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Triceps Surae Fatigue, and Its Effects on Intrinsic and Reflex Contributions of Human Ankle Dynamics

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Master of Engineering

Department of Biomedical Engineering

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Abstract

Muscle fatigue may be defined as any exercise-related reduction in the force-generating capacity of a muscle. Its effect on joint stiffness is not well understood. The purpose of this study was: (1) to characterize triceps surae muscle fatigue, and (2) to determine its effect on the dynamic stiffness of the ankle joint. To address the first question, subjects were asked to maintain constant, submaximal, isometric contractions at levels ranging from 10-50% of maximum voluntary contraction (MVC) for as long as they could. Position, torque, and elelctromyograph (EMG) signals were recorded. EMGs from ankle extensors - triceps surae group - and flexors - tibialis anterior – measured the progression of fatigue. Generally, EMG RMS, tracking error and tremor increased with fatigue. The synergistic triceps surae muscles exhibited alternate activity between two states: active and silent. Moreover, as contraction level was increased, both alternation onset time and endurance time decreased. To address the second question, pseudorandom binary sequence (PRBS) displacements were applied before, during, and after fatiguing contractions of the triceps surae. Position and torque were recorded and analyzed to identify the intrinsic and reflex contributions to ankle stiffness. Fatigue caused significant changes in both intrinsic and reflex properties. For intrinsic properties, there was a decrease in the elasticity, and an increase in the viscosity of the joint. For reflex properties, there was an increase in the gain and a change in the reflex dynamics. Thus, it appears that, during fatigue, reflex stiffness increases to compensate, at least in part, for decreased intrinsic stiffness.

Resumé

La fatigue musculaire peut être définie comme étant une réduction reliée à l'exercice de la capacité d'un muscle de générer de la force. Son effet sur (joint stiffness) est mal compris. Le but de cette étude était: (1) de charactériser la fatigue musculaire des (triceps surae) et (2) de déterminer son effet sur la (dynamic stiffness) de la cheville. En adressant le premier objectif, nous avons demandé des sujets de maintenir des contractions isométriques constantes à des niveaux allant de 10-50% de la contraction volontaire maximale aussi longtemps qu'ils pouvaient. Nous avons enregistré la position, le moment, et les signaux électromyographiques. Les signaux électromyographiques des (extensors) et (flexors) mesuraient la progression de la fatigue. En général, (EMG RMS), (tracking error) et (tremor) augmentaient avec la fatigue. Les (synergistic triceps surae muscles) démontraient de l'activité alternant entre deux états: actif et (silencieux). De plus, à mesure que le niveau de contraction augmentait, le (alternation onset time) et le temps d'endurance diminuaient. En adressant le deuxième objectif, nous avons appliqué des (pseudorandom binary sequence displacements) avant, pendant et après des contractions des (triceps surae). Nous avons enregistré et analysé la position et le moment pour identifier la contribution des mécanismes (intrinsic) et (reflex) à la (stiffness) de la cheville. Nous avons trouvé que la fatigue causait des changements importants autant aux propriétés (intrinsic) que (reflex). Pour ce qui est des propriétés (intrinsic), il y avait une diminution de l'élasticité ainsi qu'une augmentation de la viscosité du joint. Pour ce qui est des propriétés (reflex), il y avait une augmentation du gain et un changement dans (the dynamics). Ainsi, il semblerait que, pendant la fatigue musculaire, (reflex stiffness) augmente pour compenser, au moins en partie, la diminution de (intrinsic stiffness).

Acknowledgements

I would like to extend my great appreciation to all of the people and organizations that helped me complete this mission.

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Figure 5.9: The effect of contraction and fatigue on the reflex parameters. Shown here are the mean values of the normalized parameters obtained from all subjects. Circles represent the group mean values, and error bars represent the group standard error.

1. Introduction

1.1. Motivation

Why can't we run, swim, or walk forever for recreational purposes? Sometimes I wonder to myself why there seems to be a limit to my performance or ability to complete prolonged physical tasks. At some point, I get tired! My muscles get stiffer and start to cramp! Soon, I find that I am unable to perform the task any longer.

Muscle fatigue influences the way we perform tasks. Unlike robots, all living beings are subject to fatigue, which limits our capability to engage in prolonged functions. The progression of fatigue causes a decline in maximal force, loss of coordination, decrease in total reaction time, and tremor. Understanding when and how fatigue occurs and its consequences on the body's performance and capability would help us develop preconditioning exercise and work protocols to prevent the onset of fatigue, and perhaps, design pre-cautionary schemas to prevent bodily injury.

Hence, this topic has motivated me to pursue a study to quantify fatigue, and to determine its effects on dynamic joint mechanical stiffness, if any.

1.2. Objective

The objectives of the present study were: (1) to characterize fatigue in the triceps surae in humans, and (2) to use system identification techniques to examine how intrinsic and reflex components of ankle dynamic stiffness change during and after fatiguing, submaximal, isometric contractions of the triceps surae.

1.3. Outline

Chapter 2 presents the anatomical and physiological background relevant to the neuromuscular control of the ankle joint. This chapter first reviews the anatomy and physiology of skeletal muscles, the organization of joint muscles, and the proprioceptive receptors. Then, it provides a brief background on the nature and function of the central activation mechanisms and peripheral reflexes that control locomotion and posture. Finally, the chapter reviews the physiology and characteristics of muscle fatigue.

Chapter 3 first provides a detailed technical description of the experimental setup and acquisition systems. Next, it discusses subjects and experimental protocols. Finally, the chapter presents analysis techniques, including EMG analysis, ankle dynamic stiffness determination, and group averaging methodologies.

Chapter 4 and 5 present the main results of the study. Chapter 4 examines and discusses fatigue-induced alterations in muscular activity, and the effect of voluntary contraction level on endurance and the generated force. Chapter 5 examines and discusses fatigue-induced changes in EMG, intrinsic, and reflex stiffness.

Chapter 6 concludes the thesis with a discussion of possible physiological explanations and implications of the results. It also lays the ground for future work.

2. Anatomical and Physiological Background

2.1. Introduction

Chapter 2 presents the anatomical and physiological background relevant to the neuromuscular control of the ankle joint. This chapter first reviews the anatomy and physiology of skeletal muscles, the organization of joint muscles, and the proprioceptive receptors. Then, it provides a brief background on the nature and function of the central activation mechanisms and peripheral reflexes that control locomotion and posture. Finally, the chapter reviews the physiology and characteristics of muscle fatigue.

2.2. The Ankle Joint

Human locomotion and posture are results of some control and interaction mechanisms of the lower limb joints, namely: hip, knee, ankle, meta-tarsal, and tarsal joints. Hence as a first step, we need to fully understand the dynamics and functionality of each joint independently. Only then, can we study the interactions among the lower limb joints that produce locomotion and maintain posture and balance.

This study focused on the ankle joint due to the availability of an electro-hydraulic actuator, which was setup in our lab previously to investigate ankle joint dynamics [1], and because:

- The ankle joint is responsible for postural and locomotor control.
- All ground reactions are translated through the ankle.

- The ankle poses two degrees of freedom, namely: plantarflexsion-dorsiflexion and adduction-abduction. If fixed appropriately, we can restrict the motion to the sagittal plane (the former) only.
- The anatomy of synergistic and antagonistic muscles is readily known for use in electromyography.

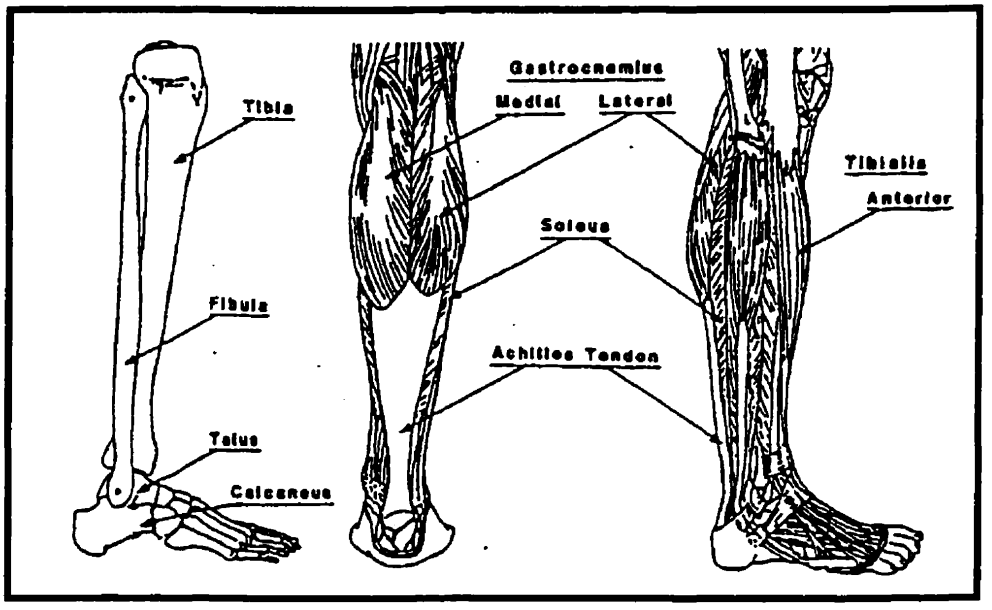

Figure 2.1: Skeletal structures of the human ankle joint [Yunan 1989 (2)].

A diagram of the ankle joint is shown in Figure 2.1. It is located between the inferior end of the fibula-tibia junction and the superior end of the talus-calceneus junction. It is a hinge-like synovial joint capable of moving in two planes: sagittal (plantarflexion and dorsiflexion) and frontal (adduction and abduction). *Plantarflexion* is the movement of the foot away from the body, while *dorsiflexion* is the movement towards the body. On the other hand, *adduction* is the movement of the ankle inwards, while *abduction* is the movement outwards. The ankle is reported to be more stable in the dorsiflexion position than the plantarflexion position [3].

2.3. Skeletal Muscle

2.3.1. Functional Anatomy

Skeletal muscles enable us to perform voluntary motion and maintain posture. Muscles comprise a complex structural hierarchy bound together by connective tissue as shown in Figure 2.2.

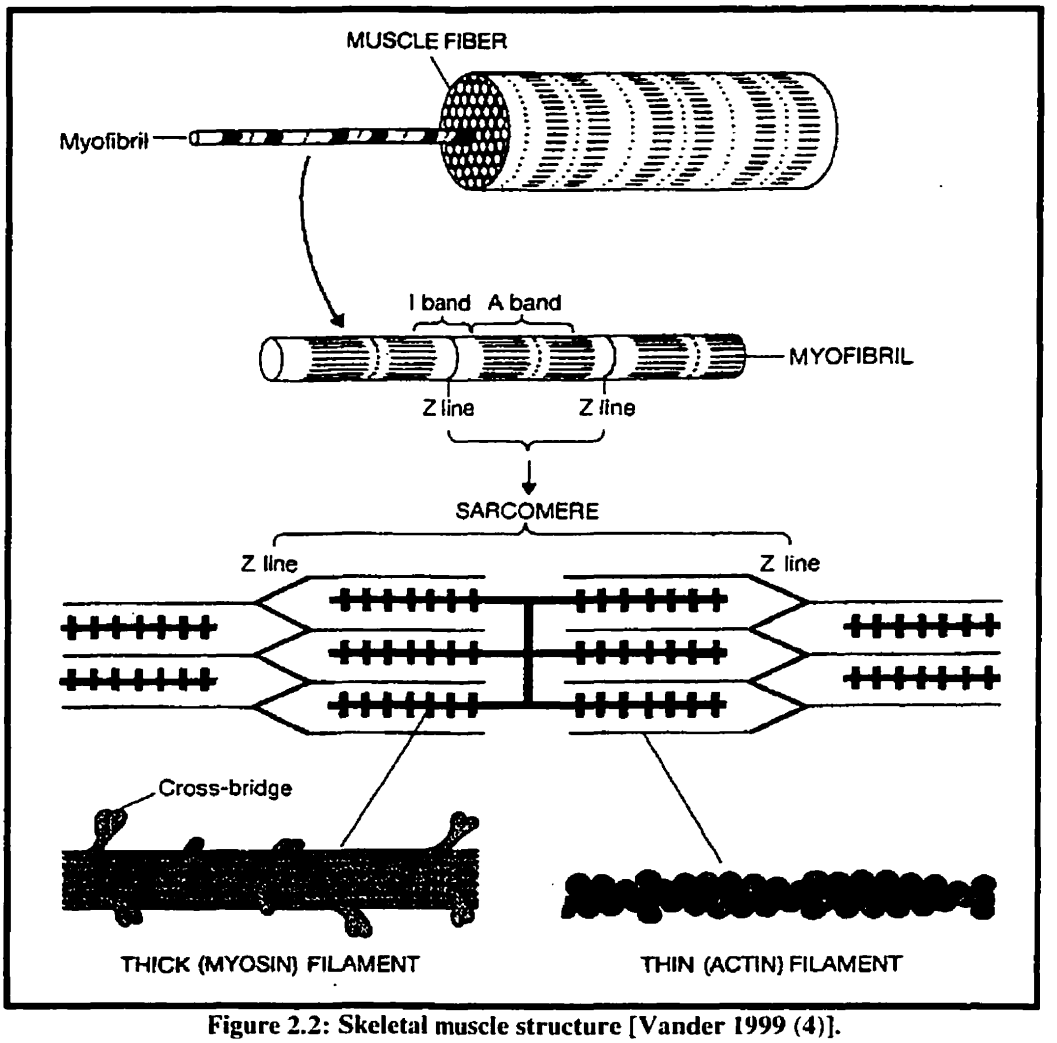

Figure 2.2: Skeletal muscle structure [Vander 1999 (4)].

Each muscle is composed of smaller contractile units called *muscle fibers*. Each muscle fiber is composed of yet smaller *myofibrils*, which exhibit striations of light and dark

bands. Each myofibril is made of many sarcomeres linked together in series. *Sarcomeres* are the functional unit of the muscle. Sarcomeres are made of interlaced thin actin filaments (light bands) and thick myosin filaments (dark bands). Myosin filaments are sandwiched in between actin filaments. Projections from myosin filaments that interact with the actin filaments, are called cross-bridges. The *sliding-filament theory* describes how the action of the cross-bridges creates muscular contraction, and hence force [4].

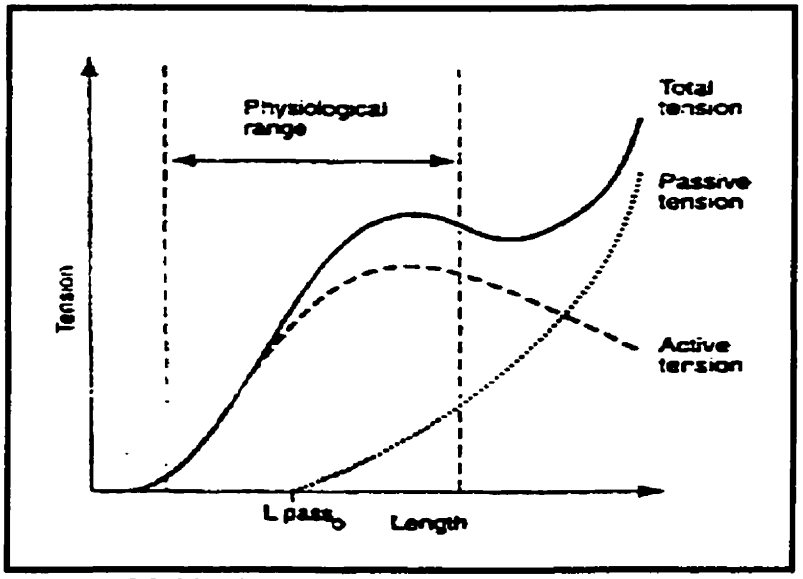

Figure 2.3: Muscle length-tension relationship [Chez 1991 (5)].

Muscles, like other biological materials, exhibit both elastic and viscous properties. Elasticity originates from the structure of the sliding filaments. According to the force-length relationship model, the total contractile force generated by the muscle depends mainly on the amount of stretching of its filaments. The active tension depends on the degree of overlap between thin and thick filaments. The more overlap, the more force will be generated until an optimal length is reached; beyond this the force decreases due to the interference of the actin filaments with each other as shown in Figure 2.3. In

contrast, passive muscle tension increases continuously following a certain length lag, which is thought to occur from crimp stretching.

On the other hand, viscosity of the muscle is believed to originate from extracellular connective tissue. The force generated depends on the stretch rate of the filaments – Figure 2.4. As the load on a muscle increases, muscle shortening velocity decreases until a point is reached at which the load is too great for the muscle to shorten. The tension at this point is known as the *maximum isometric tension*. Therefore, the response of skeletal muscles is both position and velocity dependent [6].

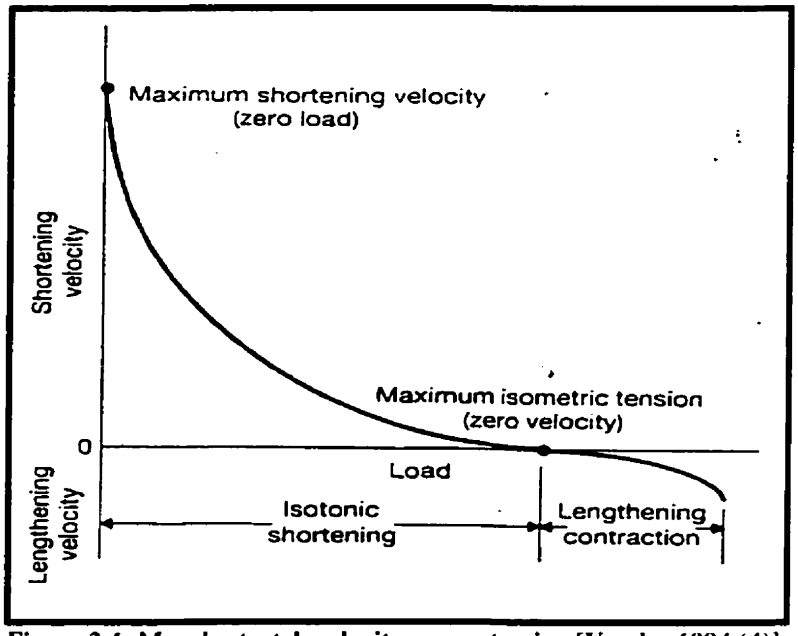

Figure 2.4: Muscle stretch velocity versus tension [Vander 1994 (4)].

2.3.2. Organization of Ankle Joint Muscles

Muscles can only '*pull*', not '*push*'; hence, at least two opposing muscles are required to control movement about a joint. Often, more muscles are present to provide redundant control and further stability to the joint.

Three muscles are mainly responsible for ankle motion in the sagittal plane: the *gastrocnemius*, the *soleus*, and the *tibialis anterior*. For a movement in the sagital plane, the first two are synergistic muscles that produce torques in the plantarflexion direction, whereas the last is their antagonist that produces torques in the dorsiflexion direction. *Triceps surae* comprises the soleus muscle, the lateral and medial heads of the gastrocnemius. While the gastrocnemius muscles span the knee and ankle, the soleus muscle and tibialis anterior span only the ankle.

2.4. Motor Units and Neuromuscular Control

2.4.1. The Functional Motor Unit

The smallest functional unit of the neuromuscular system is the *motor unit*, which as illustrated in Figure 2.5, is a group of muscle fibers, innervated by the same neuron, called the alpha motor neuron [7]. These muscle fibers are always synchronized in action. Motor units contain various numbers of muscle fibers; in addition, there are many motor units within each muscle. Depending on the muscle, motor unit size and nerve conduction velocity may vary [4,7]. *Type (I)* motor units are red, small, and are innervated by small diameter motor neurons. They possess slow conduction velocities, and highly vascularized tissue. These motor units are slow fatiguing, and are capable of generating slow twitches for long durations at low contraction levels. On the other hand, *type (II)* motor units are white, large, and are innervated by large, fast-conducting axons. They are not well vascularized; hence they are highly fatiguable, yet capable of producing large fast contractions, lasting only for short time.

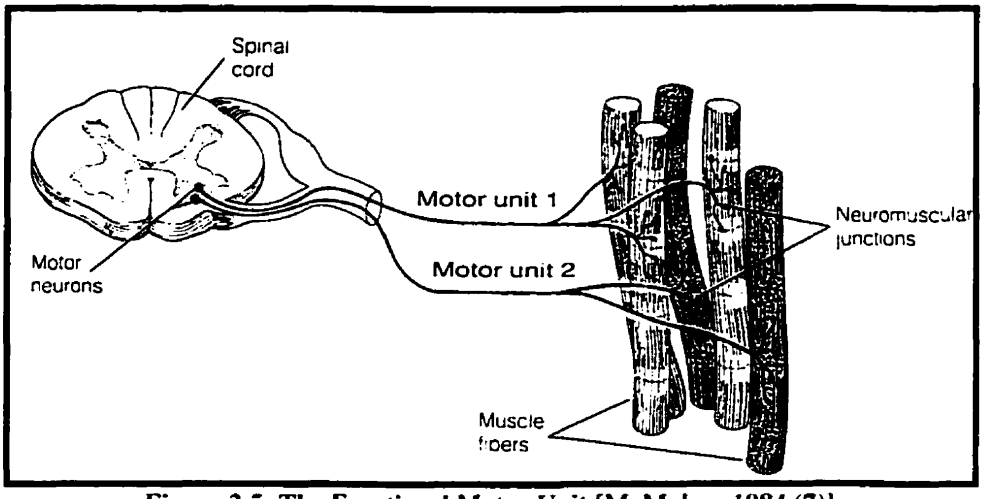

Figure 2.5: The Functional Motor Unit [McMahon 1984 (7)].

2.4.2. Central Neuromuscular Control - Muscle Activation and Force Transduction

Muscle fibers are innervated near their centers by alpha motor neurons, originating in the ventral horn of the spinal gray matter. Under steady-state conditions, a typical neuron possesses a membrane potential difference of -70 mV. An electrical signal, causing an above threshold membrane depolarization at the ganglion nucleus, will generate an action potential that travels along the ganglion ventral root and propagates down the motor neuron axon to the neuromuscular junction. Motor neuron conduction velocities may range from 40 m/s to 120 m/s.

When the action potential reaches the end of the axon, calcium (Ca^{++}) will be released extracellularily from the axon's cytoplasm. In addition a neurotrasmitter, *Acetylcholine* (AcH) will be released into the *neuromuscular synapse*, the space between the posterior part of the motor neuron and the muscle fiber. AcH will bind to receptors on the muscle fiber membrane, causing a change in membrane permeability to sodium and potassium, and producing a stimulatory electrical impulse, called *motor unit action potential*

(MUAP). Subsequently, calcium channels in the muscle fiber membrane will allow extracellular calcium to diffuse in, raising the membrane potential. This will trigger a reaction at the sarcoplasmic membrane in the muscle fiber cytoplasm, causing a further release of interior Ca^{++} into the fiber cytoplasm. Calcium ions will bind to troponin molecules on the thin filaments, causing them to shift and hence clear the way for actin-myosin interaction. These interactions are responsible for muscle contraction. The action potential propagates in both directions of the muscle fiber at speeds of 2 m/s to 6 m/s.

The neuromuscular system uses two mechanisms to modulate the force generated. The first mechanism, called *motor unit recruitment*, involves changing the number of motor units. Force is proportional to the number of active motor units. From a functional perspective, one might expect that the smaller diameter, slower conducting neurons would be stimulated after the large diameter, faster fibers. For this case, large motor units would ensure that movements were carried out instantly, and fine-tuning could be achieved by smaller motor units later. However, many investigators have observed the contrary [8]. The *size principle* indicates that smaller motor units, which have lower threshold and are more sensitive to an electrical stimulus, are activated first. Larger motor units, which have higher thresholds and are less sensitive, are activated later.

The second mechanism for neural regulation of force is *rate coding*. This refers to the adjustment of motor neuron firing rate. As the action potential frequency increases, a smooth sustained tension known as *tetanus* develops as shown in Figure 2.6. *Unfused* or

incomplete tetanus occurs when successive stimuli arrive far enough apart in time that the muscle is allowed to recover partially between stimuli. Alternatively, if the stimuli are proximate in time, recovery does not occur, and tension increases giving rise to a condition of *fused* or *complete tetanus*. Tetanic tension is 2 - 5 times greater than the maximal twitch tension.

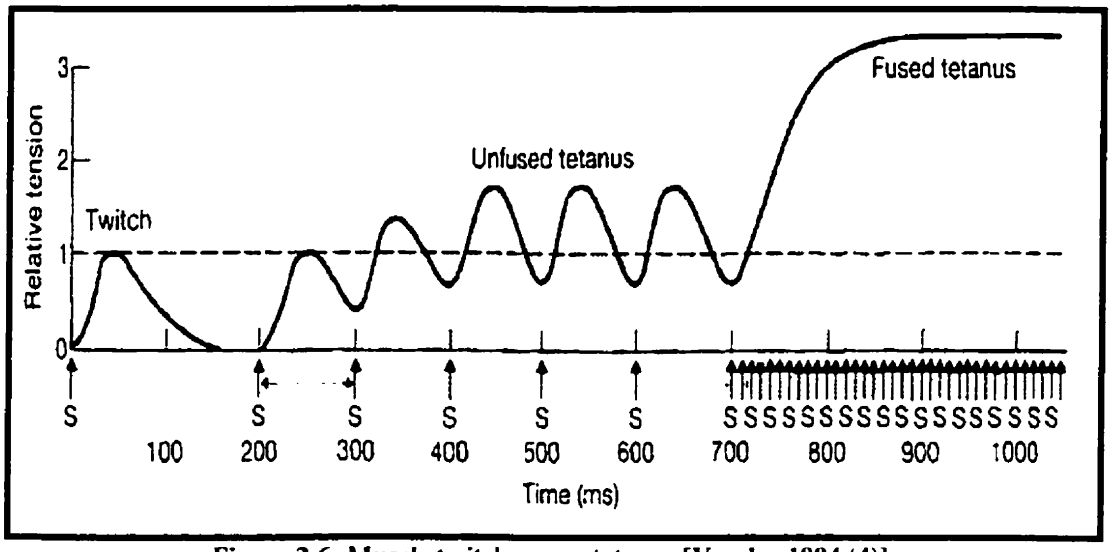

Figure 2.6: Muscle twitch versus tetanus [Vander 1994 (4)].

Determining the relative importance of these two central control mechanisms in a particular muscle for a certain task is not trivial. However, it has been established that smaller muscles rely primarily on firing-rate coding, while larger muscles rely mainly on motor unit recruitment to modulate force output [7].

2.4.3. Peripheral Neuromuscular Control

Central commands are integrated with peripheral commands to produce coordinated, balanced movements according to the diagram shown in Figure 2.7. Unlike central

commands, peripheral neuromuscular commands originate between the spinal cord and the muscle fibers, and are called *segmental reflexes* [7]. These are involuntary, compensatory, regulatory responses that maintain stability of the body, and coordinate delicate tasks.

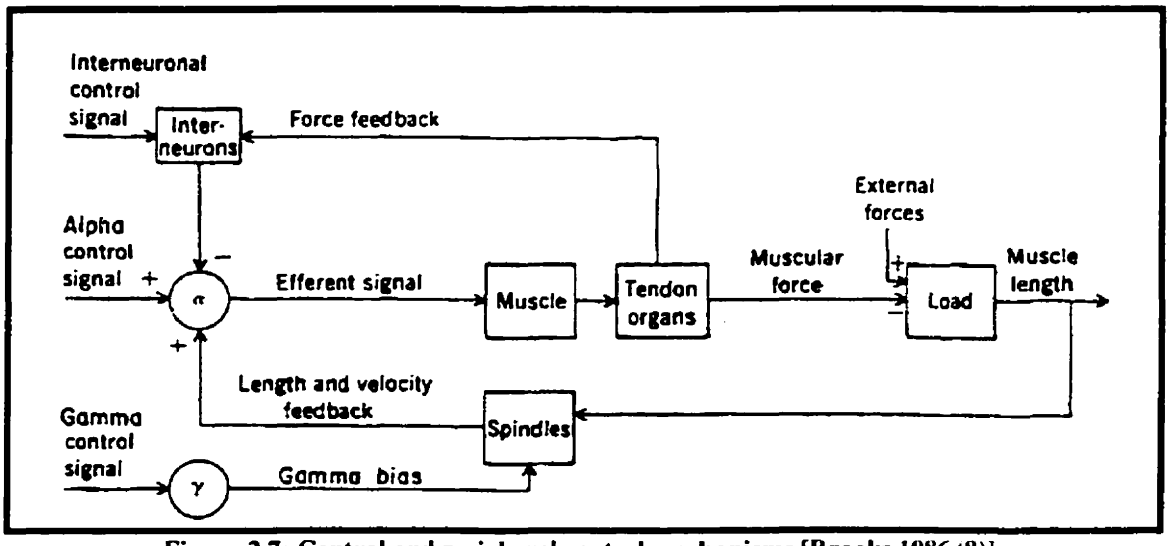

Figure 2.7: Central and peripheral control mechanisms [Brooks 1986 (8)].

In general, the anatomy of a segmental reflex consists of the *reflex arc*, comprising a set of sensory receptors, afferent and efferent neural pathways, an integrating center, and an effector. Peripheral sensory receptors or *proprioceptors* provide and co-ordinate information via afferent neurons to the integration center, located in the spinal cord. This information is integrated with other signals to generate appropriate control signals. These are then translated into signals that travel to the effector muscle fibers via motor neurons and efferent neural pathways.

The state of a muscle is constantly monitored by peripheral receptors imbedded in it. These receptors detect muscle tension, length and velocity. The two most important types of receptors are the *muscle spindles* and the *Golgi Tendon Organs*. As shown in

Figure 2.8 spindles are attached in parallel to muscle fibers, and hence provide information about muscle length and velocity. In contrast, Golgi Tendon Organs are arranged in series with bundles of muscle fibers, and hence are sensitive to muscle tension.

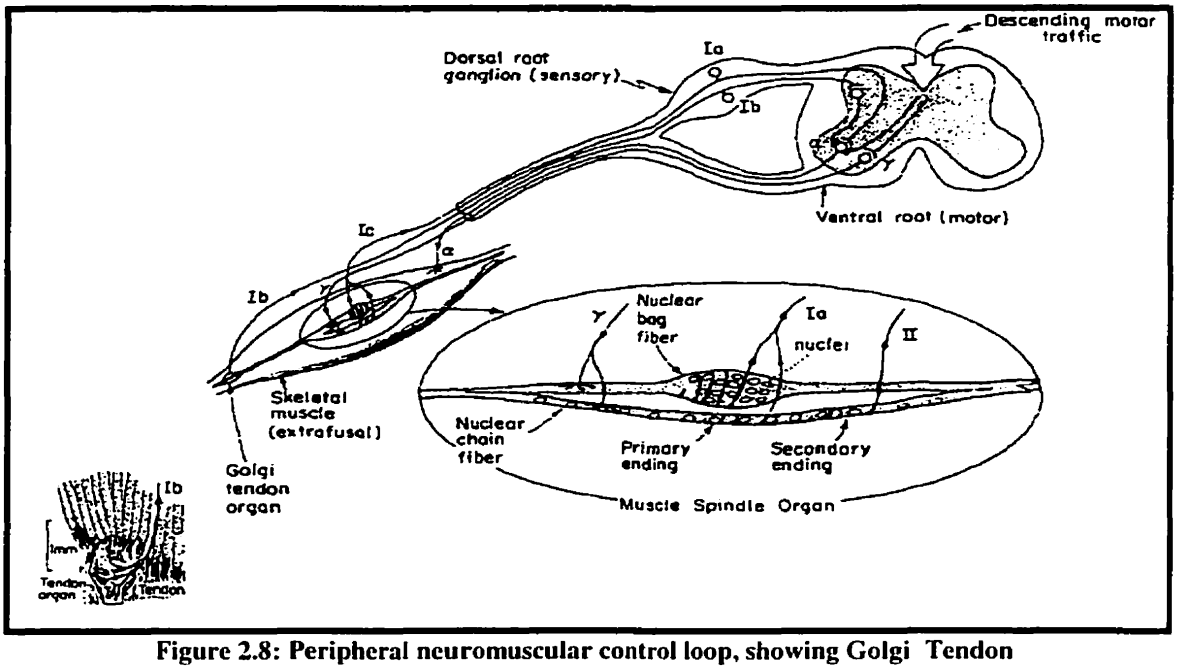

Figure 2.8: Peripheral neuromuscular control loop, showing Golgi Tendon Organ and muscle spindle orgran [McMahon 1984 (7)].

Muscle Spindles

Figure 2.8 illustrates the structure of muscle spindle. Sensory endings of afferent fibers are wrapped around modified muscle fibers, called *intrafusal muscle fibers*. These intrafusal fibers lie within muscle spindles, and can be differentiated into three functionally different types: *dynamic nuclear bag*, *static nuclear bag*, and *nuclear chain fibers* [7].

Two types of afferent fibers carry stretch information from muscle spindles to the integrating center in the spinal cord: primary and secondary. The spindle *primary sensory*

fibers (Group Ia) are large in diameter (12-20 μm), myelinated, and have unmyelinated endings that innervate intrafusal muscle fibers. There is a single primary ending per muscle spindle. The *secondary sensory fibers (Group II)* are small in diameter (4-12 μm), and terminate on the static nuclear bag and nuclear chain fibers. There are multiple secondary endings per muscle. Primary endings are more sensitive than secondary endings to changes in muscle length (static) and velocity (dynamic). Using sinusoidal inputs, the responses of both primary and secondary endings have been characterized as shown in Figure 2.9. Primary endings are more sensitive to smaller amplitude displacements, and in particular more sensitive to stretching than shortening displacements. Primary endings are also sensitive to acceleration at high perturbation frequencies [8-10]. Distortion of these fiber endings due to muscle stretch will generate action potentials in the afferent fibers. Group Ia afferents transmit nerve impulses at speeds of 70-120 m/s, while Group II afferents transmit at lower speeds of 20-70 m/s [9].

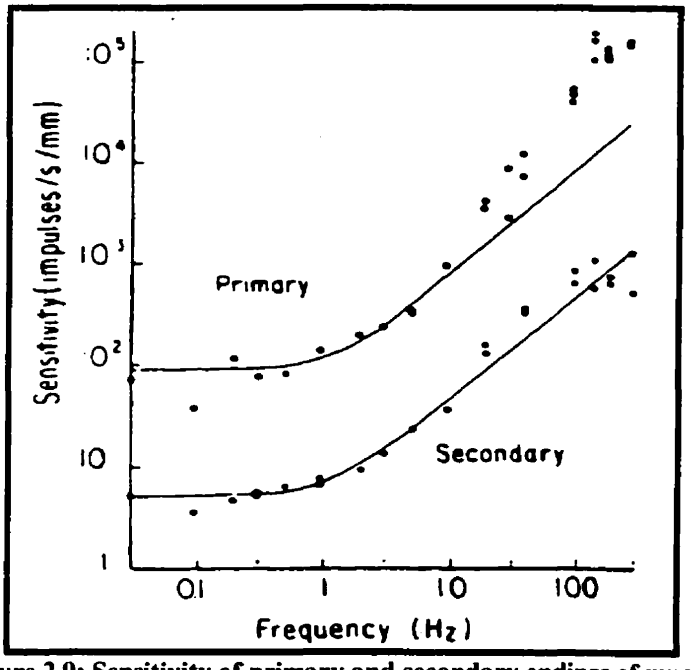

Figure 2.9: Sensitivity of primary and secondary endings of muscle spindles to sinusoidal stretching [Stein 1980 (10)].

Muscle spindles receive efferent innervation from *gamma (γ)* or *fusimotor fibers*, which synapse onto the contractile ends of the intrafusal fibers. These efferent fibers modulate the sensitivity of primary and secondary sensory endings under passive and active muscle tension conditions. There are two types of gamma fibers: static and dynamic. Activation of *static gamma fibers* increases the firing rate of both primary and secondary sensory endings, when the muscle length remains constant. On the other hand during active muscle stretching, activation of *dynamic gamma fibers* promotes firing of the primary endings only [9]. Gamma fibers conduct at speeds of 10-50 m/s.

Golgi Tendon Organs

Golgi Tendon Organs are located at the junction of the muscle and the tendon. Because they are in series with muscle fibers, and are distributed across several muscle fibers, they are very sensitive to active muscle tension, but less sensitive to passive muscle tension and external perturbations [9].

Each Golgi Tendon Organ is innervated by a single group Ib axon that looses its myelination after it enters the capsule, and branches into many neural endings. When the muscle contracts, tendon filaments are stretched and these endings are distorted. Consequently, action potentials will be generated and carried via the Ib afferent axon to the integrating center at the spinal cord.

Peripheral Reflexes

When a muscle is stretched, as shown in Figure 2.10, muscle spindles are distorted, generating an action potential that travels along group Ia afferents to the spinal cord. A secondary motor signal is generated, which travels down the alpha motor neuron axons to the stretched muscle, causing it to contract, hence counteracting the stretch. This monosynaptic reflex arc is called the *stretch reflex.*

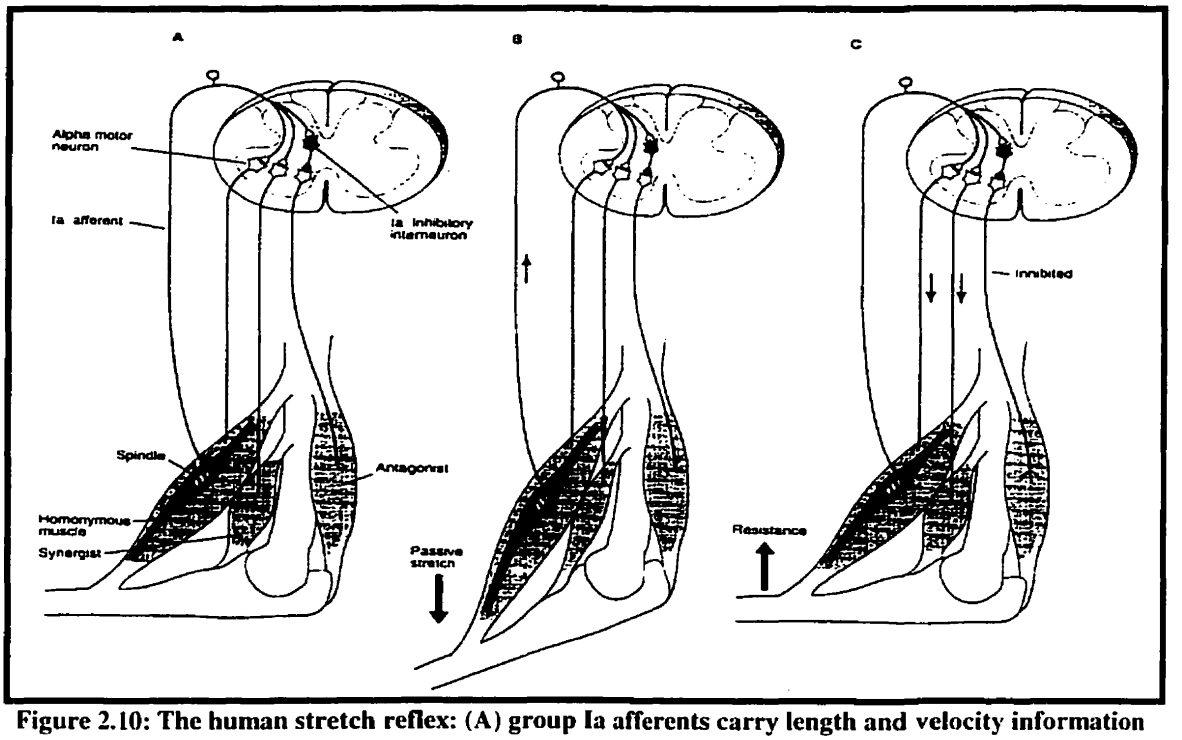

Figure 2.10: The human stretch reflex: (A) group Ia afferents carry length and velocity information from the active muscle to the spinal cord, (B) on passive stretching, the spinal cord activates synergistic muscles, and (C) inhibits antagonist muscles [Kandel 1991 (11)].

Group Ia afferents are also responsible for *agonist, synergistic muscle co-activation.* When a muscle contracts, signals from Ia afferents propagate to alpha motor neurons that innervate other synergistic muscles, causing them to contract. This is a monosynaptic signal relay at the level of the spinal cord.

In addition, Group Ia afferents are responsible for *reciprocal inhibition*. When a muscle is stretched, group Ia afferents carry relative length and velocity information to inhibitory inter-neurons at the spinal cord. This will inhibit antagonist muscle motor neurons. Unlike the two former reflexes, reciprocal inhibition is a dysnaptic reflex arc.

As illustrated in Figure 2.11, group Ib afferents relay information about muscle tension to the CNS, and are responsible for activation of antagonistic muscle motor neurons, and inhibition of synergistic muscle motor neurons. Hence, this polysynaptic reflex arc limits the contraction of a given muscle to preserve balance and posture, and is called the *inverse stretch reflex.*

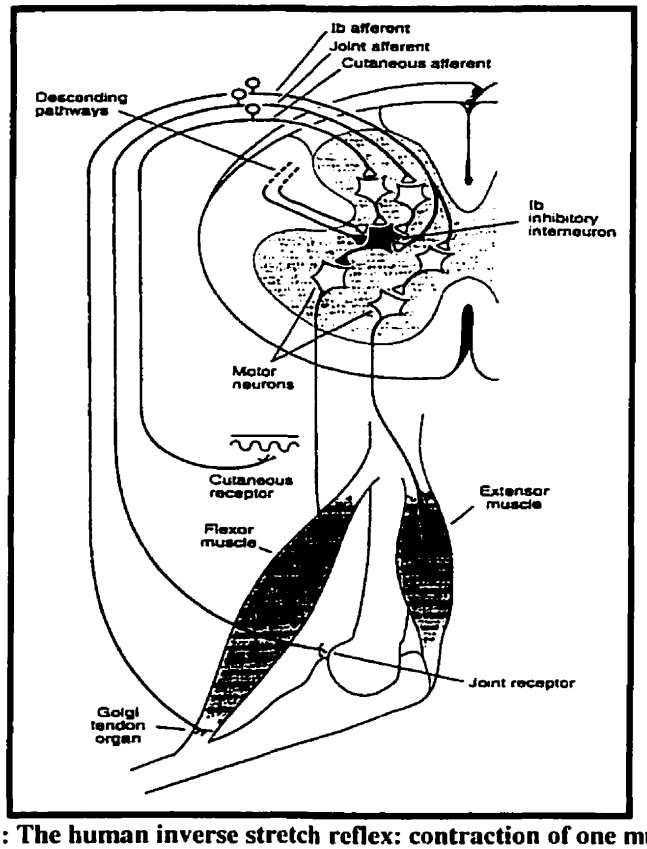

Figure 2.11: The human inverse stretch reflex: contraction of one musle causes activation of antagonist muscles and inhibition of synergistic muscles [Kandel 1991 (11)].

2.5. Muscle Fatigue

The definition of fatigue differs with the task. *Muscle fatigue* can be defined as the muscle's inability to maintain a maximal voluntary contraction. This is usually associated with a decrease in maximal electromyographic (EMG) activity in the muscle. Fatigue can also occur as a result of long-lasting submaximal contractions. This type of fatigue is accompanied by increased electromyographic activity to compensate for the decreasing force output, generated by the individual muscle fibers. Hence, the type, intensity, and duration of the muscle contraction affects the physiology of muscular fatigue [12]. The most common symptoms of fatigue are muscle soreness, muscle stiffness, and localized pain [12].

2.5.1. The Physiology of Fatigue

Three sites have been identified as potential sources of fatigue: (1) the central nervous system, (2) the neuromuscular joint , and (3) the motor unit (MU) [13-16]. Previous studies have not been able to identify a single source of fatigue for a given task [13,17,18]. However, during isometric contractions, fatigue tends to coincide with the restriction of blood flow to the muscle [13]. In contrast, during isotonic and isokinetic contractions, fatigue may be caused by central and peripheral adaptive mechanisms [12].

Central and Peripheral Factors

Central and peripheral mechanisms control the force generated by muscle fibers. These mechanisms are altered during long-lasting, fatiguing, muscular contractions. As a result, motor neuron pool activation patterns change. This involves changes in the recruitment

pattern, firing rate frequency, and the degree of synchronization of motor units.

During maximal voluntary contractions, there is a gradual decline in electromyographic (EMG) activity [18-22]. This decline can be attributed directly to a depression in the alpha-motor-neuron pool excitability [23]. Increased recurrent inhibition, exerted by Renshaw cells [24], increased group III and IV muscle afferent input [13,26,27], and decreased membrane potential of motor-neurons [24] have all been suggested as the cause of reduction in the firing frequencies of the motor-neurons [25]. This phenomenon is described as *'muscle wisdom'* [28,29]. The decline is advantageous because less activation of the motor units results in less expenditure of energy by muscle fibers, and hence preservation of energy.

On the other hand, during submaximal contractions, the central command drive has been reported to increase during fatigue [20], and then decrease at the endurance limit [18]. The increase in central motor command causes as increase in motor unit firing rates, recruitment of new fatigue-resistant motor units, and more frequent activation of active motor units. These alterations in activation mechanisms give rise to increased EMG activity [20,30-32]. In addition, it has been shown that during fatigue, the H-Reflex is suppressed due to increased inhibitory input from group III and IV muscle afferents [20,27,33,34]. However, this increase in inhibitory peripheral feedback is overridden by the increase in central drive to the alpha motor neurons [35].

Central fatigue is believed to prevent the muscle from maintaining a constant force

beyond the endurance limit [36]. It may occur as a result of: (1) supraspinal alterations, such as a decreased efficiency in the generation of the central motor command due to neurotransmitter depletion [37]; (2) reflex inhibition of the alpha motor-neuron pool by muscle afferents [26,38,39]; (3) progressive disfacilitation of excitatory Ia afferents to alpha-motor-neurons [24,26,27,33,40]; (4) late adaptation of alpha-motor-neurons [41] and neuro-modulation of synaptic efficiency [42].

One study showed that peripheral reflex inhibition of alpha motor neurons via small diameter muscle afferents is of a minor significance for the development of the central fatigue at the endurance limit [43].

Neuromuscular Factors

Fatigue has also been reported to influence neuromuscular facilitation by causing: (1) depletion of neurotransmitter at the pre-synaptic end; (2) a decrease in the motor unit membrane potential at the post-synaptic end [21,44]. These changes cause a propagation delay of the electrochemical signals that travel from alpha motor neurons to the motor units.

Intrinsic Muscular Factors

Localized muscle fatigue influences the mechanical and electrical properties of the muscle fibers of active motor units. With fatigue, the motor unit action potential amplitude decreases, and its duration increases [21,44]. In addition, there is a depression in the amplitude of the mechanical twitch, and an elongation in the recovery phase of the

MUAP [45,46]. These changes occur as a result of: (1) accumulation of metabolic by-products, such as lactic acid; (2) decreased muscle fiber conduction velocity [47,48]; (3) depletion of K^+ and Ca^{++} in active muscle fibers; (4) decreased membrane excitability, and (5) contractile apparatus sensitivity to intracellular metabolites, such as H^+, and inorganic phosphate [49].

2.5.2. Characterization of Fatigue: EMG Analysis Techniques

Objective quantitative measurements of fatigue are limited. Serum lactic acid levels have been used to measure fatigue, but have the limitation that blood samples must be obtained at regular intervals [12]. Another method relies on measuring the total force output, associated with a muscular contraction. However, this method examines fatigue after it limits performance, and not before. Another method uses electrical stimulation [50] of nerve and muscle, to characterize reflex action and to determine neural activation patterns. However, by itself this method alone cannot detect fatigue-induced changes within the muscle.

Recently, other non-invasive methods have been proposed and used to characterize fatigue, and trace its progression in a contracting muscle. These techniques rely on measuring muscular EMG signals. These signals are derived from the spatial-temporal summation of MUAPs, generated in the contracting muscle. The EMG signal depends on the firing rate of the motor units, the shape and number of MUAPs, and the degree of synchronization of these MUAPs.

Analyzing EMG signals in both time and frequency domains yields information regarding the physiological state of a muscle and/or the efficiency of a contraction.

Root Mean Square of Signal Amplitude

The *root mean square* (RMS) is a temporal EMG measure that is believed to reflect on central and intrinsic muscular factors, including recruitment of motor units, alterations in the firing frequency of individual motor units, and the degree of synchronization of motor unit firing patterns [23].

RMS values depend on the tension level as well as the extent of fatigue in a muscle. For sustained maximal contractions, there is a general decrease in the RMS EMG amplitude [19,21]. This decrease can be directly attributed to depressed alpha-motorneuron pool excitability [51]. On the other hand, during sustained submaximal contractions, there have been reports of an initial increase, followed by a plateau, and then a decrease in the RMS EMG amplitude [52]. The increase has been attributed to: (1) recruitment of new, fatigue-resistant motor units; (2) increased firing frequency of motor neurons; (3) increased synchronization of motor units; (4) increased duration of MUAP, and (5) decreased conduction velocity of muscle fibers [53]. The plateau in EMG RMS occurs as a result of two counterbalancing mechanisms. On the one hand, there is an increase in the central drive to the motor neurons, as described earlier. On the other hand, as fatigue progresses, there is a simultaneous decrease in the motor unit firing frequency and in the MUAP amplitude, as well as de-recruitment of some highly fatigable motor units. These events lead to a decrease in the EMG power. The net effect is a plateau of the EMG

RMS with time. The final decrease in the EMG RMS can be attributed to the later events dominating the former ones.

Median Frequency of Signal Frequency Spectrum

The *median frequency* (MF) is a spectral EMG measure that can be obtained by first taking the Fast-Fourier Transform (FFT) of the EMG signal, and then calculating the median of its power spectrum [23,53]. It is believed to reflect muscular activation properties such as the duration of motor unit action potentials, muscle fiber conduction velocity, and the degree of synchronization of motor units[23].

A shift in power density distribution toward lower frequencies, along with decreased MF, has been reported to occur with fatigue [44,52]. This change occurs due to an increase in the lower frequency components, and an decrease in the higher frequency components of the EMG power spectrum. These changes are attributed to a decrease in the average conduction velocity of the muscle fibers, resulting from increased intramuscular metabolites [54,55]. Unlike RMS values, MF values depend only on the extent of fatigue developed within a muscle; MF does not depend on the level of tension exerted by a muscle [24,53].

3. Methods

3.1. Apparatus

Figure 3.1 illustrates the experimental setup used in this study. Subjects lay supine with their foot attached to the pedal of a stiff, position controlled electro-hydraulic actuator. For this purpose, a rigid, low-inertia fiberglass boot was constructed for each subject. To isolate ankle movements, straps where applied just superior to the knee, which was elevated slightly using sand bags for support. An oscilloscope, mounted above the subject's table, displayed a visual torque target and a torque feedback signal, which was low-pass filtered at 1.5 Hz to reduce the perturbation-evoked component.

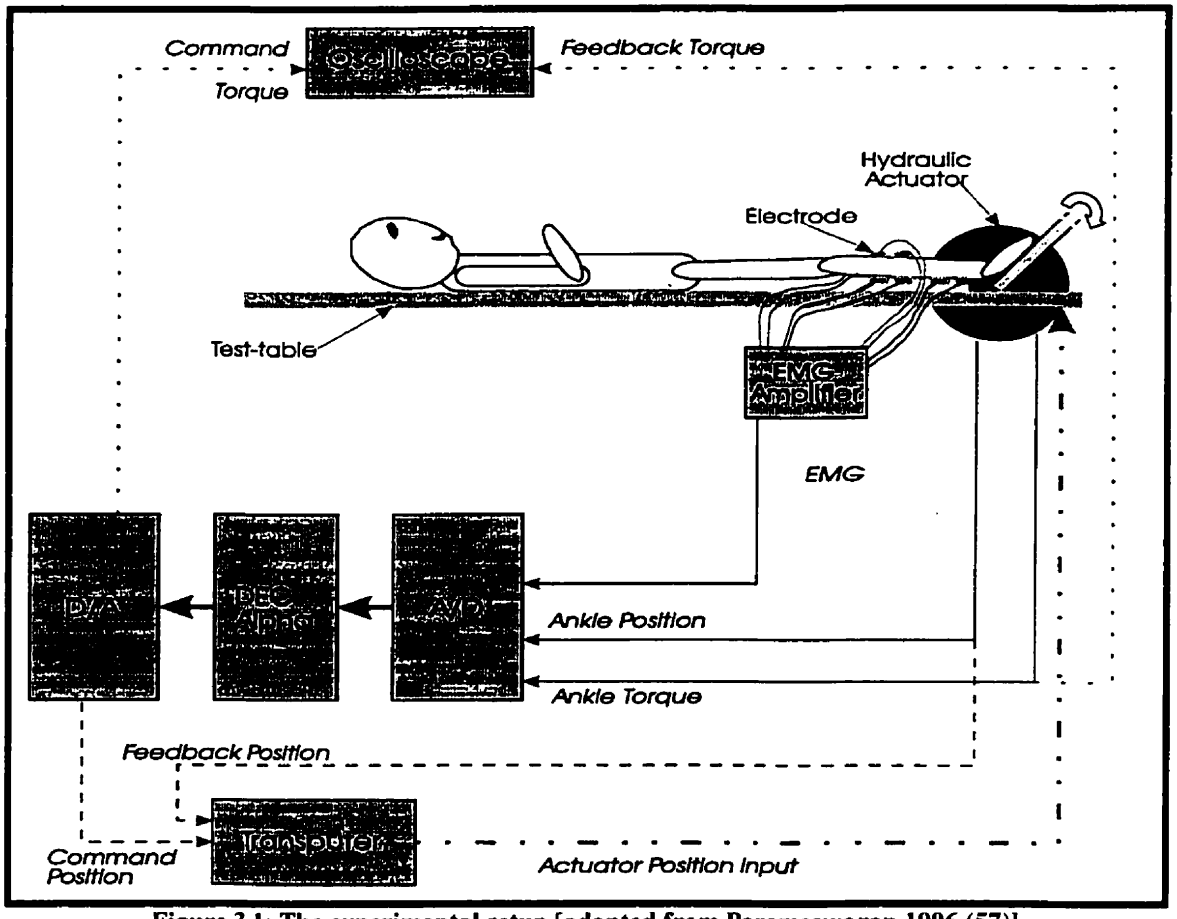

Figure 3.1: The experimental setup [adapted from Parameswaran 1996 (57)].

The following sections give details of the electro-hydraulic actuator, the fiberglass boot, and the signal acquisition system. A summary of subject selection and training protocol follows. The chapter closes with a thorough discussion of the analytical methods used to interpret the results.

3.1.1. Electro-hydraulic Actuator and Control Strategy

The ankle actuator consisted of a rotary electro-hydraulic motor controlled by a two-stage Moog® servo-valve. Proportional control, implemented on a two-processor transputer system, was used to ensure a flat frequency response up to 50 Hz. The servo-controlled system could deliver perturbations with power up to 100 Hz, which was sufficient for system identification of ankle stiffness dynamics. The combined system operated at a pressure of 3000 psi and was capable of producing torques up to 380 Nm [2]. The actuator allowed motion in the sagittal (anterior-posterior) plane only.

A potentiometer, a torque transducer, and a boot pedal were mounted in series with the actuator as shown in Figure 3.2. Four independent safety mechanisms were designed to prevent injury: *(1)* two adjustable aluminum bolts limited rotation to less than subject's range of motion (ROM); *(2)* an adjustable hydraulic cam which shuts off the flow to the actuator before reaching extreme ROM; *(3)* pre-set control parameters which limit normal actuator function; *(4)* a panic button, placed in the subject's hand, which stopped the actuator when pushed[2].

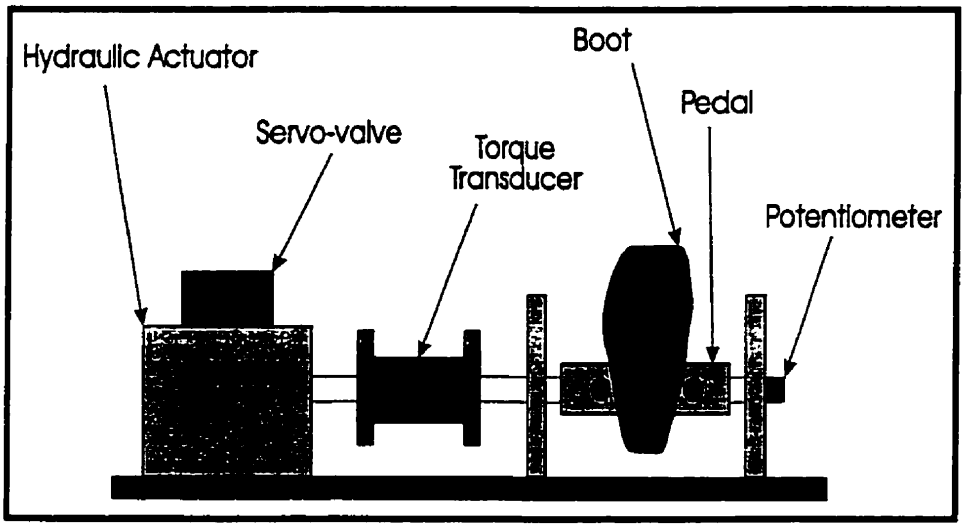

Figure 3.2: The actuator arm showing all mounted components [Parameswaran 1996 (57)].

3.1.2. Rigid Ankle Fixation

A fiberglass custom-fitted boot was constructed for each subject. The boots had the following properties:

(1) light-weight to minimize foot inertia;

(2) rigid to withstand application of high torques, and removable and durable so that it can be used in more than one experiment;

(3) comfortable and close fitting to transmit stochastic perturbations from the actuator to the foot without interfering with the joint dynamics;

(4) did not limit the range of motion of the ankle joint.

The construction procedure was straightforward and is outlined in detail elsewhere [56,57].

3.1.3. Signal Transduction and Processing

Position perturbations were applied to the ankle, and six signals were recorded: angular position (radians), angular torque (Nm), and electromyograms (mV) from the triceps surae group (MGS, LGS, and SM) and the tibialis anterior (TA).

Experimental and analytical modules were implemented using Matlab®. The operator controlled the desired perturbation signal and actuator angular position. The operator also controlled a command torque signal, which was displayed on an overhead BK Precision® (2121) digital oscilloscope. The digital position signal was converted to analog using a 16-bit, 4-channel digital-to-analog converter (DAC). Further it was low-pass filtered at 200 Hz using an 8-pole, linear-phase, constant-delay filter.

Angular position of the ankle was transduced by a Beckman® (6273-R5K) precision plastic film rotary potentiometer, located on the axis of the ankle actuator. The potentiometer had a maximum non-linearity of ± 0.2% and a maximum resistance of 5 kΩ. A custom-built potentiometer module amplified position signals. In the frequency domain, the gain of the module increased to become flat at 10 dB from 0.316 Hz-90 kHz, after which it rolled off at 2-3 dB/decade. The phase shift was 180° from 0.316 Hz-90 kHz, and then increased to 225°. The noise level was about 10 mV peak-to-peak or 0.001 radians. A reference ankle angle of zero was assigned to mid-position, i.e. the position at which the foot was perpendicular to the leg. Plantarflexed positions were assigned negative values, whereas dorsiflexed positions were assigned positive values. The range

of motion was limited to ± 0.5 radians (± 26.5 degrees). Ankle angular velocity was obtained by numerically differentiating the angular position record.

Ankle angular torque was transduced by a Lebow® (2110-5K) general-purpose reaction rotary torque sensor, mounted on the axis of the actuator. This four-arm, strain-gauge, torque sensor had a stiffness of 10^5 Nm/radian, a maximum capacity of 565 Nm, and a maximum non-linearity of ± 0.2%. In the frequency domain, the gain of the sensor increased to become flat from 0.1 Hz-20 kHz, after which it rolled off at 20 dB/decade. The phase shift was 0-10° from 0.1 Hz-20 kHz, and then increased to 90°. The noise level was about 10 mV peak-to-peak or 0.2 Nm. The torque signal was amplified by a conventional bridge amplifier module. Then, it was low-pass filtered at 1.5 Hz using an 8-pole Bessel filter, and displayed on an oscilloscope, placed above the subject's head to assist the subject in tracking the command torque signal.

Surface electromyograms (EMGs) were continuously monitored to evaluate the neural activity and the state of the ankle plantar-flexors and dorsi-flexors. EMG signals were acquired using Hewlett Packard (13951C) disposable, self-adhesive Ag/AgCl surface electrodes. Pairs of electrodes, their centers separated by 1 cm, were placed along the midline of the bellies of the medial and lateral gastrocnemius. Another pair was placed on the soleus along the mid-lateral line of the leg, 4 cm down from where the MGS and the LGS join the Achilles tendon. TA electrodes were placed on the belly of the muscle just lateral to the tibial bone, 15 cm below the patella. A reference electrode was placed

over the patella. The skin was shaved, abraded, and cleaned with alcohol to reduce the skin electrode impedance before electrodes were applied.

EMG signals were pre-amplified using custom-made, three stage pre-amplifiers [57]. These consisted of an AD625 instrumentation amplifier, a passive single-pole RC high-pass filter with 1 Hz cutoff, and an AD210 isolation amplifier. The high-pass filter removed low frequency artifacts associated with electrode polarization, cable and electrode motion. The overall gain of the pre-amplifier was set to 1000. These pre-amplified EMG signals were then high-pass filtered using a fourth-order Butterworth filter with a cutoff frequency of 5 Hz. Lastly, the signals were positively full-wave rectified.

All signals were anti-alias filtered at 200 Hz, and sampled at 1000 Hz using a 16 bit analog-to-digital converter (ADC). Given the quantization limitation of the ADC, position resolution was limited to 0.002 radians, and torque resolution to 0.006 Nm.

3.2. Subjects

Nine normal human subjects, aged 19-27 years, with no known history of neuromuscular disease or injury were examined. The experiments performed in this study received prior approval from the ethics committee at McGill University. All subjects were provided with a detailed description of the experiment, and gave informed consent prior to experiment.

Subjects were trained to maintain a tonic contraction of the triceps surae muscles by aligning a low-pass filtered subject torque signal with a target torque signal, both of which were displayed on an overhead oscilloscope. The experiments were performed at the REKLAB Neuromuscular Control Laboratory in McGill University, Montreal, Quebec.

3.3. General Procedures

ROM Determination

With the actuator off, and the subject's foot in the boot attached to the actuator, the range of motion (ROM) was determined. The ankle was manually rotated from neutral position to the maximum tolerable plantarflexing position, and then to the maximum tolerable dorsiflexing position. Position values for these extreme levels were recorded for future experiments. The mechanical safety stops were adjusted to limit actuator motion to the ROM of each subject.

MVC Determination

Three maximum voluntary contractions (MVCs) were recorded for each subject at the start of each experiment, with the ankle in neutral position. Subjects were asked to execute and hold a maximum plantarflexing contraction for 3 seconds in response to a step change in a tracking stimulus displayed on the overhead oscilloscope. MVC trials were each separated by 2 minutes to allow for recovery and eliminate possible transient effects. After the collection of three MVCs, electromyographs (EMGs) and torque were

averaged, and maximum values were used to normalize subsequent EMG and torque measurements, respectively.

3.4. Analysis Methods

3.4.1. EMG Analysis

RMS values were computed for each EMG signal for 2 second epochs. EMG RMS were normalized across subjects, by dividing the EMG RMS of each subject by the corresponding MVC EMG RMS maximum value. Variations between experiments might arise from differences in placement of the surface electrodes, body positioning on the experimental table, limb positioning, muscle state and history, and neural innervation differences. Normalization allowed us to compare different behaviors of the muscles across subjects. It also allowed us to compare the modulation of EMG of extensor and flexor muscles with fatigue across subjects.

RMS values of the EMGs, collected from the lateral and medial heads of the gastrocnemius, the soleus muscle, and from the tibialis anterior, were plotted continuously during the experiment to monitor neural activation and the state of the muscles as a function of time.

3.4.2. System Identification of Ankle Joint Dynamics: Quasi-Linear Model.

System identification techniques provide a quantitative description of an unknown system's behaviour through analysis of its input-output relationship. This methodological technique is also called *'black-box analysis'*. Although no direct structural or functional

information is provided by this analysis, a *'black-box'* model can serve as a reference against which other morphological models can be validated [58].

System identification techniques have been successfully developed in our lab to study human ankle joint dynamics [59,60]. These techniques have been used to quantify ankle joint behavior with various operating points such as: torque level [1], displacement amplitude [61], angular position [62,63], angular velocity [60], and fatiguing contractions [64]. In addition, inter-subject variability and intra-subject reliability of the system identification techniques for the ankle joint have been studied [65].

3.4.2.1. Parallel-Cascade Identification

Overall joint stiffness can be separated into two components: intrinsic stiffness arises from limb dynamics, articular mechanics, and active muscle mechanics; reflex stiffness arises from changes in muscle activation as a result of reflex effects of muscle afferents as shown in Figure 3.3.

Recently, our lab developed a parallel cascade, nonlinear identification method to estimate the intrinsic and reflex contributions to overall ankle joint stiffness simultaneously [60]. A parallel cascade model is shown in Figure 3.4. Non-parametric and parametric techniques were used to identify and quantify each pathway.

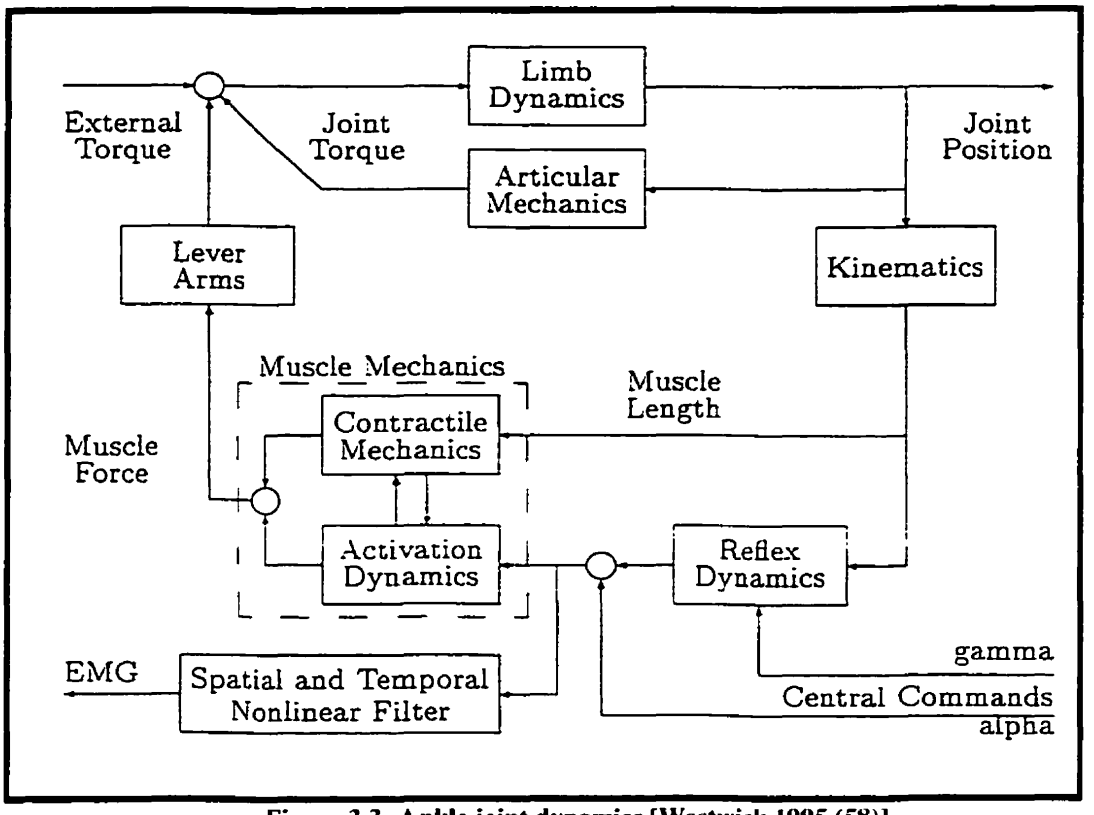

Figure 3.3: Ankle joint dynamics [Westwick 1995 (58)].

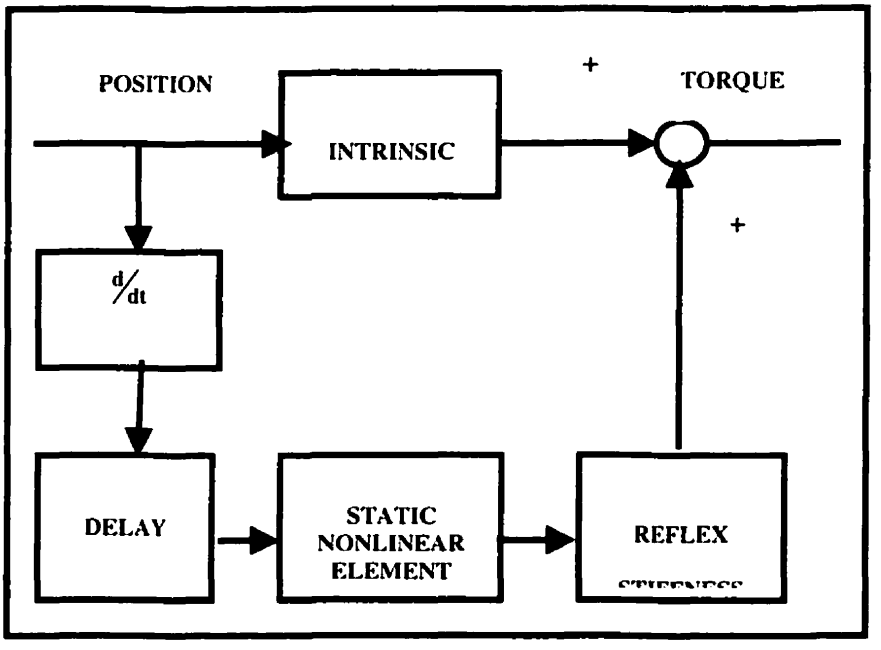

Figure 3.4: Parallel cascade model. The upper pathway represents position-dependant intrinsic stiffness, whereas the lower pathway represents the velocity-dependant reflex stiffness [Merbagheri 1998 (66)].

3.4.2.2. Non-Parametric Identification Techniques

These techniques provide a numerical description of the system, require no *'a priori'* assumptions, and provide no information regarding the order or structure of the system under investigation. The most common linear or quasi-linear models are impulse response models and frequency response models. For the purpose of this study, we were interested in impulse response models only.

The dynamic relationship between position input and torque output is called *dynamic stiffness*. The inverse of stiffness is called *dynamic compliance*. A linear non-parametric representation of joint stiffness, called the *stiffness impulse response function* (IRF) can be defined by $S(\tau)$ such that:

$$T(t) = \int_{-t_{max}}^{t_{max}} S(\tau) \cdot \theta(t - \tau) d\tau \qquad (3.1)$$

Similarly, the *compliance impulse response function* $C(\tau)$ is defined by:

$$\theta(t) = \int_{-t_{max}}^{t_{max}} C(\tau) \cdot T(t - \tau) d\tau \qquad (3.2)$$

Here, the output is computed by convolving the input signal with the IRF in the time domain. To obtain accurate estimates of the IRFs, the input signal must have a wide-band frequency content that stimulates all system modes. IRF estimation is also susceptible to noise in the input signal. The dynamic stiffness or compliance IRF can predict the response of the joint to any position or torque input, respectively.

Intrinsic compliance and reflex stiffness IRFs were computed using a parallel cascade identification procedure described in previous work [60]. The procedure was as follows:

1. *Intrinsic Dynamics:* first, intrinsic stiffness dynamics were calculated from the measured position and torque signals according to Equation 3.1. The length of the intrinsic stiffness IRF was fixed to 40 ms, less than the reflex delay to ensure that reflex stiffness dynamics did not corrupt intrinsic stiffness estimates. The corresponding intrinsic compliance IRF was calculated subsequently by taking the inverse of the intrinsic stiffness IRF.
2. *Reflex Dynamics:* reflex stiffness was then estimated from the residuals, the difference between actual measured torque and predicted intrinsic torque. The reflex stiffness was represented by a static non-linearity (half-wave rectifier), followed by a linear dynamic relationship between velocity input and residual torque output. The length of the IRF was typically from 200-400 ms.
3. *Activation Dynamics:* reflex activation IRF estimates were also calculated, using velocity as input and MGS EMG as output. The MGS was used since it was the largest muscle in the triceps surae, and EMG magnitude was the largest and most detectable. The length of the IRF was typically from 200-400 ms. The estimated IRFs provided estimates of the conduction delays associated with the reflex pathway. Reflex delays were about 40 ms.
4. *Net Predicted Torque:* Subsequently, the net torque predicted by the complete model was calculated from intrinsic and reflex torques estimates.

5. *%VAF:* The overall accuracy of the identification procedure was evaluated by calculating the percentage variance accounted for (%VAF) described as:

$$\%VAF = 100 \times \left(1 - \frac{\left(T_{obs} - T_{pre} \right)}{T_{obs}} \right) \tag{3.3}$$

where T_{obs} is the observed torque output, T_{pre} is the torque predicted by the complete model.

3.4.2.3. Parametric Identification Techniques

Unlike non-parametric techniques, parametric techniques provide insight into the order and structure of a system, given some *'a priori'* assumptions. Assumptions about the structure and order of the system can be derived from the system's non-parametric properties, like its frequency response. Parametric identification methods describe the system analytically with a "small" parameter set.

Linear and non-linear least-squares methods are commonly used in parametric system identification. Non-linear parametric methods have been found to describe joint dynamics, intrinsic and reflex contributions well, provided that the experimental operating points (mean position, angular velocity, muscle activation level, displacement amplitude) remain fixed. These techniques utilize the recursive Levenberg Marquardt algorithm to minimize the mean squared error between the predicted response of a mathematical fit and the actual response.

Intrinsic Stiffness

Ankle intrinsic stiffness has been described by a quasi-linear, second-order model, which relates ankle position to torque [59]. It has elastic (K), viscous (B) and inertial (I) parameters:

$$\text{In time Domain:} \quad TQ(t) = I(\lambda)\frac{d^2\theta(t)}{dt^2} + B(\lambda)\frac{d\theta(t)}{dt} + K(\lambda)\theta(t) \tag{3.4}$$

where (λ) defines the current operating point of the system. This can also be written in Laplace domain (or frequency domain) as:

$$TQ(s) = \theta(s)\left[Is^2 + Bs + K\right] \tag{3.5}$$

The stiffness transfer function is defined as the ratio of torque output to position input in the frequency domain:

$$\frac{TQ(s)}{\theta(s)} = Is^2 + Bs + K \tag{3.6}$$

and the compliance transfer function is just the inverse:

$$\frac{\theta(s)}{TQ(s)} = \frac{1}{Is^2 + Bs + K} \tag{3.7}$$

Reflex Stiffness

Similar quasi-linear models have been applied to describe the response of reflex stiffness, relating input velocity to torque output at the ankle joint [60]. Reflex stiffness, arising from muscle activation dynamics, has been previously described by a second-order model [60]. However, subsequent investigations revealed that this was a poor representation of muscle activation dynamics for high levels of activation, and that a third-order, low-pass filter model was required to provide good fits [67]. The reflex stiffness transfer function is defined as:

$$\frac{TQ(s)}{V(s)} = \frac{G\omega_{n1}^2\omega_{n2}}{(s^2 + 2\xi\omega_{n1}s + \omega_{n1}^2)(s + \omega_{n2})}e^{-s\tau} \qquad (3.8)$$

where G, ξ, ω_1, ω_2, and τ correspond to the static gain, damping, first and second frequency parameters, and reflex delay respectively. (G) describes the steady-state response of the system, (ξ) describes the damping in the system, (ω_1) and (ω_2) describe the length of the reflex IRF response. These parameters have functional significance because they describe muscle activation dynamics and afferent dynamics.

3.4.3. Group Averaging and Statistical Analysis

Due to inter-subject and intra-subject variability of the ankle dynamics parameters, one must normalize results to compare among different subjects, as well for different experiments for each subject. Hence, for each experiment, ankle stiffness parameters were computed, and then trials associated with spurious spikes in the parameter values – due to noise – were eliminated if the position and torque tracking error were more than 2 standard deviations away from the control mean. Typically, these spurious spikes occurred during the 30% MVC period. Noise could result from: *(1)* change in contraction level, or generated torque, and/or *(2)* movement of the toes or the limb. Following this, the computed parameters for all trials were normalized to the mean of the parameters calculated in the control trials. This procedure was repeated for all experiments and for all subjects, and a cross-subject average was computed for each parameter.

4. Experiment I: Alternate Pattern in the Triceps Surae Activity and Variation of Endurance Time with Various Submaximal Contractions

4.1. Objective

The objective of this experiment was to examine the activity of the three muscles in the triceps surae under fatiguing, submaximal, isometric contractions. The objective was: (1) to characterize endurance time as a function of contraction level, and (2) to monitor EMG activity in the synergistic triceps surae during the course of the contractions. Our motivation was to examine the adaptive physiological mechanisms related to neuromuscular control and muscle contractile mechanics. Results of this experiment were used to design the paradigm of Experiment II.

4.2. Experimental Protocol

As illustrated in Figure 4.1, the experimental protocol consisted of a series of trials in addition to the ROM and MVC determination trials discussed previously in Chapter 3:

Isometric Contraction #1

This trial was used to evaluate subjects' endurance time at various contraction levels. Subjects were asked to hold a constant, sub-maximal, isometric contraction of the triceps surae as long as possible. Root mean square values of EMG and mean torque were calculated and display every 30 seconds through the experiment to monitor the state of the muscles. The time at which subjects could no longer hold a constant torque was defined as the *endurance time*.

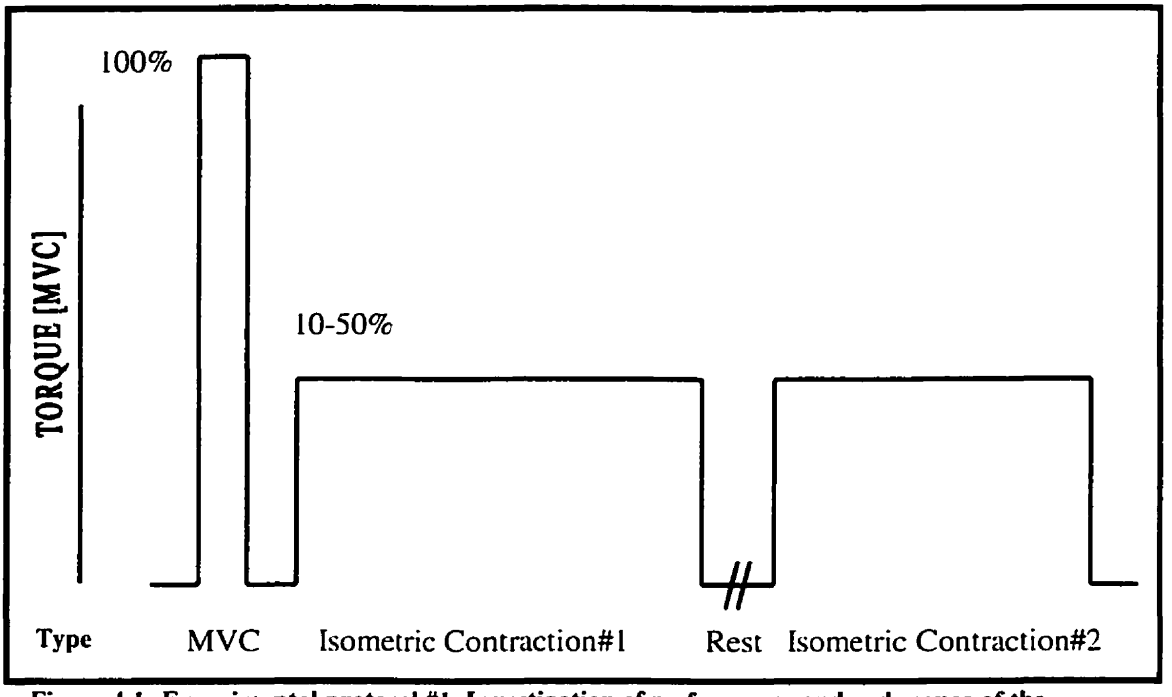

Figure 4.1: Experimental protocol #1, Investigation of performance and endurance of the triceps surae.

Rest

A 10 minute rest period at neutral position followed the first isometric contraction to allow recovery from fatigue. The subject's foot remained attached to the actuator pedal at all times, and subjects were instructed to remain quiet.

Isometric Contraction #2

Subjects were again asked to maintain a constant isometric contraction, similar to that in the first isometric contraction, for as long as possible. The objective was to evaluate recovery from fatigue after the 10 minute rest period.

4.3. Results

4.3.1. MVC

An experimental record of a maximal voluntary plantarflexing contraction (MVC) is shown in Figure 4.2.

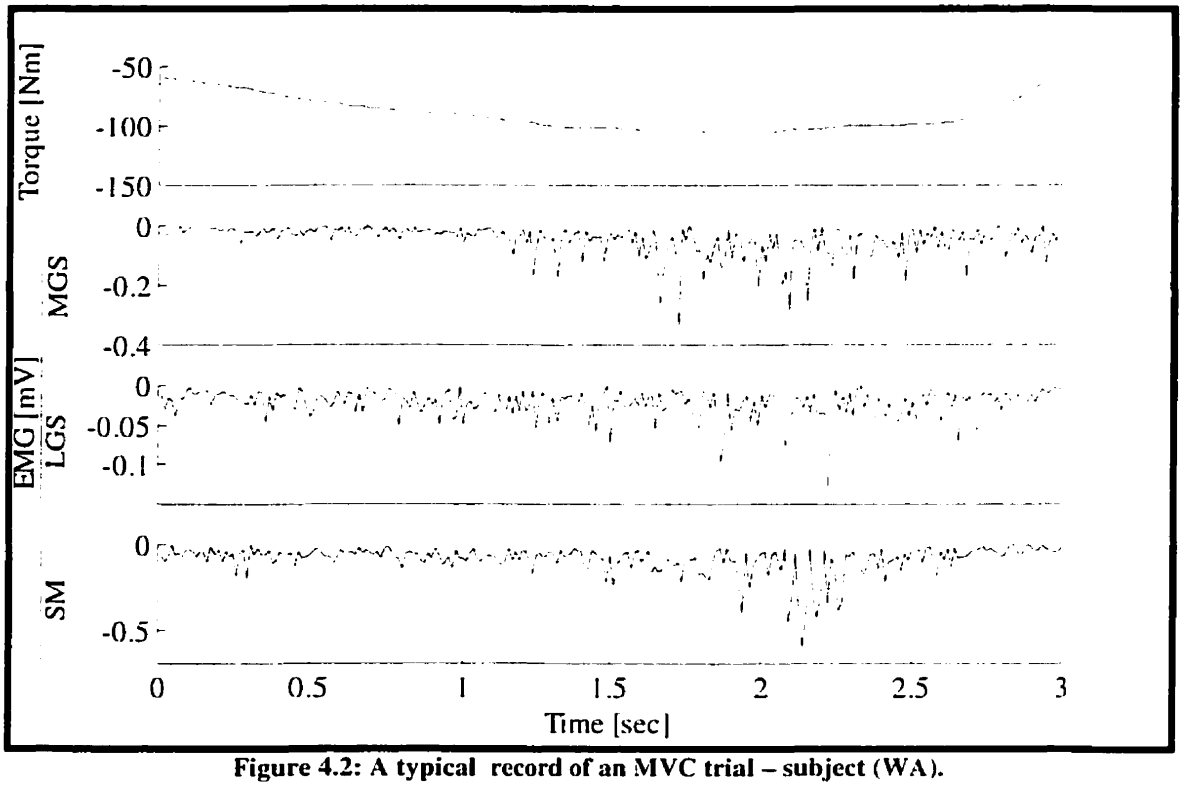

Figure 4.2: A typical record of an MVC trial – subject (WA).

Torque and EMG signals from three ankle flexors were recorded for 3 seconds, and the instantaneous maximum values determined. These values were used to normalize experimental torque and EMG values acquired in subsequent trials.

4.3.2. Isometric Contraction #1

Figure 4.3 shows an experimental record of a static, voluntary, isometric contraction (IC#1) of the triceps surae at 20% MVC. All muscles were active at the start of the

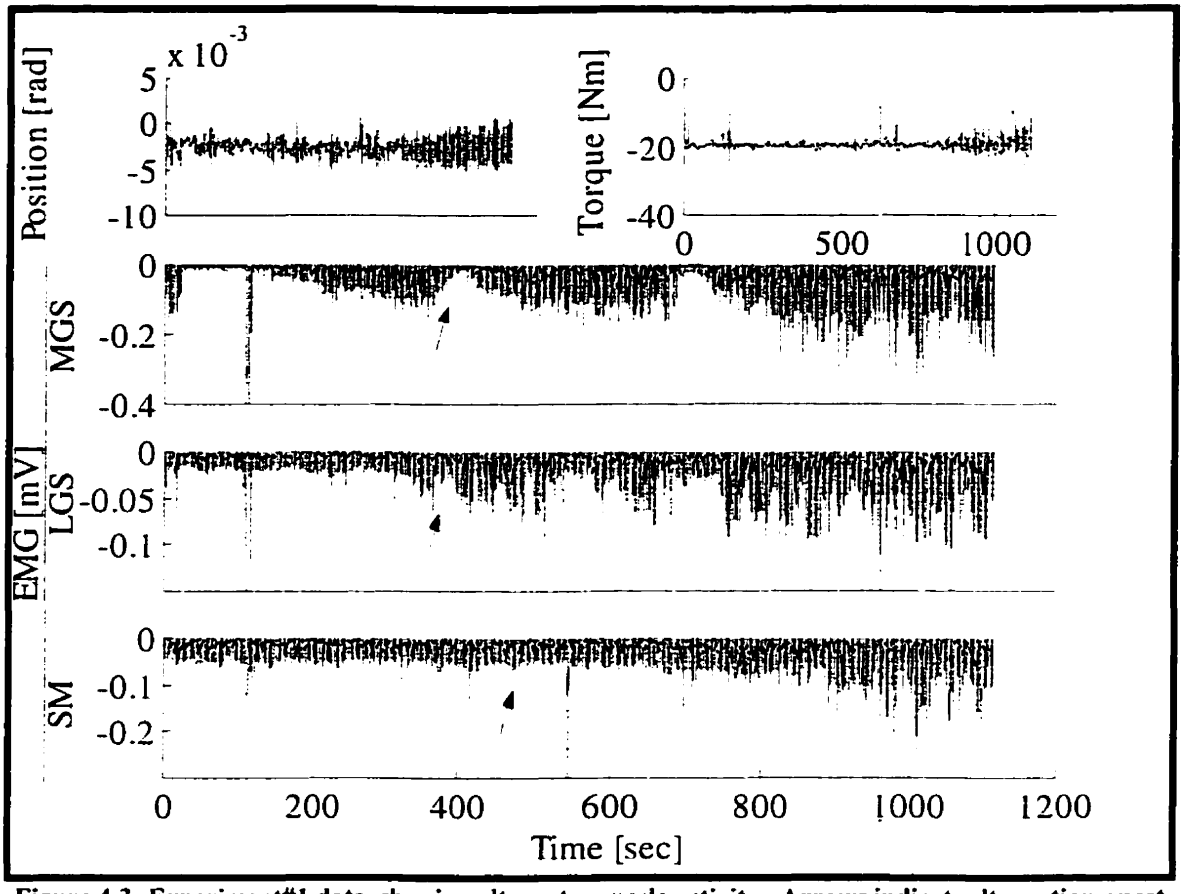

Figure 4.3: Experiment#1 data, showing alternate muscle activity. Arrows indicate alternation onset in the three muscles – subject (WA).

contraction, and as time progressed, their *activities increased* and then *decreased*. They progressively began to exhibit an *alternation pattern* between two states: active and silent. While the subject maintained relatively constant position and torque during the first 800 seconds, EMG activity was not. In general, *alternation onset*, defined as the time when the active muscle first became silent, occurred first in the medial head of the gastrocnemius (MGS), followed by the lateral gastrocnemius muscle (LGS), and lastly the soleus muscle (SM). The arrows in Figure 4.3 and Figure 4.4 indicate the alternation onset in all three muscles.

Figure 4.4 shows the mean position, torque, and RMS values of the triceps surae group for the same subject. Increasing and alternating EMG activity of the triceps surae muscles were evident. To illustrate the point, the MGS EMG increased from 1% MVC EMG to 8% MVC EMG, then decreased to 2% MVC EMG, then increased to 11% MVC EMG, then decreased to 2.2% MVC EMG, and finally increased to 16% MVC EMG. Although the MGS exhibited alternate activity patterns, the baseline EMG (1%, 2%, and 2.2% MVC EMG) showed an increasing trend. These observations were associated with fatigue of the triceps surae. Similar muscle activity patterns were seen in other experiments. In general, subjects tolerated such a contraction for 24 (± 8) minutes.

Throughout the contraction trial, muscles of the triceps surae contributed differently to the total tension generated, yet they were complementary to each other. When one muscle became silent, others became more active to compensate, hence maintaining constant torque. In figure 4.4, the MGS, being the biggest muscle, was the most active initially. However, with progression of fatigue, MGS activity decreased and the LGS and the SM EMG activities increased to maintain a constant torque. This pattern of alternate EMG activity was observed through the experiment.

In these experiments, it was important to ensure relatively constant mean position and torque (± 10%) during the contraction trials. *Tracking error* and *tremor* were calculated by taking the standard deviations of the position and torque signals respectively for 2 s epochs. Figure 4.5 shows that tracking error and tremor increased with time for one

subject. Second order fits showed the general trend in these plots. Both tracking error and tremor were relatively constant during the first 800 seconds, and increased thereafter.

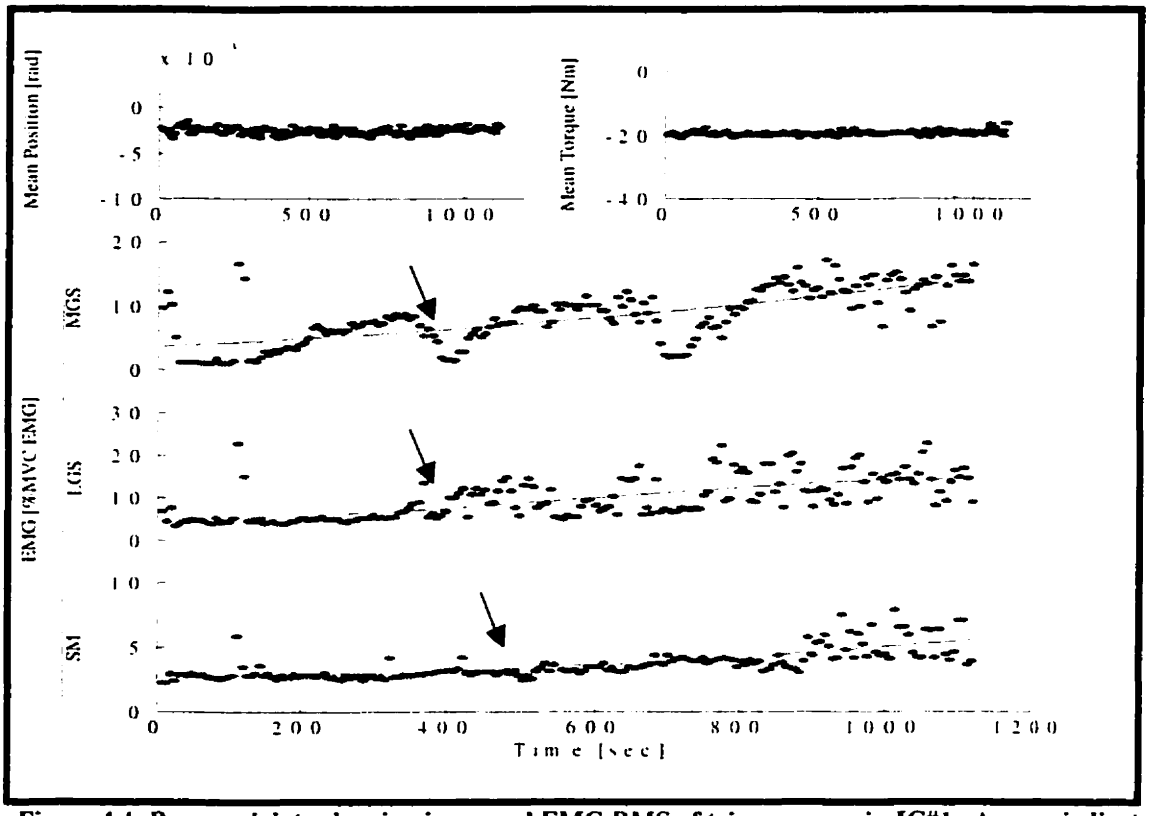

Figure 4.4: Processed data showing increased EMG RMS of triceps surae in IC#1. Arrows indicate alternation onset in the three muscles. Quadratic fits show the general trend in the plots – subject (WA).

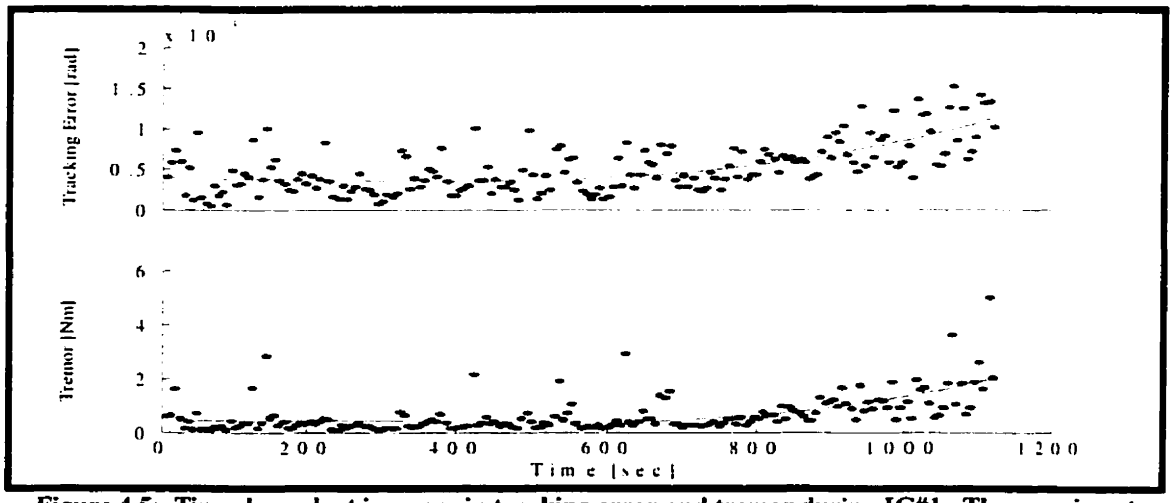

Figure 4.5: Time-dependent increase in tracking error and tremor during IC#1. The experiment was stopped once tracking error and tremor increased to more than 10% of target position and torque signals. Quadratic fits show the general trend in tracking error (R= 0.68) and tremor (R= 0.61) – (subject WA).

4.3.3. Comparison of Isometric Contraction #1 and Isometric Contraction #2

Figure 4.6 shows the mean position, torque, and the RMS values of the three muscles of the triceps surae during isometric contraction #2 (IC#2) for the same subject. Initial EMG RMS values in IC#2 were larger than initial values in IC#1. This suggested that the muscles had not completely recovered from fatigue during the 10 minute rest period. However after the 10 minute rest period, on average the MGS, LGS, and SM %MVC EMG recovered by 18%, 75% and 66.5% of the initial values in IC#1, respectively. In addition, alternation onset, a sign of fatigue, occurred earlier in IC#2 than in IC#1 – in IC#1 it occurred approximately at 400 seconds, whereas in IC#2 it occurred at 300 seconds – further reassuring that full muscle recovery was not achieved.

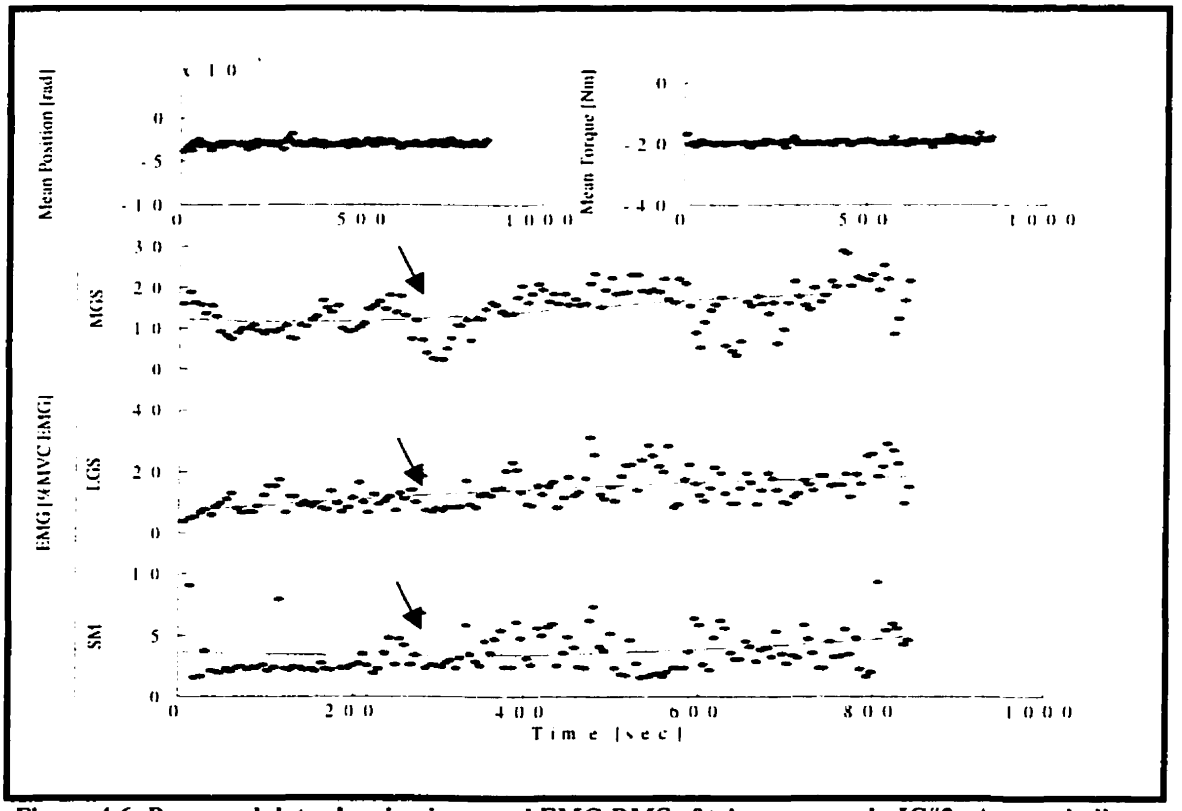

Figure 4.6: Processed data showing increased EMG RMS of triceps surae in IC#2. Arrows indicate alternation onset in the three muscles. Quadratic fits show the general trend in the plots – subject (WA).

4.3.4. Contraction Level versus Muscle Alternation Onset and Endurance Time

The isometric endurance and muscle alternation onset measured during the IC#1 at various contraction levels (10-50 %MVC) is shown in Figure 4.7.

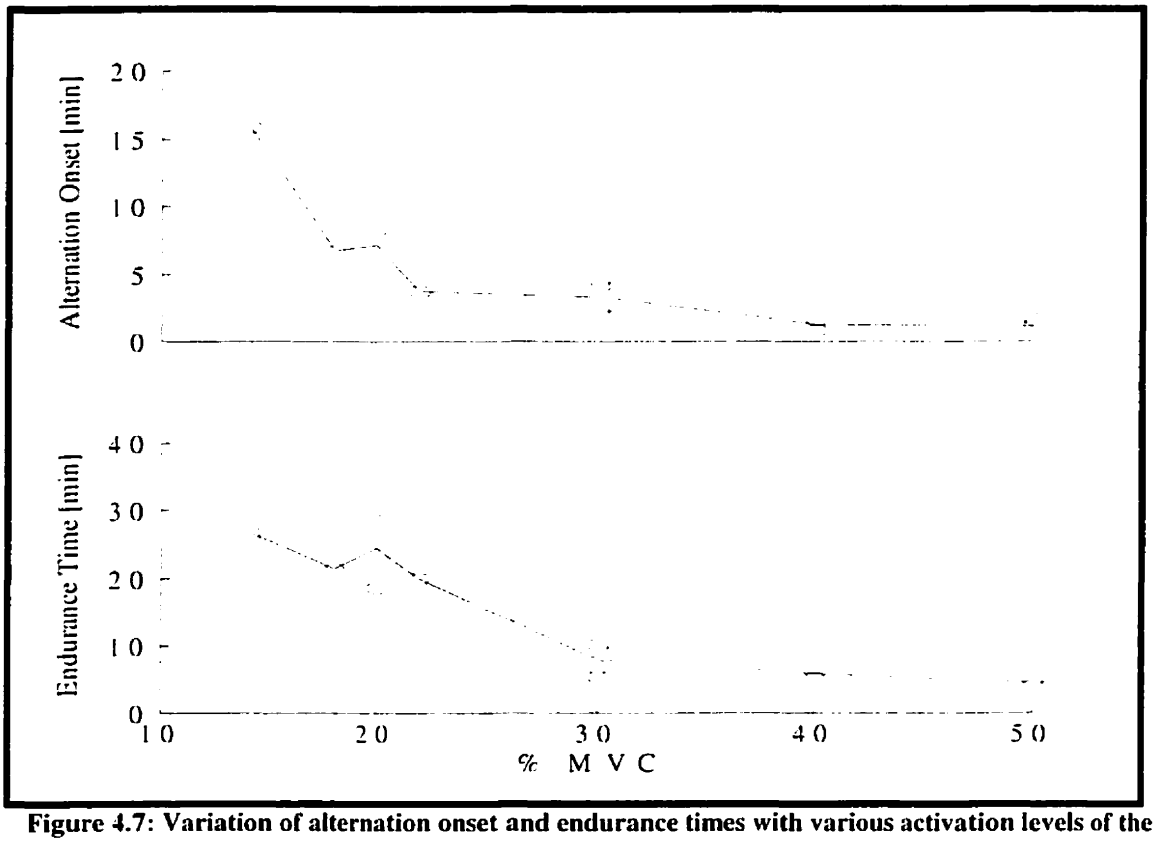

Figure 4.7: Variation of alternation onset and endurance times with various activation levels of the triceps surae. Circles represent real experimental data collected from all subjects.

As reported previously by others [68], we found that the longest isometric endurance and latest alternation onset occurred at the lowest isometric contractions. We found that endurance time decreased, and muscle alternation onset occurred earlier with increased contraction levels. Average endurance time of the subjects was (24 ± 8 min), (8 ± 2 min) and (4.6 ± 0.6 min) at 20%, 30% and 50% MVC respectively. Average time to the onset of muscle alternation was (7 ± 1.7 min), (3.3 ± 1.2 min), and (1.5 ± 0.3 min) at 20%, 30% and 50% MVC respectively.

Next, we fitted a linear model through the endurance times and alternation onset times collected for all subjects as shown in Figure 4.8. We found that the slope of the least-square line was 0.35 (R= 0.8). This indicates that in general alternation onset occurs at 35% of endurance time.

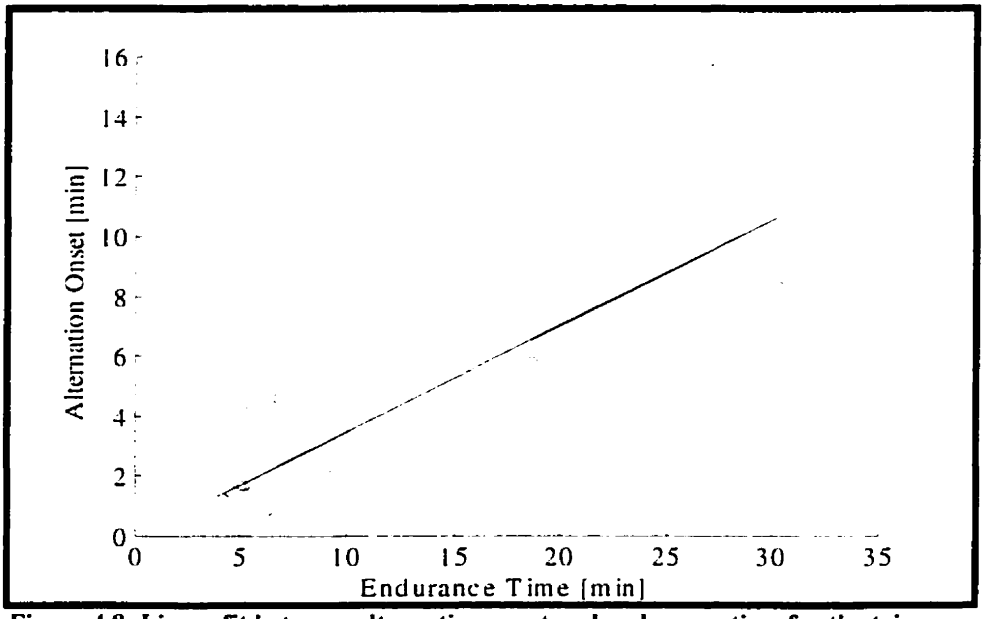

Figure 4.8: Linear fit between alternation onset and endurance time for the triceps surae (R=0.8). Circles represent real experimental data collected from all subjects.

4.4. Discussion

It is well established that the type, intensity, and duration of muscular contraction affect the physiology of muscular fatigue [23]. The main findings of this experiment were the following:

1. with progression of fatigue at the examined contraction levels (10-50% MVC), the synergistic triceps surae muscles exhibited cyclic activity between two states: active and silent, inferred from RMS EMG. EMG activity of each muscle increased and decreased continuously throughout the contraction.

2. throughout muscular contraction, the EMG activity of the triceps surae muscles varied, yet it was complementary among the synergistic muscles,
3. tracking error and tremor increased with time, indicating that subject's ability to maintain contraction was deteriorating.
4. during isometric, fatiguing contractions, the medial gastrocnemius exhibited alternation pattern the earliest, followed by the lateral gastrocnemius, and lastly the soleus muscle. However, the order of recovery during the 10 minutes rest was: LGS, SM, and then the MGS,
5. endurance time decreased and muscle alternation onset occurred earlier with increased contraction levels (10-50% MVC).

The current study, and one other [71], found a pronounced EMG cyclic activity among synergistic muscles of the triceps surae. This cyclic activity was described by two states: active and silent, comprising of continuously increasing and decreasing EMG pattern. Another study suggested that rotation of active motor-units within a fatigued muscle occurred due to changes in their relative activation thresholds [69]. Moreover, another study reported that EMG activity, collected from active motor units, disappeared and then reappeared at a later time during prolonged elbow flexion at 10% MVC [70].

These temporal changes in EMG activity may be caused by changes to the synaptic input to the motor-neurons. Synaptic inputs from red nucleus, pyramidal tract neuron, and cutaneous nerve cause a facilitative influence on fast-type motor-neurons, but cause an inhibitory influence on slow-type motor-neurons [72]. In contrast, inhibitory inputs from

Renshaw cells and excitatory inputs from group Ia afferent fibers are generally larger in fast-type motor-neurons [73,74]. The magnitude of these neural inputs may vary with progression of fatigue during prolonged contraction efforts. These alternations in recruitment patterns and rotations of muscular activity may serve to minimize fatigue of active motor units.

In this study, we found that the triceps surae muscles possessed variations in EMG activity, yet they were complementary to each other. The time-dependent increase in tracking error and tremor shows that all muscles failed at endurance limit. Another study showed similar complementary activities of the synergistic triceps surae muscles [71].

Force-regulation mechanisms ensure that appropriate measures are taken to maintain constant torque [4]. Afferent input from Golgi Tendon Organs from each muscle provide continuous force feedback to the integrating center in the spinal cord. It seems that the magnitude of these input signals are modified during fatiguing contractions of the synergistic triceps surae, which leads to the variation in individual muscle contribution to the total force. Increased inhibitory reflex input from group III and IV afferents might decrease the sensitivity of alpha-motor-neurons [90,93,94], causing a decrease in firing rate, and hence failure at endurance time.

For plantarflexing contractions, we observed that the medial gastrocnemius exhibited alternation patterns the earliest, followed by the lateral gastrocnemius, and lastly the

soleus muscle. However, EMG data showed that the order of recovery during the 10 minute rest was: LGS, SM, and then the MGS.

This can be explained in terms of variation in muscles fiber composition and homogeneity. It is well known that type (I) muscle fibers are more fatigue-resistant than type (II). However, type (I) fibers require more time to recover fully, once they are fatigued [11]. It is believed that the soleus exhibits the highest distribution of type (I) muscle fibers, followed by the LGS, and the MGS [75]. Also, the MGS, being the biggest muscle, was capable of generating the biggest torque initially. However, with progression of fatigue, the LGS and the SM took over to maintain a constant torque. This alternate muscle activity was observed through the experiments.

In this study, we showed that endurance time and EMG alternation onset depended on the level of isometric contraction of the triceps surae. For all subjects, as the level of contraction increased, endurance time decreased and EMG alternation onset occurred earlier. The former finding agrees with the results of a study conducted by Pertrofsky et al in 1981. They conducted endurance experiments on the biceps brachia at various contraction levels, and observed fatigue and recovery by monitoring EMG activity collected from the muscle. They concluded that endurance time decreased as the level of contraction increased. However, they attributed this behaviour to local, intrinsic muscle fatigue, rather than central or peripheral nervous system fatigue.

4.5. Physiological Mechanisms

The functionality of voluntary muscle activation can be assessed by the fore-mentioned EMG techniques [23,53], the twitch occlusion technique [98], and H-reflex techniques [50]. Target tracking error and tremor are further used to assess the efficiency of central supraspinal and spinal regulatory mechanisms.

The EMG amplitude is known to increase with the level of muscle activation [23,44,52,53], and this relationship was found to be quasi-linear for the human triceps surae muscles [68,99]. Previous studies by Loscher had shown that during sustained 30% MVC contractions of the triceps surae, EMG amplitude at endurance limit was only 50% of that of an unfatigued MVC [32]. Hence, he concluded that fatigue was mostly related to central factors, and not only local muscular factors.

On the other hand, the twitch occlusion technique relies on the superimposition of a supramaximal electrical stimulus onto a voluntary contraction [98]. Such stimulus is intended to recruit all motor neurons, including those not voluntarily recruited, and thereby adding an additional force to the contraction. It has been shown that superimposed twitch torque declines with increased voluntary torque exerted or increased activation level [98,100]. Also, it has been shown that during sustained 30% MVC plantar flexing contractions, twitch torque declines by 76% of the resting twitch torque [43]. Such changes were related to central factors, mainly regulation of (α) motor-neuron activity, and adaptation of fusimotor control strategies.

A further approach, that is used to assess muscle activation, involves monitoring the excitatory drive to the (α) motor-neurons pool via the H-reflex. The H-reflex is a short-latency muscular contraction that is elicited by electrical stimulation of group Ia afferent nerves from the muscle. It has been shown that the magnitude of soleus H-reflex increases with increasing contraction levels [35], but declines during sustained and intermittent maximal [26,27] and submaximal [35] contractions. This decline has been attributed to a fatigue-induced increase in the inhibitory input from group III and IV muscle spindle afferents [26,27].

It was also found that target tracking error and tremor increased with fatigue [43]. Tracking error is a precision measure of the subject's ability to track a target signal. It is controlled by supraspinal factors such as visual feedback, motor cortex, and descending neural pathways. On the other hand, tremor, defined as the oscillation in torque output, has been described to accompany isometric fatiguing muscle submaximal contractions. Several hypotheses have been made regarding its origin. While some believe it originates from unfused force ripples of active motor-neurons [101], others think it occurs due to oscillations in the stretch reflex arc [102]. Both increased active motor-neuron pool and increased reflex gain cause tremor magnitude to increase with progression of fatigue [32,82], thus leading to clonus.

5. Experiment II: The Effect of Fatigue on Ankle Stiffness Dynamics: Intrinsic and Reflex Components

5.1. Objective

The objective of this experiment was to determine the effect of triceps surae fatigue on ankle stiffness dynamics in normal human subjects, and delineate the relative contributions of intrinsic and reflex mechanisms. We applied pseudo-random binary sequence (PRBS) displacements before, during and after sub-maximal, fatiguing contractions of the triceps surae. Position, torque, and EMGs from the triceps surae and tibialis anterior were sampled. Using system identification techniques, we separated intrinsic and reflex contributions to total ankle stiffness, the dynamic relationship between ankle position and torque, and tracked how they changed with fatigue. This provided insight into neuromuscular adaptive strategies to fatigue.

5.2. Experimental Protocol

Choice of Perturbation Signal

To identify ankle dynamic stiffness and delineate its intrinsic and reflex contributions, we applied a series of PRBS displacements before, during and after sub-maximal fatiguing contractions of the triceps surae. PRBS displacements, with an amplitude 0.03 radians and a 150 ms switching rate, were used because they have enough power to identify ankle stiffness with a bandwidth up to 40 Hz, which is the range of interest for the investigated neuromuscular control system [60]. Signals with lower bandwidth (< 10 Hz) and angular velocity content, poorly identify the intrinsic dynamics of the ankle joint; whereas signals

with higher bandwidth (> 40 Hz) and angular velocity content, are known to suppress peripheral reflexes [76].

Contraction Trials

Figure 5.1 illustrates the experimental protocol that consisted of 24 trials, 30 seconds each, were applied during the experiment:

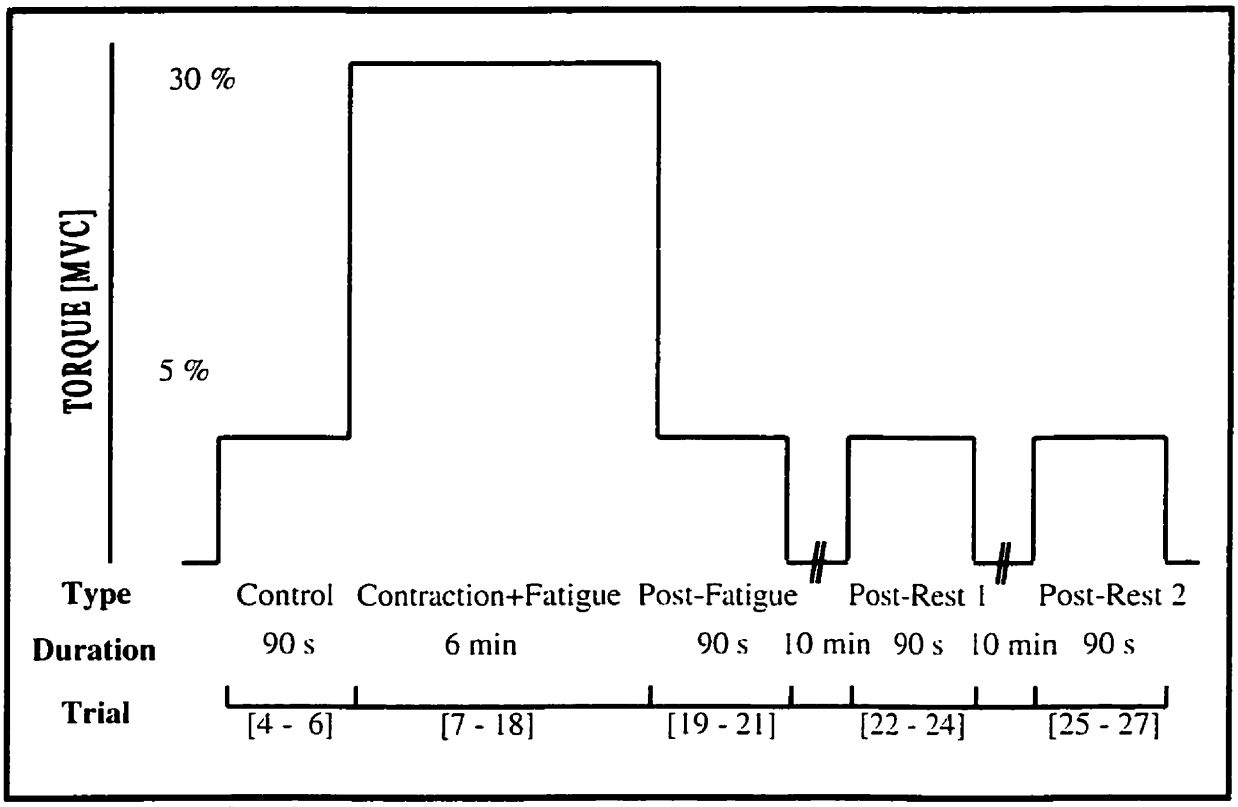

Figure 5.1: Experimental protocol #2, Investigation of the effect of fatigue on ankle stiffness dynamics.

1. Control

Initially, a 90 second control trial, performed at 5% MVC, was sampled to define the base-line ankle stiffness and intrinsic and reflex contributions prior to fatigue.

2. Contraction and Fatigue

The control trial was followed by a six minute contraction trial at 30% MVC to induce fatigue in the triceps surae.

3. Post-Fatigue

The contraction trial was followed by a 90 second trial at 5% MVC (base contraction level) to determine how stiffness parameters change due to fatigue alone, independently of contraction level.

4. Rest-Rest 1

Following a 10 minute rest period, a 90 second trial at 5% MVC was applied to determine how stiffness parameters recover after fatigue of the triceps surae.

5. Post-Rest 2

Following a second 10 minute rest period, a 90 second trial at 5% MVC was applied to determine how stiffness parameters recover further after fatigue.

5.3. Non-parametric Results

5.3.1. EMG Changes

Figure 5.2 illustrates typical PRBS displacements, torque, and four EMG signals recorded during a control trial for subject GM. The RMS values of three such control trials were used to normalize all EMG data for this experiment.

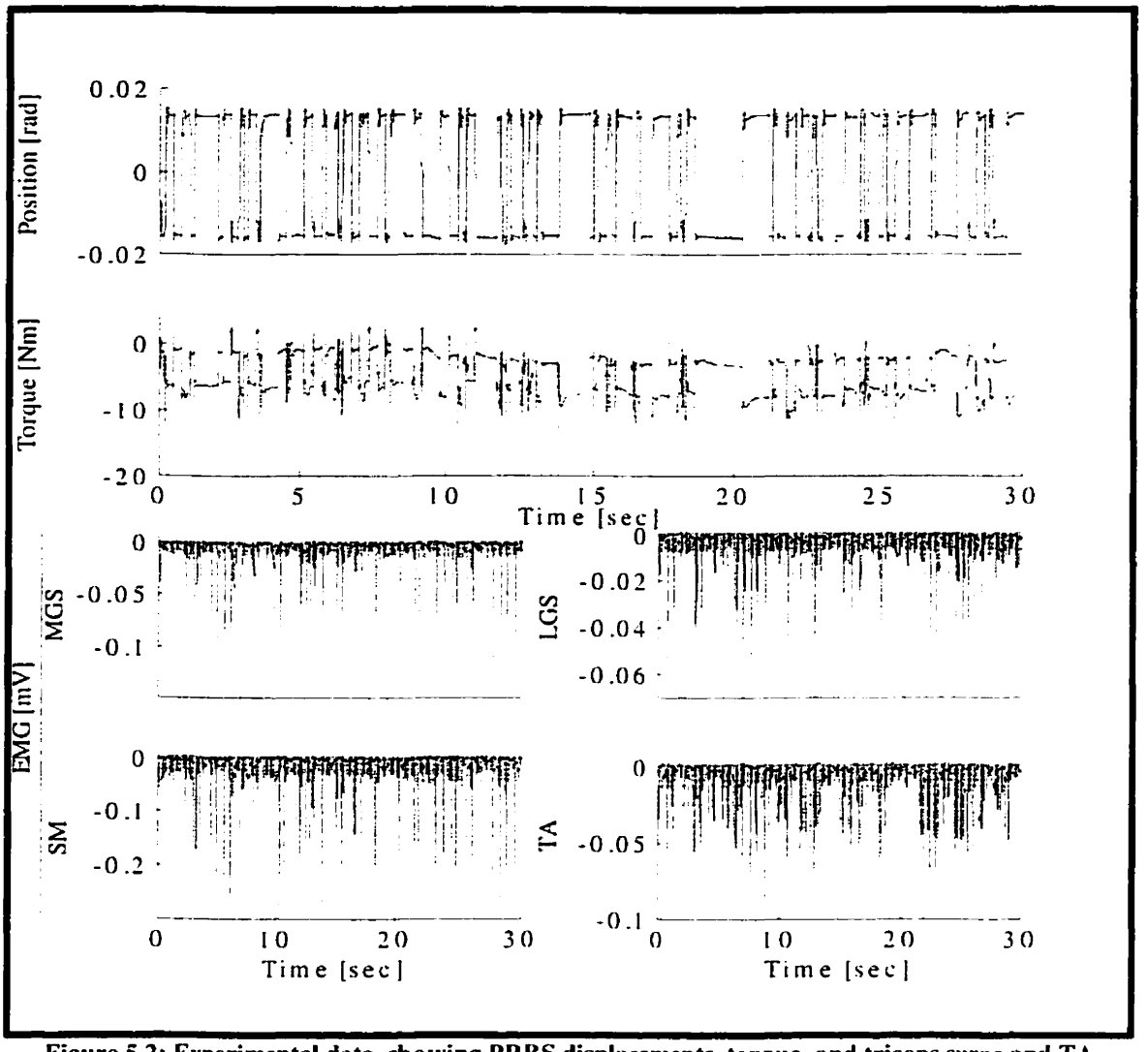

Figure 5.2: Experimental data, showing PRBS displacements, torque, and triceps surae and TA EMGs during a control trial (5% contraction, subject GM).

Figure 5.3 shows the mean ankle torque, and %MVC RMS for the MGS, LGS, SM, and TA during the course of the experiment for subject GM. The subject matched the target torque remarkably well over the course of the experiment. The mean EMG activity for all muscles increased with contraction level and with the progression of fatigue during the 30% MVC PF contraction period.

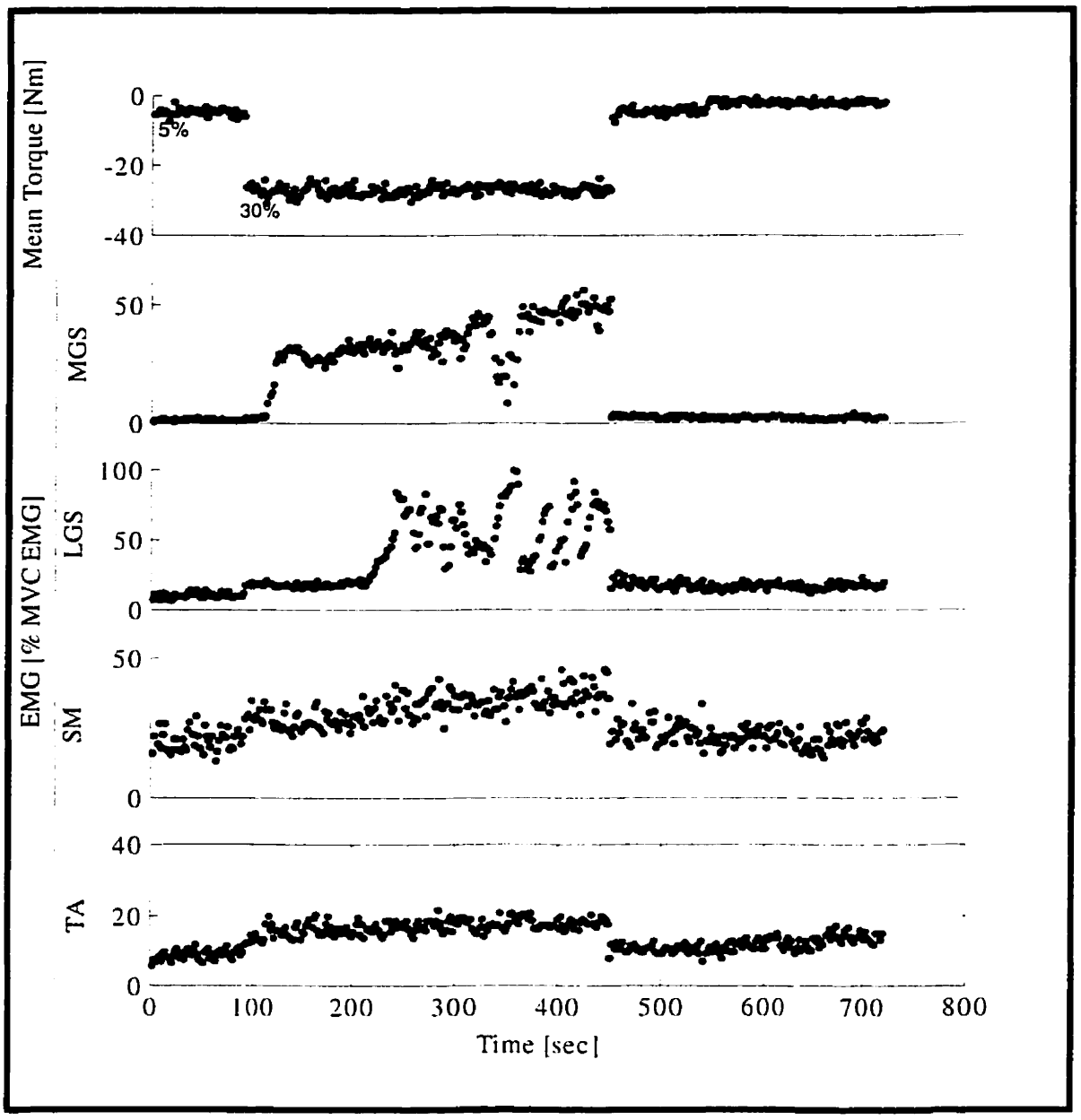

Figure 5.3:Torque and EMG variation during fatigue experiment. Each point represents the average value of a 2 second epoch (subject: GM).

Effect of Contraction on EMG Behaviour

Triceps surae and TA EMG activities increased as a result of the change in contraction level from 5% to 30% MVC. The MGS exhibited the biggest change as a result of this transition.

Effect of Fatigue on EMG Behaviour

Initially during the 30% MVC trials, the RMS EMG for the MGS, LGS, SM and TA, for subject GM, increased monotonically, then plateaued at 50%, 90%, 40% and 20% MVC EMG, respectively. The plateau level was different among the triceps surae group and across different subjects. Following the plateau, muscles' activity often decreased, as observed in the LGS, SM, and TA.

In addition, the MGS, LGS, and SM were cyclically active – EMG activity increased, decreased, then increased, etc... – during the 30% contraction period. The alternation pattern was different among subjects. TA activity increased monotonically, a sign of co-activation, since cross-talk was mostly eliminated by the applied filtering strategy.

Post-Fatigue - Recovery of EMG

EMG activity decreased during the Post-Fatigue trials (5% MVC contraction). RMS EMG continued to decrease during the subsequent recovery trials.

Similar results were observed for other subjects (AK, BA) and are shown in Figure 5.4 and Figure 5.5, respectively.

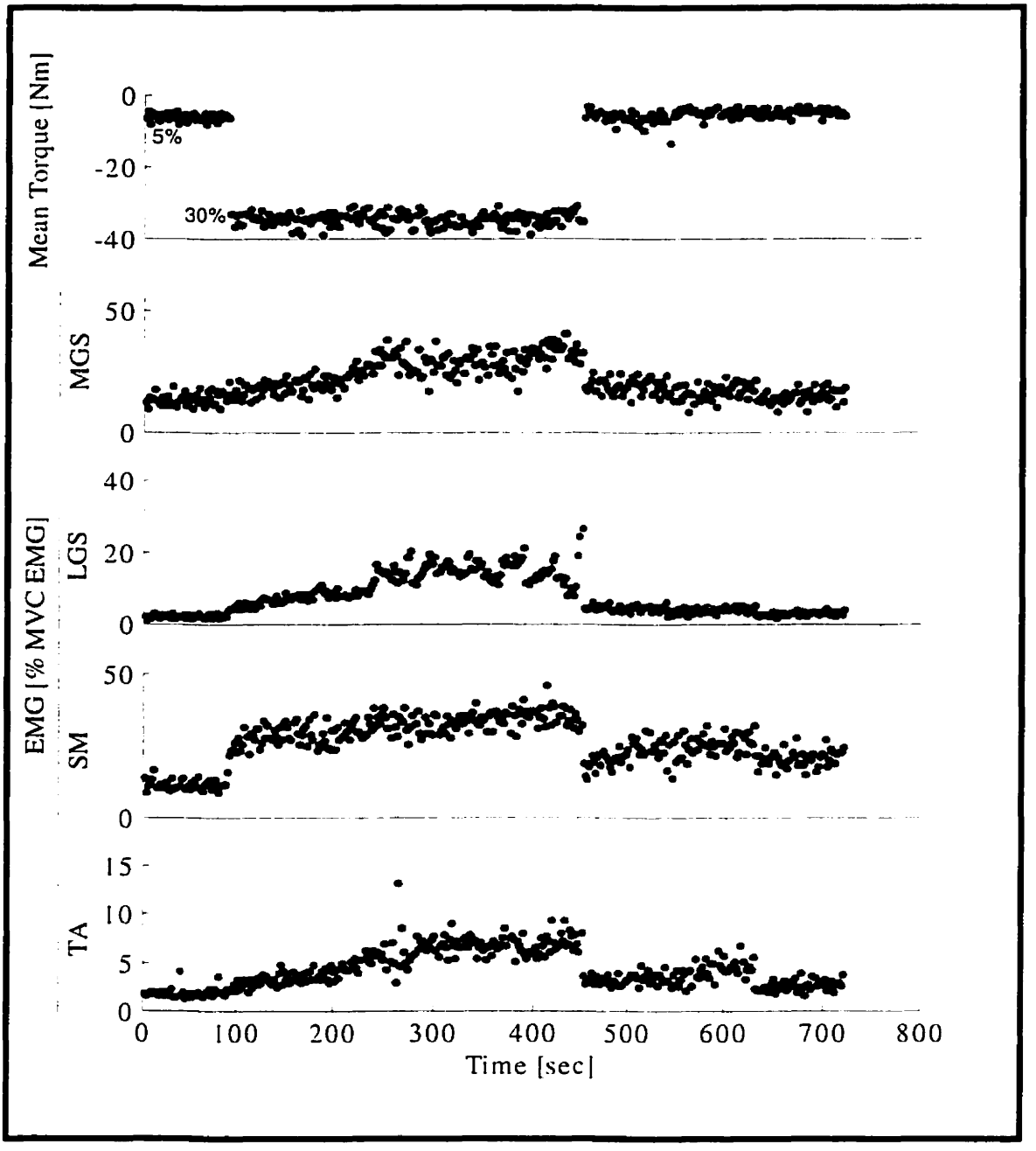

Figure 5.4: Torque and EMG variation during fatigue experiment. Each point represents the average value of a 2 second epoch (subject: AK).

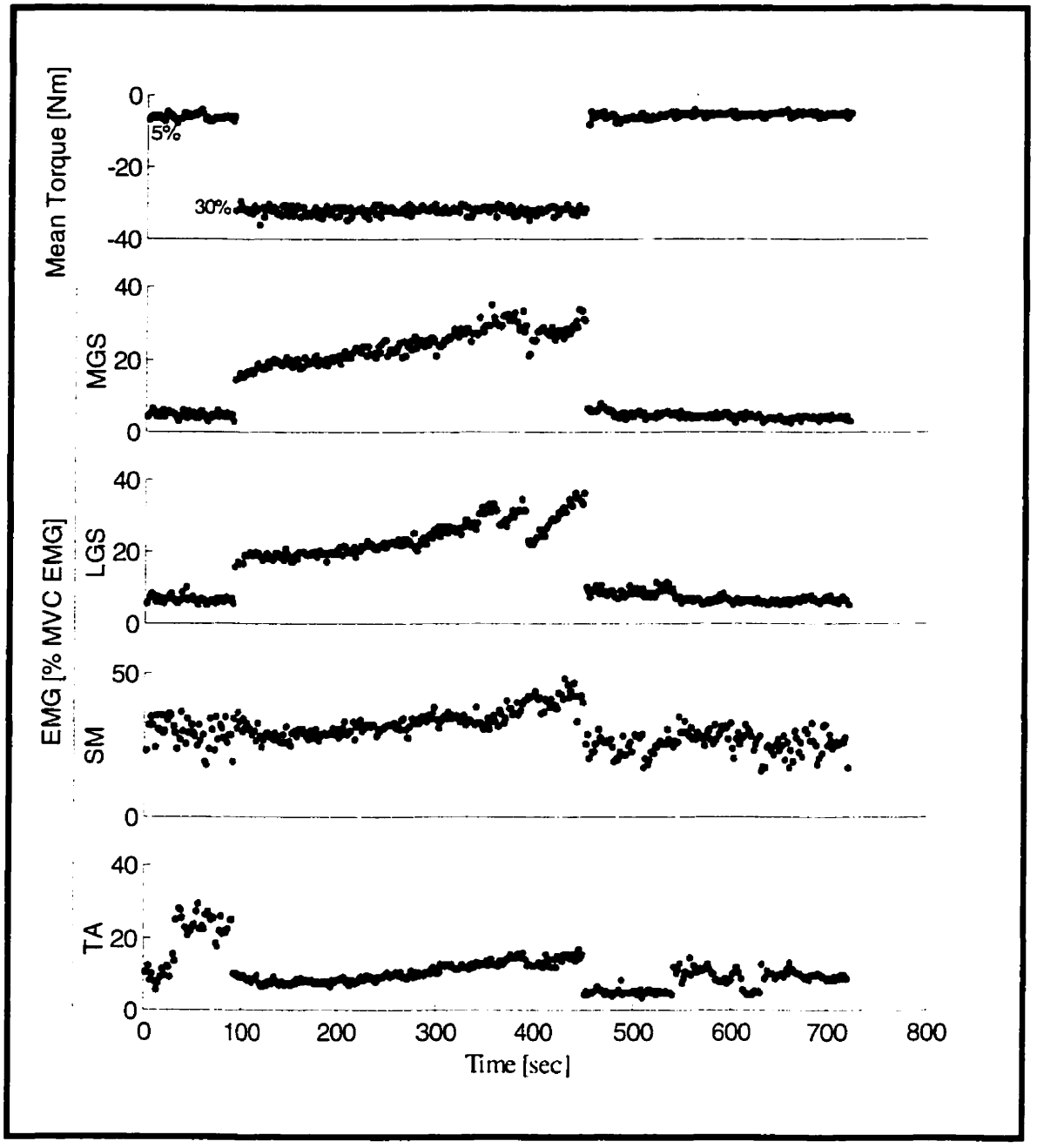

Figure 5.5: Torque and EMG variation during fatigue experiment. Each point represents the average value of a 2 second epoch (subject: BA).

5.3.2. Ankle Stiffness Changes

Intrinsic and reflex stiffness dynamics were computed and plotted at different intervals throughout the experiment. Figure 5.6 illustrates the intrinsic and reflex IRF changes through the various contraction trials. Two sets are shown in the *Contraction+Fatigue* trial: the first was taken immediately following contraction, and the second was taken after muscle cyclic activity was observed. Each intrinsic and reflex figure represents a single trial, whereas the LGS EMG activity shown is for the duration of the experiment.

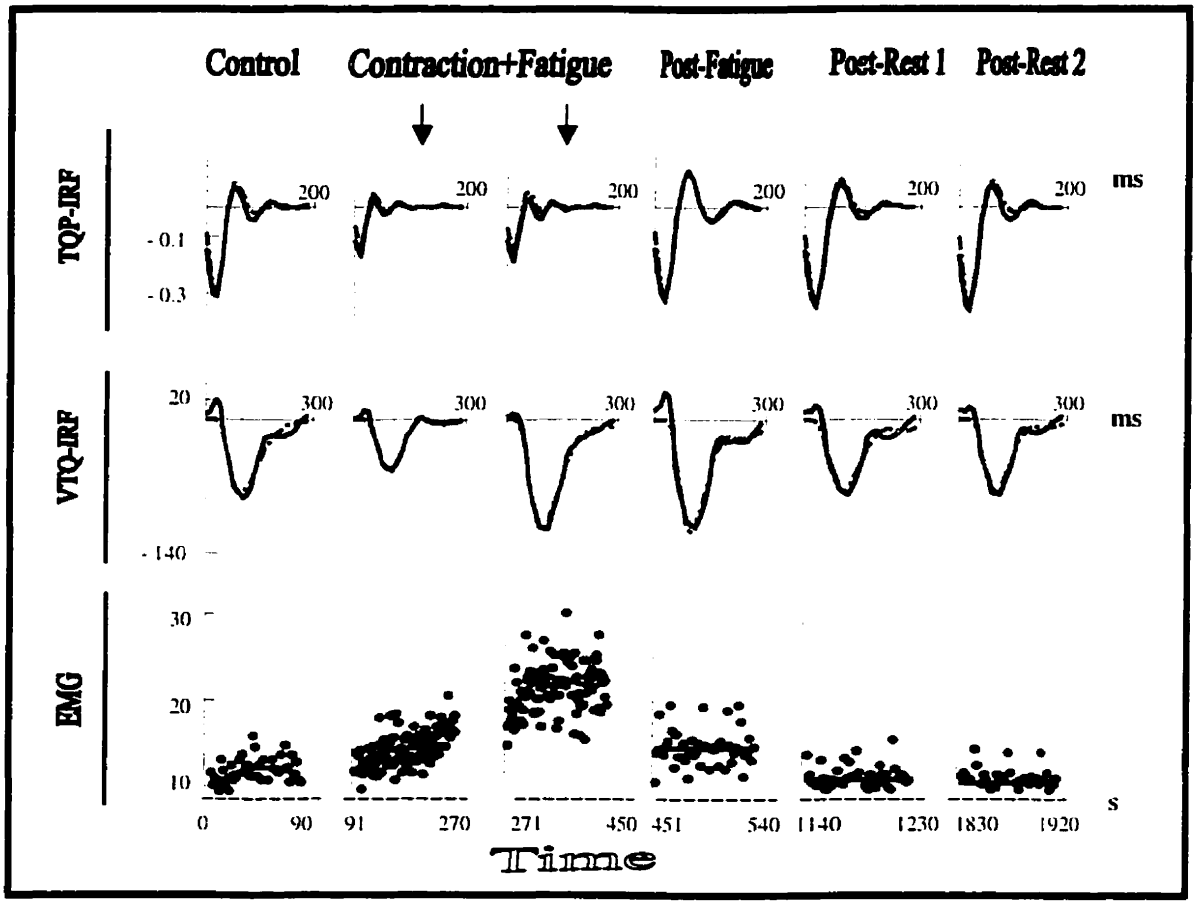

Figure 5.6: Intrinsic compliance *(top row)*, **reflex stiffness** *(middle row)*, **and normalized RMS EMG of LGS** *(bottom row)* **for different trials (subject VH).**

Intrinsic Dynamics Changes

The intrinsic compliance impulse response functions (TQP-IRFs) estimated at different intervals are shown in the top row of Figure 5.6. The following observations were made:

1. Intrinsic compliance dynamics changed with contraction level, as reported previously [77], and with triceps surae fatigue.
20 With contraction, the amplitude of the IRF decreased from -0.35 rad/Nm to -0.17 rad/Nm or by a factor of 48%.
2. With fatigue, the amplitude of the IRF increased slightly from -0.17 rad/Nm to -0.19 rad/Nm or by a factor of 12%.
3. In Post-Fatigue and both Post-Rest trials, the IRF shape remained slightly different from the Control trials.

Reflex Dynamics Changes

The reflex stiffness impulse response functions (VTQ-IRFs) estimated during the different trials are shown in the middle row of Figure 5.6. The following observations were made:

1. Reflex stiffness dynamics changed with contraction level, as reported previously [59,77], and with triceps surae fatigue.
2. With contraction, the amplitude of the IRF decreased from -82 Nm.s/rad to -52 Nm.s/rad or by a factor of 37%.
3. With fatigue, the amplitude of the IRF increased significantly from -52 Nm.s/rad to -105 Nm.s/rad or by a factor of 100%.
4. In Post-Fatigue and both Post-Rest trials, the IRF shape remained slightly different from the Control trials.

VAF Changes

Together, the intrinsic and reflex stiffness IRFs provided a good description of the linear dynamic relation between ankle torque and angular position and velocity. As shown in Figure (5.7, bottom), our parallel-cascade model accounted for over 80% of torque variance in all subjects throughout the experiment.

However, the percent variance accounted for (%VAF) by each mechanism changed with contraction and fatigue development as shown in Figure (5.7, top and middle):

1. Following the transition from 5% to 30% MVC contraction, intrinsic %VAF increased slightly, and reflex %VAF decreased significantly by more than 85%.
2. However with fatigue, intrinsic %VAF decreased from 82% to 74% or by a factor of 8%, and reflex %VAF increased from 1% to 2% or by a factor of 100%.
3. In Post-Fatigue and both Post-Rest trials, intrinsic and reflex %VAF were sightly different from the control values. This demonstrated that with fatigue, there was a shift in the total contribution of intrinsic and reflex mechanisms to the net ankle torque. While intrinsic stiffness decreased, reflex stiffness increased to maintain a constant torque output as illustrated in Figures 5.3, 5.4, and 5.5.

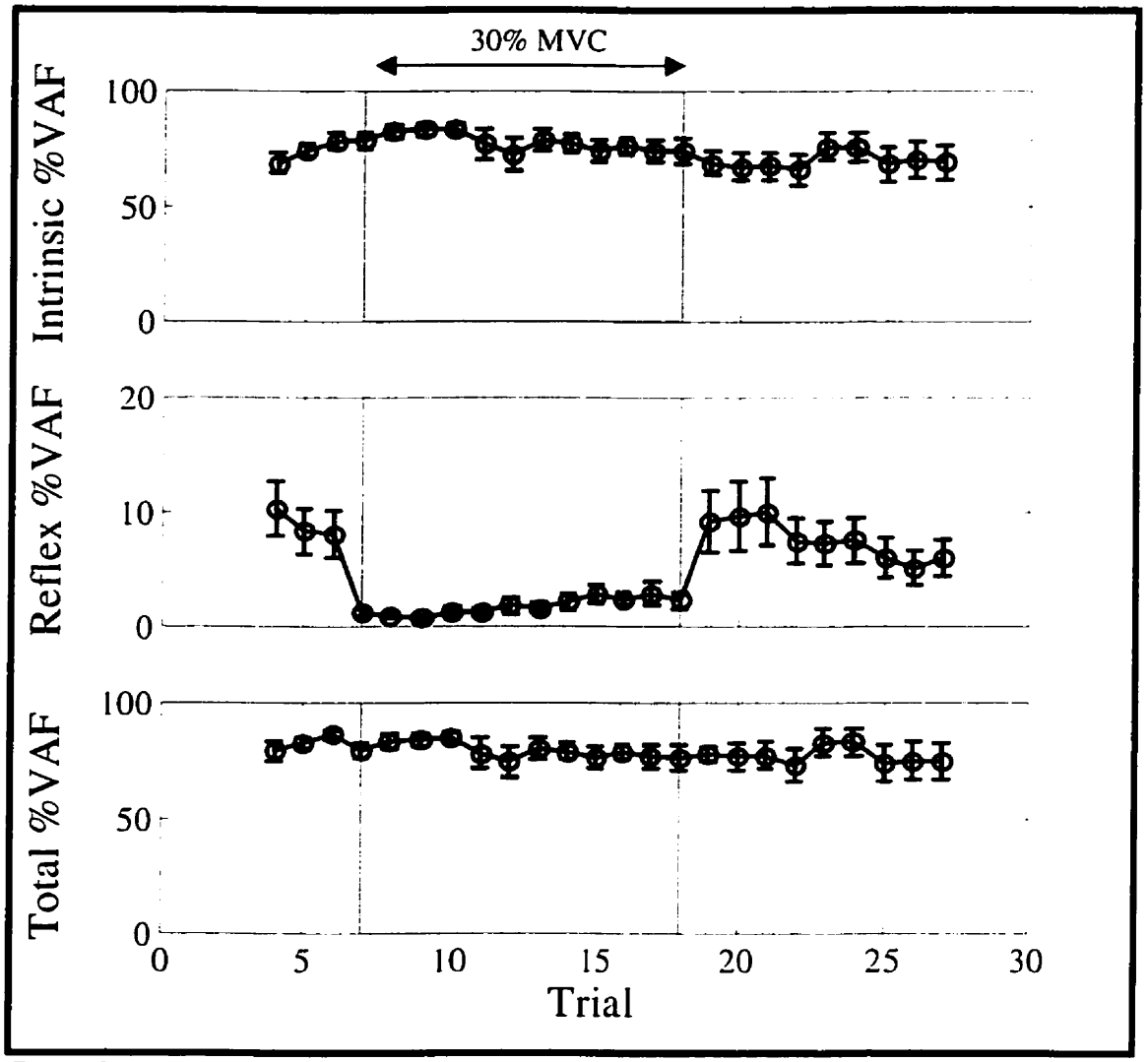

Figure 5.7: Mean variance accounted for by the intrinsic pathway *(top)*, reflex pathway *(middle)*, and total accounted for by the parallel cascade model *(bottom)*. Circles represent the group mean values, and error bars represent the group standard error.

5.4. Parametric Results

5.4.1. Intrinsic Stiffness

The intrinsic stiffness parameters (Elasticity K, Viscosity B, Inertia I) for each subject were normalized to the parameters obtained from the Control trials, and averaged. Figure 5.8 shows intrinsic stiffness parameters as a function of trial number. The following observation were made:

1. With contraction, both K and B increased, as previously observed [59,77].
2. However, with fatigue (30% MVC), K decreased progressively from 400 Nm/rad to 350 Nm/rad or by a factor of 12%, and B increased from 1.45 Nm.s/rad to 1.72 Nm.s/rad or by a factor of 19%.
3. Post-Fatigue, K returned to the control value immediately; however, B remained larger than the control value, even after 20 minutes of the contraction trial.
4. The inertial parameter (I) did not change significantly during the experiment.

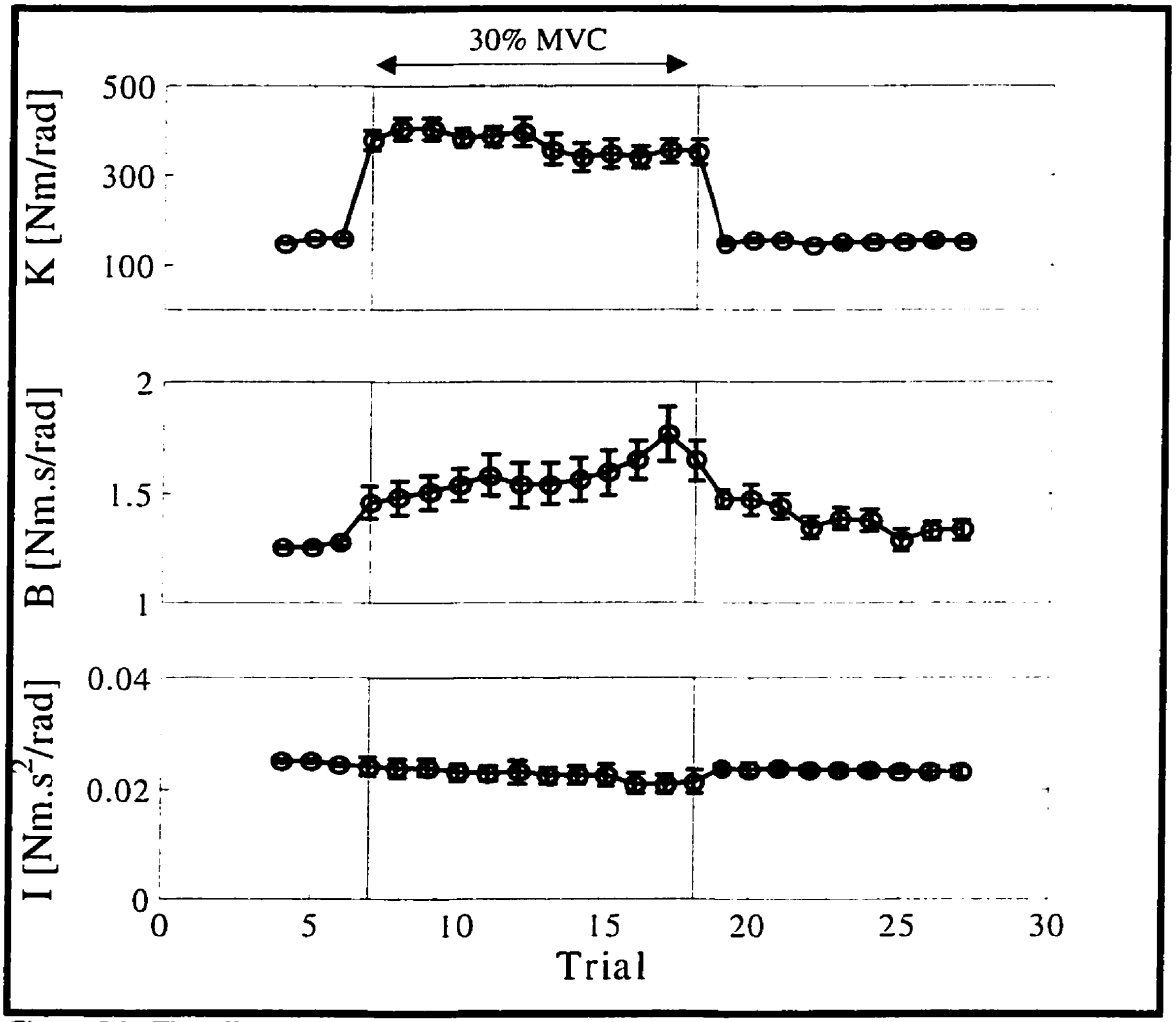

Figure 5.8: The effect of contraction and fatigue on the intrinsic parameters of the ankle joint stiffness. Circles represent the group mean values, and error bars represent the group standard error.

5.4.2. Reflex Stiffness

The reflex stiffness parameters (Gain G, Damping ξ, 1st Frequency ω_1, 2nd Frequency ω_2, and Delay D) for each subject were normalized to the parameters obtained from the Control trials, and averaged. Figure 5.9 shows these parameters as a function of trial number. The following observations were made:

1. With contraction, in general, G decreased from 12 Nm/rad to 8.5 Nm/rad or by a factor of 30%.
2. However with progression of fatigue, G increased from 8.5 Nm/rad to 19 Nm/rad or by a factor of 140%. The two frequencies (ω_1) and (ω_2) also changed - ω_1 increased by 100% while ω_2 decreased by 65%.
3. Even after 20 minutes following the contraction trial, these parameters remained significantly different from the control values.
4. The damping parameter (ξ) and delay (D) did not change with fatigue.

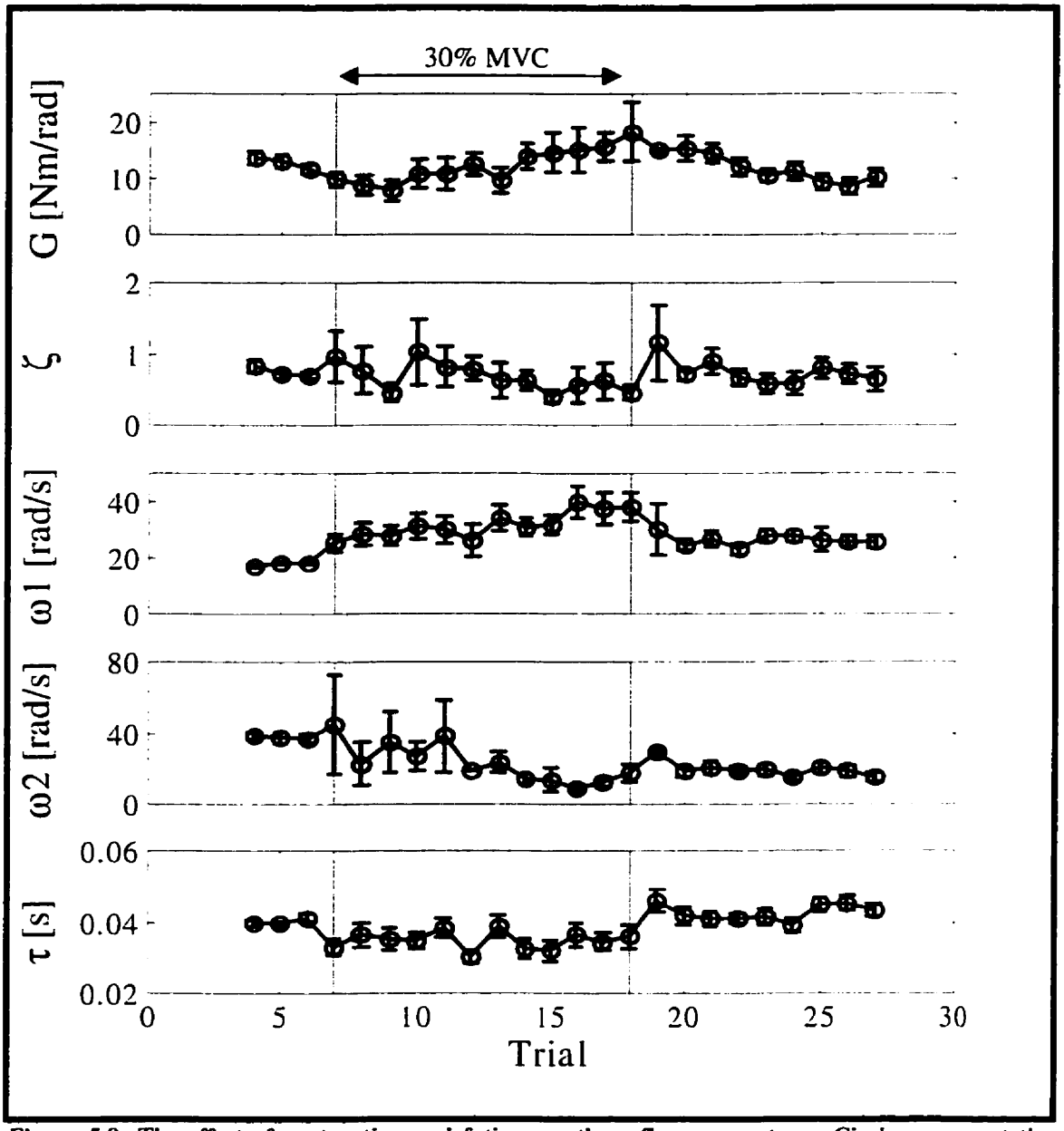

Figure 5.9: The effect of contraction and fatigue on the reflex parameters. Circles represent the group mean values, and error bars represent the group standard error.

5.5. Discussion

5.5.1. Changes in Ankle Stiffness

This study showed that as the triceps surae fatigued, there was a change in both intrinsic and reflex contributions to the total ankle stiffness. System identification revealed that

intrinsic stiffness decreased slightly as a result of decreased elasticity, and increased viscosity; whereas reflex stiffness increased significantly as a result of increased gain and the first frequency, and decreased second frequency. Overall, the total ankle stiffness, inferred from Total %VAF (Figure 5.7), decreased slightly following fatigue of the triceps surae, hence the joint became more compliant.

5.5.2. Relative Research

Vigreux showed similar conclusions relating to intrinsic, muscular compliance [88]. By combining a quick limb release paradigm with intermittent fatiguing contractions, Vigreux concluded that fatigued muscles were more compliant than unfatigued muscles. Petrofsky et al [68] studied fatigue in isolated muscles, and observed a change in the force-velocity curve which implied a change in muscle stiffness. They concluded that this change was due to fatigue or variation in activation level. This supports our finding that reflex gain typically increases during fatigue. Moreover, Avela et al found that stretch reflex sensitivity and muscle stiffness were reduced after long-lasting stretch-shortening cycle exercise in humans [94]. The test protocol included various jumps on a sledge ergometer. The interpretation of the reflex sensitivity was based on measurements of the patellar reflexes and the M1 reflex components. Muscle stiffness was indirectly calculated as a coefficient of the changes in the measured Achilles tendon force and muscle length. Fatigue caused a 14-17% reduction in the mean eccentric force, and a 17-32% reduction in the corresponding M1 area under the electromyograms, and hence reflex sensitivity. Muscle stiffness declined by 5.4-7.1%, depending on the jump condition. Hence, Avela et al concluded that decreased force output could be partly due

to decreased reflex sensitivity and modulation of the muscle, which accompanied decreased muscle stiffness.

Hunter et al [64] applied stochastic wideband (50 Hz) perturbation signal during a constant isometric contraction of the tibialis anterior muscle. They observed an incremental increase in EMG activity, yet a loss of force output at endurance time. Using system identification techniques [1], they were able to quantify torque-position relationship by a linear dynamic impulse response function (IRF). Non-parametric and parametric analysis of IRFs, collected before, during, and after fatigue revealed no significant changes in net ankle stiffness. It is known that reflex action is impaired during application of wide bandwidth stochastic perturbations [76,82]. Therefore, the study assumed that net ankle stiffness was only intrinsic stiffness, which didn't change significantly with progression of fatigue. Hence the results were valid and agreed with the results of this study.

In this study, we found that elastic stiffness decreased by 12%, and the reflex gain increased by 140%. Kirsch et al found that torque responses to a static ramp extension of the elbow flexor muscles decreased slightly after fatigue, whereas EMG responses increased, indicating the nervous system compensates, at least in part, for the decreased torque [89]. They concluded that force-sensitive reflex pathways are functionally important in force-regulation. In a later study, this group applied stochastic position perturbations and a step function to the forearm while extending the elbow flexor muscles [82]. Using system identification techniques, they identified two independent pathways:

intrinsic and reflex. The intrinsic component was computed from the isometric torque and position signals, whereas the reflex component was computed from the isometric torque and incremental EMG signals. Kirsch et al demonstrated that a substantial fatigue-induced loss of contractile force was accompanied by a smaller decrease in joint stiffness [82]. While intrinsic elastic stiffness decreased only by 13%, the magnitude of the reflex response increased, almost doubling, to mediate the substantial contractile loss of force output. These observations provide support to our findings in this study.

5.5.3. Physiological Mechanisms

It is known that length and force feedback neural pathways exist and work cooperatively to regulate muscle stiffness [79,80], and ankle stiffness [59,60,82]. The existence of peripheral length sensors, such as muscle spindles [83], and force sensors, like Golgi Tendon Organs [81] provide feedback signals that affect muscle activation. In addition, the existence of joint position receptors [84], and cutaneous receptors [85] provide continuous peripheral input into the central regulatory circuit. These sensors and receptors are sensitive to fatigue-induced alterations in the mechanical and metabolic state of the muscle [86,87].

Intrinsic stiffness changes may arise from decreased regional temperature and blood flow in the muscle and joint [23], accumulation of metabolite byproducts, such as lactic acid, and increased tissue acidity [54,55,68], local tissue damage and inflammation [23].

On the other hand, reflex stiffness changes may have occurred due to alterations in activation dynamics [94-97]. Such alterations might include increased number of active alpha motor-neurons, increased firing rate of these motor-neurons, increased membrane potential of receptors, and decreased nerve conduction velocity.

5.6. Related Behaviour

Mirbagheri et al examined the dynamic ankle over a wide range of ankle positions and tonic contraction levels in human subjects [59,77]. Using system identification techniques, they were able to separate intrinsic and reflex contributions to ankle torque. They found that both intrinsic and reflex stiffness increased with ankle dorsiflexion. However, intrinsic stiffness was more dominant at higher levels of tonic contractions. Reflex stiffness was largest at neutral positions and at low levels of muscular activity. These results were consistent with those obtained by Toft et al [105], Weiss et al [106], and Zhang et al [104].

In this study, we demonstrated the change in intrinsic and reflex properties as a result of a transition in activation level from 5% to 30% and back to 5% MVC. We noted that generally upon contraction, intrinsic stiffness increased, and reflex stiffness decreased.

On the one hand, the increase in intrinsic stiffness was mainly attributed to an increase in elastic stiffness of the joint, which occurred in all subjects. In contrast, the viscous parameter increased for some subjects (5 out of 9) and decreased for others (4 out of 9). This demonstrates the inter-subject variability in joint behaviour, observed earlier [66].

On the other hand, the decrease in reflex stiffness was attributed to a decrease in reflex gain, second frequency, and delay, and an increase in the first frequency.

6. General Discussion and Conclusion

6.1. Summary of Study

Muscle fatigue may be defined as any exercise-related reduction in the force-generating capacity of a muscle. Its effect on joint stiffness is not well understood. The purpose of this study was (1) to characterize fatigue in the triceps suare group, and (2) to determine the effect of triceps surae fatigue on the dynamic stiffness of the ankle joint. To address the first task, isometric contraction trials were applied to induce fatigue in the triceps surae. For the later task, pseudorandom binary sequence (PRBS) displacements were applied before, during, and after fatiguing contractions of the triceps surae. EMG from ankle extensors – triceps surae group – and flexors – tibialis anterior – measured the progression of fatigue. Position and torque were recorded and analyzed to identify the intrinsic and reflex contributions to ankle dynamic stiffness.

6.2. Summary of Results

In this study, we found that ankle intrinsic stiffness, comprising limb dynamics, articular and active muscle mechanics, changed with fatigue. Muscle activation patterns were modified to compensate for fatiguing synergistic muscles of the triceps surae. Complementary, cyclic EMG activity among these muscles was also observed. Both tracking error and tremor increased with fatigue. In addition, a general increase in RMS EMG was observed in all synergistic muscles, due to local muscle fatigue.

System identification techniques revealed changes in intrinsic and reflex components of ankle dynamic stiffness. For intrinsic properties, there was a decrease in joint elasticity

by a factor of 12%, and an increase in joint viscosity by a factor of 19%. These changes may have arisen from changes in limb dynamics, articular mechanics and active muscle mechanics.

For reflex properties, there was an increase in the reflex gain by 100%, which accompanied changes in reflex dynamics, seen in the shape of impulse response function. Reflex stiffness changes may have arisen from changes in the neuromuscular activation patterns.

6.3. Study Contributions

This study provides additional support to the following previously documented observations:

1. alternate activities of synergistic muscles during static and dynamics fatiguing contractions helped to minimize fatigue,
2. complementary rotations of muscular activity ensured constant torque output, at least until the endurance limit,
3. both tracking error and tremor increased with fatigue, due to decreased muscle stiffness and increased reflex potentiation.

In addition, this study provides evidence to support the following new observations:

1. as contraction level was increased, both endurance and muscle alternation onset times decreased,

2. the dynamics of ankle stiffness were modified with fatigue. Both intrinsic and reflex components changed – intrinsic stiffness decreased, and reflex stiffness increased.
3. for intrinsic changes, there was a decrease in elasticity, but an increase in viscosity.
4. for reflex changes, there was an increase in reflex gain, and change to the dynamics, resulting from changes to the natural frequencies.

Based on these results, we have shown that fatigue does, indeed, influence our performance. Thus, our capability to maintain long-lasting submaximal efforts was limited with fatigue due to possible adjustments in the force-length and force-velocity feedback mechanisms, as well as central and peripheral regulatory mechanisms.

We have also shown that both muscle and ankle – intrinsic and reflex – stiffness changed with fatiguing, submaximal contractions of the triceps surae. Decreased intrinsic stiffness might provide flexibility to the muscle and joint, yet increased reflex stiffness might provide stability, and hence postural and locomotor control.

6.4. Potential Physiological Implications of Fatigue

6.4.1. Muscle Stiffness versus Joint Stiffness

Stiffness is the dynamic relation between position of the joint and the torque produced. It is an important property in the control of posture and locomotion. The nervous system continuously modulates joint resistance in response to an external perturbation. It has

been proposed that the nervous system modulates muscle [80], joint [79,82], and/or endpoint [107] stiffness to compensate for external force perturbation or to carry out a voluntary movement. Hence, the net joint torque comprises muscle, joint, and reflex mechanics. Muscle mechanics include passive connective tissue and muscle mechanics, and active contractile muscle dynamics. This is referred to as *muscle stiffness*. On the other hand, joint mechanics include intrinsic and reflex dynamics. This is referred to as *joint stiffness*.

This study showed that joint intrinsic and reflex stiffness were altered during fatiguing, muscular contractions. While both the active muscle and passive intrinsic stiffness decreased with fatigue, the neurally-modulated reflex stiffness increased to compensate for the loss in mechanical torque output, at least until the endurance limit.

6.4.2. Joint Flexibility versus Stability

Although we require certain joint flexibility during our locomotion, this flexibility might lead to various injuries if not limited or modulated properly by the central and peripheral nervous system. Appropriate neural control mechanisms, redundant muscle strategies, and limited range of motion of limbs give rise to joint stability. Hence, this inter-play between joint flexibility and stability determines the efficiency of task performance.

This study showed that during submaximal, fatiguing contractions of the triceps surae, the ankle joint becomes more compliant (less stiff), due to a decrease in intrinsic stiffness. However to maintain joint stability, reflex stiffness increases to compensate for the loss in torque output.

6.4.3. Fatigue and Performance

Fatigue renders our ability to perform long-lasting tasks. With progression of fatigue, we soon find ourselves incapable of maintaining a muscular activation level or torque output. Performance efficiency, defined as minimal control strategies required to produce a contraction or movement, decreases with fatigue [108-112].

In contrast, one study demonstrated that the total reaction time, the time-lapse between a triggered light signal and maximal knee extension, did not change due to fatigue of the vastus medialis muscle [113].

This study showed that target tracking error and tremor increased with fatigue. We demonstrated the increase and alternation in background EMG activity of the synergists triceps surae and tibialis anterior muscles, while maintaining a constant mean torque output. However, at endurance limit, there was a general decrease in the torque output across all subjects.

6.5. Recommendation for Future Work

6.5.1. Static versus Dynamic Fatiguing Protocol

In this study, we used static, isometric contractions to fatigue the triceps surae, and independently, dynamic contractions to estimate ankle stiffness parameters.

Masuda et al showed that muscle fiber conduction velocity (MFCV) decreased significantly with static, fatiguing contractions of the knee, and was unaffected by dynamic contractions [114]. They concluded that MFCV was affected by both the muscle metabolic state and activation patterns.

Future studies might consider using combinations of static and dynamic, maximal and submaximal fatiguing contractions to characterize fatigue development with these various test conditions. Such studies might include monitoring EMG activity in the synergistic and antagonistic muscles to examine cyclic activity. Moreover, such studies might include conducting H-Reflex experiments to describe how nerve conduction velocity changes with the physical task.

6.5.2. Examination of Fatigue at Alternative Ankle Positions

In this study we investigated the effect of fatigue on ankle stiffness dynamics when the ankle was in neutral position. This position provided optimal stretch reflex response [77], and the most selective triceps surae activation [115].

Future extensions of this study might investigate various dorsiflexion positions, at which reflex gain increases [77]. However, the drawback to this is that antagonist co-activation increases with dorsiflexion, and might influence muscle fatigue, reflex potentiation, and ankle stiffness.

6.5.3. Examination of Fatigue with Alternative Physical Tasks

In this study, both fatiguing, submaximal, isometric and intermittent perturbation contractions were applied to the triceps surae. To isolate ankle dynamics properly, the following procedures were followed:

1. a fiberglass custom-fitted boot was constructed for each subject to restrict motion in the sagittal plane only,
2. subjects were asked to lie prone on the test table, and were instructed to remain still during the experiments,
3. straps were applied to isolate ankle motion from lower limb motion,

We can clearly see that although these procedures were successful in isolating ankle dynamics, they were not practical for everyday life. Results from this study might or might not be extended for alternative normal physical tasks like walking or running. Yet, they provide an initial guide to the methodologies used to investigate the effect of fatigue on muscle and dynamic joint stiffness.

Central - spinal and supra-spinal - and peripheral control mechanisms are complex. Modulation strategies of these regulatory mechanisms with normal physical tasks are not fully understood. Therefore, in this study, we did not examine the effect of fatigue on joint operation in normal conditions. When the fore-mentioned control strategies are understood in detail, this study may be extended to examine ankle dynamics under normal, fatiguing, physical tasks.

6.5.4. Investigation of Short- and Long-Term Effects of Physical Training on Fatigue and Ankle Joint Dynamics

It has been postulated that training enhances the efficiency of the neuromuscular system, increases the capacity of a muscle to generate force, and improves the ability to cope with fatigue resulting from submaximal loads. There is some evidence of improved fatigue resistance with maximal [116] and submaximal [27,117,118] contractions, which could be attributed to [119]:

1. conversion of type II muscle fibers from fast-fatigable to fast and fatigue-resistant,
2. increased synchronization of motor unit impulses,
3. prolongation of membrane excitation,
4. decreased antagonist and inhibitory afferent activity,
5. increased central drive,
6. alterations in the contractions of agonistic and synergistic muscles.

On the other hand, fatigue might enhance performance during maximal voluntary contractions (MVC), but not during submaximal contractions [119]. Moreover, the changes in muscle and reflex activities are task-dependent [120], and may vary differently with strength and endurance exercises [119]. Hence, to resolve these controversies, further studies are needed in this field.

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