Rise Time (ms)	31.54	5.67	34.47	3.02	26.61	7.19	45.33	31.92	29.79	12.46	50.33	20.86	33.24	5.15	45.65	22.75	31.91	9.34	40.95	24.33
Stretch reflex Duration (ms)	65.77	11.60	96.48	34.23	66.50	33.21	103.23	40.04	59.40	24.11	114.83	37.85	76.28	21.59	122.19	47.77	100.93	38.56	118.44	46.96

Appendix B

Descriptive Statistics

Descriptive Statistics

	status	Starting Angle	Force Applied	Voluntary Activity	Head Position	Mean	Std. Deviation	N
Stretch Reflex Amplitude μV	non-impaired	90	Low	Relax	Centre	296.27	118.585	14
				Triceps	Centre	245.94	144.581	12
				Biceps	Centre	200.34	136.026	13
			High	Relax	Centre	264.79	158.642	17
					Left	303.74	122.996	17
					Right	314.42	98.941	17
				Triceps	Centre	232.62	155.422	16
				Biceps	Centre	293.77	105.342	16
		120	Low	Relax	Centre	244.11	149.427	16
				Triceps	Centre	148.91	128.948	8
				Biceps	Centre	267.19	115.758	8
			High	Relax	Centre	140.76	140.973	12
				Triceps	Centre	132.74	120.901	11
				Biceps	Centre	243.79	115.547	14
	stroke	90	Low	Relax	Centre	97.8	112.664	11
				Triceps	Centre	205.71	146.357	5
				Biceps	Centre	239.37	141.445	6
			High	Relax	Centre	121.52	83.15	14
					Left	136.26	105.797	11
					Right	124.37	101.059	13
				Triceps	Centre	212.45	166.616	5
				Biceps	Centre	202.81	159.513	7
		120	Low	Relax	Centre	119.3	105.742	12
				Triceps	Centre	205.95	123.708	5
				Biceps	Centre	286.57	137.796	6
			High	Relax	Centre	148.21	127.394	14
				Triceps	Centre	268.38	154.501	5
				Biceps	Centre	255.21	114.554	6
Stretch Reflex Latency ms	non-impaired	90	Low	Relax	Centre	23.5247	10.31125	14
				Triceps	Centre	44.7591	20.13513	12
				Biceps	Centre	43.5885	14.89412	13
			High	Relax	Centre	37.5833	15.91683	17
					Left	27.9182	16.80916	17
					Right	39.7806	13.47412	17
				Triceps	Centre	31.8756	19.38216	16
				Biceps	Centre	34.3323	18.46665	16
		120	Low	Relax	Centre	41.4429	16.53405	16
				Triceps	Centre	42.3584	22.89126	8
				Biceps	Centre	32.6538	22.10354	8
			High	Relax	Centre	21.6471	15.94267	12
				Triceps	Centre	29.0305	19.04687	11
				Biceps	Centre	19.3743	7.46166	14
	stroke	90	Low	Relax	Centre	18.3324	19.97658	11
				Triceps	Centre	25.5377	11.83364	5
				Biceps	Centre	18.717	15.50414	6
			High	Relax	Centre	18.0664	11.72931	14
					Left	15.5141	6.97111	11
					Right	16.3765	9.77409	13
				Triceps	Centre	16.9435	9.64375	5
				Biceps	Centre	14.9629	12.04356	7
		120	Low	Relax	Centre	28.8289	23.94874	12
				Triceps	Centre	30.5671	12.43278	5
				Biceps	Centre	31.2902	29.37132	6
			High	Relax	Centre	23.3503	20.81946	14
				Triceps	Centre	19.0925	15.16656	5
				Biceps	Centre	33.7318	23.73193	6

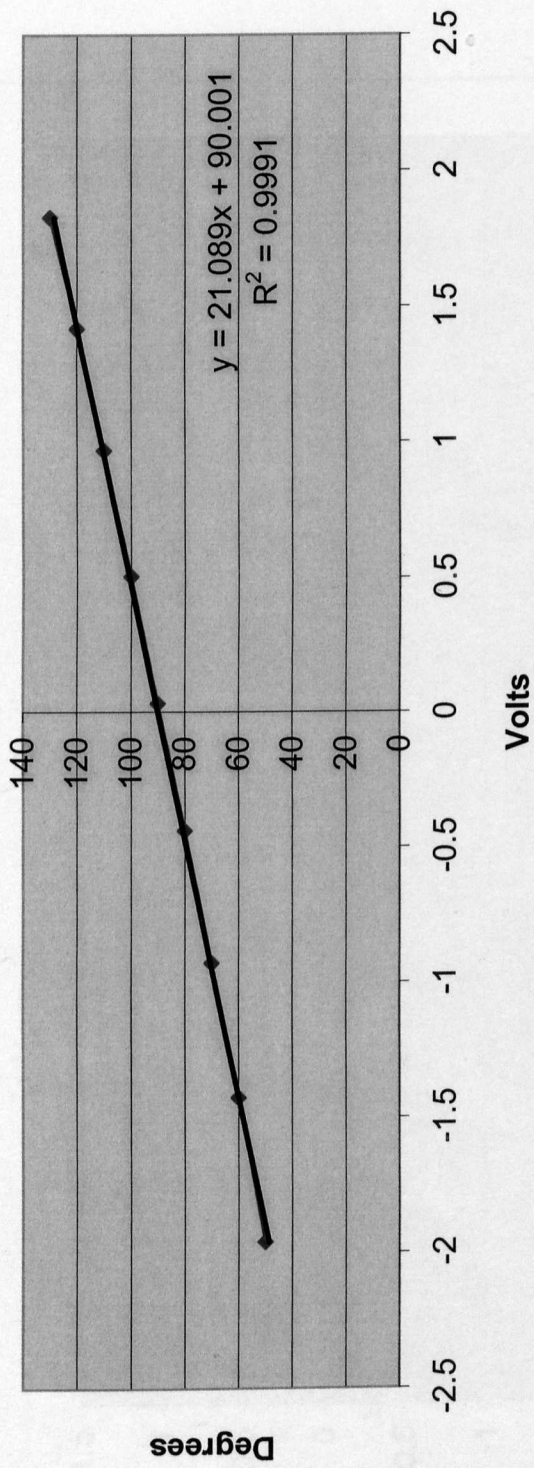
Descriptive Statistics

	status	Starting Angle	Force Applied	Voluntary Activity	Head Position	Mean	Std. Deviation	N
Stretch Reflex Rise Time (ms)	non-impaired	90	Low	Relax	Centre	32.959	9.87395	14
				Triceps	Centre	52.9175	23.13456	12
				Biceps	Centre	39.6447	5.13812	13
			High	Relax	Centre	36.6355	7.75241	17
				Relax	Left	34.0935	14.70528	17
				Relax	Right	49.2733	20.63173	17
				Triceps	Centre	52.597	22.38917	16
				Biceps	Centre	31.4178	8.86501	16
		120	Low	Relax	Centre	37.4298	8.08874	16
				Triceps	Centre	35.0952	9.72609	8
				Biceps	Centre	39.2151	9.93757	8
			High	Relax	Centre	44.8405	16.76052	12
				Triceps	Centre	37.2203	5.43438	11
				Biceps	Centre	28.6516	13.82747	14
	stroke	90	Low	Relax	Centre	34.2684	13.01029	11
				Triceps	Centre	36.3274	17.38593	5
				Biceps	Centre	33.5279	6.03172	6
			High	Relax	Centre	35.7143	12.82554	14
				Relax	Left	32.7145	7.70772	11
				Relax	Right	32.3204	8.47397	13
				Triceps	Centre	28.7589	6.12272	5
				Biceps	Centre	35.1565	7.35053	7
		120	Low	Relax	Centre	44.8816	15.31791	12
				Triceps	Centre	36.4259	19.18905	5
				Biceps	Centre	33.977	4.14876	6
			High	Relax	Centre	41.6608	22.38233	14
				Triceps	Centre	30.7617	16.15579	5
				Biceps	Centre	37.842	8.99186	6
Stretch Reflex Duration (ms)	non-impaired	90	Low	Relax	Centre	65.5169	17.32237	14
				Triceps	Centre	107.5439	36.59675	12
				Biceps	Centre	124.399	109.52792	13
			High	Relax	Centre	90.1741	31.12984	17
				Relax	Left	67.7418	23.19129	17
				Relax	Right	115.1051	48.23393	17
				Triceps	Centre	107.605	37.70262	16
				Biceps	Centre	71.3501	25.13495	16
		120	Low	Relax	Centre	88.5925	25.32871	16
				Triceps	Centre	71.7163	20.9684	8
				Biceps	Centre	103.4241	55.78553	8
			High	Relax	Centre	131.1239	61.58123	12
				Triceps	Centre	112.2825	55.30143	11
				Biceps	Centre	54.2864	23.25971	14
	stroke	90	Low	Relax	Centre	75.0619	33.27229	11
				Triceps	Centre	64.9417	17.50689	5
				Biceps	Centre	100.1377	30.76949	6
			High	Relax	Centre	96.7494	38.87914	14
				Relax	Left	94.1049	52.19495	11
				Relax	Right	89.7683	39.48854	13
				Triceps	Centre	64.6492	11.5075	5
				Biceps	Centre	94.3429	29.96565	7
		120	Low	Relax	Centre	131.5716	77.05935	12
				Triceps	Centre	91.6994	76.14437	5
				Biceps	Centre	125.9767	45.27405	6
			High	Relax	Centre	114.7462	59.35409	14
				Triceps	Centre	73.4378	31.43154	5
				Biceps	Centre	83.8223	22.13418	6

Appendix C

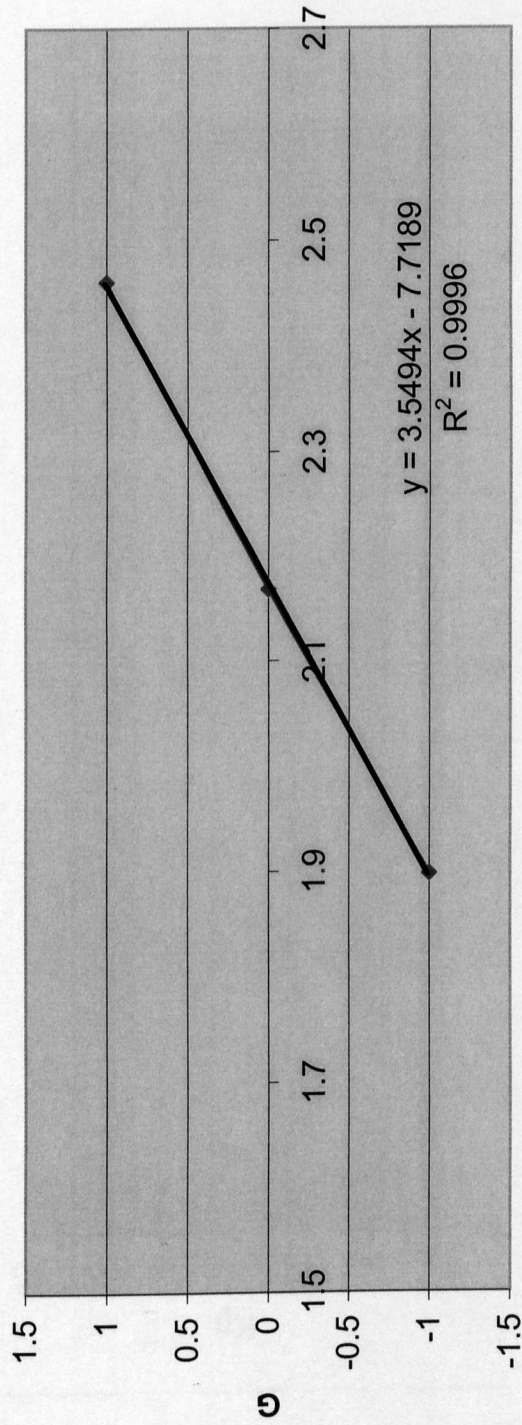
Sensor calibration

Potentiometer Calibration



Angle (deg)	Voltage (V)	
	Mean	SD
50	-1.958917	0.002519
60	-1.425378	0.002565
70	-0.929408	0.002551
80	-0.440011	0.002584
90	0.031995	0.002524
100	0.505219	0.002574
110	0.967665	0.002558
120	1.419548	0.002549
130	1.828856	0.002544

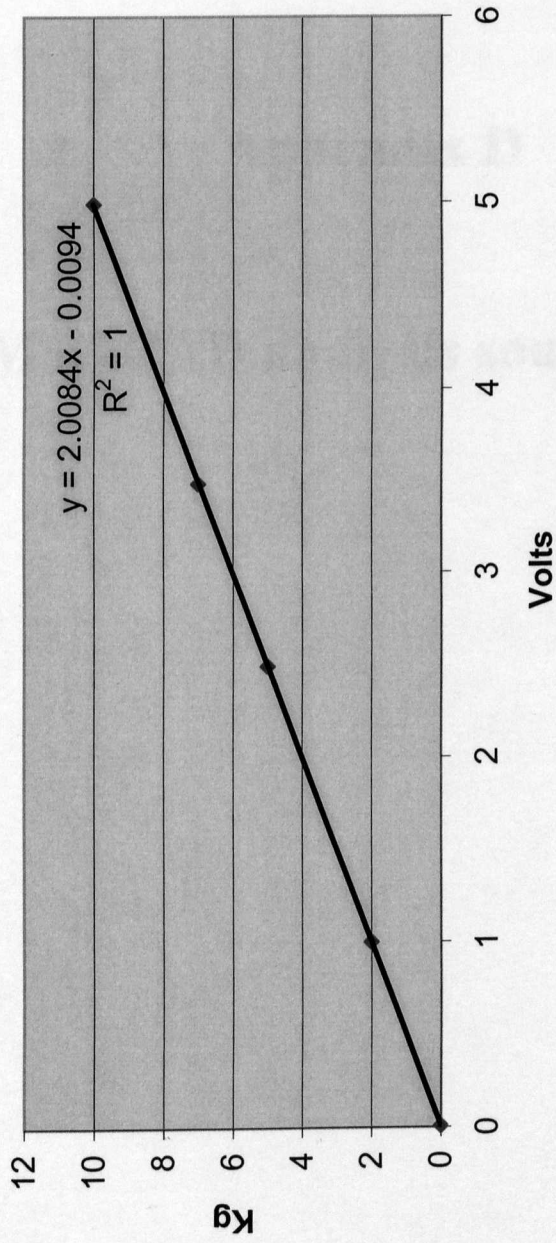
Accelerometer calibration



Volts

G	Voltage	
	Mean	SD
-1	1.896568888	0.0137034
0	2.167841857	0.013929153
1	2.459799083	0.013276915

Load Cell Calibration



Mass (kg)	Voltage (V)	
	Mean	SD
0	0.0064096	0.00250634
2	1.00188651	0.00200675
5	2.49458165	0.0022743
7	3.47980845	0.00225709
10	4.99026679	0.0020082

Appendix D

MathCAD analysis source code

Smoothing frequency $FC2 := 20$

Enter sample frequency $SF := 4096$

Sensor linearization

Loadcell (Newton)

slopeforce := 19.8022701

interceptforce := -0.2682097

Potentiometer (degrees)

slopepot := 21.0886335

interceptpot := 90.0010110

Accelerometer (G)

slopeacc := 3.54935034

interceptacc := -7.71890212

Electrogoniometer

slope := 41.7277364

intercept := 3.70906076

Filters

50 Hz Filter

fcl := 48

fch := 52

Order := 8

Order2 := 20

analogb := butter(Order)

analogb2 := butter(Order2)

filtb := iirstop $\left(\text{analogb}, \frac{fcl}{SF}, \frac{fch}{SF}\right)$

filtb2 := bandstop $\left(\frac{fcl}{SF}, \frac{fch}{SF}, \text{Order}\right)$

Bandpass filter

fcl1 := 20

fch1 := 45

filtb2 := iirpass $\left(\text{analogb}, \frac{fcl1}{SF}, \frac{fch1}{SF}\right)$

filtb2b := bandpass $\left(\frac{fcl1}{SF}, \frac{fch1}{SF}, \text{Order}\right)$

Lowpass filter (Smoothing)

f1 := 15

filtc := iirlow $\left(\text{analogb}, \frac{f1}{SF}\right)$

filtc2 := lowpass $\left(\frac{f1}{SF}, \text{Order}\right)$

Highpass filter (acceleration offset)

f1h := 10

filtacc := iirhigh $\left(\text{analogb2}, \frac{f1h}{SF}\right)$

$$\text{filtacc2} := \text{highpass}\left(\frac{f_h}{\text{SF}}, \text{Order}, 4\right)$$

Standard deviation

$$\text{SD}(x) := \text{stdev}(x) \cdot \sqrt{\frac{\text{length}(x)}{\text{length}(x) - 1}}$$

Range limits

$$\begin{aligned} \text{rangepos}(x) &:= (\text{mean}(x) + 6 \text{SD}(x)) \\ \text{rangeneg}(x) &:= (\text{mean}(x) - 6 \text{SD}(x)) \\ \text{rangeposb}(x) &:= (\text{mean}(x) + 3 \text{SD}(x)) \\ \text{rangenegb}(x) &:= (\text{mean}(x) - 3 \text{SD}(x)) \end{aligned}$$

Enter patient code $\sqrt{A := \text{PSI}a}$

Algorithms

Root Mean Square algorithm

$$\begin{aligned} \text{RMSFS}(\text{signal}) := & \begin{array}{|l} \text{SFD} \leftarrow \text{signal}^2 \\ \text{MSFD} \leftarrow \text{movavg}(\text{SFD}, 11) \\ \text{RMSFD} \leftarrow \sqrt{\text{MSFD}} \end{array} \end{aligned}$$

Numerical integration algorithms

Graphic

$$\begin{aligned} \text{graphicintegral}(\text{signal}) := & \begin{array}{|l} \text{area} \leftarrow 0 \\ \text{for } i \in 2.. \text{length}(\text{signal}) - 3 \\ \quad \text{partial_area} \leftarrow \text{signal}_i \\ \quad \text{partial_area2} \leftarrow \frac{|\text{signal}_{i+1} - \text{signal}_i|}{2} \\ \quad \text{area}_i \leftarrow \text{partial_area} + \text{partial_area2} \\ \quad \text{area} \leftarrow \text{area}_i + \text{area} \\ \text{area} \end{array} \end{aligned}$$

Area under the curve

$$\begin{aligned} \text{integral}(\text{signal}) := & \begin{array}{|l} \text{for } i \in 1.. \text{length}(\text{signal}) - 3 \\ \quad \text{partial_area2} \leftarrow \frac{|\text{signal}_{i+1} - \text{signal}_i|}{2} \\ \quad \text{partial_area} \leftarrow \text{signal}_i \text{ if } \text{signal}_i < \text{signal}_{i+1} \\ \quad \text{partial_area} \leftarrow \text{signal}_{i+1} \text{ otherwise} \\ \quad \text{area}_i \leftarrow \text{partial_area} + \text{partial_area2} \\ \sum \text{area} \end{array} \end{aligned}$$

Numerical differentiation algorithm

```
graphicdifferential(signal) :=
  for h ∈ 1..length(signal) - 2
    slope_h ← (signal_{h+1} - signal_{h-1}) / ((1/SF) · 2)
  slope
```

Signal rectification algorithm

```
rectification(signal) :=
  for i ∈ 0..length(signal) - 1
    R_i ← |signal_i|
  R
```

Starting and ending points algorithms

```
prestart(sig, stpnt) := for i ∈ 0..length(sig) - 1
  start ← i if (sig_i = stpnt)
preend(sig, endpnt) := for i ∈ 0..length(sig) - 1
  end ← i if (sig_i = endpnt)
start(sig, stpnt) := for i ∈ 0..length(sig) - 1
  start ← i if (sig_i ≥ stpnt)
start2(sig, stpnt) := for i ∈ 0..length(sig) - 1
  start ← i if sig_i ≤ stpnt
end(sig, endpnt) := for i ∈ length(sig) - 1..0
  end ← i if sig_i ≤ endpnt
end2(sig, endpnt) := for i ∈ length(sig) - 1..0
  end ← i if sig_i ≥ endpnt
samp(sig, endpnt) := for i ∈ 0..length(sig) - 1
  end ← i if sig_i = endpnt
```

Signal differentiation

First Differential

fstdiff(sig) := $\frac{\text{graphicdifferential(sig)}}{100}$

Second Differential

scndiff(sig) := $\frac{\text{graphicdifferential(fstdiff(sig))}}{100}$

Signal selection, linearisation and rounding algorithms

Signal selection

$\text{signalselection}(\text{file}, \text{col}) :=$ $\left| \begin{array}{l} \text{for } i \in 0.. \text{length}(\text{file}) - 1 \\ \quad \text{signal}_i \leftarrow (\text{file}_i)^{\langle \text{col} \rangle} \\ \text{signal} \end{array} \right|$

Signal linearisation

$\text{lin}(\text{signal}, \text{slope}, \text{intercept}) :=$ $\left| \begin{array}{l} \text{for } i \in 0.. \text{length}(\text{signal}) - 1 \\ \quad \text{signal}_i \leftarrow (\text{signal}_i \cdot \text{slope}) + \text{intercept} \\ \text{signal} \end{array} \right|$

Rounding

$\text{rounding}(\text{signal}, \text{dec}) :=$ $\left| \begin{array}{l} \text{for } i \in 0.. \text{length}(\text{signal}) - 1 \\ \quad \text{signal}_i \leftarrow \text{round}(\text{signal}_i, \text{dec}) \\ \text{signal} \end{array} \right|$

Finite and infinite impulse response filters

Finite (fir) and Infinite (iir) impulse response filter

Low pass

$\text{firlp}(\text{signal}, \text{fc}, \text{order}) :=$ $\left| \begin{array}{l} l \leftarrow \text{lowpass}\left(\frac{\text{fc}}{\text{SF}}, \text{order}\right) \\ \text{for } i \in 0.. \text{length}(\text{signal}) - 1 \\ \quad \text{signal}_i \leftarrow \text{convol}(\text{signal}_i, l) \\ \text{signal} \end{array} \right|$

$\text{iirlp}(\text{signal}, \text{fc}, \text{order}) :=$ $\left| \begin{array}{l} \text{filter} \leftarrow \text{butter}(\text{order}) \\ l \leftarrow \text{iirlow}\left(\text{filter}, \frac{\text{fc}}{\text{SF}}\right) \\ \text{for } i \in 0.. \text{length}(\text{signal}) - 1 \\ \quad \text{signal}_i \leftarrow \text{filtfil}(\text{signal}_i, l) \\ \text{signal} \end{array} \right|$

High pass

$\text{iirhp}(\text{signal}, \text{fc}, \text{order}) :=$ $\left| \begin{array}{l} \text{filter} \leftarrow \text{butter}(\text{order}) \\ h \leftarrow \text{iirhigh}\left(\text{filter}, \frac{\text{fc}}{\text{SF}}\right) \\ \text{for } i \in 0.. \text{length}(\text{signal}) - 1 \\ \quad \text{signal}_i \leftarrow \text{filtfil}(\text{signal}_i, h) \\ \text{signal} \end{array} \right|$

```

firhp(signal, fc, order) :=
  h ← highpass( $\frac{fc}{SF}$ , order)
  for i ∈ 0..length(signal) - 1
    signali ← convol(signali, h)
  signal

```

Band pass

```

firbp(signal, fl, fh, order) :=
  bp ← bandpass( $\frac{fl}{SF}, \frac{fh}{SF}$ , order)
  for i ∈ 0..length(signal) - 1
    signali ← convol(signali, bp)
  signal

iirbp(signal, fl, fh, order) :=
  filter ← butter(order)
  bp ← iirpass( $\frac{fl}{SF}, \frac{fh}{SF}$ , filter)
  for i ∈ 0..length(signal) - 1
    signali ← filtfil(signali, bp)
  signal

```

Stop band

```

firbs(signal, fl, fh, order) :=
  bs ← bandstop( $\frac{fl}{SF}, \frac{fh}{SF}$ , order)
  gain ← gain( $\frac{fh-fl}{SF}, bs$ )
  for i ∈ 0..length(signal) - 1
    signali ← fftfil(signali, bs)
  signal·gain

iirbs(signal, fl, fh, order) :=
  filter ← butter(order)
  bs ← iirstop( $\frac{fl}{SF}, \frac{fh}{SF}$ , filter)
  for i ∈ 0..length(signal) - 1
    signali ← filtfil(signali, bs)
  signal

```

Signal acquisition

```
file:= for i ∈ 0..length(A) - 1
      | filei ← READPRN(Ai)
      | file
```

Signal Selection

```
Time := signalselection (file, 0)
Position := signalselection (file, 3)
Biceps := signalselection (file, 1)
Force := signalselection (file, 4)
Triceps := signalselection (file, 2)
Acceleration := signalselection (file, 5)
FootSwitch := signalselection (file, 7)
Angle := signalselection (file, 6)
```

Linearisation

```
Position := lin(Position, slope, intercept)
Force := lin(Force, slopeforce, intercept)
Acceleration := lin(Acceleration, slopeacc, interceptacc)
Angle := lin(Angle, slopepot, interceptpot)
```

(compensating the alignment with the centre of rotation

```
Acceleration := for i ∈ 0..length(file) - 1
                | Accelerationi ←  $\frac{\text{Acceleration}_i \cdot 9.807}{0.16 \cdot 9}$ 
                | Acceleration
```

```
Force := for i ∈ 0..length(file) - 1
        | Forcei ← Forcei · 16
        | Force
```

Signal Filtering

Filtering

```
FC := 50
Order := 20
FAngle := iirlp(Angle, FC, Order)
FForce := iirlp(Force, FC, Order)
FAcceleration := iirlp(Acceleration, FC, Order)
FPosition := iirlp(Position, FC, Order)
```

```

FFootSwitch := iirlp(FootSwitch,FC,Order)
FBiceps := Biceps
FTriceps := Triceps

```

Filter gain

```

gainiirlp(signal,fc,order) :=
    filter ← butter(order)
    l ← iirlow( filter,  $\frac{fc}{SF}$  )
    for i ∈ 0..length(signal) - 1
        signali ← gain( l,  $\frac{fc}{SF}$  )
    signal

```

Biceps and Triceps Rectification

```

RBiceps := rectification(FBiceps)
RTriceps := rectification(FTriceps)

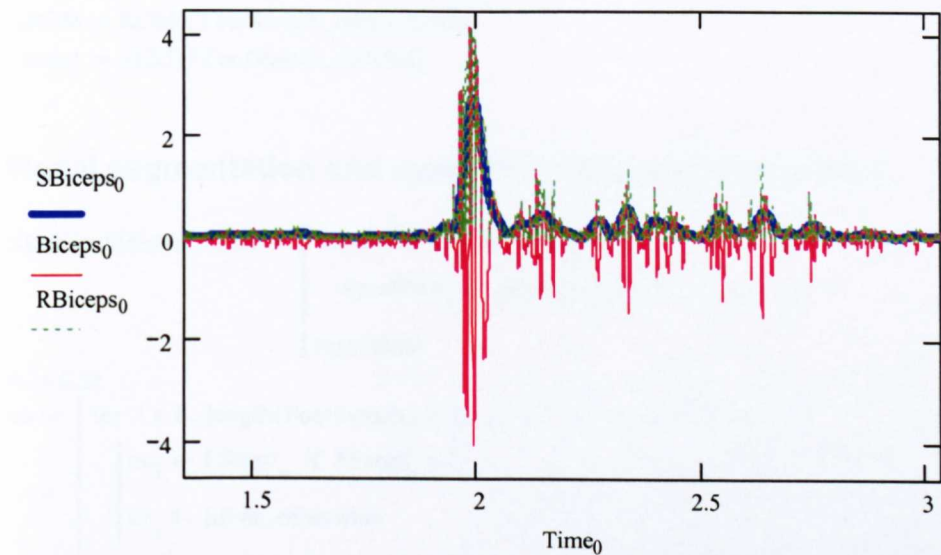
```

Biceps and Triceps envelope

```

SBiceps := iirlp(RBiceps,FC2,Order)
SBicepsgain := gainiirlp(RBiceps,FC2,Order)

```



```

STriceps := iirlp(RTriceps,FC2,Order)
STricepsgain := gainiirlp(RTriceps,FC2,Order)

```

Maximum and Minimums, Sample and Range

```

maxsignal(signal) :=
    for i ∈ 0..length(signal) - 1
        maxsignali ← max( signali )
    maxsignal
ROM(signal,mx,mn) :=
    for i ∈ 0..length(signal) - 1
        ROM ← mx - mn
    ROM

```

```

minsignal(signal) := for i ∈ 0.. length(signal) - 1
                     | minsignali ← min(signali)
                     | minsignal

signalsampl(signal, value) := for i ∈ 0.. length(FFootSwitch) - 1
                              | maxsignalsampli ← samp(signali, valuei)
                              | maxsignalsampl

STRT(signal, ROM) := for j ∈ 0.. length(signal) - 1
                    | for i ∈ length(signalj) - 1.. 0
                    | | STRTj ← i - 1 if (signalj)i ≥  $\frac{ROM_j}{2}$ 
                    | | STRTj ←  $\frac{\text{length}(\text{signal}_j)}{2}$  if  $ROM_j \leq 3$ 
                    | STRT

```

Foot Switch analysis

```

maxfs := maxsignal(FFootSwitch)
minfs := minsignal(FFootSwitch)
maxfssampl := signalsampl(FFootSwitch, maxfs)
minfssampl := signalsampl(FFootSwitch, minfs)
fsROM := ROM(FFootSwitch, maxfs, minfs)
FSstart := STRT(FFootSwitch, fsROM)

```

Signal segmentation and synchronization with Footswitch

```

signalfract(signal, ll, hl) := for i ∈ 0.. length(signal) - 1
                              | signalfracti ← submatrix(signali, lli, hli, 0, 0)
                              | signalfract

```

```

fs := 0.25
no := for i ∈ 0.. length(FootSwitch) - 1
      | noi ← FSstarti if FSstarti > 0
      | noi ← SF·fs otherwise
      | no
b := for i ∈ 0.. length(FootSwitch) - 1
    | bi ← round(noi + .69SF)
    | b
a := for i ∈ 0.. length(FootSwitch) - 1
    | ai ← round(noi - SF·fs)
    | a
i := 0.. length(FAnglei)

```

```

SBic := signalfract(SBiceps,a,b)
Bic1 := signalfract(FBiceps,a,b)
STric := signalfract(STriceps,a,b)
Tric1 := signalfract(FTriceps,a,b)
Bic := signalfract(RBiceps,a,b)
SAcc := signalfract(FAcceleration,a,b)
Tric := signalfract(RTriceps,a,b)
SFor := signalfract(FForce,a,b)
STim := | for i ∈ 0..length(Time) - 1
        |   STimi ← submatrix(Timei,0,bi - ai,0,0)
        | STim

```

```

SPos := signalfract(FPosition,a,b)
SAng := signalfract(FAngle,a,b)
SFS := signalfract(FFootSwitch,a,b)

```

Pre-perturbation values

```

silPos := signalfract(FPosition,a,no)
silRBic := signalfract(RBiceps,a,no)
silAcc := signalfract(FAcceleration,a,no)
silRTric := signalfract(RTriceps,a,no)
silFor := signalfract(FForce,a,no)
silBic := signalfract(SBiceps,a,no)
silAng := signalfract(FAngle,a,no)
silTric := signalfract(STriceps,a,no)
silsignavg(signal) := | for i ∈ 0..length(signal) - 1
                     |   silsignalavgi ← mean(signali)
                     | silsignalavg

```

```

silTim := signalfract(Time,a,no)
silsignal(signal1,signal2) := | for i ∈ 0..length(signal1) - 1
                             |   silsignali ← submatrix(signal1i - signal2i,ai,noi,0,0)
                             | silsignal
Ssignal(signal1,signal2) := | for i ∈ 0..length(signal1) - 1
                           |   SAngi ← (signal1i - signal2i)
                           | SAng

```

```

silAngavg := silsignavg(silAng)
silAccavg := silsignavg(silAcc)
silAng := silsignal(FAngle,silAngavg)
silAcc := silsignal(FAcceleration,silAccavg)
SAng := Ssignal(SAng,silAngavg)
SAcc := Ssignal(SAcc,silAccavg)

```

Raw EMG silent range

```

Rawrange(signal) := | for i ∈ 0..length(signal) - 1
                    |   Rawrangesignali ← max(signali)
                    | Rawrangesignal

```

```

RBicrangepos := Rawrange(silRBic)
RTricrangepos := Rawrange(silTric)

```


Silent range

```

silentrangepos3sd (signal) := for i ∈ 0.. length(signal) - 1
                                silentranglei ← rangeposb (signali)
                                silentrangle
silentrangeneg3sd (signal) := for i ∈ 0.. length(signal) - 1
                                silentranglei ← rangenegb (signali)
                                silentrangle
silentrangepos6sd (signal) := for i ∈ 0.. length(signal) - 1
                                silentranglei ← rangepos (signali)
                                silentrangle
silentrangeneg6sd (signal) := for i ∈ 0.. length(signal) - 1
                                silentranglei ← rangeneg (signali)
                                silentrangle

```

Bicrange_{pos} := silentrangepos3sd (silBic)

Bicrange_{neg} := silentrangeneg3sd (silBic)

Tricrange_{pos} := silentrangepos3sd (silTric)

Tricrange_{neg} := silentrangeneg3sd (silTric)

STric := for i ∈ 0.. length(STriceps) - 1
 STric_i ← STric_i - Tricrange_{pos}_i

SBic := STric
 for i ∈ 0.. length(SBiceps) - 1
 SBic_i ← SBic_i - Bicrange_{pos}_i
 SBic

Accrangeneg := silentrangeneg6sd (silAcc)

Posrange_{pos} := silentrangepos6sd (silPos)

Forrange_{pos} := silentrangepos6sd (silFor)

Posrange_{neg} := silentrangeneg6sd (silPos)

Forrange_{neg} := silentrangeneg6sd (silFor)

Accrange_{pos} := silentrangepos6sd (silAcc)

Angrange_{pos} := 0.2⁴

Angrange_{neg} := -0.2⁴

Accrange_{pos}₀ = 8.103

Accrange_{neg}₀ = -8.103

Signal differentiation

```

fdif(signal) := for i ∈ 0.. length(signal) - 1
                  fdifi ← fstdiff(signali) if length(signal) > 0
                  fdifi ←  $\begin{pmatrix} 0 \\ 0 \end{pmatrix}$  otherwise
                  fdif

```

```

sdif(signal) :=
  for i ∈ 0..length(signal) - 1
    | sdifi ← scndiff(signali) if length(signal) > 0
    | sdifi ←  $\begin{pmatrix} 0 \\ 0 \end{pmatrix}$  otherwise
  | sdif

```

fdifang := fdif(SAng)

sdifang := sdif(SAng)

fdifang := iirlp $\left(\text{fdifang}, \frac{\text{FC}}{3}, \text{Order}\right)$

sdifang := iirlp $\left(\text{sdifang}, \frac{\text{FC}}{3}, \text{Order}\right)$

maximums and minimums detectors

```

minimum(signal, fdif, sdif) :=
  for i ∈ 0..length(signal) - 1
    | j ← 0
    | for n ∈ 10..length(sdifi) - 10
      | if  $\left[ \left( \text{fdif}_i \right)_{n-3} < 0 \wedge \left( \text{fdif}_i \right)_{n-4} < 0 \wedge \left( \text{fdif}_i \right)_{n-5} < 0 \wedge \left( \text{fdif}_i \right)_{n-6} < 0 \wedge \left( \text{fdif}_i \right)_n > 0 \wedge \left( \text{fdif}_i \right)_{n+3} > 0 \wedge \left( \text{fdif}_i \right)_{n+4} > 0 \wedge \left( \text{fdif}_i \right)_{n+5} > 0 \wedge \left( \text{fdif}_i \right)_{n+6} > 0 \right]$ 
        | minimumsj,i ← n
        | j ← j + 1
        | minimums
    | minimums

```

```

maximum(signal, fdif, sdif) :=
  for i ∈ 0..length(signal) - 1
    | j ← 0
    | for n ∈ 10..length(sdifi) - 10
      | if  $\left[ \left( \text{fdif}_i \right)_{n-3} > 0 \wedge \left( \text{fdif}_i \right)_{n-4} > 0 \wedge \left( \text{fdif}_i \right)_{n-5} > 0 \wedge \left( \text{fdif}_i \right)_{n-6} > 0 \wedge \left( \text{fdif}_i \right)_n \leq 0 \wedge \left( \text{fdif}_i \right)_{n+3} < 0 \wedge \left( \text{fdif}_i \right)_{n+4} < 0 \wedge \left( \text{fdif}_i \right)_{n+5} < 0 \wedge \left( \text{fdif}_i \right)_{n+6} < 0 \right]$ 
        | maximumsj,i ← n
        | j ← j + 1
        | maximums
    | maximums

```

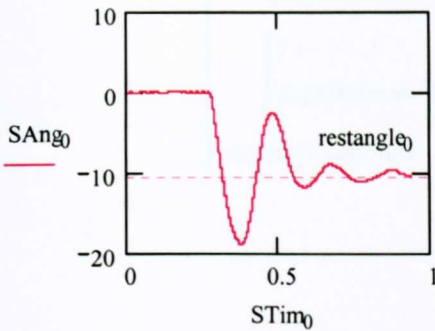
Angle maximums and minimums

angmaximums := maximum(SAng, fdifang, sdifang)

angminimums := minimum(SAng, fdifang, sdifang)

Resting angle (steady state estimation)

```
restangle :=
  k ← 1
  for i ∈ 0..length(file) - 1
    for j ∈ length(SAngi) - 1..length(SAngi) - 100
      restj-(length(SAngi)-100),i ← (SAngi)j
      restanglei ← mean(rest(i))
  restangle
```



```
restanglesd :=
  k ← 1
  for i ∈ 0..length(file) - 1
    for j ∈ length(SAngi) - 1..length(SAngi) - 100
      restj-(length(SAngi)-100),i ← (SAngi)j
      restanglei ← SD(rest(i))
  restangle
```

```
simun := for k ∈ 0..length(SAng) - 1
  j ← 0
  x ← length(angminimums(ψ)) if length(angmaximums(ψ)) > length(angminimums(ψ))
  x ← length(angmaximums(ψ)) otherwise
  for i ∈ 0..x - 1
    if [((SAngk)(angmaximums(ψ))i < Angrangeneg] ∧ [((SAngk)(angmaximums(ψ))i < Angrangepos] ∨ [((SAngk)(angmaximums(ψ))i > Angrangeneg - 0 ∧ (SAngk)(angmaximums(ψ))i > Angrangepos] ∧ [(angmaximums(ψ))i > [(angmaximums(ψ))i-1 + 5] ∧ (angmaximums(ψ))i < [(angmaximums(ψ))i-1 - 5]]
      angmaximumsj,k ← (angmaximums(ψ))i
      j ← j + 1
    otherwise
      j ← j
      angmaximumsj,k ← 0
  angmaximums
```

```

angmaximumval:= for k ∈ 0..rows(angmaximum) - 2
  for i ∈ 0..cols(angmaximum) - 1
    angmaximumvalk,i ← (SAngi)(angmaximumk,i)
  angmaximumval
angmaximumval
angmaximumcount:= for i ∈ 0..length(SAcc) - 1
  j ← 0
  for m ∈ 0..rows(angmaximum⟨i⟩) - 1
    if (angmaximum⟨i⟩)m ≠ 0
      j ← j + 1
      angmaximumcounti ← j
  angmaximumcount

```

```

angminimum = for k ∈ 0..length(SAng) - 1
  j ← 0
  x ← length(angmaximum(ω)) if length(angmaximum(ω)) < length(angminimum(ω))
  x ← length(angminimum(ω)) otherwise
  for i ∈ 0..(x - 1)
    if (angminimum(ω))i > 0 ∧ [((SAngk)(angminimum(ω))i ≤ Angrangelow) ∧ ((SAngk)(angminimum(ω))i < Angrangepos)] ∨ ((SAngk)(angminimum(ω))i > Angrangelow ∧ ((SAngk)(angminimum(ω))i > Angrangepos)] ∧ (angminimum(ω))i > [(angminimum(ω))j-1 + 1]
      angminimumj,k ← (angminimum(ω))i
      j ← j + 1
    otherwise
      j ← j
      angminimumj,k ← 0
  angminimum

```

```

angminimumval:= for k ∈ 0..rows(angminimum) - 1
  for i ∈ 0..cols(angminimum) - 1
    angminimumvalk,i ← (SAngi)(angminimumk,i)
  angminimumval
angminimumval

```

```

angminimumcount:= for i ∈ 0..length(SAcc) - 1
    j ← 0
    for m ∈ 0..rows(angminimum⟨i⟩) - 1
        if (angminimum⟨i⟩)m ≠ 0
            j ← j + 1
            angminimumcounti ← j
    angminimumcount
maxisignal(signal, point) := for i ∈ 0..length(signal) - 1
    maxsignali ← (signal)i[(point⟨i⟩)0]
    maxsignal
minisignal(signal, point) := for i ∈ 0..length(signal) - 1
    minsignali ← (signal)i[(point⟨i⟩)0]
    minsignal

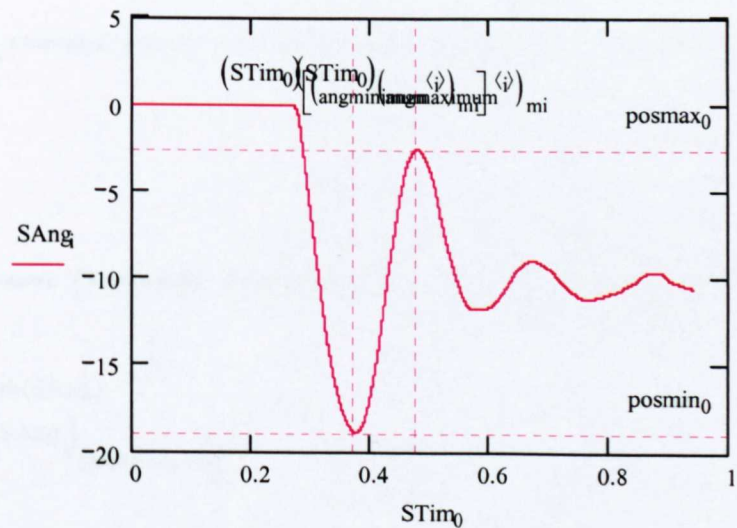
```

Position analysis

```

firstswing := minisignal(SAng)
startpos := maxsignal(SAng)
posmin := minisignal(SAng, angminimum)
posmax := maxsignal(SAng, angmaximum)
i := 0
minsampl := signalsampl(SAng, posmin)
maxsampl := signalsampl(SAng, posmax)
mi := 0

```



First Maximum and minimum detector

```

movmp := for i ∈ 0..length(SAng) - 1
        | movmpi ← posmini if minsampi < maxsampi
        | movmpi ← posmaxi otherwise
        | movmp
        | minsampmi
mini :=  $\frac{\text{minsamp}_{mi}}{100SF}$ 

movmaxpeak := for i ∈ 0..length(SAng) - 1
              | movendi ← minsampi if minsampi < maxsampi
              | movendi ← maxsampi otherwise
              | movend
              | maxsampmi
maxi :=  $\frac{\text{maxsamp}_{mi}}{100SF}$ 

movstartang := for i ∈ 0..length(SAng) - 1
               | for j ∈ movmaxpeak...0
               | movstarti ← (SAng)i,j if  $\left[ \left[ (SAng)_{i,j} < \text{Angrangeneg} \wedge \text{maxsamp}_i > \text{minsamp}_i \right] \vee \left[ (SAng)_{i,j} > \text{Angrangepos} \wedge \text{maxsamp}_i < \text{minsamp}_i \right] \right] \wedge \text{movmaxpeak}_i \neq 0$ 
               | movstarti ← 0 if  $(\text{movmaxpeak}_i \leq 1 \wedge \text{movmaxpeak}_i \geq 0) \vee (\text{movmaxpeak}_i \leq 0 \wedge \text{movmaxpeak}_i \geq -1)$ 
               | movstart

movstart := for i ∈ 0..length(SAng) - 1
            | for j ∈ movmaxpeak...0
            | movstarti ← j + 1 if  $\left[ (SAng)_{i,j} < \text{Angrangeneg} \wedge \text{maxsamp}_i > \text{minsamp}_i \right] \vee \left[ (SAng)_{i,j} > \text{Angrangepos} \wedge \text{maxsamp}_i < \text{minsamp}_i \right]$ 
            | movstarti ← movstarti-1 if  $(\text{movmaxpeak}_i \leq 1 \wedge \text{movmaxpeak}_i \geq 0) \vee (\text{movmaxpeak}_i \leq 0 \wedge \text{movmaxpeak}_i \geq -1)$ 
            | movstart

movendang := for i ∈ 0..length(SAng) - 1
             | for j ∈ movmaxpeak...0
             | movendangi ← (SAng)i,j if  $\left[ (SAng)_{i,j} \leq \text{Angrangeneg} - \left[ .99(\text{Angrangeneg} - \text{movmp}_i) \right] \wedge \text{maxsamp}_i > \text{minsamp}_i \right] \vee \left[ (SAng)_{i,j} \geq \text{Angrangepos} + \left[ .99(\text{movmp}_i - \text{Angrangepos}) \right] \wedge \text{maxsamp}_i < \text{minsamp}_i \right]$ 
             | movendangi ← 0 if movmaxpeaki = 0
             | movendang

movend := for i ∈ 0..length(SAng) - 1
          | for j ∈ movmaxpeak...0
          | movendi ← j + 1 if  $\left[ (SAng)_{i,j} \leq \text{Angrangeneg} - \left[ .99(\text{Angrangeneg} - \text{movmp}_i) \right] \wedge \text{maxsamp}_i > \text{minsamp}_i \right] \vee \left[ (SAng)_{i,j} \geq \text{Angrangepos} + \left[ .99(\text{movmp}_i - \text{Angrangepos}) \right] \wedge \text{maxsamp}_i < \text{minsamp}_i \right]$ 
          | movendi ← 0 if movmaxpeaki = 0
          | movend

movmaxpeakang := for i ∈ 0..length(SAng) - 1
                 | movendi ← (SAng)i,(\text{movmaxpeak}_i)
                 | movend
    
```



```

presamplstart := for i ∈ 0..length(SAng) - 1
                  presamplstarti ← prestart(SAngi, movstartangi)
                  presamplstart
presamplmaxpeak := for i ∈ 0..length(SAng) - 1
                    presamplendi ← preend(SAngi, movmaxpeakangi)
                    presamplend
RL1 := for i ∈ 0..length(SAng) - 1
        RL1i ← movstarti - 10
        RL1

```

phase shift

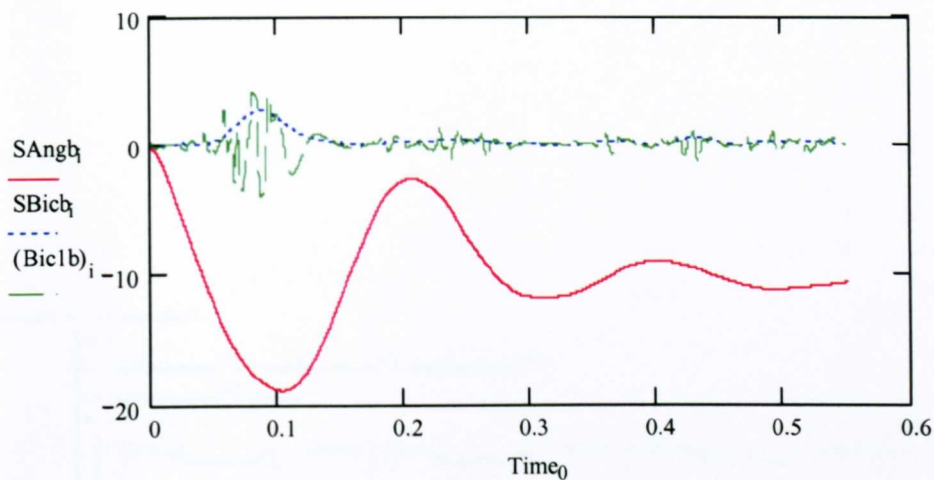
This routine synchronises simultaneous signals

```

fs := .15
no := for i ∈ 0..length(FootSwitch) - 1
      noi ← RL1i if FSstarti > 0
      noi ← fs·SF·1000 otherwise
      no
endfs := .55
b := for i ∈ 0..length(FootSwitch) - 1
     bi ← round(noi + endfs SF)
     b
a := for i ∈ 0..length(FootSwitch) - 1
     ai ← round(noi - SF fs)
     a

Bic1b := signalfract(Bic1, no, b)
Bic1 := signalfract(Bic1, a, b)
Tric1b := signalfract(Tric1, no, b)
Tric1 := signalfract(Tric1, a, b)
Bicb := signalfract(Bic, no, b)
Bic := signalfract(Bic, a, b)
Tricb := signalfract(Tric, no, b)
Tric := signalfract(Tric, a, b)
SPosb := signalfract(SPos, no, b)
SPos := signalfract(SPos, a, b)
SBicb := signalfract(SBic, no, b)
i := 0
SBic := signalfract(SBic, a, b)
STricb := signalfract(STric, no, b)
SAccb := signalfract(SAcc, no, b)
STric := signalfract(STric, a, b)
SAcc := signalfract(SAcc, a, b)
SForb := signalfract(SFor, no, b)
SFor := signalfract(SFor, a, b)
SAngb := signalfract(SAng, no, b)

```

```

SAng := signalfract(SAng,a,b)
SFS := signalfract(SFS,a,b)
STim:= for i ∈ 0..length(Time) - 1
        Stim_i ← (submatrix(STim_i,0,b_i - a_i,0,0))
        Stim
STimd:= for i ∈ 0..length(Time) - 1
        Stim_i ← (submatrix(STim_i,0,b_i - no_i,0,0))
        Stim

```

Signal differentiation

```

fdifang := fdif(SAng)
sdifang := sdif(SAng)
fdifacc := fdif(SAcc)
sdifacc := sdif(SAcc)
fdiffor := fdif(SFor)
sdiffor := sdif(SFor)
fdifang := iirlp(fdifang, FC/3, Order)
sdifang := iirlp(sdifang, FC/3, Order)
angmaximums:= maximums(SAng, fdifang, sdifang)
angminimums:= minimums(SAng, fdifang, sdifang)

```

```

angmaximum:= for k ∈ 0..length(SAng) - 1
| j ← 0
| x ← length(angminimums(ψ)) if length(angmaximums(ψ)) > length(angminimums(ψ))
| x ← length(angmaximums(ψ)) otherwise
| for i ∈ 0..x - 1
| | if [ [ [ (SAngk)(angmaximums(ψ))i < Angrangneg ] ∧ [ (SAngk)(angmaximums(ψ))i < Angrangepos ] ] ∨ [ (SAngk)(angmaximums(ψ))i > Angrangneg - .0 ∧ (SAngk)(angmaximums(ψ))i > Angrangepos ] ] ] ∧ (angmaximums(ψ))i > [ (angmaximums(ψ))i-1 + 3 ]
| | | angmaximumsj,k ← (angmaximums(ψ))i
| | | j ← j + 1
| | otherwise
| | | j ← j
| | | angmaximumsj,k ← 0
| | angmaximums
| angmaximums
angmaximumval:= for k ∈ 0..rows(angmaximum) - 1
| for i ∈ 0..cols(angmaximum) - 1
| | angmaximumvalk,i ← (SAngi)(angmaximumk,i)
| | angmaximumval
| angmaximumval
angmaximumcount:= for i ∈ 0..length(SAcc) - 1
| j ← 0
| for m ∈ 0..rows(angmaximum(ψ)) - 1
| | if (angmaximum(ψ))m ≠ 0
| | | j ← j + 1
| | | angmaximumcounti ← j
| | angmaximumcount

```

```

angminimum:= for k ∈ 0..length(SAng) - 1
    j ← 0
    x ← length(angmaximum(ω)) if length(angmaximum(ω)) > length(angminimum(ω))
    x ← length(angminimum(ω)) otherwise
    for i ∈ 0..(x - 5)
        if (angminimum(ω))i > 0 ∧ [ [ (SAngk)(angminimum(ω))i ≤ Angrangebeg ] ∧ [ (SAngk)(angminimum(ω))i < Angrangepos ] ] ∨ (SAngk)(angminimum(ω))i > Angrangebeg ∧ (SAngk)(angminimum(ω))i > Angrangepos ] ∧ (angminimum(ω))i > [ (angminimum(ω))i-1 + 5 ]
            angminimumj,k ← (angmaximum(ω))i
            j ← j + 1
        otherwise
            j ← j
            angminimumj,k ← 0
    angminimum

```

```

angminimumval:= for k ∈ 0..rows(angminimum) - 1
    for i ∈ 0..cols(angminimum) - 1
        angminimumvalk,i ← (SAngi)(angminimumk,i)
    angminimumval
angminimumval

```

```

angminimumcount:= for i ∈ 0..length(SAcc) - 1
    j ← 0
    for m ∈ 0..rows(angminimum(ψ)) - 1
        if (angminimum(ψ))m ≠ 0
            j ← j + 1
            angminimumcounti ← j
    angminimumcount

```

```

movmp:= for i ∈ 0..length(SAng) - 1
    movmpi ← posmini if minsampli < maxsampli
    movmpi ← posmaxi otherwise
    movmp
    minsamplmi
mini:=  $\frac{\text{minsampl}_{mi}}{1000SF}$ 

```

```

movmaxpeak:= for i ∈ 0..length(SAng) - 1
    | movendi ← minsampi if minsampi < maxsampi
    | movendi ← maxsampi otherwise
    | movend
    | maxsampmi
maxi:=  $\frac{\text{maxsamp}_{mi}}{100SF}$ 

movstartang := for i ∈ 0..length(SAng) - 1
    | for j ∈ movmaxpeaki..0
    | | movstarti ← (SAngi)j if  $\left[ \left[ (SAng_i)_j < \text{Angrangeneg} \wedge \text{maxsamp}_i > \text{minsamp}_i \right] \vee \left[ (SAng_i)_j > \text{Angrangepos} \wedge \text{maxsamp}_i < \text{minsamp}_i \right] \right] \wedge \text{movmaxpeak}_i \neq 0$ 
    | | movstarti ← 0 if  $(\text{movmaxpeak}_i \leq 1 \wedge \text{movmaxpeak}_i \geq 0) \vee (\text{movmaxpeak}_i \leq 0 \wedge \text{movmaxpeak}_i \geq -1)$ 
    | | movstart
movstart := for i ∈ 0..length(SAng) - 1
    | for j ∈ movmaxpeaki..0
    | | movstarti ← j + 1 if  $\left[ (SAng_i)_j < \text{Angrangeneg} \wedge \text{maxsamp}_i > \text{minsamp}_i \right] \vee \left[ (SAng_i)_j > \text{Angrangepos} \wedge \text{maxsamp}_i < \text{minsamp}_i \right]$ 
    | | movstarti ← movstarti-1 if  $(\text{movmaxpeak}_i \leq 1 \wedge \text{movmaxpeak}_i \geq 0) \vee (\text{movmaxpeak}_i \leq 0 \wedge \text{movmaxpeak}_i \geq -1)$ 
    | | movstart

movendang := for i ∈ 0..length(SAng) - 1
    | for j ∈ movmaxpeaki..0
    | | movendangi ← (SAngi)j if  $\left[ (SAng_i)_j \leq \text{Angrangeneg} - \left[ .99(\text{Angrangeneg} - \text{movmp}_i) \right] \wedge \text{maxsamp}_i > \text{minsamp}_i \right] \vee \left[ (SAng_i)_j \geq \text{Angrangepos} + \left[ .99(\text{movmp}_i - \text{Angrangepos}) \right] \wedge \text{maxsamp}_i < \text{minsamp}_i \right]$ 
    | | movendangi ← 0 if movmaxpeaki = 0
    | | movendang
movend := for i ∈ 0..length(SAng) - 1
    | for j ∈ movmaxpeaki..0
    | | movendi ← j + 1 if  $\left[ (SAng_i)_j \leq \text{Angrangeneg} - \left[ .99(\text{Angrangeneg} - \text{movmp}_i) \right] \wedge \text{maxsamp}_i > \text{minsamp}_i \right] \vee \left[ (SAng_i)_j \geq \text{Angrangepos} + \left[ .99(\text{movmp}_i - \text{Angrangepos}) \right] \wedge \text{maxsamp}_i < \text{minsamp}_i \right]$ 
    | | movendi ← 0 if movmaxpeaki = 0
    | | movend

movmaxpeakang:= for i ∈ 0..length(SAng) - 1
    | movendi ← (SAngi)(movmaxpeaki)
    | movend

presamplstart := for i ∈ 0..length(SAng) - 1
    | presamplstarti ← prestart(SAngi, movstartangi)
    | presamplstart

presamplmaxpeak:= for i ∈ 0..length(SAng) - 1
    | presamplendi ← preend(SAngi, movmaxpeakangi)
    | presamplend

RL1:= for i ∈ 0..length(SAng) - 1
    | RL1i ← movstarti - 10
    | RL1

```

Position analysis II

```
firstswing := minsignal(SAng)
startpos := maxsignal(SAng)
posmin := minsignal(SAng, angminimum)
posmax := maxsignal(SAng, angmaximum)
minsampl:= signalsampl(SAng, posmin)
maxsampl:= signalsampl(SAng, posmax)
```

Position parameters after synchronisation

```
maxROM:= movmaxpeakang – movstartang
absROM := movendang – movstartang
ST :=  $\frac{\text{movend} - \text{movstart}}{\text{SF}}$ 
RT :=  $\frac{\text{movmaxpeak} - \text{movstart}}{\text{SF}}$ 
delay :=  $\frac{\text{movstart} - \text{SF} \cdot l}{\text{SF}}$ 
meanmovend := mean(movend)
meanmovmaxpeakang := mean(movmaxpeakang)
meanmovstart := mean(movstart)
meanmovstartang := mean(movstartang)
meanmovmaxpeak:= mean(movmaxpeak)
meanmovendang := mean(movendang)
meanmaxROM:= mean(maxROM)
meanangmaximumcount:= mean(angmaximumcount)
meanabsROM := mean(absROM)
meanangminimumcount:= mean(angminimumcount)
meanRT := mean(RT)
```

Goniogram period

Determines the parameters for the model

```

T := for j ∈ 0..length(movstart) - 1
  for i ∈ 1..2
    T0,j ←  $\frac{(\text{angmaximum}^{\langle \dot{\psi} \rangle})_0 - (\text{movstart})_j}{\text{SF}}$  if  $\left[ \text{movstartang}_j > (\text{angmaximumval}^{\langle \dot{\psi} \rangle})_0 \right]$ 
    T0,j ←  $\frac{(\text{angminimum}^{\langle \dot{\psi} \rangle})_0 - (\text{movstart})_j}{\text{SF}}$  otherwise
    (T)i,j ←  $\frac{(\text{angmaximum}^{\langle \dot{\psi} \rangle})_i - (\text{angmaximum}^{\langle \dot{\psi} \rangle})_{i-1}}{\text{SF}}$  if  $(\text{angmaximum}^{\langle \dot{\psi} \rangle})_i > 0 \wedge \text{movstartang}_j > (\text{angmaximumval}^{\langle \dot{\psi} \rangle})_0$ 
    (T)i,j ←  $\frac{(\text{angminimum}^{\langle \dot{\psi} \rangle})_i - (\text{angminimum}^{\langle \dot{\psi} \rangle})_{i-1}}{\text{SF}}$  otherwise
  T
cycles := 1..angmaximumcount0
rows(angmaximum) = 4
movstartang0 = -0.258
Tmean := for j ∈ 0..25
  Tmeanj ← mean(T⟨ $\dot{\psi}$ ⟩)
  (Tmean) - .011

```

Damping ratio

```

Dmax:= for j ∈ 0..25
  for i ∈ 1..rows(T) - 1
    (Dmax)0,j ←  $\frac{(\text{angmaximumval}^{\langle \dot{\psi} \rangle})_0 - \text{restangle}_j}{\text{movstartang}_j - \text{restangle}_j}$ 
    (Dmax)i,j ←  $\left[ \frac{(\text{angmaximumval}^{\langle \dot{\psi} \rangle})_i - \text{restangle}_j}{(\text{angmaximumval}^{\langle \dot{\psi} \rangle})_{i-1} - \text{restangle}_j} \right]$  if  $(\text{angminimum}^{\langle \dot{\psi} \rangle})_i > 0$ 
  Dmax
Dmin:= for j ∈ 0..25
  for i ∈ 1..rows(T) - 1
    (Dmin)i-1,j ←  $\left[ \frac{\text{restangle}_j - (\text{angminimumval}^{\langle \dot{\psi} \rangle})_{i-1}}{\text{restangle}_j - (\text{angminimumval}^{\langle \dot{\psi} \rangle})_i} \right]$  if  $(\text{angminimum}^{\langle \dot{\psi} \rangle})_i > 0$ 
  Dmin
angmaximumvalseg:= submatrix(angmaximumval,0,rows(T) - 2,5,25)
angmaximumseg:= submatrix(angmaximum,0,rows(T) - 2,5,25)

```

```

ζ := for j ∈ 0..0
| for i ∈ 0..rows(T) - 2
|   ζi,j ←  $\frac{\ln(D_{\max_{1,j}})^2}{(2\pi)^2 + \ln(D_{\max_{1,j}})^2}$ 
| ζ
ζ =  $\begin{pmatrix} 0.041 \\ 0.26 \end{pmatrix}$ 
ζ := for j ∈ 0..29
| for i ∈ 0..rows(T) - 2
|   ζi,j ←  $\frac{\ln(D_{\min_{1,j}})^2}{(2\pi)^2 + \ln(D_{\min_{1,j}})^2}$ 
| ζ
ζmean := (ζ0)
ζmean = 0.041
i := 0
ζmean := mean(ζ)

```

Natural frequency

```

ω :=  $\frac{\pi}{T_{\text{mean}}}$ 
ω := for j ∈ 0..0
| for i ∈ 0..rows(T) - 1
|   (ω)i,j ←  $\frac{2\pi}{T_{i,j}}$ 
| ω
ωmean := mean(ω)
ωn := for i ∈ 0..cols(ω) - 1
| for j ∈ 0..rows(T) - 2
|   ωnj,i ←  $\frac{\omega_{j,i}}{\sqrt{1 - (\zeta_{j,i})^2}}$ 
| ωn
ωn2 := for i ∈ 0..cols(ω) - 1
| for j ∈ 0..rows(T) - 2
|   ωn2j,i ←  $\left[ \frac{\omega_{j,i}}{\sqrt{1 - (\zeta_{j,i})^2}} \right]^2$ 
| ωn2

```



```

ωn2mean := | for i ∈ 0..cols(ω) - 1
              for j ∈ 0..rows(T) - 1
                ωn2meani ← mean(ωn2<ψ>)
              | mean(ωn2mean)
ωn2mean := ωn20

```

calculated from an estimated forearm moment of inertia and a previously calculated device parameters

I := .032
 Elastic constant from the springs configuration

```

K := 27.34
ωnmean := | for i ∈ 0..cols(ω) - 1
              for j ∈ 0..rows(T) - 1
                ωnmeani ← mean(ωn<ψ>)
              | mean(ωnmean)

```

Elastic constant (Device + upper limb)

Kn := I·ωn2mean
 Kn = 33.8

Second order equation

$$\frac{d}{dt} \frac{d}{dt} y + 2 \cdot \zeta \cdot \omega_n \cdot \frac{d}{dt} y + \omega_n^2 \cdot y = \omega_n^2 \cdot u$$

```

Bn := | for i ∈ 0..cols(ω) - 1
        for j ∈ 0..rows(ωn) - 1
          Bnj, i ← (ζj, i) · 2 · √I · (Kn)
        | Bn

```

```

ζmean = 0.151
Bnmean := | for i ∈ 0..cols(ω) - 1
              for j ∈ 0..rows(T) - 1
                Bnmeani ← mean(Bn<ψ>)
              | mean(Bnmean)

```

ωn := ωn

Moment of inertia

I = 0.032

Elastic constant

Kn = 33.8

Damping constant

Bnmean = 0.313

ωnmean := (ωn₀) - 2

ωnmean := ωn

$$\tau := \frac{1}{\left(\zeta^{\langle \dot{\varphi} \rangle}\right)_0 \cdot \left(\omega n^{\langle \dot{\varphi} \rangle}\right)_0}$$

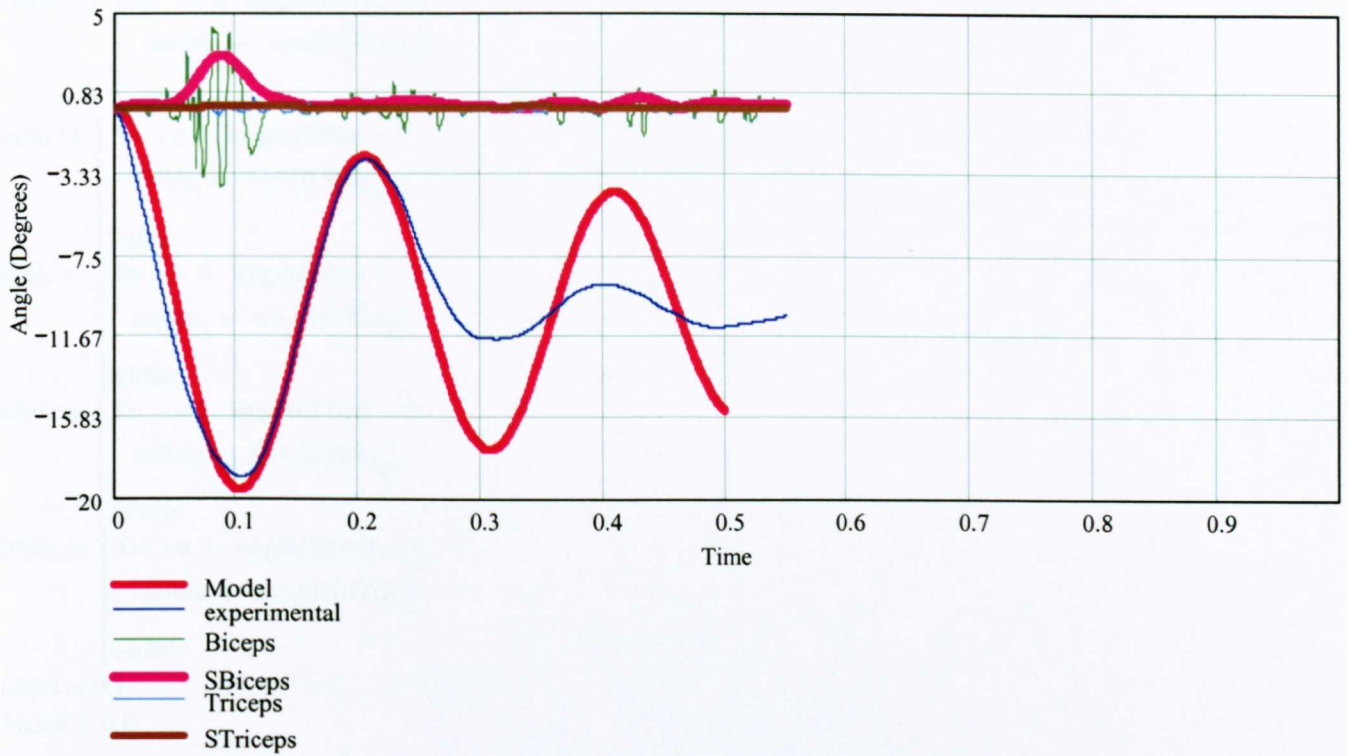
$$t := 0, .00002441. 0.5$$

Second order equation

$$y(t) := \left[1 - e^{-\left[\left(\omega n^{\langle \dot{\varphi} \rangle}\right)_0 \cdot \left[\left(\zeta^{\langle \dot{\varphi} \rangle}\right)_0\right] \cdot t} \cdot \cos \left[\sqrt{1 - \left[\left(\zeta^{\langle \dot{\varphi} \rangle}\right)_0\right]^2} \cdot \left(\omega n^{\langle \dot{\varphi} \rangle}\right)_0 \cdot t - \operatorname{atan} \left[\frac{\left(\zeta^{\langle \dot{\varphi} \rangle}\right)_0}{\sqrt{1 - \left[\left(\zeta^{\langle \dot{\varphi} \rangle}\right)_0\right]^2}} \right] \right] \right]$$

$$\operatorname{vel}(t) := \frac{d}{dt} y(t) \qquad \operatorname{acc}(t) := \frac{d}{dt} \operatorname{vel}(t)$$

Biomechanical model and measured data from a post-stroke subject



EMG differentiation

```
fdifbic1 := for i ∈ 0..length(SBic) - 1
              fdifbici ← fstdiff(Bic1i)
              fdifbic
sdifbic1 := for i ∈ 0..length(SBic) - 1
              sdifbici ← scndiff(Bic1i)
              sdifbic
```

EMG signals differentiation

```
fdiftric1 := for i ∈ 0..length(STric) - 1
               fdiftrici ← fstdiff(Tric1i)
               fdiftric
sdiftric1 := for i ∈ 0..length(STric) - 1
               sdiftrici ← scndiff(Tric1i)
               sdiftric
fdifbic := for i ∈ 0..length(SBic) - 1
            fdifbici ← fstdiff(SBici)
            fdifbic
sdifbic := for i ∈ 0..length(SBic) - 1
            sdifbici ← scndiff(SBici)
            sdifbic
fdiftric := for i ∈ 0..length(STric) - 1
             fdiftrici ← fstdiff(STrici)
             fdiftric
sdiftric := for i ∈ 0..length(STric) - 1
             sdiftrici ← scndiff(STrici)
             sdiftric
```

```
EMGst := 0.1
EMGnd := 0.84
length(sdifbic0) - 1 = 2.865 × 103
size := length(sdifbici) - 1
```

Biceps Minimums Detector

```
bicminimums:= for i ∈ 0..length(SBic) - 1
| j ← 0
| for n ∈ 1..length(sdifbici) - 1
|   if (fdifbici)n-1 < 0 ∧ (fdifbici)n > 0 ∧ (fdifbici)n+1 > 0 ∧ [[(sdifbici)n ∨ (sdifbici)n+1 ∨ (sdifbici)n-1 = 0]]
|     | minimumsj,i ← n
|     | j ← j + 1
|     | minimums
| minimums
```

Biceps Maximums Detector

```
bicmaximums:= for i ∈ 0..length(SBic) - 1
| j ← 0
| for n ∈ 1..length(sdifbici) - 1
|   if (STimi)n > 0.1 ∧ (fdifbici)n-1 > 0 ∧ (fdifbici)n < 0 ∧ (fdifbici)n+1 < 0 ∧ [(sdifbici)n ∨ (sdifbici)n+1 ∨ (sdifbici)n-1 = 0]
|     | maximumsj,i ← n
|     | j ← j + 1
|     | maximums
| maximums
```

im:= 0

Biceps Maximums and Minimums discriminator

```
maxpeakbic:= for k ∈ 0..length(SBic) - 1
|   maxpeakbick ← max(SBick)
| maxpeakbic
minpeakbic:= for k ∈ 0..length(SBic) - 1
|   minpeakbick ← (min(silBick))
| minpeakbic
ROB:= for k ∈ 0..length(SBic) - 1
|   ROBk ← maxpeakbick - minpeakbick
| ROB
ROB:= for k ∈ 0..length(SBic) - 1
|   ROBk ← maxpeakbick
| ROB
maxpeakbicsampl:= for k ∈ 0..length(SBic) - 1
|   maxpeakbicsamplk ← samp(SBick,maxpeakbick)
| maxpeakbicsampl
```

bicmaximum= for $k \in 0.. \text{length}(\text{SBic}) - 1$

```

| j ← 0
| x ← length(bicmaximums(k))
| for i ∈ 0.. x - 1
|   if  $\left[ (\text{SBic}_k)(\text{bicmaximums}^{(k)})_i > 1 \cdot \text{EMGnd} \cdot \text{ROB}_k \right] \wedge (\text{SBic}_k)(\text{bicmaximums}^{(k)})_i > 0 \wedge \left[ \left[ (\text{bicmaximums}^{(k)})_i > \text{RL}_k \right] \right]$ 
|     | bicmaximumsj,k ← (bicmaximums(k))i
|     | j ← j + 1
|   otherwise
|     | j ← j
|     | bicmaximumsj,k ← 0
|   bicmaximums
| bicmaximums

```

bicmaximumcount:= for $i \in 0.. \text{length}(\text{SBic}) - 1$

```

| j ← 0
| for m ∈ 0.. rows(bicmaximum(i)) - 1
|   if (bicmaximum(i))m ≠ 0
|     | j ← j + 1
|     | bicmaximumcounti ← j
|   bicmaximumcounti ← j otherwise
| bicmaximumcount

```

bicmaximumamp= for $k \in 0.. \text{length}(\text{bicmaximumcount}) - 1$

```

| for j ∈ 0
|   bicmaximumampk,j ← (SBick)bicmaximumj,k if bicmaximumcountk > 0
| bicmaximumamp

```

bicminimum:= for $k \in 0.. \text{length}(\text{SBic}) - 1$

$j \leftarrow 0$

$x \leftarrow \text{length}(\text{bicminimums}^{(k)})$ if $\text{length}(\text{bicmaximums}^{(k)}) > \text{length}(\text{bicminimums}^{(k)})$

$x \leftarrow \text{length}(\text{bicmaximums}^{(k)})$ otherwise

 for $i \in 0.. (x - 1)$

 if $(\text{bicminimums}^{(k)})_i > (\text{bicmaximums}^{(k)})_i$

$\text{bicminimums}_{j,k} \leftarrow (\text{bicminimums}^{(k)})_i$

$j \leftarrow j + 1$

 otherwise

$j \leftarrow j$

$\text{bicminimums}_{j,k} \leftarrow 0$

 bicmaximums

 bicminimums

bicminimumcount:= for $i \in 0.. \text{cols}(\text{bicminimum}) - 1$

$j \leftarrow 0$

 for $m \in 0.. \text{rows}(\text{bicminimum}^{(i)}) - 1$

 if $(\text{bicminimum}^{(i)})_m \neq 0$

$j \leftarrow j + 1$

$\text{bicminimumcount}_i \leftarrow j$

 bicminimumcount_i $\leftarrow 0$ otherwise

 bicminimumcount - 1

beginrefbic := for $k \in 0.. \text{length}(\text{bicmaximumcount}) - 1$

 for $m \in 0$

 if $\text{bicmaximumcount}_k > 0$

$i_m \leftarrow (\text{bicmaximum}^{(k)})_m$

 while $\left[(\text{SBic}_k)_{(i_m)} \geq \text{EMGst} \cdot \text{ROB}_k \wedge i_m > 0 \right]$ if $i_m > \text{movstart}_m$

$i_m \leftarrow i_m - 1$

$i_m \leftarrow 0$ otherwise

$\text{beginref}_k \leftarrow i$

beginref

```

finrefbic:= for k ∈ 0..length(bicmaximumcount) - 1
    for m ∈ 0
        if bicmaximumcountk > 0
            im ← (bicmaximum(k))m
            while [(SBick)im ≥ 1EMGst-ROBk] ∧ im < length(SBick) - 1 if im ≠ 0
                im ← im + 1
            im ← 0 otherwise
            finrefk ← i
    finref

rawbicmaximum:= for i ∈ 0..length(SBic) - 1
    rawbicmaximumi ← submatrix[Bici, (beginrefbic)i, (finrefbic)i, 0, 0] if bicmaximumcounti > 0
    rawbicmaximumi ← max(rawbicmaximumi) if bicmaximumcounti > 0
rawbicmaximum

beginrefbicmax:= for k ∈ 0..length(SBic) - 1
    i ← maxpeakbicsampk
    while (SBick)i > Bicrangeposk + EMGst-ROBk ∧ (SBick)i > movstartk if i ≠ 0
        i ← i - 1
    i ← 0 otherwise
    beginrefk ← i
beginref

beginrefbicmax0 = 830

finrefbicmax:= for k ∈ 0..length(SBic) - 1
    i ← maxpeakbicsampk
    while (SBick)i > Bicrangeposk + EMGst-ROBk ∧ i < length(SBick) - 1 if i ≠ 0
        i ← i + 1
    i ← 0 otherwise
    beginrefk ← i
beginref

```

Biceps Analysis

```

Risereflex:= for i ∈ 0..length(bicmaximumcount) - 1
    for k ∈ 0
        Risereflexi,k ← submatrix[SBici, 0, (bicmaximum(i))k, 0, 0] if bicmaximumcounti > 0
    Risereflex

Risereflexmax:= for i ∈ 0..length(bicmaximumcount) - 1
    Risereflexmaxi ← submatrix[SBici, 0, maxpeakbicsampi, 0, 0]
Risereflexmax

```



```

Reflexamplitude:= for i ∈ 0..length(bicmaximumcount) - 1
                  for k ∈ 0
                    Reflexamplitudei,k ←  $\left[ \left( \text{SBic}_i \right) \left[ \left( \text{bicmaximum}^{(i)} \right)_k \right] - \text{EMGstROB}_i \right]$  if bicmaximumcounti > 0
                  Reflexamplitude
Reflexamplitudemax:= for i ∈ 0..length(SBic) - 1
                    Reflexamplitudemaxi ← maxpeakbici -  $\left( \text{SBic}_i \right) \left[ \left( \text{bicminimums}^{(i)} \right)_0 \right]$ 
                    Reflexamplitudemax
Reflexstart:= for i ∈ 0..length(bicmaximumcount) - 1
              for k ∈ 0
                Reflexstarti,k ←  $\left( \text{SBic}_i \right) \left[ \left( \text{beginrefbic} \right)_k \right]$  if bicmaximumcounti > 0
              Reflexstart
Reflexstartmax:= for i ∈ 0..length(SBic) - 1
                 Reflexstartmaxi ←  $\left( \text{SBic}_i \right) \left( \text{beginrefbicmax} \right)$ 
                 Reflexstartmax
samplrefstart := for i ∈ 0..length(bicmaximumcount) - 1
                 for k ∈ 0
                   Reflexstarti,k ←  $\left( \text{beginrefbic} \right)_i$  if bicmaximumcounti > 0
                 Reflexstart
Reflexendmax:= for i ∈ 0..length(bicmaximumcount) - 1
               for k ∈ 0..bicmaximumcounti - 1
                 Reflexendmaxi ← EMGnd Reflexamplitudemaxi + EMGstROBi if bicmaximumcounti > 0
               Reflexendmax
Reflexend:= for i ∈ 0..length(bicmaximumcount) - 1
            for k ∈ 0
              Reflexendi,k ← EMGnd(Reflexamplitudei,k) + EMGstROBi if bicmaximumcounti > 0
            Reflexend
samplrefend := bicmaximumT
samplrefstartmax:= for i ∈ 0..length(bicmaximumcount) - 1
                   samplrefstartmaxi ← start2(Risereflexmaxi, Reflexstartmaxi)
                   samplrefstartmax
samplrefendmax:= maxpeakbicsamp
Risetimemov:= for i ∈ 0..length(bicmaximumcount) - 1
              for k ∈ 0
                Risetimemov0,i ←  $\frac{\text{samplrefend}_{i,k} - \text{samplrefstart}_{i,k}}{\text{SF}}$  if bicmaximumcounti > 0
              Risetimemov
samplstartang := movstartang

```

Reflexlatencymov:=
 for $i \in 0.. \text{length}(\text{bicmaximumcount}) - 1$
 for $k \in 0$
 $\text{Risetimemov}_{0,i} \leftarrow \frac{\text{samplrefstart}_{i,k} - \text{samplstartang}_i}{\text{SF}}$ if $\text{bicmaximumcount}_i > 0$
 Risetimemov

$\text{length}(\text{bicmaximumcount}) = 26$
 RL1:=
 for $i \in 0.. \text{length}(\text{SBic}) - 1$
 $\text{RL1}_i \leftarrow \text{movstart}_i$
 RL1

RL2max:=
 for $i \in 0.. \text{length}(\text{bicmaximumcount}) - 1$
 $\text{RL2max}_i \leftarrow \text{samplrefstartmax}_i$
 RL2max

RL2:=
 for $i \in 0.. \text{length}(\text{bicmaximumcount}) - 1$
 for $k \in 0$
 $\text{RL2}_{i,k} \leftarrow \text{samplrefstart}_{i,k}$ if $\text{bicmaximumcount}_i > 0$
 $\text{RL2}_{i,k} \leftarrow 0$ otherwise
 RL2

latencymax:=
 for $i \in 0.. \text{length}(\text{bicmaximumcount}) - 1$
 $\text{latencymax}_i \leftarrow \text{RL2max}_i - \text{RL1}_i$
 $\frac{\text{latencymax}}{\text{SF}}$

latency:=
 for $i \in 0.. \text{length}(\text{bicmaximumcount}) - 1$
 for $k \in 0$
 $\text{latency}_{i,k} \leftarrow \text{RL2}_{i,k} - \text{RL1}_i$ if $\text{bicmaximumcount}_i > 0$
 $\frac{\text{latency}}{\text{SF}}$

RTS:=
 for $i \in 0.. \text{length}(\text{bicmaximumcount}) - 1$
 for $k \in 0$
 $\text{RTS}_{i,k} \leftarrow \text{samplrefstart}_{i,k}$ if $\text{bicmaximumcount}_i > 0$
 RTS

RTE:=
 for $i \in 0.. \text{length}(\text{bicmaximumcount}) - 1$
 for $k \in 0$
 $\text{RTE}_{i,k} \leftarrow \text{samplrefend}_{i,0}$ if $\text{bicmaximumcount}_i > 0$
 $\text{RTE}_{i,k} \leftarrow 0$ otherwise
 RTE

RTEmax:=
 for $i \in 0.. \text{length}(\text{SBic}) - 1$
 $\text{RTEmax}_i \leftarrow \text{samplrefendmax}_i$
 RTEmax

```

RTSmax:= | for i ∈ 0..length(bicmaximumcount) - 1
          |   RTSmaxi ← samplerefstartmaxi
          | RTSmax
Risetime:= | for i ∈ 0..length(bicmaximumcount) - 1
            |   for k ∈ 0
            |     Risetimei,k ← RTEi,k - RTSi,k if bicmaximumcounti > 0
            |   Risetime
            |   SF
Risetimemax:= | for i ∈ 0..length(SBic) - 1
               |   Risetimemaxi ← RTEmaxi - RTSmaxi
               |   Risetimemax
               |   SF
RMax:= | for i ∈ 0..length(bicmaximumcount) - 1
        |   RMaxi ← (SBici)(bicmaximumi)0
        |   RMax
FiR:= | for i ∈ 0..length(bicmaximumcount) - 1
      |   for k ∈ 0
      |     | FiRi,k ← (finrefbici)k if bicmaximumcounti > 0
      |     | FiRi,k ← 0 otherwise
      |   FiR
BR:= | for i ∈ 0..length(bicmaximumcount) - 1
     |   for k ∈ 0
     |     | BRi,k ← (beginrefbici)k if bicmaximumcounti > 0
     |     | BRi,k ← 0 otherwise
     |   BR
Activesignal:= | for i ∈ 0..length(bicmaximumcount) - 1
                |   for k ∈ 0..bicmaximumcounti - 1
                |     Activesignal0,i ←  $\frac{FiR_{i,0} - BR_{i,0}}{SF}$  if bicmaximumcounti > 0
                |   Activesignal
FiRmax:= | for i ∈ 0..length(SBic) - 1
          |   FiRmaxi ← finrefbicmaxi
          |   FiRmax
BRmax:= | for i ∈ 0..length(SBic) - 1
         |   BRmaxi ← beginrefbicmaxi
         |   BRmax

```

$\text{Activesignalmax} :=$

for $i \in 0.. \text{length}(\text{bicmaximumcount}) - 1$ $\text{Activesignalmax}_i \leftarrow \frac{\text{FiRmax}_i - \text{BRmax}_i}{\text{SF}}$ if $\text{bicmaximumcount}_i > 0$	Activesignalmax
---	--------------------------

$\text{Activesignalmatrix} :=$

for $i \in 0.. \text{length}(\text{bicmaximumcount}) - 1$ for $k \in 0$ $\text{Activesignalmatrix}_{i,k} \leftarrow \text{submatrix}(\text{SBic}_i, \text{BR}_{i,k}, \text{FiR}_{i,k}, 0, 0)$ if $\text{bicmaximumcount}_i > 0$	$\text{Activesignalmatrix}$
---	-----------------------------

$\text{Activesignalmaxmatrix} :=$

for $i \in 0.. \text{length}(\text{bicmaximumcount}) - 1$ $\text{Activesignalmaxmatrix}_i \leftarrow \text{submatrix}(\text{SBic}_i, \text{BRmax}_i, \text{FiRmax}_i, 0, 0)$	$\text{Activesignalmaxmatrix}$
---	--------------------------------

$\text{Activesignalarea} :=$

for $i \in 0.. \text{length}(\text{bicmaximumcount}) - 1$ for $k \in 0$ $\text{Activesignalarea}_{i,k} \leftarrow \text{integral}(\text{Activesignalmatrix}_{i,k})$ if $\text{bicmaximumcount}_i > 0 \wedge \text{BR}_{i,k} \neq \text{FiR}_{i,k}$	Activesignalarea
--	---------------------------

Triceps Minimums Detector

$\text{size} := \text{length}(\text{sdiftric}_i) - 1$
 $\text{size} := \text{size}$
 $\text{tricminimums} :=$ for $i \in 0.. \text{length}(\text{STric}) - 1$

$j \leftarrow 0$ for $n \in 1.. \text{length}(\text{sdiftric}_i) - 1$ if $(\text{fdiftric}_i)_{n-1} < 0 \wedge (\text{fdiftric}_i)_n > 0 \wedge (\text{fdiftric}_i)_{n+1} > 0 \wedge \left[\left[(\text{sdiftric}_i)_n \vee (\text{sdiftric}_i)_{n+1} \vee (\text{sdiftric}_i)_{n-1} = 0 \right] \right]$ <table border="0" style="border-collapse: collapse;"> <tr> <td style="border-left: 1px solid black; padding-left: 10px; vertical-align: top;"> $\text{minimums}_{j,i} \leftarrow n$ $j \leftarrow j + 1$ minimums </td> </tr> </table>	$\text{minimums}_{j,i} \leftarrow n$ $j \leftarrow j + 1$ minimums	minimums
$\text{minimums}_{j,i} \leftarrow n$ $j \leftarrow j + 1$ minimums		

Triceps Maximums Detector

$\text{tricmaximums} :=$ for $i \in 0.. \text{length}(\text{STric}) - 1$

$j \leftarrow 0$ for $n \in 1.. \text{length}(\text{sdiftric}_i) - 1$ if $(\text{STim}_i)_n > \text{fs} \wedge (\text{fdiftric}_i)_{n-1} > 0 \wedge (\text{fdiftric}_i)_n < 0 \wedge (\text{fdiftric}_i)_{n+1} < 0 \wedge \left[(\text{sdiftric}_i)_n \vee (\text{sdiftric}_i)_{n+1} \vee (\text{sdiftric}_i)_n \right]$ <table border="0" style="border-collapse: collapse;"> <tr> <td style="border-left: 1px solid black; padding-left: 10px; vertical-align: top;"> $\text{maximums}_{j,i} \leftarrow n$ $j \leftarrow j + 1$ maximums </td> </tr> </table>	$\text{maximums}_{j,i} \leftarrow n$ $j \leftarrow j + 1$ maximums	maximums
$\text{maximums}_{j,i} \leftarrow n$ $j \leftarrow j + 1$ maximums		

Triceps Maximums and Minimums discriminator

```

maxpeaktric:= for k ∈ 0..length(STric) - 1
               maxpeaktrick ← max(STrick)
minpeaktric:= for k ∈ 0..length(STric) - 1
               minpeaktrick ← (mean(silTrick))
minpeaktric
ROT:= for k ∈ 0..length(STric) - 1
       ROTk ← maxpeaktrick
ROT
ROT:= for k ∈ 0..length(STric) - 1
       ROTk ← maxpeaktrick - minpeaktrick
ROT
maxpeaktricsampl:= for k ∈ 0..length(STric) - 1
                    maxpeaktricsamplk ← samp(STrick, maxpeaktrick)
maxpeaktricsampl
tricmaximum:= for k ∈ 0..length(STric) - 1
               j ← 0
               x ← length(tricminimums(k)) if length(tricmaximums(k)) > length(tricminimums(k))
               x ← length(tricmaximums(k)) otherwise
               for i ∈ 0..x - 1
                   if (STrick)(tricmaximums(k))i > EMGndROTk ∧ (STrick)(tricmaximums(k))i > 0 ∧ (tricmaximums(k))i ≥ RLk
                       tricmaximumsj,k ← (tricmaximums(k))i
                       j ← j + 1
                   otherwise
                       j ← j
                       tricmaximumsj,k ← 0
               tricmaximums
tricmaximums
tricmaximumcount:= for i ∈ 0..length(STric) - 1
                   j ← 0
                   for m ∈ 0..rows(tricmaximum(i)) - 1
                       if (tricmaximum(i))m ≠ 0
                           j ← j + 1
                           tricmaximumcounti ← j
                       tricmaximumcounti ← j otherwise
                   tricmaximumcount

```

```

tricmaximumamp= for k ∈ 0..length(tricmaximumcount) - 1
    for j ∈ 0..tricmaximumcountk - 1
        tricmaximumampj,k ← (STrick)tricmaximumj,k if tricmaximumcountk > 0
    tricmaximumamp
tricminimum:= for k ∈ 0..length(STric) - 1
    j ← 0
    x ← length(tricminimums(k)) if length(tricmaximums(k)) > length(tricminimums(k))
    x ← length(tricmaximums(k)) otherwise
    for i ∈ 0..(x - 1)
        if (STrick)(tricminimums(k))i < EMGst·ROTk
            tricminimumsj,k ← (tricminimums(k))i
            j ← j + 1
        otherwise
            j ← j + 1
            tricminimumsj,k ← 0
    tricminimums
tricminimumcount:= for i ∈ 0..length(tricmaximumcount) - 1
    j ← 0
    for m ∈ 0..rows(tricminimum(i)) - 1
        if (tricminimum(i))m ≠ 0
            j ← j + 1
            tricminimumcounti ← j
    tricminimumcount - 1
beginreftric := for k ∈ 0..length(tricmaximumcount) - 1
    for m ∈ 0
        if tricmaximumcountk > 0
            im ← (tricmaximum(k))m
            while [(STrick)(im) ≥ EMGst·ROTk] ∧ im ≥ movstartk if im ≠ 0
                im ← im - 1
            im ← 0 otherwise
            beginrefk ← i
    beginref
beginrefbic0 = (820)
(beginreftric)0 = (842)

```

```

finreftric:= for k ∈ 0..length(tricmaximumcount) - 1
  for m ∈ 0
    if tricmaximumcountk > 0
      im ← (tricmaximum(k))m
      while [(STrick)im ≥ EMGst·ROTk] ∧ im < length(STrick) - 1 if im ≠ 0
        im ← im + 1
      im ← 0 otherwise
      finrefk ← i
    finref
beginreftricmax:= for k ∈ 0..length(STric) - 1
  i ← maxpeaktricsampk
  while (STrick)i > EMGst·ROTk ∧ (STrick)i > 0 if i ≠ 0
    i ← i - 1
  i ← 0 otherwise
  beginrefk ← i
  beginref
finreftricmax:= for k ∈ 0..length(SBic) - 1
  i ← maxpeaktricsampk
  while (STrick)i > EMGst·ROTk ∧ i < length(SBick) - 1 if i ≠ 0
    i ← i + 1
  i ← 0 otherwise
  beginrefk ← i
  beginref

```

Triceps Analysis

```

RisereflexT:= for i ∈ 0..length(tricmaximumcount) - 1
  for k ∈ 0
    RisereflexTi,k ← submatrix[STrici, 0, (tricmaximum(i))k, 0, 0] if tricmaximumcounti > 0
  RisereflexT
RisereflexmaxT:= for i ∈ 0..length(tricmaximumcount) - 1
  RisereflexmaxTi ← submatrix(STrici, 0, maxpeaktricsampi, 0, 0)
  RisereflexmaxT

```



```

ReflexamplitudeT:=
    for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
            ReflexamplitudeTi,k ←  $\left[ \left( \text{STric}_i \right) \left[ \left( \text{tricmaximum}^{(i)} \right)_0 \right] - \text{EMGstROT}_i \right]$  if tricmaximumcounti > 0
        ReflexamplitudeT
ReflexamplitudemaxT:=
    for i ∈ 0..length(STric) - 1
        ReflexamplitudemaxTi ←  $\text{maxpeaktric}_i - \left( \text{STric}_i \right) \left[ \left( \text{tricminimums}^{(i)} \right)_0 \right]$ 
    ReflexamplitudemaxT
ReflexstartmaxT:=
    for i ∈ 0..length(STric) - 1
        ReflexstartmaxTi ←  $\left( \text{STric}_i \right)_{\left( \text{beginreftricmax} \right)}$ 
    ReflexstartmaxT
ReflexstartT:=
    for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
            ReflexstartTi,k ←  $\left( \text{STric}_i \right) \left[ \left( \text{beginreftriq} \right)_k \right]$  if tricmaximumcounti > 0
        ReflexstartT
ReflexendmaxT:=
    for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0..tricmaximumcounti - 1
            ReflexendmaxTi ←  $0.9 \text{ReflexamplitudemaxT}_i + \text{EMGstROT}_i$ 
        ReflexendmaxT
ReflexendT:=
    for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
            ReflexendTi,k ←  $0.9 \left( \text{ReflexamplitudeT}_{i,k} \right) + \text{EMGstROT}_i$  if tricmaximumcounti > 0
        ReflexendT
samplrefstartT :=
    for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
            samplrefstartTi,k ←  $\text{start2} \left( \text{RisereflexT}_{i,k}, \text{ReflexstartT}_{i,k} \right)$  if tricmaximumcounti > 0
        samplrefstartT
samplrefendT := tricmaximumT
samplrefstartmaxT:=
    for i ∈ 0..length(tricmaximumcount) - 1
        samplrefstartmaxTi ←  $\text{start2} \left( \text{RisereflexmaxT}_i, \text{ReflexstartmaxT}_i \right)$ 
    samplrefstartmaxT
RisetimemovT:=
    for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
            RisetimemovTk,i ←  $\frac{\text{samplrefendT}_{i,k} - \text{samplrefstartT}_{i,k}}{\text{SF}}$  if tricmaximumcounti > 0
        RisetimemovT

```

```

ReflexlatencymovT:=
    for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
            RisetimemovTi,k ←  $\frac{\text{samplestartT}_{i,k} - \text{movstartang}_i}{\text{SF}}$  if tricmaximumcounti > 0
        Risetimemov

```

```

RL1T:=
    for i ∈ 0..length(STric) - 1
        RL1Ti ← samplestartangi

```

```

RL2T:=
    RL1T
    for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
            RL2Ti,k ← samplestartTi,k if tricmaximumcounti > 0
            RL2Ti,k ← 0 otherwise
        RL2T

```

```

sampleprefendmaxT:= maxpeaktricsamp

```

```

latencyT :=
    for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
            latencyTi,k ← RL2Ti,k - RL1Ti if tricmaximumcounti > 0
         $\frac{\text{latencyT}}{\text{SF}}$ 

```

```

RL2maxT:=
    for i ∈ 0..length(tricmaximumcount) - 1
        RL2maxTi ← samplestartmaxTi
    RL2maxT

```

```

RTST:=
    for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
            RTSTi,k ← samplestartTi,k if tricmaximumcounti > 0
        RTST

```

```

RTET:=
    for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
            RTETi,k ← sampleprefendTi,k if tricmaximumcounti > 0
            RTETi,k ← 0 otherwise
        RTET

```

```

RTSTmax:=
    for i ∈ 0..length(STric) - 1
        RL2maxTi ← samplestartmaxTi
    RL2maxT

```

```

latencyTmax:=
    for i ∈ 0..length(tricmaximumcount) - 1
        latencyTmaxi ← RL2maxTi - RL1Ti
         $\frac{\text{latencyTmax}}{\text{SF}}$ 

```

```

RisetimeT:= for i ∈ 0..length(tricmaximumcount) - 1
              for k ∈ 0
                RisetimeTi,k ← RTETi,k - RTSTi,k if tricmaximumcounti > 0
              RisetimeT
              SF

RTETmax:= for i ∈ 0..length(tricmaximumcount) - 1
            RTETmaxi ← samplrefendmaxTi
          RTETmax

RisetimemaxT:= for i ∈ 0..length(tricmaximumcount) - 1
                RisetimemaxTi ← RTETmaxi - RTSTmaxi
              RisetimemaxT
              SF

RMinT:= for i ∈ 0..length(tricmaximumcount) - 1
          RMinTi ← (STrici)[ (tricminimum<math>\langle \dot{v} \rangle</math>)0 ]
        RMinT

RMaxT:= for i ∈ 0..length(STric) - 1
          RMaxTi ← (STrici)(tricmaximum<math>\langle \dot{v} \rangle</math>)0
        RMaxT

BRT:= for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
          BRTi,k ← (beginreftrici)k if tricmaximumcounti > 0
          BRTi,k ← 0 otherwise
        BRT

FiRT:= for i ∈ 0..length(tricmaximumcount) - 1
        for k ∈ 0
          FiRTi,k ← (finreftrici)k if tricmaximumcounti > 0
          FiRTi,k ← 0 otherwise
        FiRT

BRTmax:= for i ∈ 0..length(STric) - 1
          BRTmaxi ← beginreftricmaxi
        BRTmax

FiRTmax:= for i ∈ 0..length(STric) - 1
          FiRTmaxi ← finreftricmaxi
        FiRTmax

ActivesignalmaxT:= for i ∈ 0..length(tricmaximumcount) - 1
                    ActivesignalmaxTi ←  $\frac{\text{FiRTmax}_i - \text{BRTmax}_i}{\text{SF}}$ 
                  ActivesignalmaxT

```

$\text{ActivesignalT} := \begin{array}{|l} \text{for } i \in 0.. \text{length}(\text{tricmaximumcount}) - 1 \\ \quad \text{for } k \in 0 \\ \quad \quad \text{ActivesignalT}_{i,k} \leftarrow \frac{\text{FiRT}_{i,k} - \text{BRT}_{i,k}}{\text{SF}} \text{ if } \text{tricmaximumcount}_i > 0 \\ \text{ActivesignalT} \end{array}$

$\text{ActivesignalmatrixT} := \begin{array}{|l} \text{for } i \in 0.. \text{length}(\text{tricmaximumcount}) - 1 \\ \quad \text{for } k \in 0 \\ \quad \quad \text{ActivesignalmatrixT}_{i,k} \leftarrow \text{submatrix}(\text{STric}_i, \text{BRT}_{i,k}, \text{FiRT}_{i,k}, 0, 0) \text{ if } \text{tricmaximumcount}_i > 0 \\ \text{ActivesignalmatrixT} \end{array}$

$\text{ActivesignalmaxmatrixT} := \begin{array}{|l} \text{for } i \in 0.. \text{length}(\text{tricmaximumcount}) - 1 \\ \quad \text{ActivesignalmaxmatrixT}_i \leftarrow \text{submatrix}(\text{STric}_i, \text{BRTmax}_i, \text{FiRTmax}_i, 0, 0) \\ \text{ActivesignalmaxmatrixT} \end{array}$

$\text{ActivesignalareaT} := \begin{array}{|l} \text{for } i \in 0.. \text{length}(\text{tricmaximumcount}) - 1 \\ \quad \text{for } k \in 0 \\ \quad \quad \text{ActivesignalareaT}_{i,k} \leftarrow \text{integral}(\text{ActivesignalmatrixT}_{i,k}) \text{ if } \text{tricmaximumcount}_i > 0 \wedge \text{BRT}_{i,k} \neq \text{FiRT}_{i,k} \\ \text{ActivesignalareaT} \end{array}$

$\text{ActivesignalmaxareaT} := \begin{array}{|l} \text{for } i \in 0.. \text{length}(\text{tricmaximumcount}) - 1 \\ \quad \text{ActivesignalmaxareaT}_i \leftarrow \text{integral}(\text{ActivesignalmaxmatrixT}_i) \text{ if } \text{rows}(\text{ActivesignalmaxmatrixT}_i) > 0 \\ \text{ActivesignalmaxareaT} \end{array}$

Raw Biceps Analysis

$\text{RawBicsub} := \begin{array}{|l} \text{for } i \in 0.. \text{length}(\text{Bic}) - 1 \\ \quad \text{RawBicsub}_i \leftarrow \text{submatrix}(\text{Bic}_i, \text{BR}_i, \text{FiR}_i, 0, 0) \\ \text{RawBicsub} \end{array}$

$\text{Rawbicamp} := \begin{array}{|l} \text{for } i \in 0.. \text{length}(\text{RawBicsub}) - 1 \\ \quad \text{Rawbicamp}_i \leftarrow \max(\text{RawBicsub}_i) \\ \text{Rawbicamp} \end{array}$

$\text{Rawbicampsamp} := \begin{array}{|l} \text{for } i \in 0.. \text{length}(\text{Bic}) - 1 \\ \quad \text{Rawbicampsamp}_i \leftarrow \text{samp}(\text{Bic}_i, \text{Rawbicamp}_i) \\ \text{Rawbicampsamp} \end{array}$

Raw Triceps Analysis

$\text{RawTricsub} := \begin{array}{|l} \text{for } i \in 0.. \text{length}(\text{Tric}) - 1 \\ \quad \text{RawTricsub}_i \leftarrow \text{submatrix}(\text{Tric}_i, \text{BRT}_i, \text{FiRT}_i, 0, 0) \\ \text{RawTricsub} \end{array}$

Rawtricamp := $\left\{ \begin{array}{l} \text{for } i \in 0.. \text{length}(\text{RawTricsub}) - 1 \\ \quad \text{Rawtricamp}_i \leftarrow \max(\text{RawTricsub}_i) \\ \text{Rawtricamp} \end{array} \right.$

Final parameters and markers

Raw Biceps

Rawbicamp

Bicrangepos

Smooth Biceps

SBicrt := $\frac{\text{RTE} - \text{RL2}}{\text{SF}}$

SBicdur := $\frac{\text{FiR} - \text{RL2}}{\text{SF}}$

SBiclat := $\frac{\text{RL2} - \text{RL1}}{\text{SF}}$

Raw Triceps

Rawtricamp

Tricrangepos

Smooth Triceps

STricrt := $\frac{\text{RTET} - \text{RL2T}}{\text{SF}}$

STricdur := $\frac{\text{FiRT} - \text{RL2T}}{\text{SF}}$

STriclat := $\frac{\text{RL2T} - \text{RL1}}{\text{SF}}$

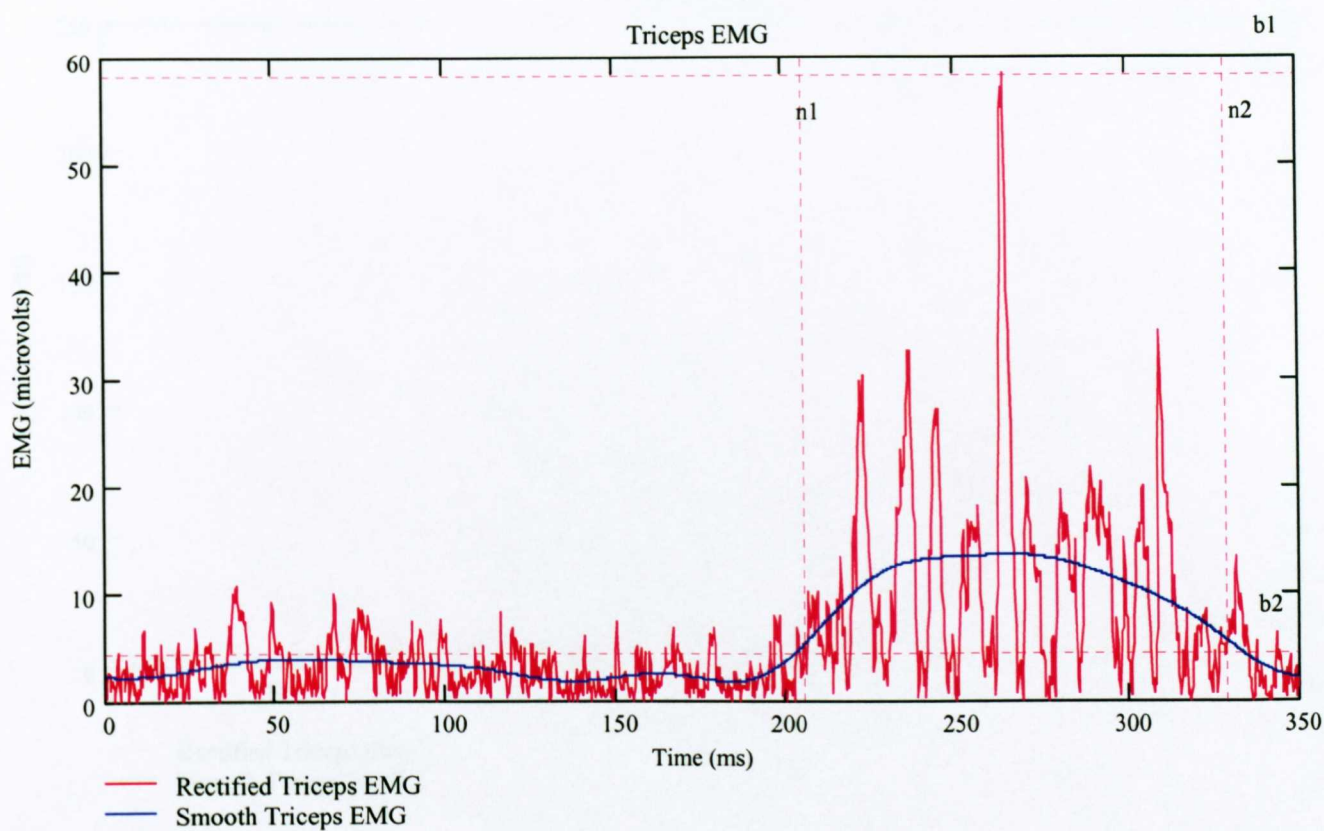
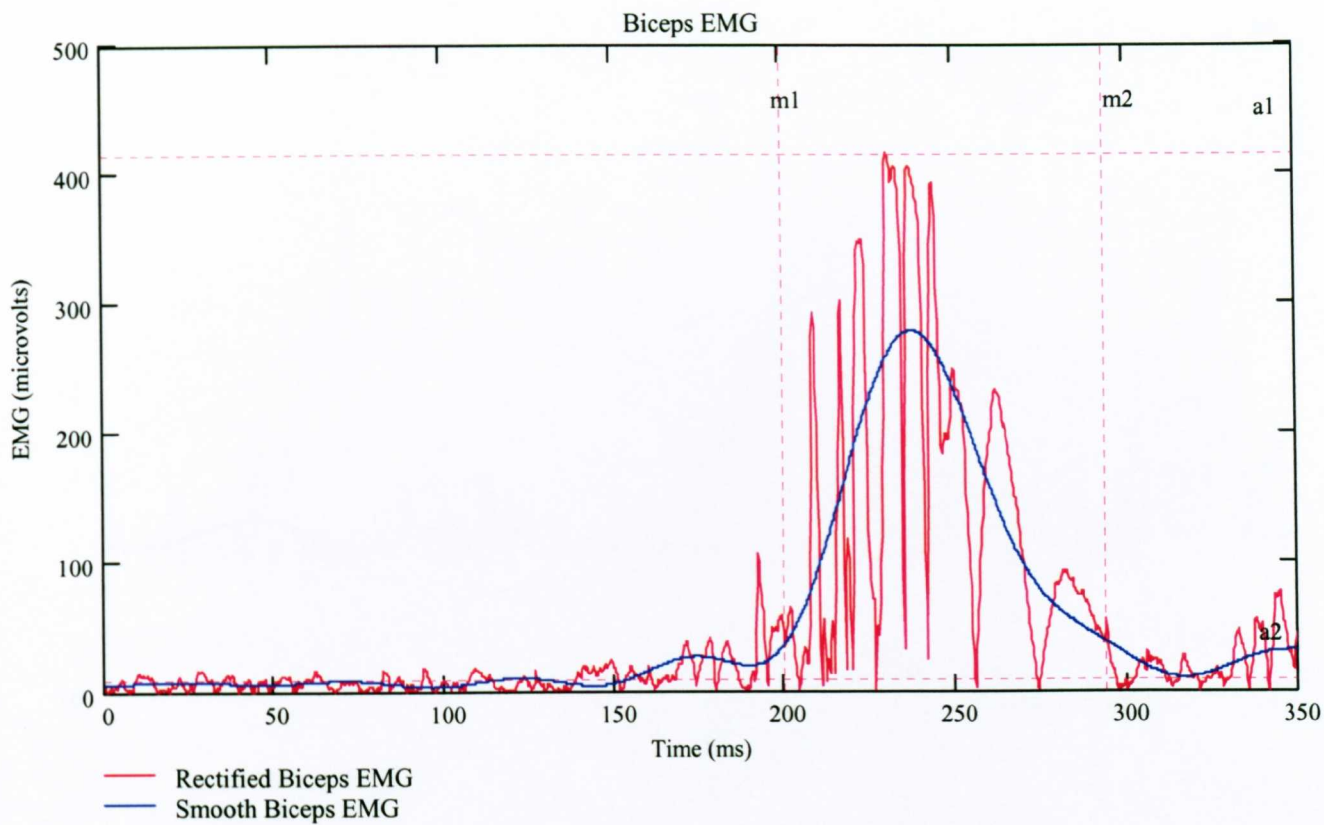
Angular position

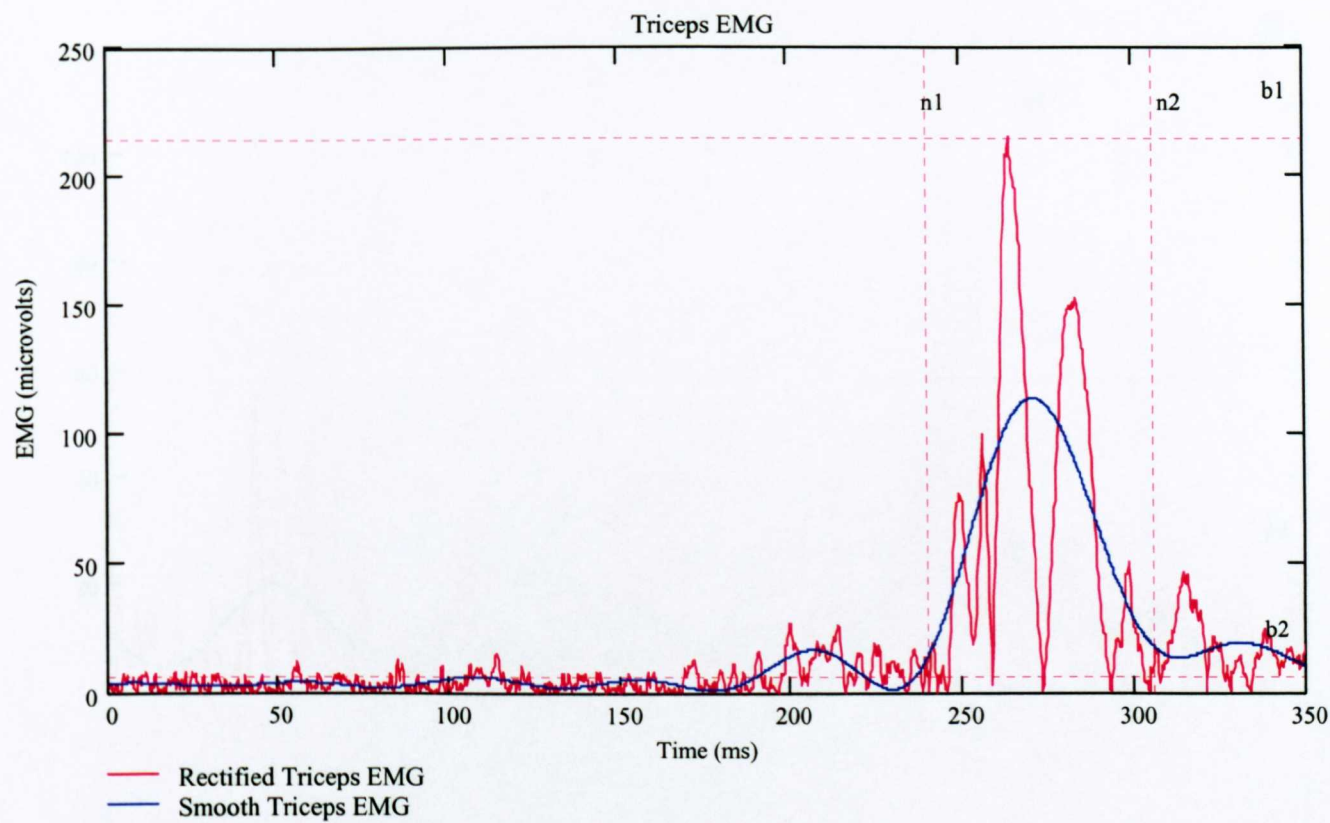
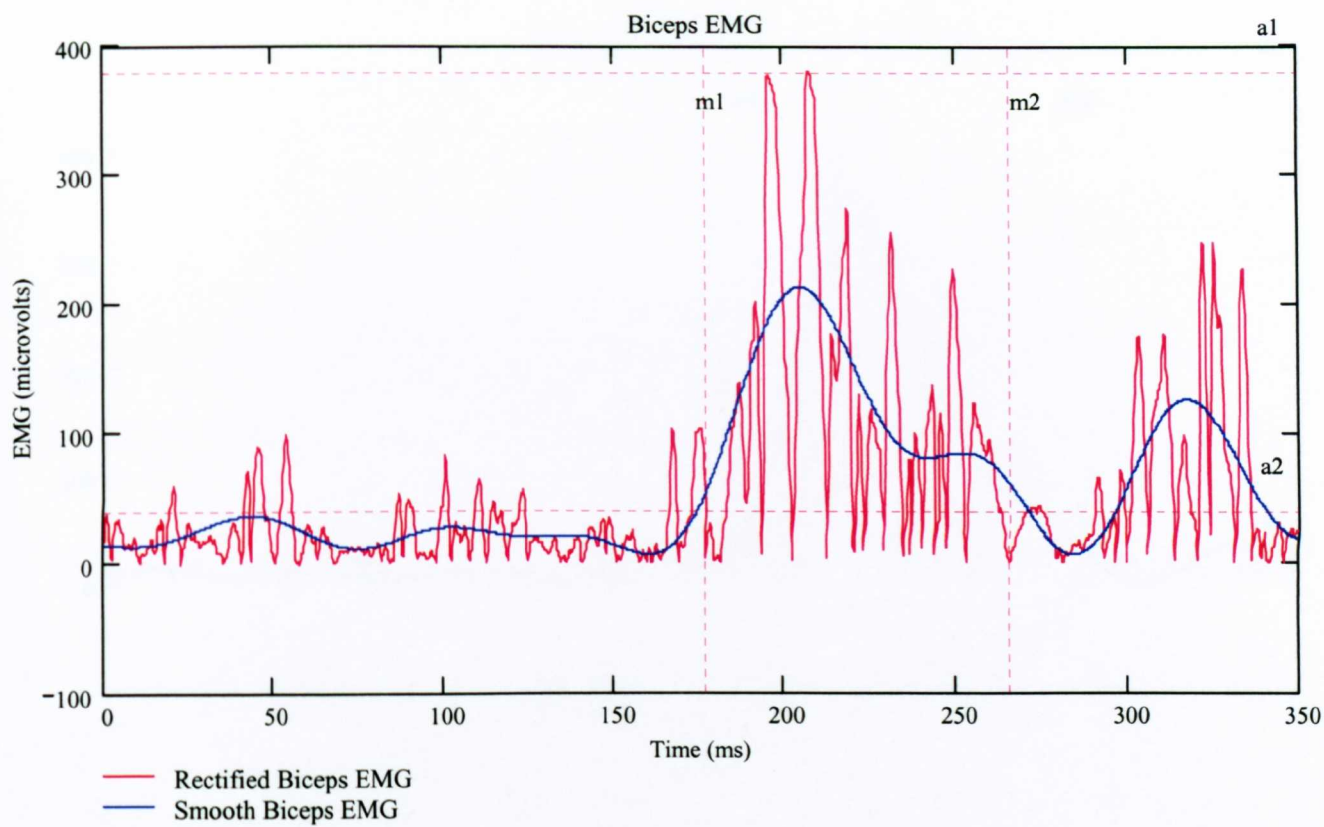
RT := $\frac{\text{movend} - \text{movstart}}{\text{SF}}$

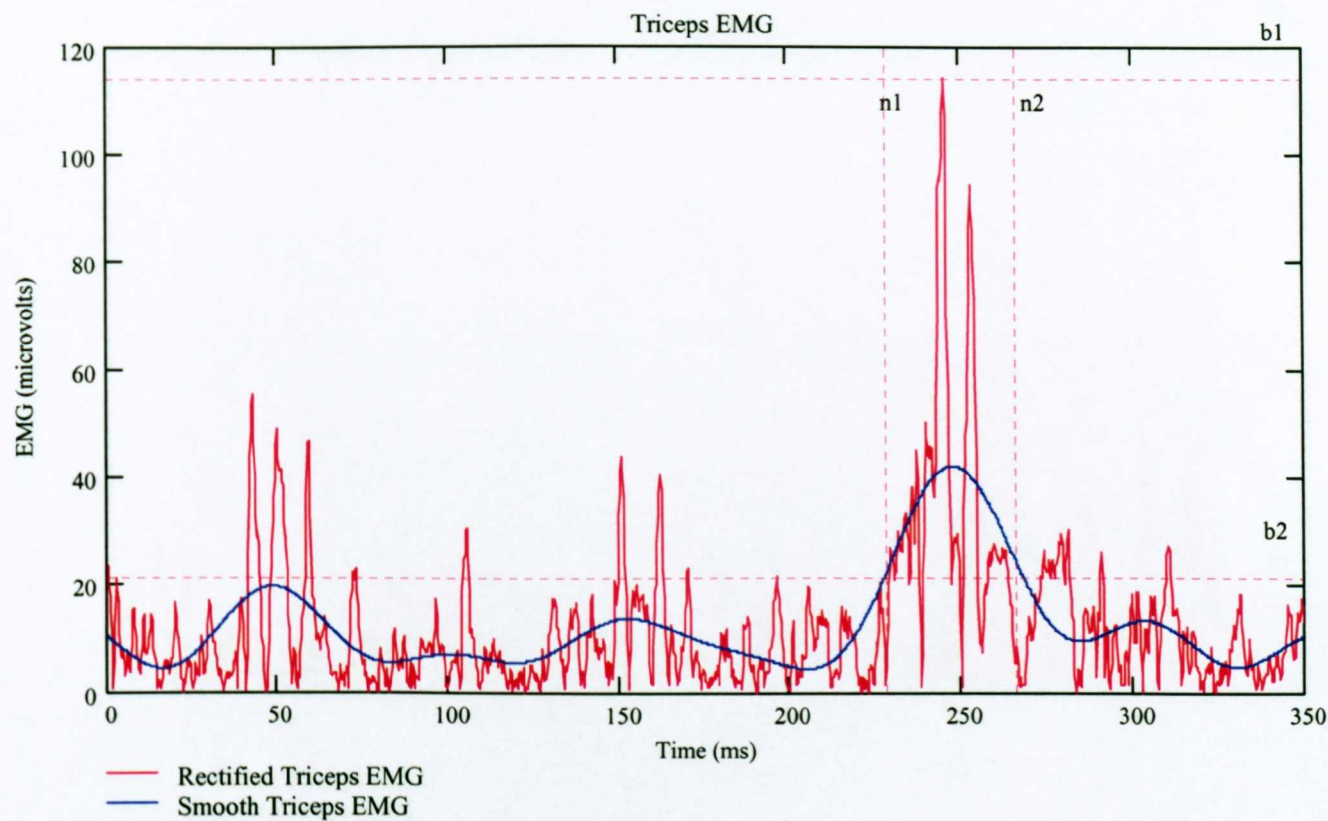
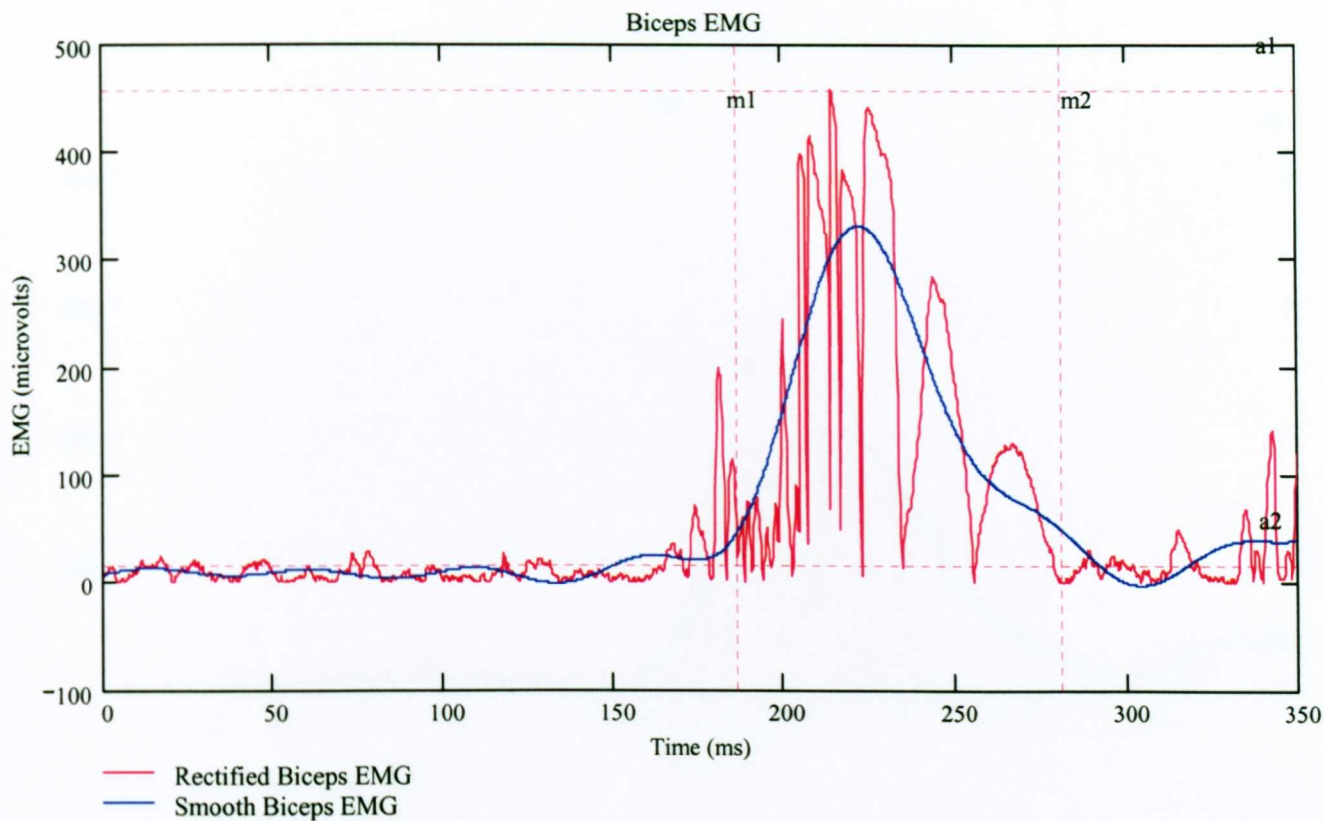
ROM := movendang - movstartang

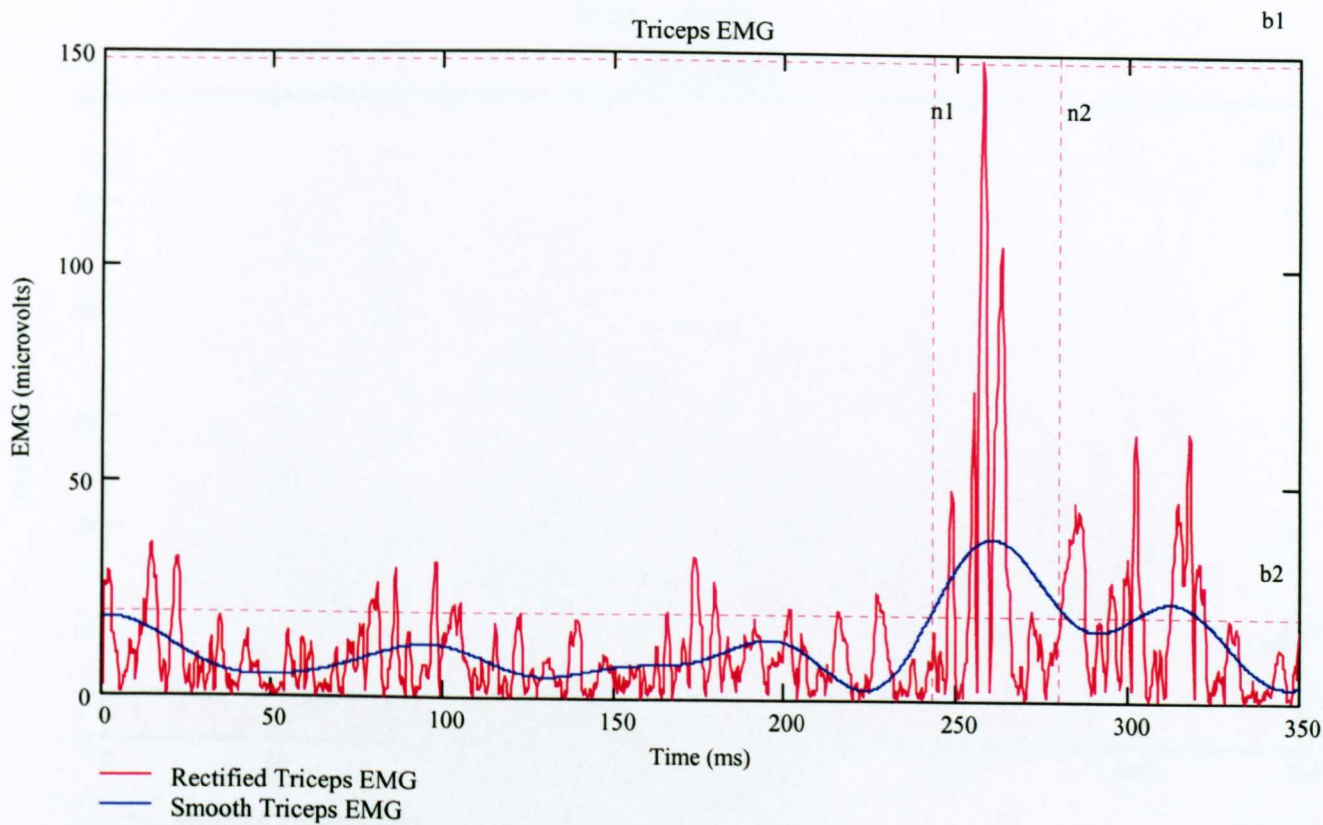
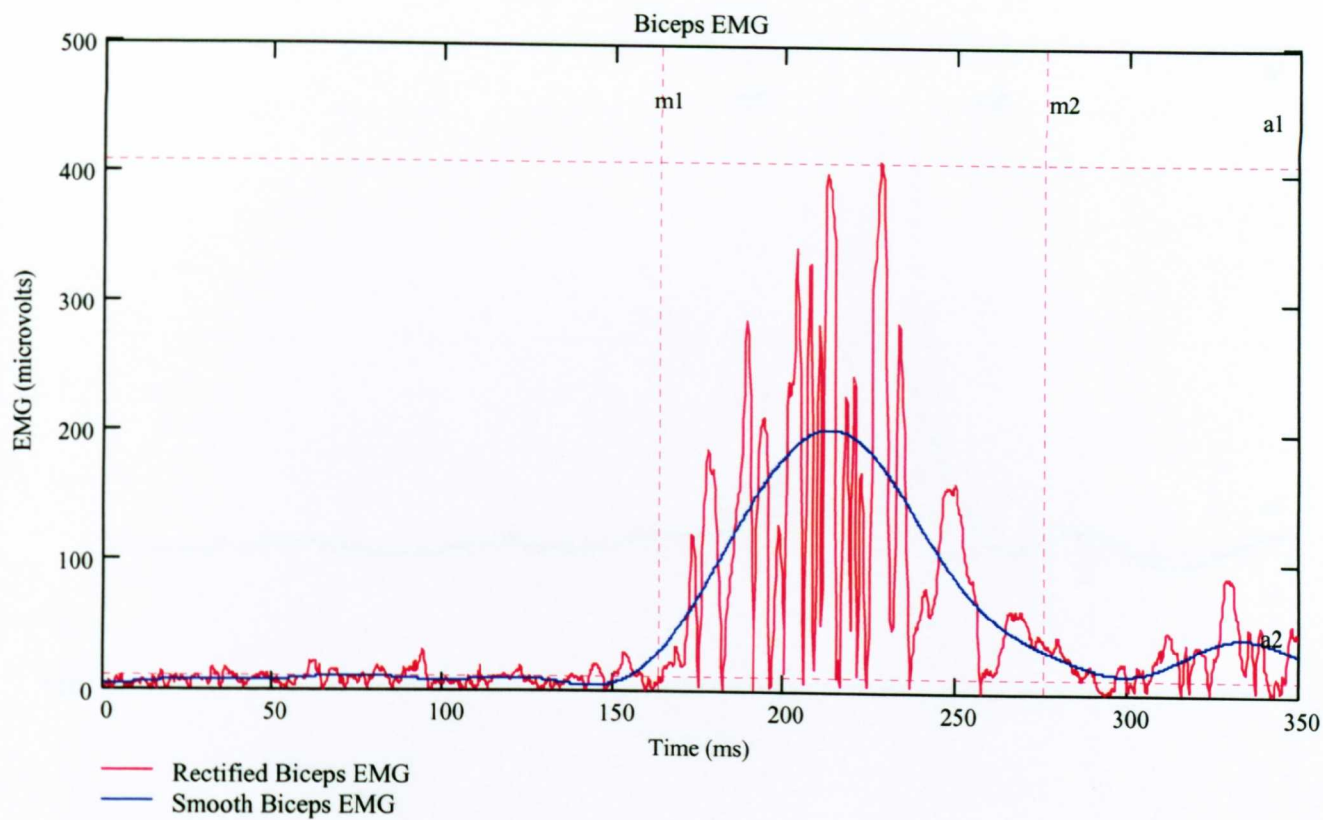
Average angular velocity

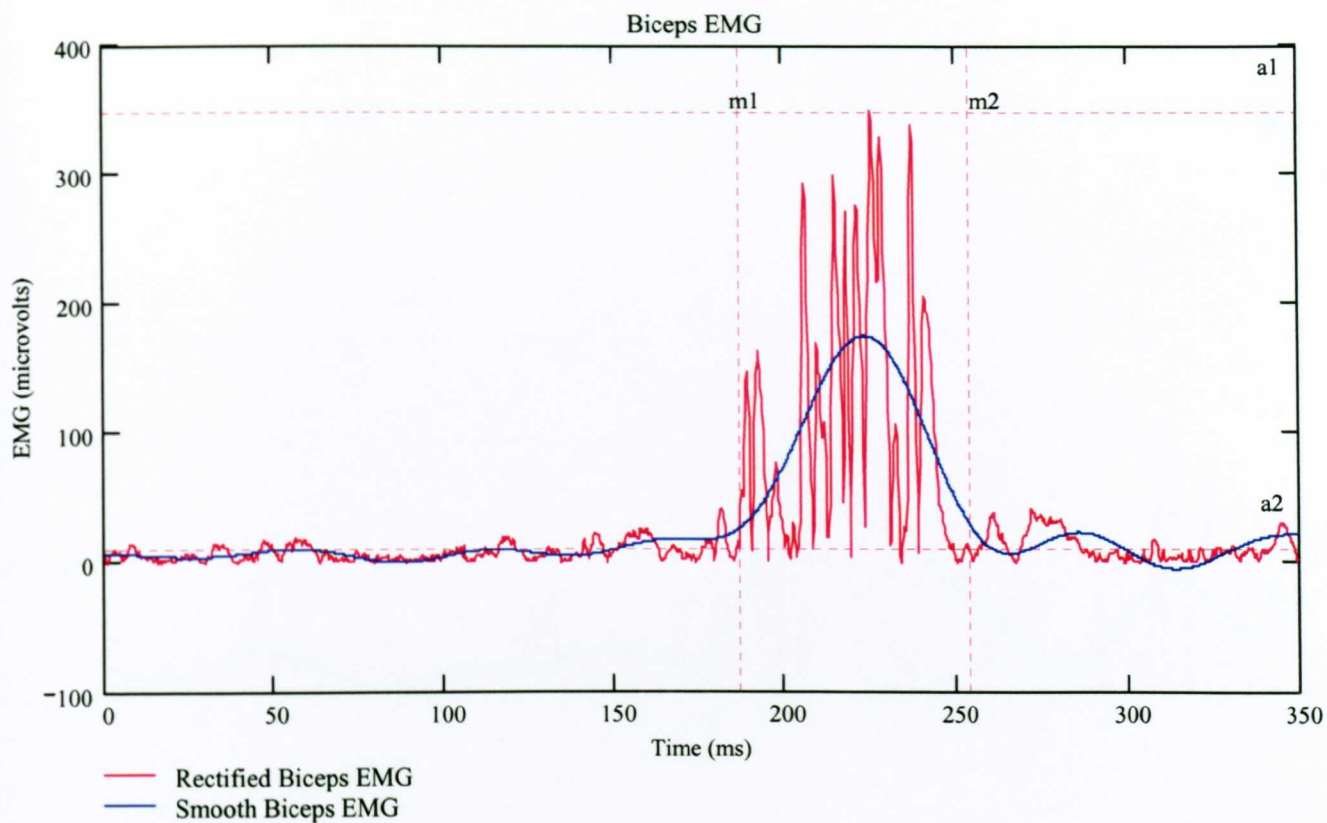
ANGVE := $\left\{ \begin{array}{l} \text{for } k \in 0.. \text{length}(\text{SBic}) - 1 \\ \quad \text{ANGVE}_k \leftarrow \frac{\text{ROM}_k}{\text{RT}_k} \\ \text{ANGVE} \end{array} \right.$



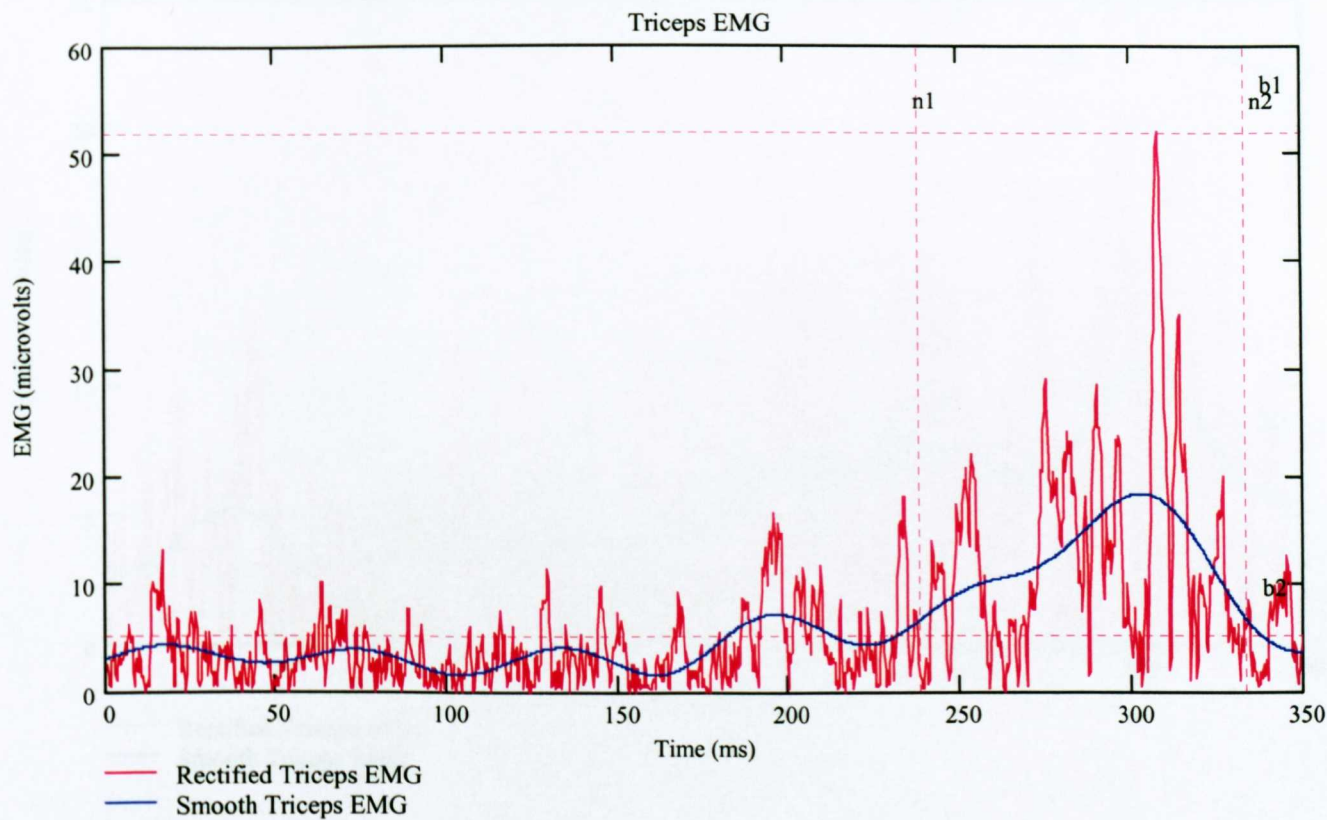


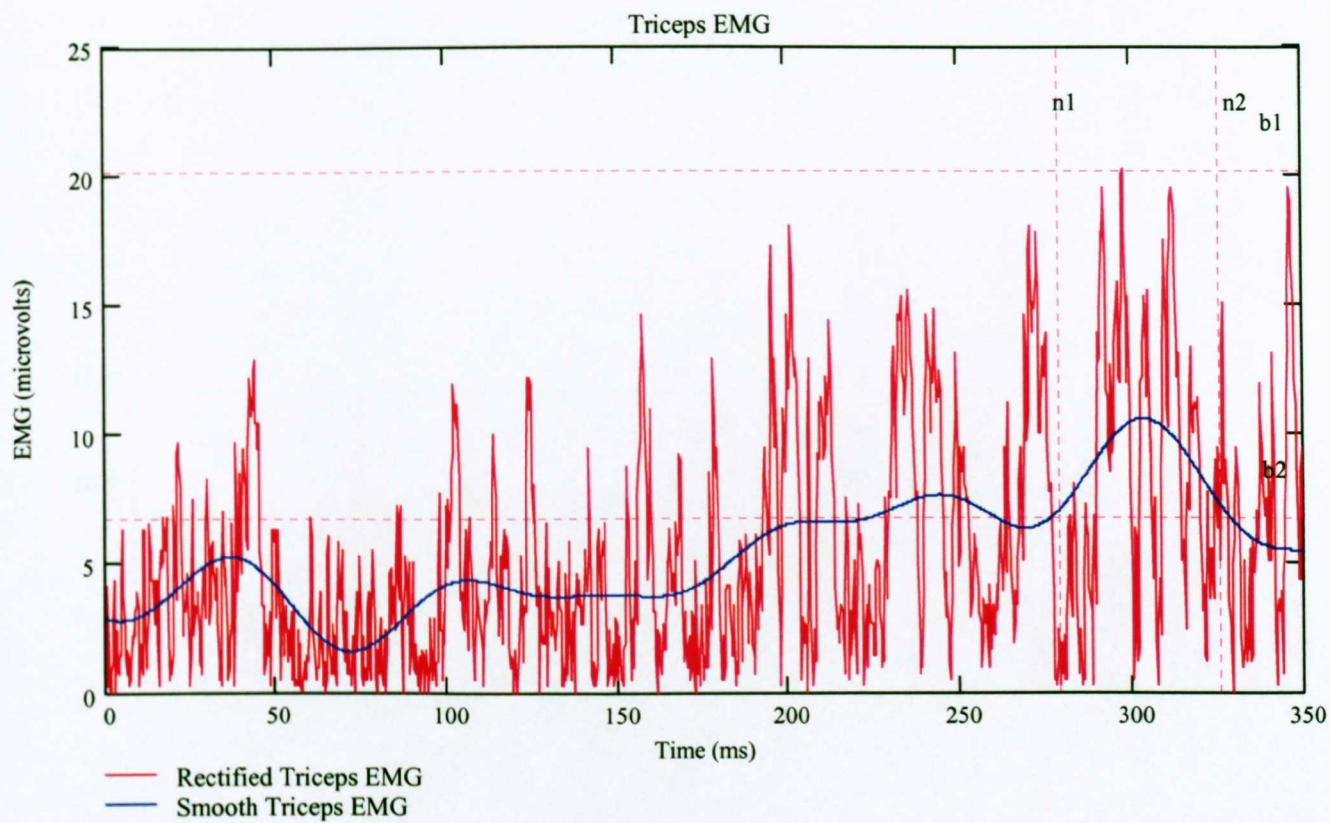
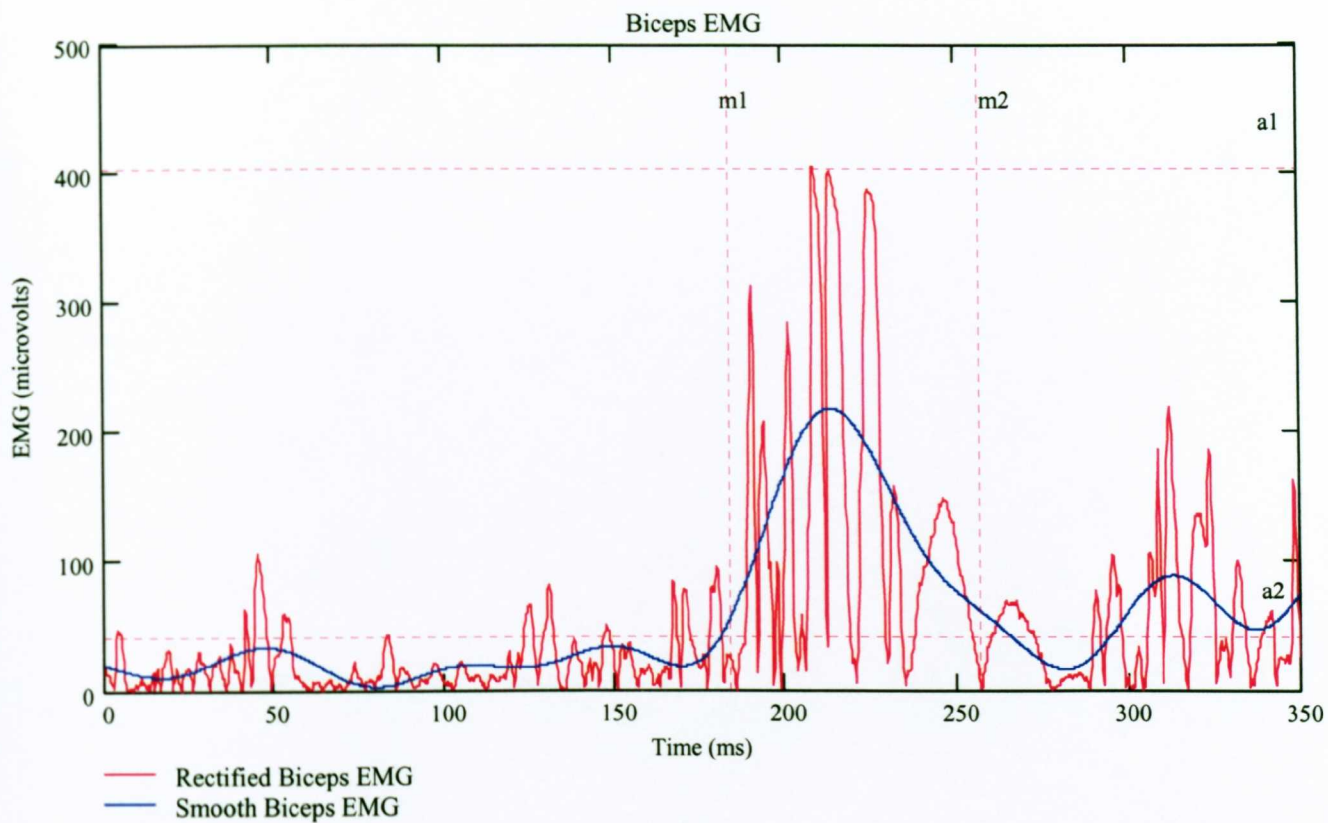


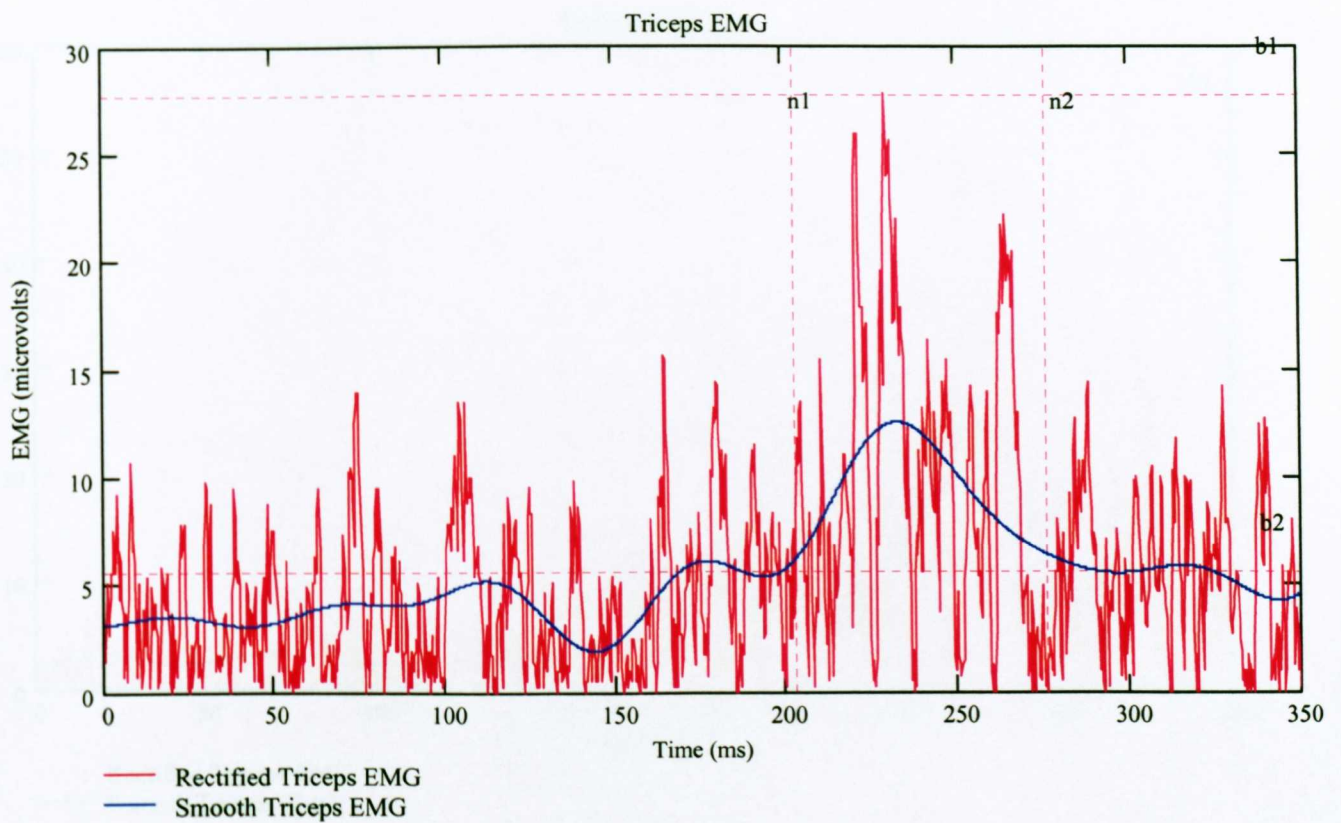
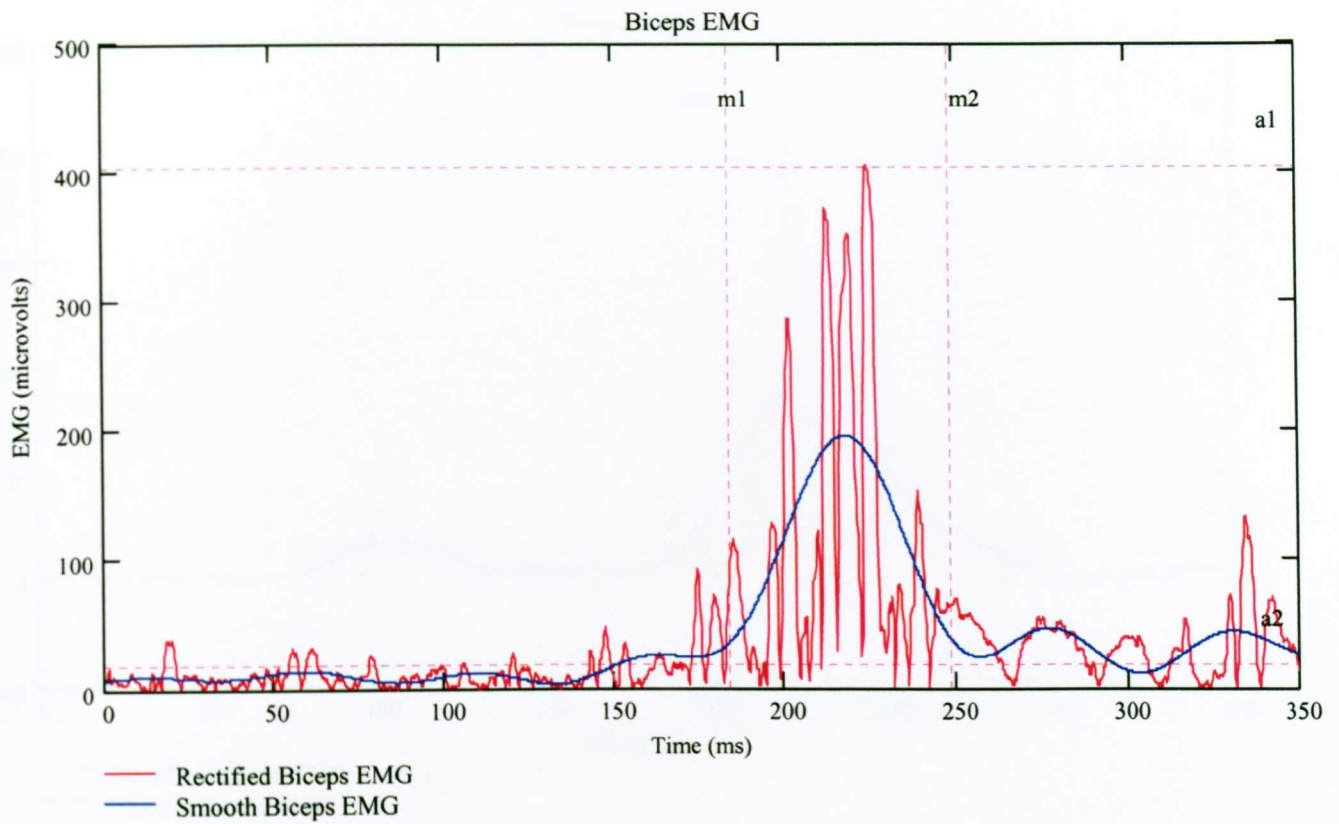


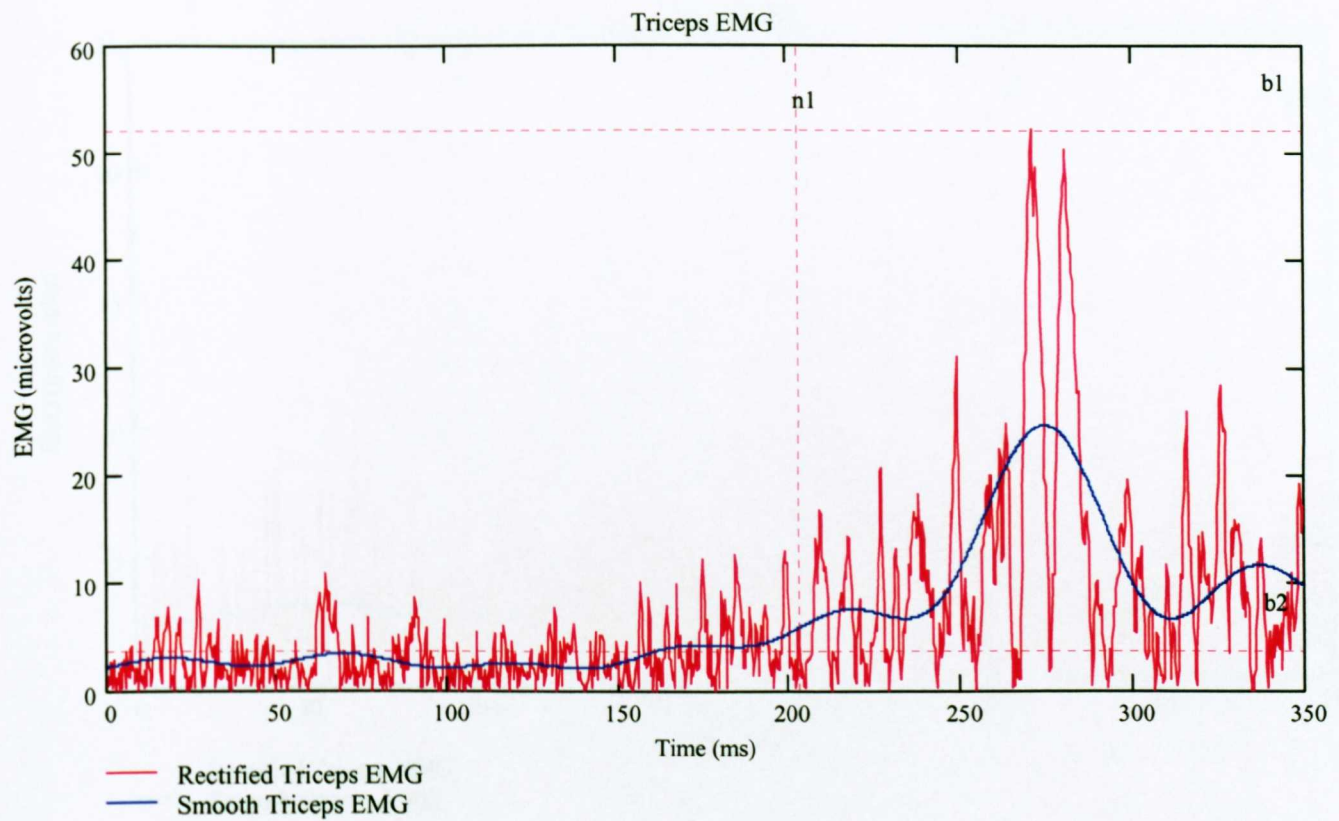
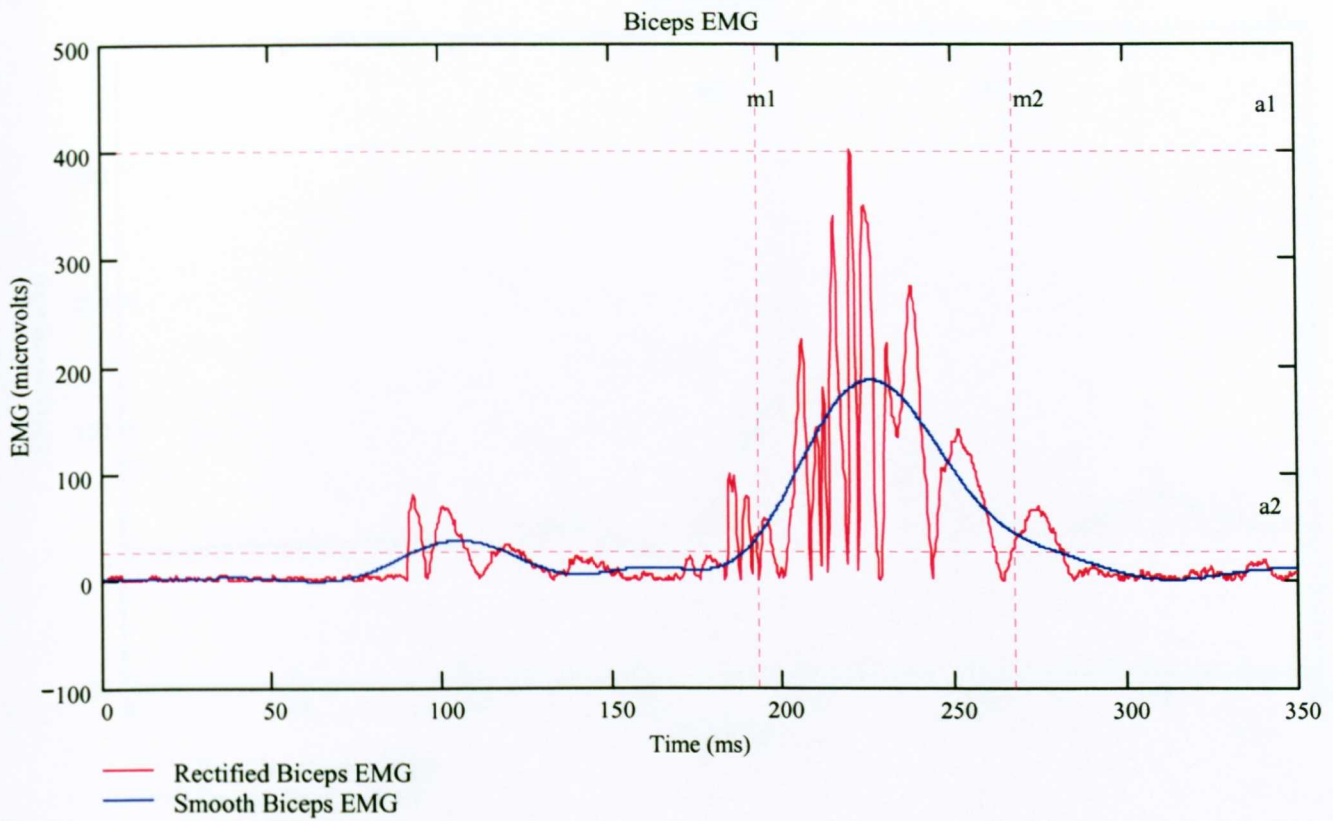


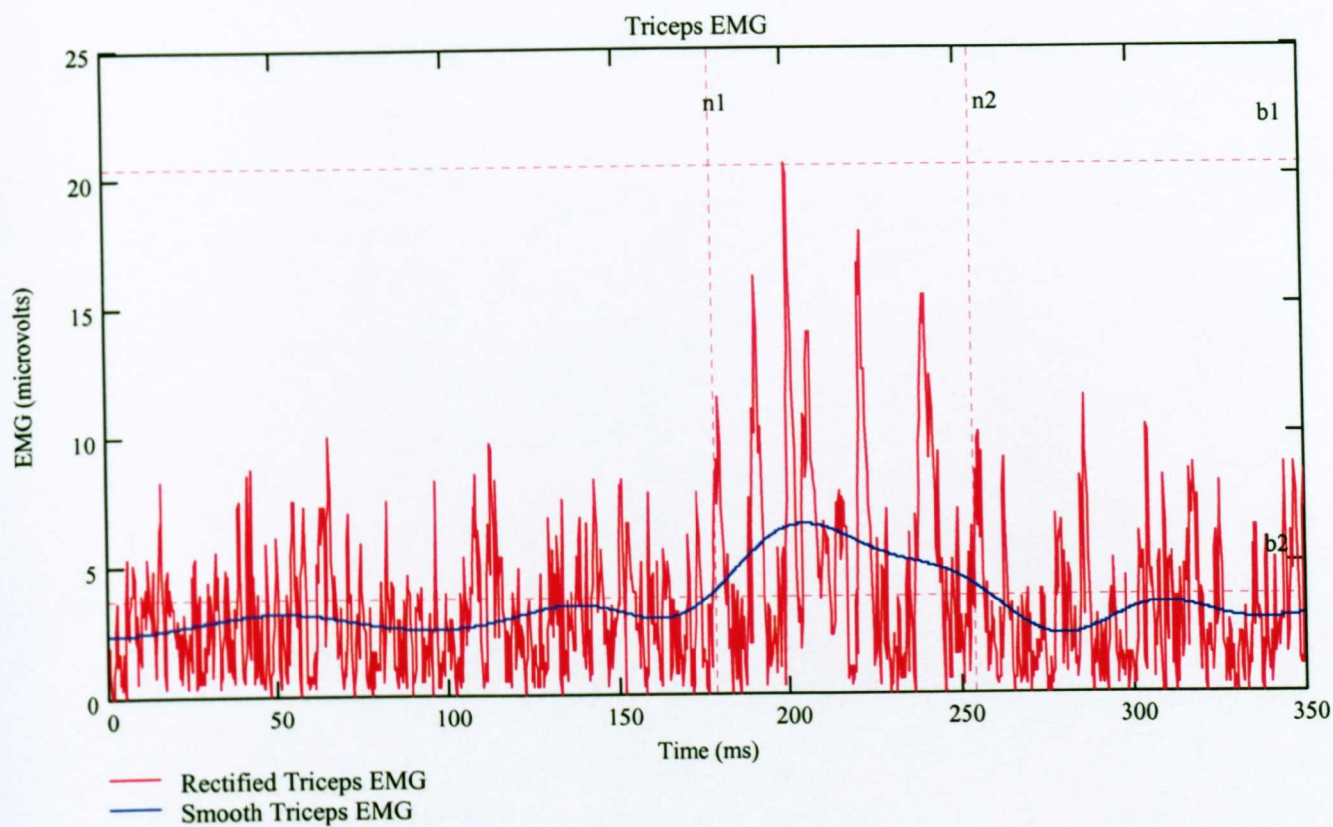
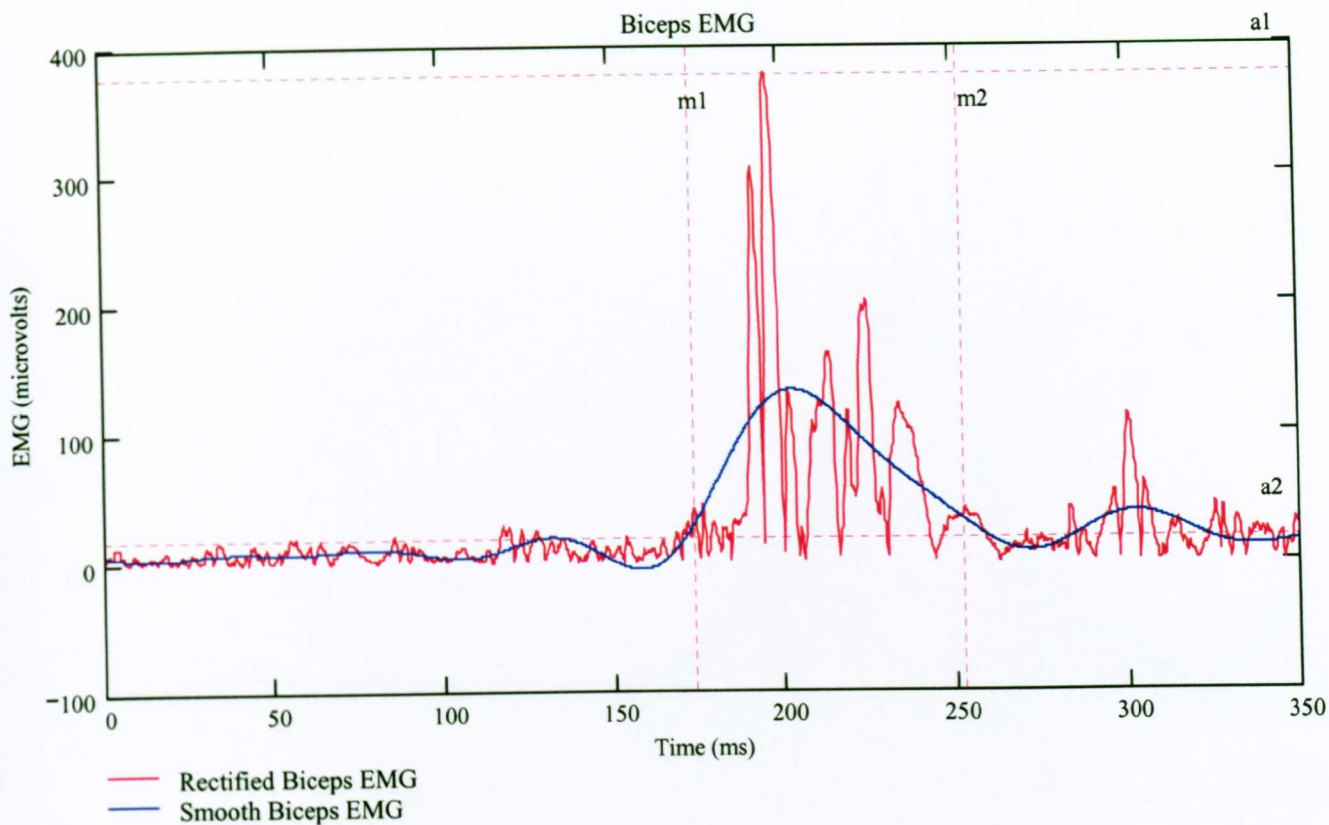
$$ROM_i = -26.442$$

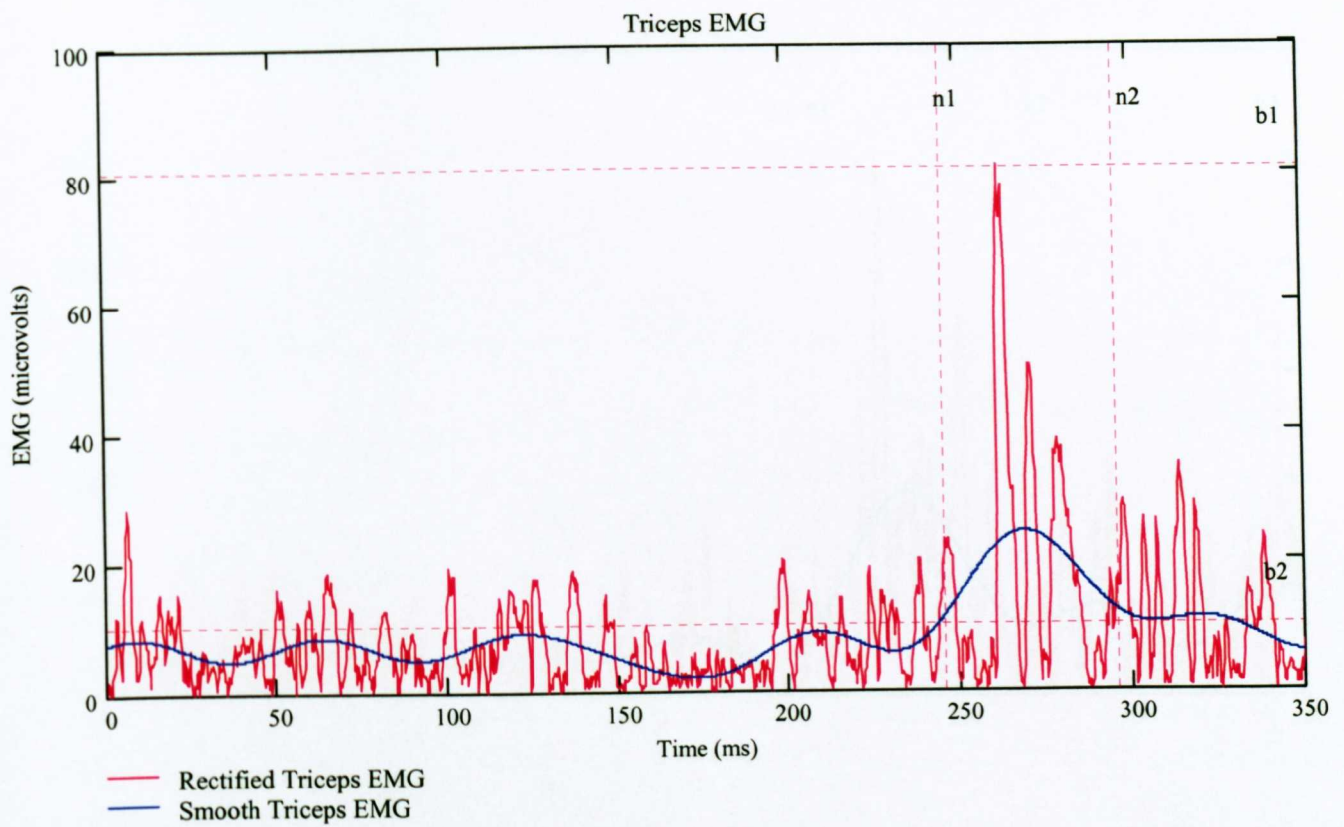
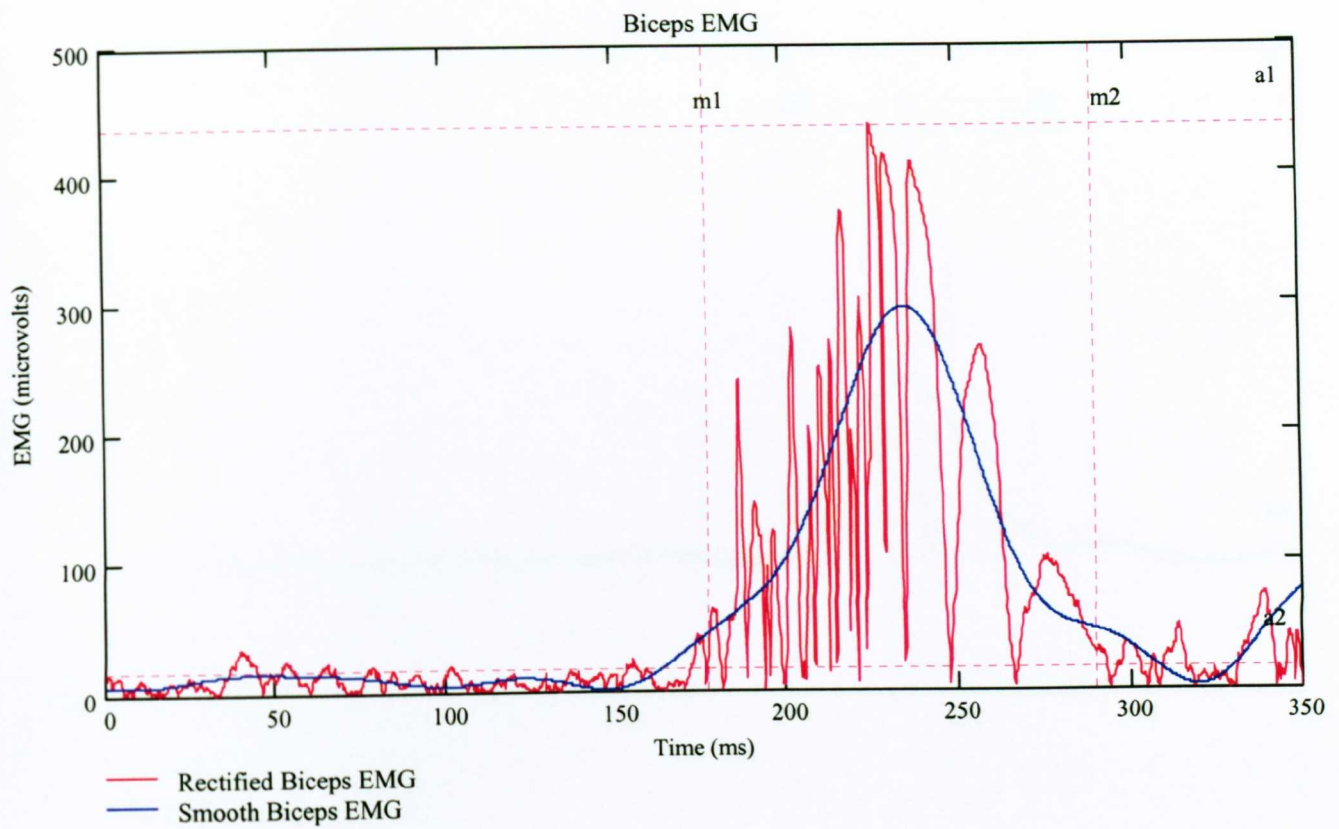


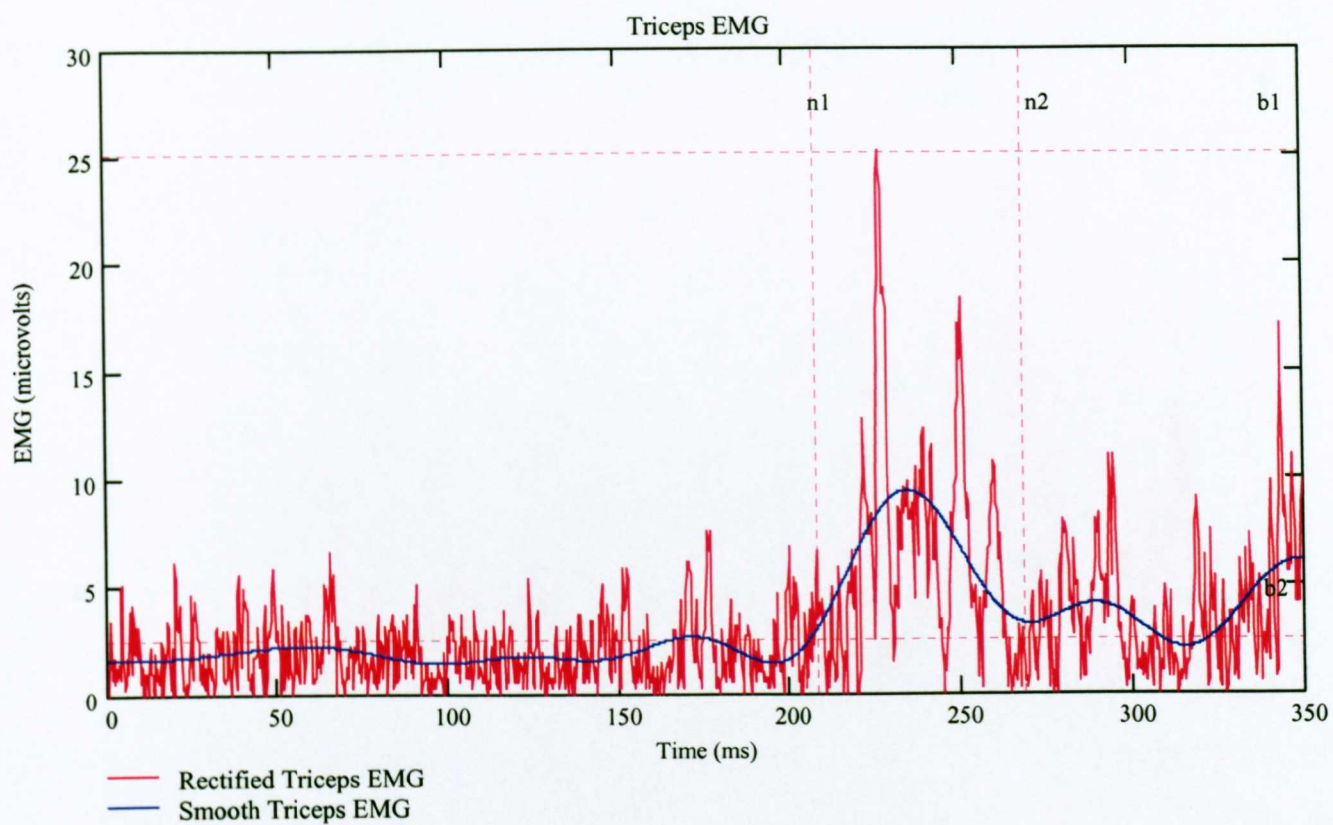
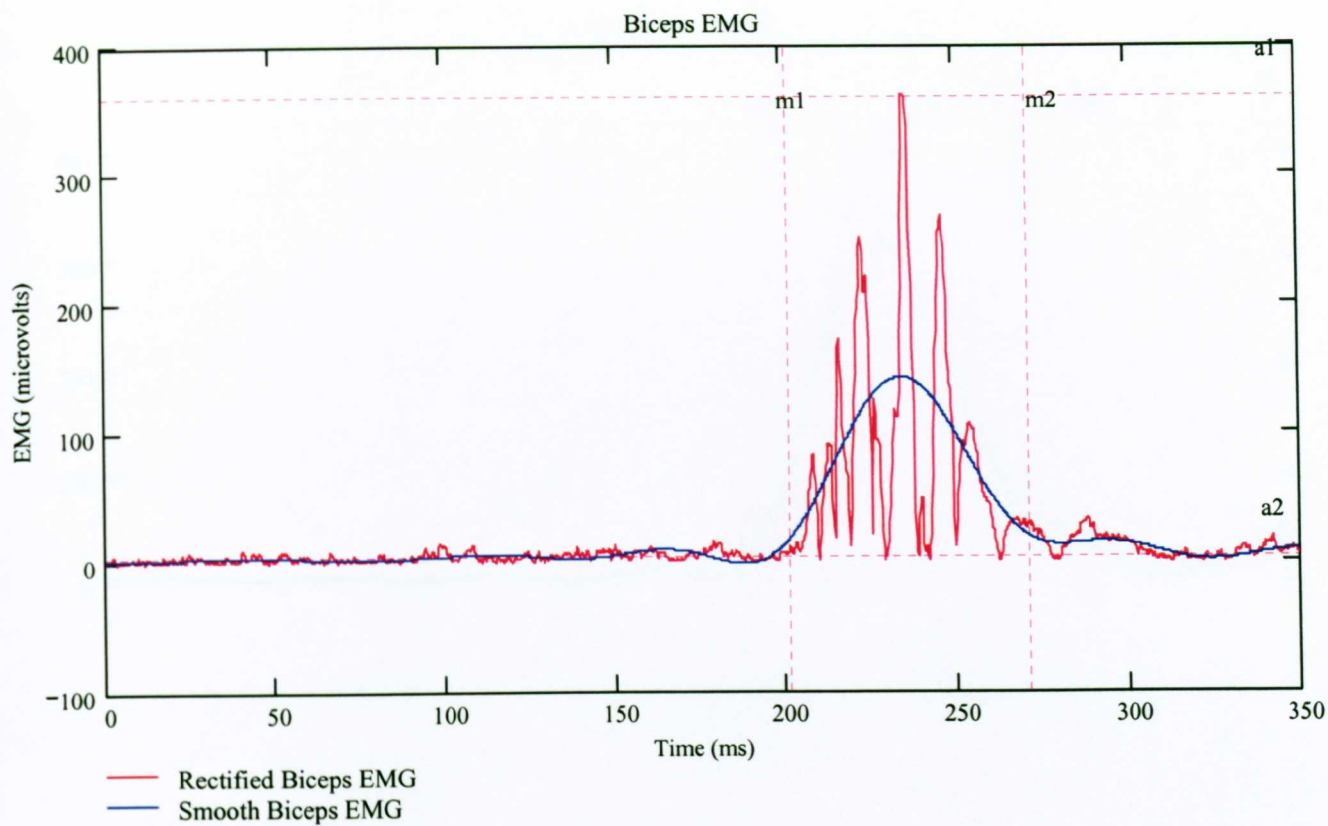


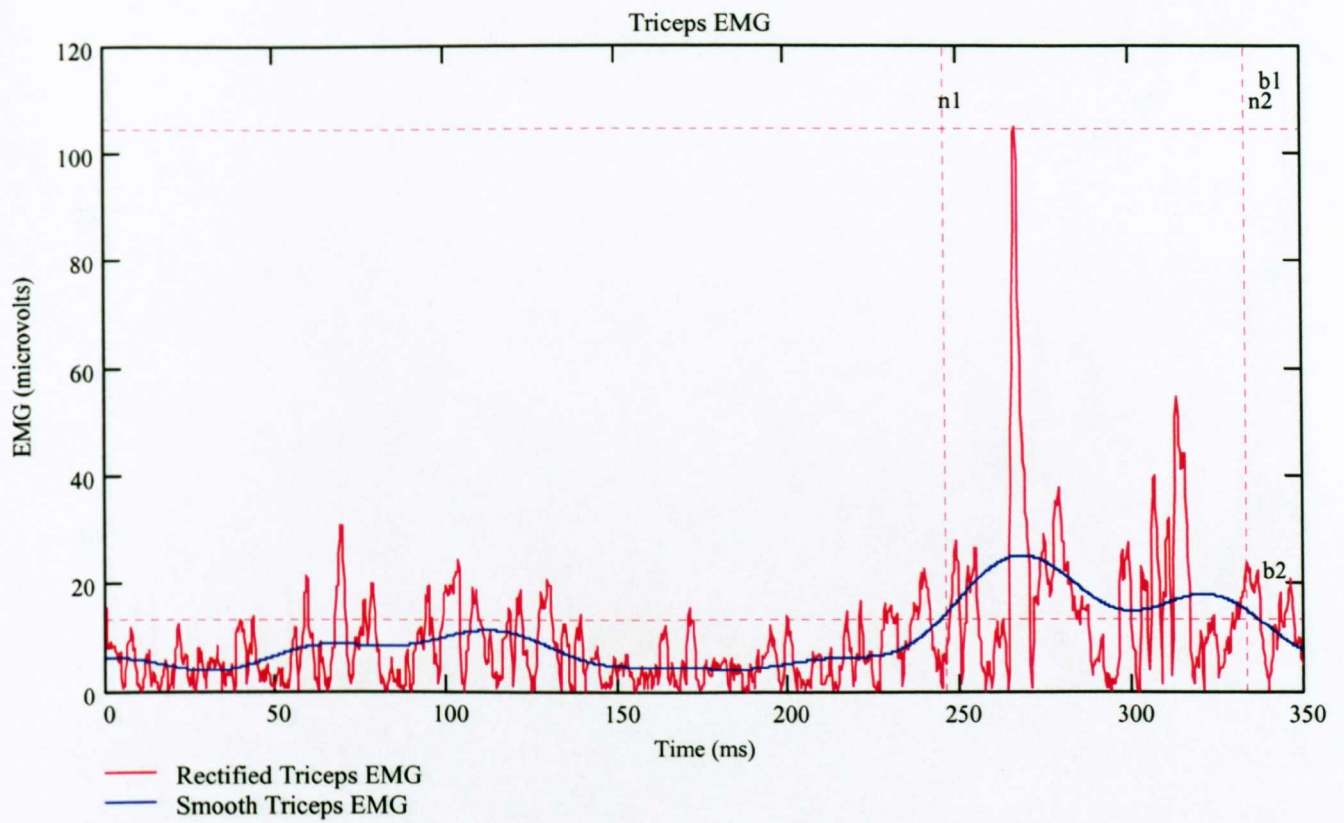
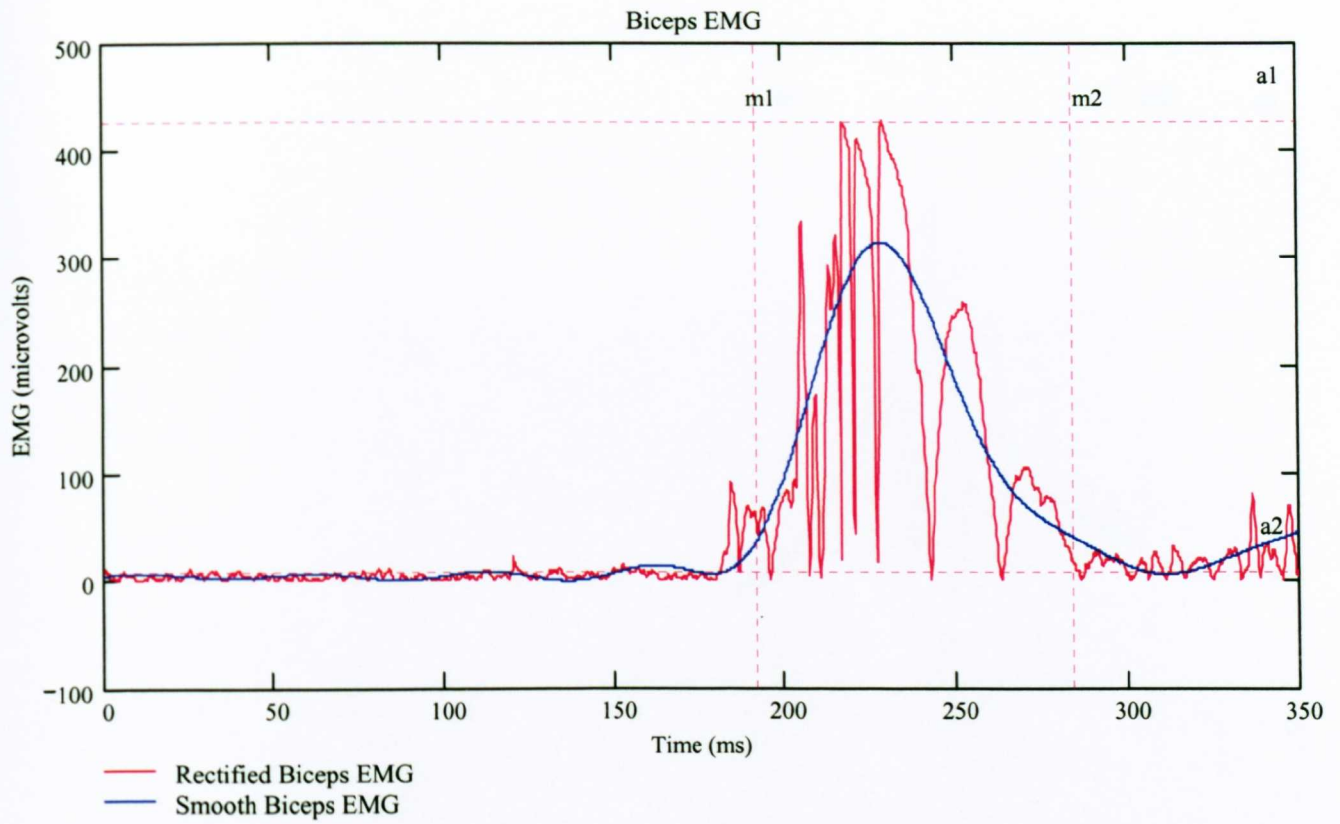


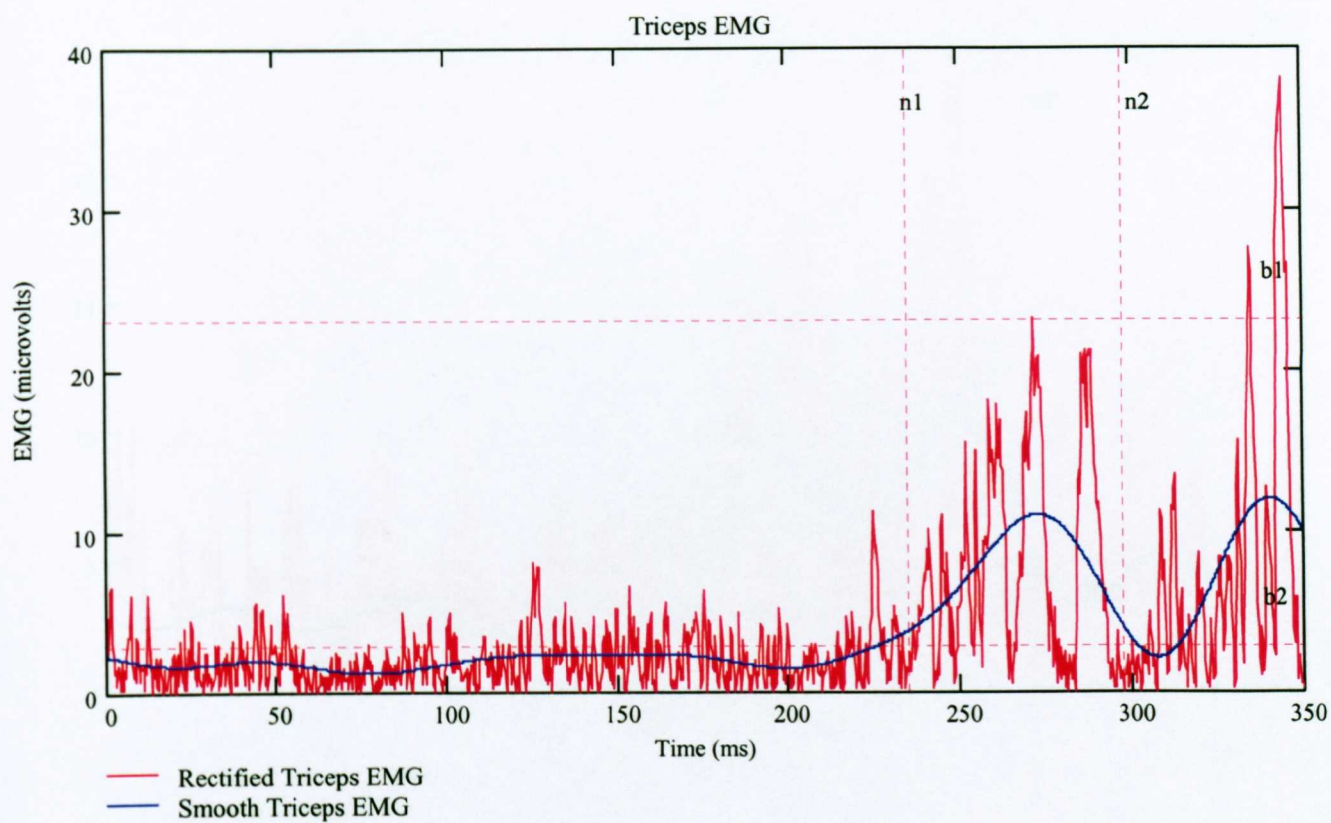
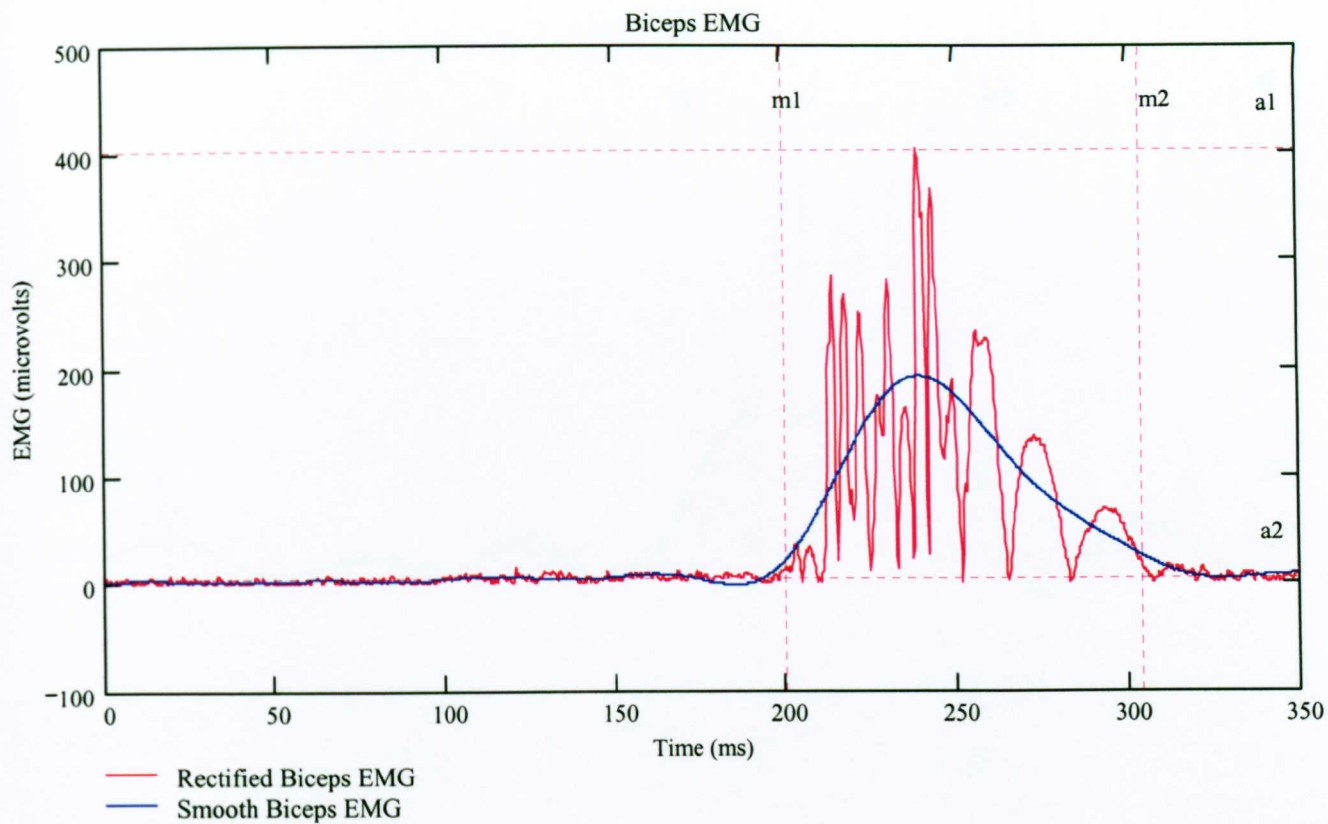


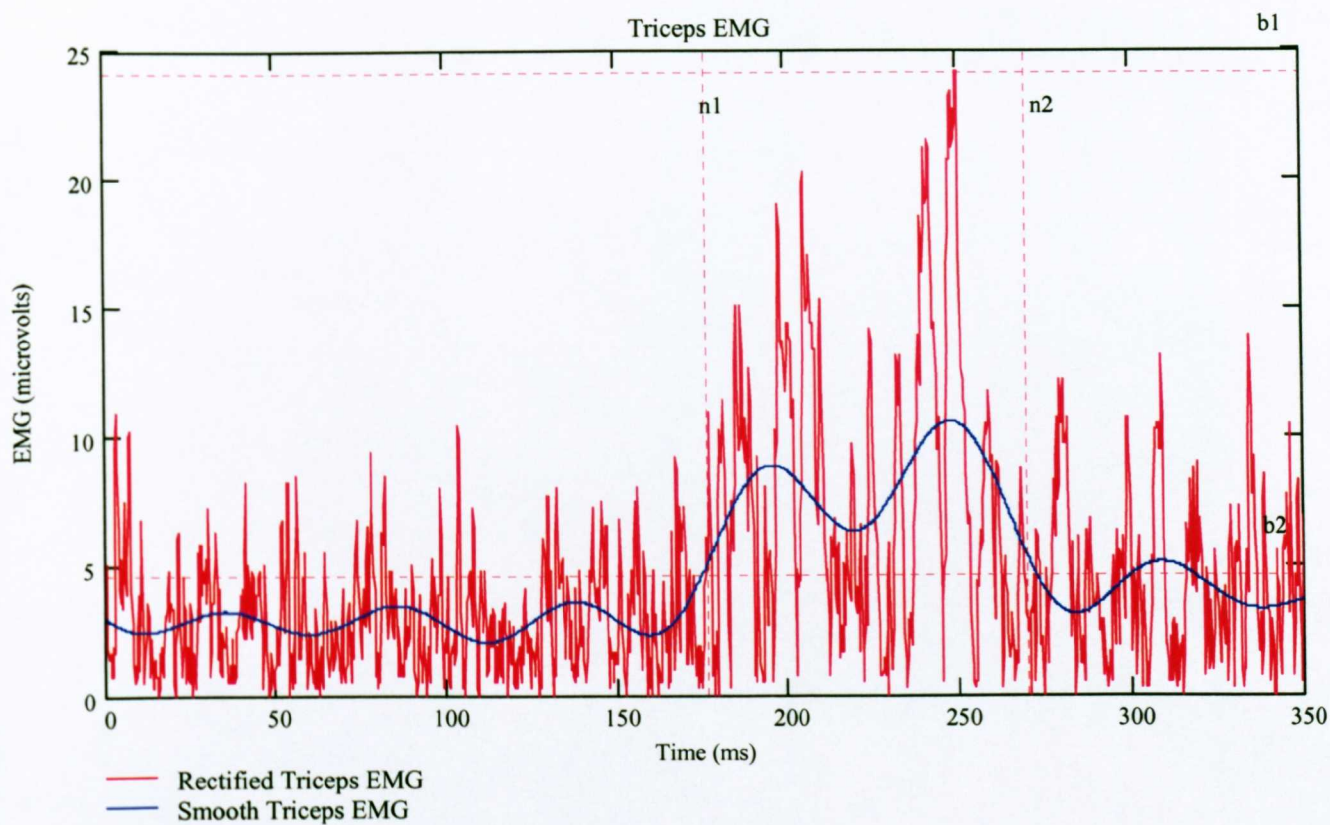
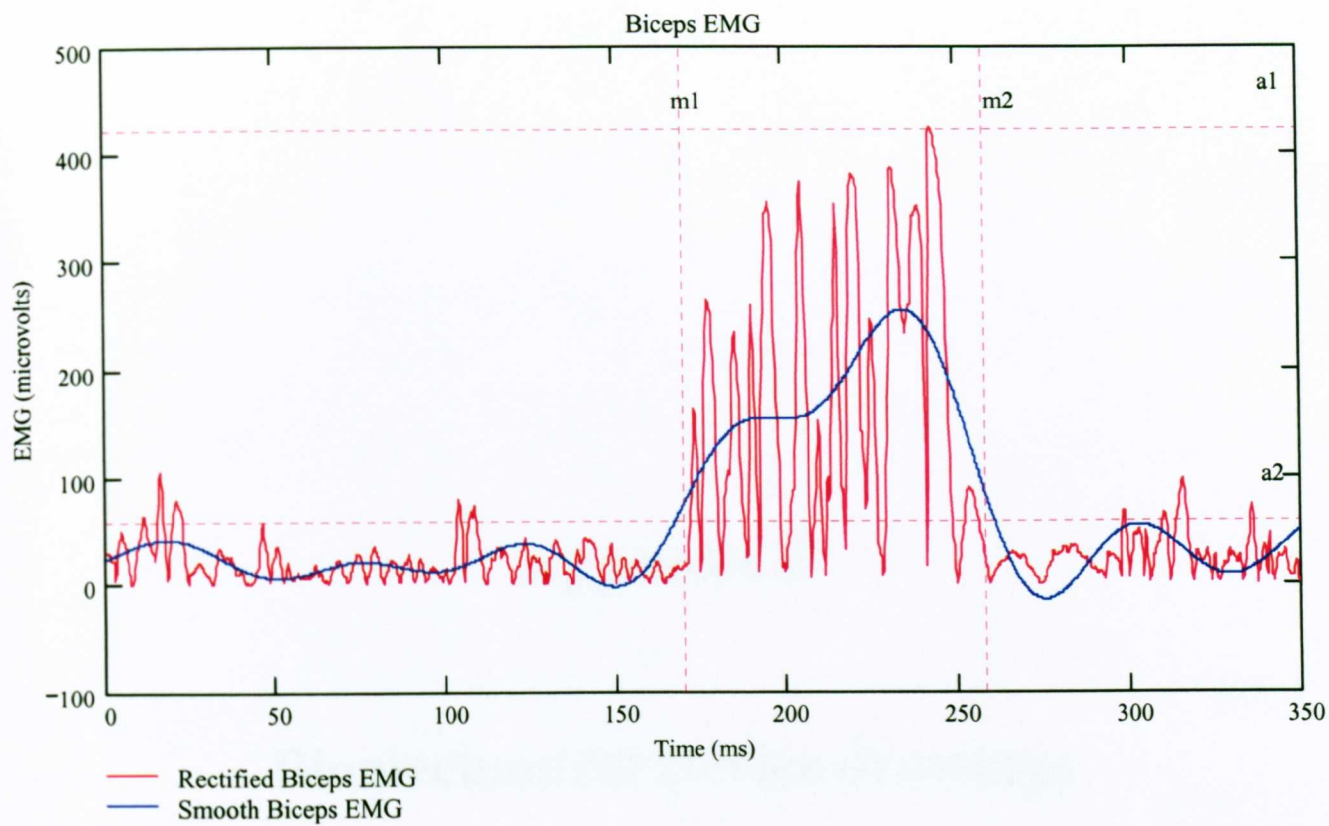












Appendix E

Biomechanical Device drawings

