Analysis of Physiological Tremor Mechanisms in Surface Electromyogram

A thesis submitted in fulfilment of the requirement for the degree of Master of Engineering by Research

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Declaration

I certify that except where due acknowledgement has been made, the work is that of the author alone; the work has not been submitted previously, in whole or in part, to qualify for any other academic award; the content of the thesis is the result of work which has been carried out since the official commencement date of the approved research program; any editorial work, paid or unpaid, carried out by a third party is acknowledged; and, ethics procedures and guidelines have been followed.

Audrey Lee Yen Tan
(27th March 2013)
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Abstract

Rhythmical grouping of action potential in the low frequency band have been observed during steady, constant force contractions. The origins of this rhythmical pattern have always been associated to tremor oscillations. However, literature concerning physiological tremor has shown that the origin of tremor remains inconclusive with various works providing valid arguments and counter-arguments for the various mechanisms proposed. Physiological tremor oscillation has consistently been associated with the following four mechanisms: a) synchronisation of motor units; b) mechanical feedback resonance; c) feedback resonance from neural delay and d) central oscillations. It is largely believed that the overall tremor oscillation is either the interaction effect of various oscillatory mechanisms acting in parallel with each other or one or other mechanisms acting predominantly in various situations. The objective of this research was to determine the role of neural feedback and muscle mechanics in the generation of physiological tremor oscillation.

This research developed a neuromuscular model to simulate the individual and resulting oscillatory mechanisms of tremor. The oscillations found in EMG signal were also analysed empirically from EMG data from 54 subjects using Hilbert Transform and rectification to obtain the envelope of the signal.

The time between rhythmical waveform patterns in the EMG signal, $T_C$ was found to be 78ms. This value corresponded to the supraspinal neural delay, 50-80ms reported in literature and the theoretical value of the supraspinal feedback simulated from the model. The theoretical model also reported a predominant mechanical resonance frequency in the
resulting oscillation between muscle and spinal feedback and the resulting oscillation between muscle, spinal and supraspinal feedback.

These observations coincide with the physiological tremor oscillations reported in literature: the mechanical-reflex and the 8-12Hz. The mechanical-reflex oscillation is defined to be the product of spinal feedback and muscle mechanics and is found to be highly influenced by the mechanical properties of muscle. The 8-12Hz component is reported to originate from the interaction of supraspinal and central mechanisms. $T_c$ has shown to coincide with the supraspinal neural delay time and the theoretical supraspinal value suggesting that $T_c$ correlates to the 8-12Hz component.
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Nomenclature

100MVC  100% of maximum voluntary contraction
25MVC  25% of maximum voluntary contraction
50MVC  50% of maximum voluntary contraction
75MVC  75% of maximum voluntary contraction
A  Spinal afferent gain
a  Thermal constant of cross sectional area of muscle
a(s)  Neural signal to active CE in s-domain
A(t)  Instantaneous amplitude of signal
a(t)  Neural signal to active CE in time domain
ac  Neural signal of central command
acCNS  Central nervous system neural signal
apNS  Peripheral neuromuscular system neural signal
As  Supraspinal afferent gain
b  Shape parameter of Hill’s hyperbole
B/Bh  Viscous element damping coefficient for force-velocity
Bi  Damping element of intrafusal fibre
Cm  Motoneuron membrane attenuation factor
CNS  Central nervous system
E  Spinal alpha efferent gain
EMG  Electromyography
Es  Supraspinal alpha efferent gain
f  Frequency of oscillation of system in Hz
Fb  Damping force
FCE  Contractile element force
<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$f_{\text{CNS}}$</td>
<td>CNS input</td>
</tr>
<tr>
<td>$F_{\text{IN}}/F_{\text{act}}$</td>
<td>Active contractile element force</td>
</tr>
<tr>
<td>$F_L$</td>
<td>External loading force</td>
</tr>
<tr>
<td>$F_m$</td>
<td>Muscular force</td>
</tr>
<tr>
<td>$F_{o_m}$</td>
<td>Maximum muscular force</td>
</tr>
<tr>
<td>$F_{\gamma/\gamma}$</td>
<td>Gamma innervation</td>
</tr>
<tr>
<td>$G$</td>
<td>Spinal gamma efferent gain</td>
</tr>
<tr>
<td>$H$</td>
<td>Shortening heat</td>
</tr>
<tr>
<td>$I_{\text{leak}}$</td>
<td>Leaky current of motoneuron membrane</td>
</tr>
<tr>
<td>$I_s$</td>
<td>Synaptic current of motoneuron membrane</td>
</tr>
<tr>
<td>$k$</td>
<td>Series elasticity of SE component</td>
</tr>
<tr>
<td>$K_a$</td>
<td>Membrane attenuation factor of $a_c$</td>
</tr>
<tr>
<td>$K_{\text{AG}}$</td>
<td>Overall alpha gamma gain</td>
</tr>
<tr>
<td>$K_b$</td>
<td>Membrane attenuation factor of spinal input</td>
</tr>
<tr>
<td>$K_{b_{\text{b}}}$</td>
<td>Series elasticity of reticulate zone</td>
</tr>
<tr>
<td>$K_c$</td>
<td>Membrane attenuation factor of supraspinal input</td>
</tr>
<tr>
<td>$k_e$</td>
<td>Effective series elastic constant</td>
</tr>
<tr>
<td>$K_i$</td>
<td>Attenuation faction of $i$th synaptic event</td>
</tr>
<tr>
<td>$K_{\text{SR}}$</td>
<td>Overall spinal gain</td>
</tr>
<tr>
<td>$K_{SS}$</td>
<td>Overall supraspinal gain</td>
</tr>
<tr>
<td>$K_{SSR}$</td>
<td>Overall supraspinal and alpha efferent gain</td>
</tr>
<tr>
<td>$r_1$</td>
<td>Closest root to origin</td>
</tr>
<tr>
<td>$r_2$</td>
<td>Next closest root to origin</td>
</tr>
<tr>
<td>std</td>
<td>Standard deviation</td>
</tr>
<tr>
<td>$T$</td>
<td>Ratio of damping element to series elasticity</td>
</tr>
<tr>
<td>$T_C$</td>
<td>Time constant between waveforms in EMG signal</td>
</tr>
</tbody>
</table>
\( v_b \) Rate of change of damper displacement

\( v_c \) Shape parameter of Hill’s hyperbole

\( V_{E,i} \) Excitatory input pulse of the \( i \)th synaptic

\( V_{I,i} \) Inhibitory input pulse of the \( i \)th synaptic

\( V_m \) Motoneuron membrane potential

\( v_m \) Rate of muscle length change

\( v_{\text{max}} \) Maximum rate of muscle length change

\( V_{\text{reset}} \) Motoneuron membrane reset potential

\( x \) Muscle length

\( x_{\text{is}}/X_b \) Damper element displacement

\( x_d/X_d \) Dense zone length

\( x_{d1}/X_{d1} \) Dense zone 1 length

\( x_{d2}/X_{d2} \) Dense zone 2 length

\( x_e/X_e \) SE component displacement

\( x_i/X_i \) Intrafusal length

\( x_m/X_m \) Muscle length

\( x_r/X_r \) Reticulate zone length

\( X_{ri} \) Transduced reticulate length

\( x_s/X_s \) Spindle length

\( \beta \) Skeletomotor efferent fibre

\( \gamma_d \) Dynamic gamma efferent fibre

\( \gamma_s \) Static gamma efferent fibre

\( \delta(t-t_i) \) Synaptic input train

\( \delta(t-t_{\text{out}}) \) Synaptic output train

\( \Delta V_i \) Change in potential due to \( i \)th synaptic event

\( \zeta \) Damping ratio of system
<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\eta$</td>
<td>Ratio of intrafusal damper to effective series elasticity</td>
</tr>
<tr>
<td>$\lambda$</td>
<td>Ratio of spindle length to muscle length</td>
</tr>
<tr>
<td>$\lambda_i$</td>
<td>Eigenvalue of system</td>
</tr>
<tr>
<td>$\tau_A$</td>
<td>Spinal afferent feedback delay</td>
</tr>
<tr>
<td>$\tau_{AG}$</td>
<td>Alpha gamma feedback delay</td>
</tr>
<tr>
<td>$\tau_{AS}$</td>
<td>Supraspinal afferent feedback delay</td>
</tr>
<tr>
<td>$\tau_E$</td>
<td>Spinal alpha efferent feedback delay</td>
</tr>
<tr>
<td>$\tau_{ES}$</td>
<td>Supraspinal alpha efferent feedback delay</td>
</tr>
<tr>
<td>$\tau_G$</td>
<td>Spinal gamma efferent feedback delay</td>
</tr>
<tr>
<td>$\tau_m$</td>
<td>Motoneuron membrane time constant</td>
</tr>
<tr>
<td>$\tau_{SR}$</td>
<td>Spinal feedback delay</td>
</tr>
<tr>
<td>$\tau_{SS}$</td>
<td>Supraspinal feedback delay</td>
</tr>
<tr>
<td>$\tau_{SSR}$</td>
<td>Supraspinal and alpha efferent feedback delay</td>
</tr>
<tr>
<td>$\phi(t)$</td>
<td>Cos and sine representation of signal</td>
</tr>
<tr>
<td>$\omega_n$</td>
<td>Natural frequency of system in rad/s</td>
</tr>
</tbody>
</table>
1. Introduction

1.1 Introduction

Electromyography (EMG) is the measure of the electrical currents generated in muscles during contraction. The shape and firing rates of active motor units found in EMG signals provide important information about muscle and neuromuscular activity which is used to aid in the diagnosis of neuromuscular disorders or understanding the neuromuscular system of the healthy, pathological, ageing or fatiguing subjects [1]. Extensive effort has been made to develop and improve algorithms and existing methodologies in signal processing, to improve detection and acquisition techniques for EMG and to develop pattern recognition technique [2]. It is important to understand the composition of the EMG signal in order to obtain pertinent information from it.

The EMG signal is composed of the superposition of the activity of individual motor units. Electrical potential is generated in the muscle fibre when an action potential is propagated along its excitable membrane by an alpha-motoneuron. Generally, an alpha-motoneuron will
innervate a group of muscle fibres. This group of muscle fibres along with its axon and alpha-motoneuron is termed a motor unit (MU) and the summation of its action potentials is known as the motor unit action potential (MUAP). During force activation, MUs fire continuously to maintain or increase the force generated, creating a motor unit action potential train (MUAPT). The summation of the collection of MUAPs from each motor unit in a muscle body forms the composite EMG signal detected by electromyography [1, 3-5]. To illustrate, Figure 1 below from Basmajian and de Luca [6] gives a schematic representation of the generation of EMG signal.

Figure 1 Composite EMG signal generated from the summation of MUAPT resulting from the direct delta impulse train fired by the alpha-motoneuron (Source: Basmajian and de Luca [6])
The MUAPT is descended from the impulse trains fired by the alpha-motoneuron which in turn is controlled by the nervous system. Information regarding the neural drive can be quantified from the discharge rate of individual MUs in the EMG signal which is used to shed light on the neuromuscular system and neuromuscular disorders. Consequently, EMG signal decomposition has received considerable attention and interest over the last few years [7]. However EMG decomposition techniques commonly believe MU firings to be primarily neural output from spinal cord in order to simplify the complex nature of the pattern of neural drive modulation seen in EMG [8, 9]. Though the influence of neuromuscular activity and physiological properties of the muscle in the EMG signal has been acknowledged [4, 6, 10], it is not generally considered.

When a set of MUs fires continuously at a similar rate, there exists a tendency towards a rhythmical grouping of action potential. During steady, constant force contractions, this rhythmical pattern has been observed in force waveform which shows significant components in the low frequency band [11, 12]. This low frequency component is often detected as a clear local peak during the power spectrum analysis of EMG signal and is termed the peak frequency. It is commonly known to appear at frequencies of 8-12Hz but higher frequencies of 20Hz or 40Hz have been noted before. In subjects with pathological tremor, a lower frequency 3-5Hz is often attributed to Parkinson’s tremor [11, 13].

Initially, this synchronisation of MUs was believed to be caused by self-oscillation in the spinal stretch reflex due to the delays around the loop [14, 15] but in later works, this synchronisation was proposed to be a result of random coincidences of the MUAP. The latter hypothesis proposes that during force generation, the successive groups of twitches and
MUAPs fuse and because of the relatively long duration of individual twitches this forms a continuous and fairly regular oscillation. However the contribution of the spinal stretch reflex to tremor was not entirely discounted and may exist in certain situations [16]. In recent years, the existence of this rhythmical component is widely acknowledge and generally attributed to the fusing of successive twitches. Consequently, EMG signal is mainly related to the neural output from the spinal cord and thus the oscillation present is associated with the discharge rate, recruitment range and synchronisation in the excitatory drive. In actuality, the cause of this oscillatory element still remains inconclusive in the field of tremor research.

The EMG signal oscillation (henceforth termed tremor) is considered to originate from a either an interaction between neuronal and mechanical oscillations or a prominent oscillation resulting from one of the two [17]. Though there is no clear consensus for the origin of the tremor, the association of the neuromuscular system in EMG signal is indisputable [18]. In fact, this potential origin has been alluded since the first recordings of periodicity in early myograms.

The understanding of peripheral neuromuscular tremor in EMG signal may prove to be useful in EMG signal analysis. A large part of EMG signal analysis is dedicated on extracting information on the firing rates of MUAP which are believed to have important information regarding neuromuscular disorder or contribute to the understanding of neuromuscular system for the healthy, pathological, ageing or fatiguing [1]. The association of peripheral neuromuscular system in EMG signal will bring a new insight to relation of EMG signals and neuromuscular disorders.
In healthy subjects, tremor is known as physiological tremor and has a complex signal resulting from interactions between several mechanical and neural factors [19]. Physiological tremor is believed to consist of two distinct oscillations, mechanical-reflex and 8- to 12- Hz oscillations [17, 20] wherein the mechanical-reflex refers to the neuromuscular system consisting of the mechanical muscle properties (mechanical) and spinal stretch reflex feedback components respectively.

The oscillations due to the spinal stretch reflex are believed to be a result of the delays in the spinal feedback loop. These loops operate over time which suggests the frequency of the spinal feedback oscillation to be inversely proportional to the loop delay [4]. On the other hand, the mechanical oscillations are believed to originate from the mechanical properties of the muscle commonly modelled in tremor studies as a mass-spring system [2, 4].

1.2 Aim and objectives

The aim of this research is to investigate the contribution of the peripheral feedback loop and the muscle mechanical properties in the oscillation present in the EMG signal of healthy subjects. The study will consider steady, constant force contractions where group rhythmical oscillations are known to be present in its EMG signal. As these oscillation frequencies can be determined through modelling the peripheral feedback loop and its components, this research will attempt to develop a theoretical model of the peripheral neuromuscular system (PNS). The results obtained from this model will be compared to the experimental values analysed from a set of experimental data available.
In order to achieve this aim, the following objectives must be met:

- Conduct a literature review on physiological tremor
- Conduct a literature review on the components found in the neuromuscular system
- Develop a neuromuscular system from the components reviewed and simulate the model.
- Obtain theoretical values of oscillations of the mechanical and peripheral feedback mechanisms
- Obtain experimental values of oscillations found in EMG signals of healthy subjects
- Compare and analyse the theoretical and experimental values

### 1.3 Research Questions

To achieve the objectives above, this research will address the following questions:

a) What is physiological tremor and what is the work done in this field?

b) What are the components involved in the neuromuscular system and how are these components modelled mechanically?

c) What is the frequency and amplitude of the peripheral stretch reflex and myogenic oscillation from the neuromuscular mathematical model and the experimental results?

d) How to distinguish the oscillations in the EMG signal of a steady, constant contraction (isometric contraction)?

e) How do the results verify the role of the peripheral feedback oscillations in the EMG signal?
f) What conclusion can be drawn from this and subsequently what are the future works and that can be proposed for this?

1.4 Structure of Thesis

This thesis contains seven chapters. The outline of each chapter can be seen below:

1. Chapter 1: An introduction into the current research and a declaration of the aims, objectives and research questions of the research

2. Chapter 2: Literature review providing a brief history of physiological tremor and its various debates followed by a brief review on the literature concerning neuromuscular system modelling in the study of tremor

3. Chapter 3: Literature review on the components involved in the neuromuscular system: the muscle, muscle spindle and motor neuron. The mathematical expression of each component is derived.

4. Chapter 4: Development of the theoretical peripheral neuromuscular model and its simulated oscillations. The peripheral feedback components, the spinal feedback and the supraspinal feedback was briefly discussed

5. Chapter 5: An explanation on the experimental methodology, signal analysis performed on the EMG signal obtained and the statistical analysis of the results

6. Chapter 6: Comparison between the experimental and theoretical values followed by a discussion on the role of peripheral feedback and muscle mechanics in physiological tremor based on the results obtained

7. Chapter 7: Conclusion of work
2. Literature Review on Physiological Tremor

As mentioned in Chapter 1, rhythmic grouping of the action potential results in oscillation in the EMG signal (known as physiological or pathological tremor). Physiological tremor exists in every person and can be seen in the low amplitude and frequency of around 10Hz in the EMG signal [13]. Physiological tremor can occur during isometric contraction (isometric tremor) or during isotonic load condition produced by gravity (postural tremor) [21]. Pathological tremor on the other hand, represents a higher amplitude and boarder frequency range and is often associated with Parkinson’s tremor (PD), cerebellar dysfunction, essential tremor (ET) and other pathologies. Loscher and Gallasch [21] noted that peripheral feedback is an active component in the generation of physiological tremor particularly for postural tremor. In order to understand the role of neuromuscular system in the physiological tremor, this chapter will provide a literature review of the work done in the field of physiological tremor.
The chapter will be divided into three main sections:

- Introduction to physiological tremor
- Mechanisms of physiological tremor oscillation
- Literature review on neuromuscular modelling in studying physiological tremor

2.1 Introduction to Physiological Tremor

The earliest observation of physiological tremor was by Schafer who noted a natural periodicity in the recordings of his myograms but discarded these findings as artefacts. Later, Schafer together with Horsley [22] found a 10 c/s rhythm in the ongoing muscle activity of the monkey following stimulation of the motor cortex which descended motor pathways at various frequencies. These authors concluded that the rhythmicity was determined at a spinal rather than at a cortical level.

This hypothesis was challenged years later when electroencephalogram (EEG) became available. Marshall and Walsh [23] recorded the EEG of two subjects with prominent alpha waves and found that the tremor movements occurred at all phases of the alpha waves. Based on their observations, they hypothesised that muscle acts as a low pass filter which converts rate discharges below 15 c/s into mechanical ripples but blocks or filters discharges at higher rates.

In 1956, the origins of physiological tremor was further expanded by Halliday and Redfearn [19]. They suggested that 9 c/s rhythm may result from any of the four following: a) from the
motor cortex or brain-stem reticular formation; b) the rhythmical property of motoneurones themselves or of the spinal system of neurons and interneurons; c) rhythmical propensity of the stretch reflex resulting from the delay between the stretch stimulus and the development of the resulting corrective force or d) purely mechanical resonant properties of the moving parts of the body. From their results, Halliday and Redfearn concluded that the 5 and 15 c/s oscillations were either an inherent spinal rhythmicity or due to the properties of the stretch reflex.

In 1970, Lippold [24] devised three experimental methods to distinguish the presence of the stretch reflex in physiological tremor. First, he introduced a mechanical perturbation and found the frequency of the waves to be the same as that of the tremor oscillation. Second, he studied the effects of temperature by warming and cooling the arm which he found raised and lowered the frequency of tremor oscillation respectively. Finally he attempted to induce the effects of deafferentation by applying an arterial cuff to the arm which led to the depression of all frequencies of normal physiological tremor oscillation. Based on these observations, he concluded that the 8-12 c/s component was due to the spinal stretch reflex loop. The results of Lippold’s paper subsequently caused the spinal stretch reflex hypothesis to gain favour.

In a later paper [25], Lippold further found there was an increase in overall tremor amplitude when the subject performs the “Jendrassik’s manoeuvre” which is known to enhance reflex action. Lippold countered the theory of asynchronous motor unit firing rate with the following: a) fatigue which lowers the frequency threshold at which individual twitches merge should lower the tremor frequency and b) the results of his experiments contradict this theory [25].
Lippold’s hypothesis remained inconclusive as following literature argued against his findings. Allum et al [26] who supported Marshall and Walsh’s hypothesis that physiological tremor is a result of the asynchronous discharge of motor nerve fibres pointed out that a) the tremor rates do not depend on the length of the spinal reflex feedback arc as tremor frequency is similar for different muscles located at different distances from the spinal cord; b) tremor is still present after deafferentation and c) the broad range of physiological tremor frequency cannot be accounted by the spinal stretch reflex hypothesis. However these arguments too have also been countered by works from subsequent literature.

2.2 Mechanisms of Tremor Oscillation

While the origins of physiological tremor remain a highly contentious area, the origins of physiological tremor are commonly referred to as one or a combination of the following:

1) Synchronised MU oscillations [26]
   - The frequency of the oscillation may correspond to the range of firing rates occurring from a combination of recruitment and fusion of twitch contractions of MUs.
   - The frequency of oscillation may be due to a common driving oscillation from the motoneuron

2) Mechanical resonance [27]
   - This oscillation arises from the mechanical properties of the joint-muscle tendon system. These parts form a complex skeletomusculotendon often
compared to a mass-spring system with the following frequency,
\[ \omega = \sqrt{\frac{k_e}{M}} \] where \( k_e \) is the spring constant and \( M \) is the mass of the system.

3) Peripheral feedback resonance [27]

* The peripheral neuromuscular feedback behaves like a servo mechanism and may oscillate at a particular frequency related to the delay around the loop. In the peripheral feedback, the stretch response may occur from:
  i. *Spinal stretch reflex* where the signal travels from the muscle to the spinal cord and back to the muscle again
  ii. *Supraspinal stretch reflex* where the signal travels from the muscle to the supraspinal segments (centres such as the cerebral cortex and thalamus) and back

4) Central oscillations [28]

* The neural activities recorded by early EEG were found to be rhythmic. It is hypothesised that EMG oscillations originate from oscillatory parts of the central nervous system (CNS) like the olive or cerebral cortex [29]

The four mechanisms of tremor have been studied with various experimental methods. Each method has produced noteworthy results which justify the existence and validity of their proposed oscillatory mechanism. To give a brief overview, the work for each mechanism has been summarised in Table 1 below.
## Table 1 Literature review on physiological tremor grouped according to proposed origin

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Proposed Origin</th>
<th>Methodology</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Motor Unit Synchronisation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marshall and Walsh [23]</td>
<td>1956</td>
<td>Fusion of twitches at higher rate</td>
<td>Electroencephalogram (EEG)</td>
<td>10c/s</td>
</tr>
<tr>
<td>Christakos [16]</td>
<td>1982</td>
<td>Asynchronous activity of the motor properties</td>
<td>Population model</td>
<td>-</td>
</tr>
<tr>
<td>Kilner et al [30]</td>
<td>2002</td>
<td>Oscillatory synchrony between motor units (between populations of premotoneuronal inputs including corticospinal neurons)</td>
<td>Detecting and discriminating different single motor unit (SMU)</td>
<td>6-12Hz; 15-30Hz</td>
</tr>
<tr>
<td><strong>Central (neurogenic) oscillations</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Salenius et al [31]</td>
<td>1997</td>
<td>Motor cortex driving the spinal motoneuronal pool</td>
<td>Magnetoencephalography (MEG)</td>
<td>15-33Hz</td>
</tr>
<tr>
<td>McAuley et al [32]</td>
<td>1997</td>
<td>Central origin</td>
<td>Mechanical and electrical perturbations; effect of limb anaesthesia; effect of loading of finger; effect of varying strength contraction</td>
<td>10, 20 and 40 Hz</td>
</tr>
<tr>
<td>Marsden et al [33]</td>
<td>1967</td>
<td>Presence of 9.5c/s tremor in deafferentated subject indicates that tremor frequency is not solely the result of stretch reflex delay</td>
<td>Deafferentated subject</td>
<td>9.5c/s</td>
</tr>
<tr>
<td><strong>Muscle load mechanics and mechanical-reflex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stein and Oguztoreli [34]</td>
<td>1975</td>
<td>Interactions between muscle and load; stretch reflex oscillations</td>
<td>Neuromuscular modelling</td>
<td>8-12Hz</td>
</tr>
<tr>
<td><strong>Stretch reflex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Halliday and Redfearn [19]</td>
<td>1956</td>
<td>Spinal cord or stretch reflex</td>
<td>Measuring natural frequency of finger and the effects of adding weight</td>
<td>5-15c/s</td>
</tr>
<tr>
<td>Lippold [33]</td>
<td>1970</td>
<td>Stretch reflex</td>
<td>Mechanical perturbation, effects of temperate and effects of ischemia</td>
<td>8-12c/s</td>
</tr>
<tr>
<td>Hagbarth and Young [35]</td>
<td>1979</td>
<td>Stretch reflex</td>
<td>Comparison of spindle afferents readings to EMG in the light of fatigue, mechanical and electrical perturbations and passive length changes to contracting muscle</td>
<td>-</td>
</tr>
<tr>
<td>Lippold [25]</td>
<td>1971</td>
<td>&quot;Hunting&quot; mechanism in the reflex arc</td>
<td>Inflated cuff to interrupt the efferent signal; Jendrassik manoeuvre; effect of temperature; mechanical perturbations</td>
<td>8-12c/s</td>
</tr>
<tr>
<td>Hagbarth and Young [36]</td>
<td>1980</td>
<td>Segmental stretch reflex</td>
<td>Jendrassik manoeuvre; vibration enchanched tremor</td>
<td>Enhanced amplitude up to 1/2 or 3/4 maximum</td>
</tr>
<tr>
<td>Takanokura and Sakamoto [37]</td>
<td>2001</td>
<td>Stretch reflex of supraspinal (8-12Hz) and spinal (higher frequency) origins</td>
<td>Neuromuscular modelling; increasing weight of component by studying finger, hand, forearm and arm</td>
<td>8-12Hz</td>
</tr>
<tr>
<td>Horsley and Schäfer [22]</td>
<td>1886</td>
<td>Lower nerve centre (ie the motor-nerve cells in the spinal cord, medulla oblongata, pons and mesencephalon)</td>
<td>Electrical excitation to nerve centres</td>
<td>10c/s</td>
</tr>
</tbody>
</table>

As shown in Table 1 above the existence of each origin has been noted in separate accounts by various authors using different experimental techniques. Arguments for and against each
mechanism have been presented by all sides equally leaving the true origins of tremor to be inconclusive. Consequently, these results led many to believe that physiological tremor is a product of peripheral and central mechanism which could lead to either a single common oscillation or a tremor with several independent frequency components [27, 32, 38]. The complex interactions between these generators have been diagrammatically represented by McAuley [28] and can be seen in the Figure 2 below:

![Figure 2 Oscillatory mechanism generating physiological tremor and its interactions (Source: McAuley [28])](image)

The prevalent opinion favoured identifies MU properties as the main source of physiological tremor but many works (particularly those delving in physiological tremor) do not exclude the spinal stretch reflex system. In fact, several researches have hypothesised (through mechanical perturbations, inertial loading and spring loading experiments [15, 20]) that
central oscillatory frequency and mechanical and spinal stretch reflex system are inextricable coupled rendering it quite difficult to identify the contribution of each resultant tremor [17].

2.2.1 Literature Review on Neuromuscular Modelling of Tremor Oscillations

Recently, neuromuscular modelling has begun gaining favour in an attempt to extricate the various oscillatory origins. The earliest attempt in this field was from Stein and Oguztoreli [39] who developed a mechanical model of the neuromuscular system based on Bawa et al’s [40] visco-elastic muscle model. They attempted to consider, from a theoretical viewpoint, the conditions under which reflexes can interact with skeletal muscle to give rise to oscillations. Later, the original model was further expanded to include and study the following:

a) the effects of central and peripheral oscillations and their interactions [41]

b) the effects of multiple pathways of the stretch reflex (spinal and supraspinal) [42]

c) the effects of neuromuscular resetting and entrainment [43]. Both are methods commonly employed to distinguish stretch reflex oscillations from central oscillations.

However the aforementioned models never attempted to compare their theoretical results with any experimental results. Instead it was used to model tremor oscillation arising from the neuromuscular system during muscle contraction. Nevertheless, Stein and Oguztoreli’s work became a popular basis for future mathematical modelling of peripheral feedback oscillations.
Though Stein and Oguztoreli’s work had not attempted to compare experimental to theoretical results, several proceeding works have adopted their model and expanded on this area. One such work is Takanokura and Sakamoto’s [37] study of physiological tremor in upper limbs. For their work, these authors adopted Stein and Oguztoreli’s multiple reflex pathways model [42] to take into account spinal and supraspinal reflex loops. Their results showed two peak frequencies whose theoretical values and experimental values corresponded closely. In order to distinguish the origins of the two frequencies, they varied the weight of the theoretical model and did experimental testing on four different limb segments (finger, hand, forearm, upper limb) respectively. The component of supraspinal reflex which exists in the range of 8-12Hz is found to be unaffected by the change in mass of limb segment but the peak frequency of the spinal reflex varies with the mass of limb segments. From this, they concluded that the spinal component represents the mechanical system of the limb segment and the supraspinal component largely involves the upper centres such as the cerebral cortex and the thalamus. They suggested 8-12Hz frequency of the supraspinal system is predominant and it indirectly entrains the oscillation of the spinal system irrespective of the mass of the limb. This hypothesis is in accordance with previous works that have suggested that there exists a coupling of mechanical and central oscillations. In addition, their results verifies Allum’s [11] previous argument which states that frequency values do not vary irrespective of limb segments or distance from spinal cord.

From Takanokura and Sakamoto’s [33] results, it can be seen there exists a mechanical-reflex oscillation that is dependent on the peripheral neural delay of the spinal loop and the mechanical properties of the muscle. However the effects of the mechanical muscle properties and the spinal stretch reflex delay have not been distinguished but have been modelled as an entity. It is not clear if this change in frequency observed in the theoretical
and experimental model is representative of the effects of either muscle-loading or stretch reflex.

Consequently, this paper is largely concerned with the role of peripheral neuromuscular feedback and the muscle mechanical properties in generating the oscillations in EMG signal. The research has developed and adapted Stein and Oguzoreli and Takanokura and Sakamoto’s model [37, 39]. In accordance to both works the mathematical neuromuscular system was modelled as a feedback system where the change in length occurring in a muscle during contraction is relayed to the spinal cord and supraspinal centres through corresponding muscle spindle receptors and neural feedback loop. For an experimental comparison, a large number of experimental EMG signal from subjects ranging from ages 20 to 40 were gathered and the EMG signal oscillations were obtained. Subjects were told to maintain an isometric contraction to ensure the mechanical effects of muscle properties are excluded in the EMG signal.

The following chapter largely concerns the theoretical neuromuscular model. It will look into the anatomical and mechanical properties of each component involved in the neuromuscular system.
The term neuromuscular composes of two components: the “neuro” which indicates the peripheral feedback system comprising of the lower and higher reflex loop and “muscular” which represents the muscle which will be described in the next section [13]. The lower level reflex loops consists of a MU (a spinal motoneuron, its associated muscle fibres and connecting axons) and proprioceptive receptors and is thus known as the spinal reflex loop. In this lower sensory loop, the action potential activity from the motoneuron activates the muscular contraction resulting in changes in muscle length. In response, muscle spindles send the afferent signal of the detected force and changes to muscle length to the motoneuron, completing the lower sensory loop (ref Figure 3). This afferent signal also ascends the tract to the cerebellum where the sensory information is modulated by the supraspinal CNS regions and thus forming the higher reflex loop [44]. In neuromuscular modelling, this higher reflexive loop is commonly termed as the supraspinal reflex [39].
This chapter will focus on the components involved in the lower sensory loop which consists of the following:

- Muscle
- Muscle spindle
- Motoneuron

The respective mechanical and anatomical properties of each component is presented and reviewed to obtain the mathematical expression of each component.
3.1 Muscle model

The early foundations behind muscle modelling were laid down in works such as Hill [45] and Fenn which utilises a concept termed “systems engineering” wherein the system is modelled as a “black box” with fairly well defined input and output [46]. Using systems engineering, they were able to build models that could fairly predict the output given a certain input and consequently this enabled mechanical representations of the system to be built.

The early experiments and observations viewed muscle as a spring like structure in a linearly viscous medium [47]. They defined the output of muscle contraction as a summation of active and passive forces. Active muscle contraction occurs when thin actin filaments and thick myosin filaments slide past one another in a rowing motion due to crossbridges that extend from the myosin filament to the actin filament [48]. Passive muscle contraction occurs due to the inherent elastic properties of the muscle during lengthening or shortening. Together, the active and passive forces give the total force generated by muscle contraction.

The earlier works in modelling muscular contraction [49] considered a purely tension-length relationship between muscle and force of contraction by hypothesising that the active muscle acts as a stretched spring. This was later proven inaccurate when Blix and later Fick showed that at any length, tension developed by the muscle was greater than the corresponding after tension [45, 49]. However this problem was never considered in detail until Hill performed his experiment on the heat generated by the shortening of frog leg muscle [45].
In Hill’s experiment with frog leg muscle he noted that at higher velocities of muscle shortening, the total force generated is notably less. He attributed this to the “visco-elasticity” of the muscle and introduced a viscous element to compensate for this force-velocity relationship. He suggested that the passive properties of the contracting muscle are a two-component system; one being the known undamped elastic element which contributes to the length-tension and the other is a viscous element which contributes to the force-velocity dependent forces. He grouped this in the contractile element (CE) which contains the active force generator, a length-tension property and a force-velocity property.

Using the “black box” method, Hill looked at the total energy of the system and found, experimentally, that when a muscle was shortened a distance $x$, it gave off a “shortening heat”, $H$ (where $a$ is the thermal constant related to the cross sectional area of the muscle) [50]:

$$H = ax$$

(1.1)

He then described the total energy of the system as the sum of the heat shortening energy, $H$ and work, $F^m x$ which he differentiated to obtain the rate of energy liberation:

$$\text{total energy} = F^m x + H = (F^m + a)x$$

(2.1)

$$\text{rate of total energy liberation} = (F^m + a)\nu_m$$

(3.1)

He proposed that this rate is proportional to the change in force where muscle forces start at a maximal value of $F^m_0$:

$$\left(F^m + a\right)\nu_m = b\left(F^m_0 - F^m\right)$$

(4.1)
This hyperbolic Hill’s equation (ref equation 4.1) can be used to describe the force-velocity property of the CE component. The variable $a$ and $b$ are dimensionless “shape” parameters which define the hyperbolic concavity of the force-velocity [46]. This hyperbolic function is widely considered to be the most practical and accurate approach and is a classic standard for modelling the CE force-velocity of muscle contraction [51, 52]. Consequently this has led to the widely employed three-element muscle model.

The three basic elements of skeletal muscle (muscle fibre) model are: the active and controllable contractile element (CE), the passive series elastic element (SE) and the passive parallel elastic element (PE) [45, 53-56]. Simply stated, Hill considers the skeletal muscle model to consist of CE that is surrounded both in series and in parallel by “passive” connective tissue. CE is furthermore characterised by two fundamental relationships: CE tension-length and CE force-velocity which he describes using his hyperbolic function in Equation 4.1 and an active component. To illustrate, Figure 4 shows the classical structural arrangement of Hill’s muscle model:

- **Series elastic element (SE):** Models the muscle’s passive forces due to inherent muscle elasticity when lengthening or shortening.
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- **Contractile element (CE):** Models the active force generated by the “ratcheting” [57] mechanism of the actin and myosin filaments and the length-tension and force-velocity relation of active contraction.

- **Parallel elastic element (PE):** Models the muscle’s passive forces due to inherent muscle elasticity when lengthening or shortening.

### 3.1.1 Series and parallel elastic element (SE and PE)

The CE of the muscle is modelled to be in series and in parallel with the PE and SE components. The latter two elements represent the mechanical properties of passive soft connective tissue. This biological connective tissue can range from tendon to skin to blood vessel and have quasi-static mechanical properties which can be described mathematically by equation 5.1 below [46] (where $F$ is force, $x$ is extension and $K_1$ and $K_2$ are constants [often $K_2 \approx 0$]):

$$\frac{dF}{dx} = K_1 F + K_2$$  \hspace{1cm} (5.1)

In most works, the PE element is neglected or assumed linear as the force developed by this element is insignificant except in extreme lengths which are beyond the physiological range of skeletal muscle [46].

For the SE component, its mechanical properties have been generally attributed to tendon as it was believed that the length-tension extension of the CE is virtually insignificant relative to tendon extension [46, 58]. There are a variety of ways to model the SE element but it is typically modelled as a single linear spring element. Works concerned neuromuscular
modelling for tremor such as Stein, Bawa and Takanokura [37, 39, 40] utilises a spring element. Similarly, the current model will adopt a linear spring element representation of the SE element.

### 3.1.2 Contractile element

Hill’s hyperbolic function is a good fit for shortening muscle thanks to its intuitive parameters. This relation can be rewritten in many forms including a CE force equation, $F_{CE}$ described by Cook and Stark [46, 59] to be:

$$F_{CE} = F_{IN} - \frac{F_{IN} (1 + a)}{a v_{max} + v_c} v_c$$  \hspace{1cm} (6.1)

Cook and Stark modelled equation 6.1 and found that this describes the behaviour of a force generator in parallel with a dashpot (as seen in Figure 5) where the PE and SE elements are modelled as linear springs.

![Figure 5](image.png)

**Figure 5** Mechanical representation of the muscle model shown by Winters (originally Cook and Stark)

The viscous element, $B_h$ is defined as the following and the resulting contractile force, $F_{CE}$ is defined as:
Chapter 3: Components of the neuromuscular model

\[ B_h = \frac{F_{IN} (1 + a)}{av_{max} + v_e} \]
\[ F_{CE} = F_{IN} - B_h v_{CE} \]

The muscle model above coincides with Stein and Oguztoreli’s muscle mechanics model as can be seen in Figure 6 [39, 40]

![Figure 6](image)

**Figure 6** Bawa and Stein’s mechanical neuromuscular model of muscle and muscle spindle (Source: Bawa and Stein [40])

As mentioned previously, the PE element is considered negligible except in the case of extreme lengths [46]. During experimental procedures, the subject is instructed to pull a cord which leads to the shortening of the biceps brachii when it contracts. As such, the current model will neglect the PE element and consequently model the muscle as a CE in series with an SE as shown in Figure 7 below. Here the mass, \( M \) represents the mass of the muscle in contraction and the nomenclature \( F_{act} \) is used to denote the active force generated by CE (where \( F_{act} \) is equivalent to \( F_{IN} \) nomenclature used by Cook and Stark denoted in equation 7.2) and \( B \) as the viscous element.
When the muscle contracts, the contractile element generates an active force from the cyclical interaction between actin and myosin and a passive force from the inherent elastic and plastic properties of the muscle. This passive force is driven by the change in muscle length caused when the muscle shortens. As such, muscular force, $F_M$ is considered a function of displacement, $x$.

The muscular force, $F_M$ generated by the above model during contraction, can be obtained in three ways:

- **The parallel active-passive element**

Muscular force, $F_M$ is the sum of active generated force, $F_{act}$ and damping force, $B$ that is generated by the viscous element. The damping force, $F_b$ is generally expressed as:

$$F_b = -Bv_b$$

$$F_b = -B \frac{dx_b}{dt}$$

The variable $B$ generally denotes the viscous damping coefficient of the damper. In this system, $B$ represents the visco-elasticity of the muscle. The variable $v_b$ is the
velocity of the damped element and can be expressed in terms of \( x_b \) using \( \frac{dx_b}{dt} \) where 

\( x_b \) is the displacement of damper. Summing the damping force, \( F_b \) to the active generated force, \( F_{act} \) will give the muscular contraction, \( F_M \)

\[
F_M = F_{act} - B \frac{dx_b}{dt}
\]

(9.1)

- **The series elastic element**

Muscular force, \( F_M \) is an element of passive elastic force generated by the elastic properties of the muscle when lengthening and shortening. Here, the formula for the potential energy of a spring is used to define \( F_M \).

\[
F_M = k x_e
\]

(10.1)

The variable \( k \) denotes the elastic coefficient of the muscle and can be negative or positive depending on the direction of displacement (shortening or lengthening) and \( x_e \) denotes the displacement of the elastic element. The value of \( x_e \) can be found from the shortening of muscle, \( x_m \):

\[
x_m = x_e + x_b
\]

\[
F_M = k(x_m - x_b)
\]

(11.1-11.2)

- **Muscular force, \( F_M \)** is the resultant force from any existing external, \( F_L \) loading and the displacement of mass of biceps brachii muscle, \( M \)

\[
F_M = Ma - F_L
\]

\[
F_M = M \frac{d^2 x_m}{dt^2} - F_L
\]

(12.1-12.2)
To obtain $F_M$ as a function of $x_m$, Equations 10, 11 and 12 are Laplace transformed:

$$F_M(s) = k(X_m(s) - X_b(s))$$
$$F_M(s) = F_{ac}(s) - BsX_b(s)$$
$$F_M(s) = Ms^2X_m(s) + F_L(s)$$

(13.1-13.3)

Using linear equation, Equation 13.1 is substituted into Equation 13.2 to gain an expression of $F_M$ in terms of $X(s)$ giving the following:

$$X_b(s) = X_m(s) - \frac{F_M(s)}{k}$$
$$F_M(s) = F_{ac}(s) - Bs\left(X_m(s) - \frac{F_M(s)}{k}\right)$$
$$F_M(s) = \frac{F_{ac}(s) - BsX_m(s)}{1 - \frac{Bs}{k}}$$

(14.1-14.3)

Then the muscular force, $F_M$ in equation 14.3 can be equated to equation 13.3 to give the following expression of $X_m(s)$:

$$Ms^2X_m(s) + F_L(s) = \frac{F_{ac}(s) - BsX_m(s)}{1 - \frac{Bs}{k}}$$
$$X_m(s) = \frac{F_{ac}(s) + F_L(s)}{s\left(\frac{MB}{k}s^2 + Ms + B\right)}$$

(15.1-15.2)

The changes in muscle length, $X_m$ are detected by muscle spindles that lie in parallel to the extrafusal fibres. The transducer properties of these muscle spindles allow these stretch receptors to relay information of the change in muscle length back to the CNS in the form of impulses. The next section will cover the physiological and mechanical properties of muscle spindle in further detail.
3.2 Muscle Spindle and its Properties

The muscle spindles provide proprioceptive feedback and reflexive adjustments to movement and perturbations. They contain a number of intrafusal muscle fibres that are mechanically connected in parallel to the extrafusal muscle fibres at both polar ends which allows them to experience the same relative length changes as the overall muscle [60] like a sophisticated kind of strain gauge [61]. Typically a general muscle spindle consists of the following:

- Intrafusal fibres [60, 62, 63]
  - *Nuclear bag fibres* which are characterised by a central aggregation of nuclei, forming a bag-like structure
    - *Bag 1* is the only fibre with a dynamic fusimotor efferent endings located in it and is primarily responsible for velocity sensitivity of the spindle
    - *Bag 2* receives static fusimotor endings and contributes to length sensitivity
  - *Nuclear chain fibres* which are characterised by an aligned chain of nuclei fairly distributed over a length of the equatorial region
    - *Chain fibres* receive static fusimotor endings and contribute to length sensitivity
- Neural outputs
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- **Primary (group Ia afferent) endings** are largely sensitive to the rate of change in muscle length and to a lesser degree, change in muscle length. They have large axons and therefore relatively high conduction velocities [61]. They have branches on the nuclear bag and upon chain fibres.

- **Secondary (group II afferent) endings** have smaller axons and are largely sensitive to change in muscle length. They are mostly found on nuclear-chain fibres.

- However there exists some overlap in sensitivity between the two sensory endings [64].

- **Neural inputs** [65]
  - *Dynamic gamma efferent fibres, $\gamma_d$* shifts the sensitivity of the dynamic sensory ending to velocity changes
  - *Static gamma efferent fibres, $\gamma_s$* shifts the sensitivity of the static sensory ending to length changes
  - *Skeletofusimotor efferent fibres, $\beta$* innervates both intrafusal and extrafusal fibres

These components can be modelled diagrammatically to give the following muscle spindle control model [65]:

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The intrafusal fibre behaves like an advance strain gauge and is attached in parallel to the extrafusal fibre. When the muscle contracts the changes in the length of the muscle will reflect on the intrafusal fibre. Sensory elements of the afferent nerves wrapped around the equatorial region of the muscle spindle will detect the changes in length and relay this information to the CNS in the form of impulses [65]. As the spindle supplies the CNS with information, it also receives continuous motor innervation through $\gamma_s$ and $\gamma_d$ efferent. These gamma efferent modulate the respective static and dynamic sensitivity of the sensory ending according to the length and velocity changes occurring in various tasks [62, 63]. The schematic diagram in Figure 8 and the anatomical and mechanical properties identified above can be used to generate the muscle spindle model.
The sensory region, or better known, as the polar region consists of striated contractile muscles of dense myofilaments arrangements with little supporting connective tissues which leads to visco-elastic properties [66]. The nuclear bag region, at the spindle’s equator lacks the myofilaments and is instead reinforced by a dense extracellular network of connective tissue fibres leading to elastic properties [64]. This can be described by Figure 9 below:

Houk [64] terms the equatorial region and the polar region the “reticulate” zone and the dense zone respectively. The reticulate zone acts as the sensory region due to the afferent nerve endings wrapped around the nuclear bag region generating the corresponding neural output during muscle lengthening or shortening.

Figure 9 Anatomical view of the muscle spindle regions (Source: Houk [64])

Figure 10 Muscle spindle attached to the extrafusal muscle on its polar ends (Source: Houk [64])
A mechanical model of the muscle spindle is developed based on the Figure 10 and the discussed anatomical properties. The muscle spindle model used for this paper is adapted from McMahon and Mileusnic et al [61, 62]. While second order muscle spindle models have been explored [60], this research is interested in how the spindle is related to the length and proprioceptive changes of the extrafusal muscle and how it operates internally to provide a neuromuscular feedback control [62, 65] as such a simple linear first order system will be adequate. Figure 11 below shows McMahon’s lumped, linear model of the spindle [61].

As shown in Figure 10 the muscle spindle consists of two polar contractile zone which is called dense zone 1, \( x_{d1} \) and dense zone 2, \( x_{d2} \) and an equatorial elastic region called the reticulate zone, \( x_r \). The visco-elastic properties of the polar contractile zone are represented by a parallel elastic spring, \( K_i \) and a damper system, \( B_i \) with a parallel active-state force generator, \( F_\gamma \) whose force level is controlled by the gamma efferent input.

The total length of the dense zone can be represented by \( x_d \) as shown in Figure 11 above.

\[
x_d = x_{d1} + x_{d2}
\]  

(16.1)
As seen from Figure 11 the total spindle length and muscle length can be related to the following:

\[ x_s = x_a + x_r \]
\[ x_s = \lambda x_m \] (17.1-17.2)

The variable \( \lambda \) is the magnitude of the spindle length relative to the muscle length and is a constant less than unity. From Figure 11 the force generated by the mechanical model can be balanced by equation 18.1:

\[ F_y + B_i \frac{dx_d}{dt} + K_i x_d = K_d x_r \] (18.1)

In order to express \( x_r \) in terms of \( x_s \) equation 17.1 and 17.2 are Laplace transformed and substituted into the following:

\[ X_d = X_s - X_r \]
\[ F_y + B_i s X_d + K_i X_d = K_d X_r \]
\[ F_y + B_i s (X_s - X_r) + K_i (X_s - X_r) = K_d X_r \] (19.1-19.4)
\[ X_r = \frac{(B_i s + K_i)X_s + F_y}{(K_d + B_i s + K_i)} \]

Considering the effective series elasticity, \( K_e \)

\[ \frac{1}{K_e} = \frac{1}{K_i} + \frac{1}{K_d} \]
\[ \frac{K_d}{K_e} = \frac{K_d + K_i}{K_i} \] (20.1-20.2)

Let
\[
\eta = \frac{K_d}{K_i} = \frac{K_d + K_i}{K_i} \quad (21.1-21.2)
\]
\[
T = \frac{B_i}{K_i + K_d}
\]

Substitute equation 21.1 and equation 21.2 and the Laplace transform of equation 17.2 into equation 19.4 gives the following:

\[
X_r = \frac{\lambda(\eta Ts + 1)}{Ts + 1} X_m + \frac{1}{Ts + 1} F_r \quad (22.1)
\]

The linear, first order model of a muscle spindle is generally modelled as a lead-lag filter type [61, 63, 64, 67] whose transfer properties is described by the general transfer function:

\[
H(s) = K \left( \frac{\eta Ts + 1}{Ts + 1} \right) \quad (23.1)
\]

As shown above, the derived equation 22.1 reflects the general lead-lag transfer function for a linear, first order spindle model shown in equation 23.1.

The sensory region is believed to act as a transducer which converts the mechanical deformation of the reticulate zone into electrical impulses [60, 65]. The stretch of this region results in the distortion of the afferent endings wrapped around it. Once the afferent endings are stretched beyond its threshold length, the ion channels open and depolarisation of the membranes/impulse generation takes place at the nerve endings [62, 65]. The impulse rate is assumed to be directly proportional to the degree of extension [63, 66] where \( C_f \) is the proportionality constant of the impulse rate.
3.3 Motoneuron and its properties

The resulting afferent neural output is sent to the CNS where it terminates at the dorsal root ganglion of the peripheral nerve [61]. It forms direct synaptic connections with the alpha motoneurons of the muscle in which the spindle is embedded. When a pulse reaches the synapse (the specialised ending of axon), it releases a small quantity of chemical substance which diffuses on the post synaptic neuron surface. This alters the permeability of the postsynaptic membrane to ions affecting the current flow in or out of the postsynaptic neuron and therefore altering the potential difference across its membrane [68]. The input pulses (excitatory, $V_{E,Ne}$ or inhibitory, $V_{I,Ni}$) are summed linearly and the resulting output spike (action potential) is generated (Figure 12) when the membrane potential crosses its threshold reminiscence of an integrate-and-fire mechanism [69].

![Figure 12 Schematic representation of the intergrate-and-fire neuron mechanism (Source: Burkitt [71])](image)

This mechanism was first proposed by Lapicque [70] who postulated that the cell membrane acts as a capacitor which is charged until a certain threshold potential before the action potential is generated.
potential is released causing the membrane potential reset. Today, his model has been established to be a canonical model capable of mathematically describing the complexity of the neural processing [69].

Lapicque’s adapted leaky intebrate-and-fire model can be described as [69]:

\[
C_m \frac{dV_m}{dt} = I_{\text{leak}} + I_s
\]  

(24.1)

\( I_{\text{leak}} \) is the leaky current due to the passive leak of the membrane and \( I_s \) is the synaptic input current to the membrane [71]. These variables are defined to be:

\[
I_{\text{leak}} = -\frac{C_m}{\tau_m}(V_m - V_{\text{rest}})
\]

\[
I_s = C_m \sum_i \Delta V_i \delta(t - t_i)
\]  

(25.1-25.3)

\[
C_m \frac{dV_m}{dt} = -\frac{C_m}{\tau_m}(V_m - V_{\text{rest}}) + C_m \sum_i \Delta V_i \delta(t - t_i)
\]

where \( C_m \) is the membrane capacitance, \( \tau_m \) is the membrane time constant, \( V_m \) is the membrane potential and \( V_{\text{reset}} \) is the reset potential after \( V_m \) discharges, \( \delta(t-t_i) \) is the synaptic input train and \( \Delta V_i \) is the change in potential due to the \( i \)th synaptic event.

This paper is interested in the resulting impulse of the input thus the integrate-and-fire model is considered a perfect integrator and the decay of the membrane potential over time is neglected leaving equation 26.1:

\[
C_m \frac{dV_m}{dt} = C_m \sum_i \Delta V_i \delta(t - t_i)
\]  

(26.1)
From equation 26.1 above, the alpha motor neuron junction can be modelled into equation 27.1 where the variable $K_i$ where represents $C_m\Delta V_i$:

$$\delta(t - t_{out}) = K_i\delta(t - t_i) \quad (27.1)$$

### 3.4 Muscle Activation Dynamics

The resulting impulse train of action potential travels down the alpha efferent nerves to innervate the active contractile element in the form of neural signal, $a(t)$ forming a closed neuromuscular feedback loop.

During muscle activation, the action potential transmitted from the alpha-motoneuron depolarises along the muscle fibre membrane. This depolarisation is conveyed to the interior of the muscle fibre by means of transverse tubules called T-tubules causing the release of calcium from the sarcoplasmic reticulum (compartments in the muscle fibre in which all calcium of the muscle is sequestered) [72]. The released calcium into the sarcoplasm results in a sequence of chemical reactions that manifests itself as the active contraction found in CE.
When the calcium is released, it exposes the actin binding sites where myosin heads at an initial angle of 90° can attach (A+M$_{90}$→A·M$_{90}$). The attachment of actin and myosin molecules results in action myosin complexes being formed with the head of the myosin subtending an angle of 45° to the backbone (A·M$_{90}$→A·M$_{45}$). This change in angle of the myosin head is what provides the mechanical response (length-tension and force-velocity) of the contractile element. The detachment of the actin-myosin complex is achieved with the splitting of an adenosine triphosphate (ATP) molecule into adenosine diphosphate (ADP) and phosphate (P$_i$) (ATP→ADP+P$_i$). Once detached, the myosin head returns to its initial 90° angle. This cycle can be diagrammatically represented below [72]

![Figure 14 Active component of CE energy generation cycle (Source: Devasahayam [72])]}

In Hill’s model, the activation element is defined as the tension that the CE would generate without lengthening or shortening after the beginning of excitation and is considered to be virtually instantaneous [46]. In Buchanan’s [50, 58] muscle activation modelling, he expresses this signal as a number between 0 and 1 which represents the ratio of the active contractile force generated to the maximum active contractile possible. In EMG signal, this number is the product of the EMG signal that is rectified, smoothed and normalised. In the current model, the neural signal will be expressed based on Buchanan’s mechanism.

3.5  Peripheral Neuromuscular Model
Chapter 3: Components of the neuromuscular model

The loop described above is representative of the lower sensory loop. In neuromuscular modelling of tremor oscillation, this loop is indicated as the spinal feedback loop as the afferent information is passed to the alpha-motoneuron situated at the basal ganglia in the spinal cord. The second loop, the higher reflexive loop, is indicated as the supraspinal loop because afferent information travels up to the higher supraspinal centres such as the cerebellum. In the next chapter, the lower sensory loop and the higher reflexive loop are reviewed in more detail. Then this feedback loop analysis along with the neuromuscular component reviewed from this chapter are brought together to form the neuromuscular model described by the block diagram in Figure 15.

![Block diagram of neuromuscular control model with supraspinal and spinal feedback adapted from Stein and Oguztoreli [42]](image)

The control model above is adapted from Takanokura’s and Stein as well as Oguztoreli’s neuromuscular model for tremor oscillations. It can be seen that this neuromuscular model reflects the individual and resulting oscillatory mechanism shown in Chapter 2, Figure 2. In the next chapter, the following oscillatory mechanisms are simulated: a) muscle mechanical resonance b) spinal feedback resonance c) supraspinal feedback resonance. Following that, the interaction between the mechanisms a) muscle and spinal feedback b) muscle, spinal and supraspinal feedback is simulated.
4. Theoretical Analysis of the neuromuscular model

This section will be focused on generating the neuromuscular model and simulating the respective and overall oscillations of the mechanisms identified in Figure 2, Chapter 2 with the exception of central oscillations. To generate the model, the peripheral feedback loops of the neuromuscular system will be reviewed in the following sections.

4.1 Neuromuscular system and muscle contraction

Anatomically, movement is a multilevel sensory motor process that largely involves the neuromuscular system. It is initiated by the motor cortex which sends a command down the spinal cord to the actuator level of motoneurons, muscle and receptors while simultaneously
activating several sensory motor loops in the cerebellum brain stem and spinal cord in a hierarchical fashion [44]. These loops have been explained briefly in Chapter 3 and can be seen in Figure 18. This chapter will study these loops in more detail and how they come together to form the neuromuscular system.

The lower sensory motor loop is involved in motor control and is entirely contained within the spinal cord hence it is referred as the spinal loop. One of the simplest and most prominent motor loops is the stretch reflex which is responsible for mediating the stiffness of stretch and muscle. It consists of the monosynaptic connections from group Ia afferents to homonymous alpha-motoneurons and the concurrent activation of gamma-motoneurons acting in parallel to it [73]. (ref Figure 16).

The mechanics of this loop can be explained by the servo-assist hypothesis which proposes that primary muscle activation occurs from alpha-motoneuron innervation which receives
feedback from the spindle afferent that is being excited in parallel by gamma-motoneurons. Gamma-motoneurons function to maintain the tension in muscle spindle during active contraction ensuring muscle spindle responsiveness across all ranges of lengths. In many normal movements, gamma-motoneuron is activated in parallel with alpha-motoneuron and is often termed alpha-gamma coactivation [74].

However, various studies into reflex response have found that afferent response do not conform exclusively to the servo-assistance hypothesis above. Different movements have been shown to induce different responses to the activity of muscle spindle afferents. For example, the discharge of static or dynamic gamma-motoneuron which is rhythmically modulated with the activity of the alpha-motoneuron is functionally related to whether the muscle undergoes concentric, eccentric or near-isometric contractions [73]. This led to the suggestion that there is an involvement of descending commands from the supraspinal CNS regions which can be known as the supraspinal loop. The supraspinal loop is postulated to involve multiple long-loop pathways from muscle spindle to cerebellum [75].

Muscle spindle afferent signal is projected to higher levels of CNS where the appropriate central commands of muscle activation, force level production and movement are mediated based on the afferent information and the intended movement [76]. The resulting command is sent down various descending pathways depending on the supraspinal region involved. Traditionally descending systems include the cortico-, rubro-, reticulo-, vestibule-, tecto- and interstitions spinal- systems which terminate mono- or poly- synaptically on alpha-motoneurons in various patterns depending on the muscle type, position and motor unit [73]. This descending signal is believed to converge at the alpha-motoneuron where the corresponding neural signal for muscle activation is relayed to the respective muscle [73, 76]. The CNS
neural response is shown to excite both the alpha- and the gamma- motoneuron (ref Figure 16) where signals to alpha-motoneuron control extrafusal muscle fibres and signals to gamma-motoneuron control the intrafusal fibres. In the neuromuscular system, these two motoneurons do not activate concurrently and the excitation of either motoneuron is independent of each other [76]. The control block model of supraspinal feedback in Figure 17 is independent of gamma-motoneuron coactivation.

The resulting alpha-motoneuron activation will be either a direct response to the spinal feedback loop or the descending commands from the supraspinal centres or a combination of both [75]. Figure 18 shows the control block diagram representing a simplified interaction between the spinal and supraspinal reflex loop and the mathematical components reviewed in Chapter 3. The exponential functions, $e^{-ST_{AS}}$ and $e^{-ST_{AE}}$ represent the neural delay of the supraspinal and spinal afferent neural delay respectively. Subsequently $e^{-ST_{AE}}$ and $e^{-ST_{FS}}$ represent the supraspinal and spinal efferent neural delay respectively.
The muscle activation can be seen to be a result of the neural signal, $a(s)$ which, as mentioned previously in the muscle activation section of Chapter 3, represents the rectified, smoothed and normalised EMG initiating the active CE in the muscle [50]. This neural signal can be defined as a summation of the signal from the central nervous system, $a_{CNS}$ and a peripheral neuromuscular (PNS) oscillation, $a_{PNS}$ at the alpha-motoneuron junction, and its corresponding neural delay:

$$a(s) = e^{-\tau_s}E(a_{CNS} + a_{PNS})$$  \hspace{1cm} (28.1)

It can be seen in Figure 18 that the alpha-motoneuron receives two input signals:

a) A central command, $a_c$ which is a summation of the supraspinal feedback and the CNS command, $f_{CNS}$

b) The mechanical-reflex feedback response which is a function of $X_{ri}$ (where $X_{ri}$ is the transduced reticulate length, $X_r$) and spinal feedback delay, $e^{-\tau_s}A$
These two inputs are summed linearly in the alpha-motoneuron junction to give the neural signal, \( a(s) \):

\[
a(s) = e^{-st} E \left( K_a a_c + e^{-st} A K_b X_{ri} \right)
\]  

(29.1)

Comparing equation 28.1 to equation 29.1

\[
a(s) = e^{-st} E (a_{CNS} + a_{PNS}) = e^{-st} E \left( K_a a_c + e^{-st} A K_b X_{ri} \right)
\]  

(30.1)

Therefore \( a_{CNS} \) and \( a_{PNS} \) can be defined as the following:

\[
e^{-st} (a_{CNS}) = e^{-st} E K_a a_c
\]

\[
e^{-st} (a_{PNS}) = e^{-st} E e^{-st} A K_b X_{ri}
\]

(31.1-31.2)

It can be seen that the PNS neural signal describes the muscle mechanics and spinal stretch reflex interactions (hereafter referred as mechanical-reflex) aspect of the neuromuscular model and the CNS neural signal describes the central nervous commands and the supraspinal reflex interactions. This research intends to analyse the contributions of the neural feedback mechanisms and the muscle mechanics which can be seen in the PNS neural signal and the supraspinal feedback in the CNS neural signal. It will simulate the individual and resulting oscillations of these peripheral mechanisms as such the following sections will be grouped into:

- Individual mechanism
  - Muscle
  - Spinal feedback
  - Supraspinal feedback

- Interaction of mechanisms
  - Mechanical reflex
  - Mechanical reflex and supraspinal feedback
4.2 Individual mechanisms

In Chapter 3, the output of the muscle, muscle length, $X_m$, was found to be:

$$X_m(s) = \frac{F_{act}(s) + F_L(s)(1 + \frac{Bs}{k})}{s\left(\frac{MB}{k}s^2 + Ms + B\right)} \quad (32.1)$$

This output is relayed to the muscle spindle receptor which receives two inputs, muscle length, $X_m$ and gamma innervation, $F_\gamma$

$$X_r = \frac{\lambda(\eta Ts + 1)}{Ts + 1} X_m + \frac{1}{Ts + 1} F_\gamma e^{-Ts}G \quad (33.1)$$

Reticulate length, $X_r$, is then transduced into a series of impulses that represent the change in length detected by muscle spindle. As noted in equation 31.2 the PNS neural signal is a product of the impulse signal, $X_{ri}$, its afferent feedback delay and the attenuation factor of the alpha-motoneuron.

Based on Figure 16 equation 33.1 can be substituted into equation 31.2 to give the following representation of the PNS neural signal:

$$a_{PNS} = K_s e^{-Ts} A e^{-Ts} E \left\{ \frac{\lambda(\eta Ts + 1)}{Ts + 1} \left[ \frac{F_{act}(s) + F_L(s)(1 + \frac{Bs}{k})}{s\left(\frac{MB}{k}s^2 + Ms + B\right)} \right] + \frac{1}{Ts + 1} F_\gamma e^{-Ts}G \right\} \quad (34.1)$$

The equation above describes the mechanical-reflex oscillation. Within it, are the equations describing the muscle mechanics and the spinal reflex which can be extracted to the following:
4.2.1 Muscle mechanics

From equation 34.1 and Figure 16 the muscle mechanics is defined by the following equation (where $v_m$ is the rate of muscle length change):

$$v_m = \frac{F_{act}(s)}{\left(\frac{MB}{k} s^2 + Ms + B\right)}$$

(35.1)

This shows that the muscle is a second-order system with the following system poles:

$$\lambda_i = -\frac{k}{2B} \pm j \sqrt{\frac{4kB^2 - Mk^2}{4B^2M}}$$

(36.1)

The natural frequency, $\omega_n$ and damping ratio, $\zeta$ is found to be:

$$\omega_n = \sqrt{\frac{k}{M}}$$

$$\zeta = \frac{k}{2B} \sqrt{\frac{M}{k}}$$

(37.1-37.2)

The values for $k$, and $B$ are taken from Stein and Oguztoreli’s[77] neuromuscular modelling and the value of $M$ which in this case represents the mass of the biceps brachii is taken from Watson and Wilson [78]. These values have been commonly referred in existing work on neuromuscular modelling for tremor:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>B</td>
<td>40 Nsm$^{-1}$</td>
<td></td>
</tr>
<tr>
<td>M</td>
<td>0.8 Kg</td>
<td></td>
</tr>
<tr>
<td>K</td>
<td>647 Nm$^{-1}$</td>
<td></td>
</tr>
</tbody>
</table>

Based on these known parameters, the roots of the eigenvalue, $\lambda_i$ natural frequency, $\omega_n$ and damping ratio, $\zeta$ can be calculated from equation 36.1, 37.1 and 37.2 respectively:
Table 3 Frequency response of muscle mechanics system

<table>
<thead>
<tr>
<th>ROOTS</th>
<th>DAMPING, ζ</th>
<th>FREQUENCY, ω</th>
<th>FREQUENCY, f</th>
</tr>
</thead>
<tbody>
<tr>
<td>-8.09e+000 + 2.73e+001i</td>
<td>2.84E-01</td>
<td>2.84E+01</td>
<td>4.52</td>
</tr>
<tr>
<td>-8.09e+000 - 2.73e+001i</td>
<td>2.84E-01</td>
<td>2.84E+01</td>
<td>4.52</td>
</tr>
</tbody>
</table>

Table 3 indicate two root values in the complex region which can be seen in corresponding poles (x-marker) plotted in Figure 19. As can be seen in Table 3 above, these roots will cause an oscillation at the frequency, $f$ of 4.52Hz. A smaller value of the real part of the root indicates a slower oscillatory decay. The damping ratio, $ζ$ is a measure of the system’s ability to oppose its oscillatory behaviour. Smaller values of damping ratio indicate a lesser ability to damp the system’s oscillations. The values in Table 3 indicate the muscle acts as an underdamped system oscillating at the freq of 4.52Hz. The step response of the system is plotted in Appendix Figure A1.

Figure 19 Pole zero map of the muscle mechanical system
4.2.2 Spinal feedback

The PNS neural signal, \( a_{PNS} \) represented in equation 34.1 is the resulting mechanical-reflex of the muscle system and spinal system. In order to obtain an equation of the spinal loop component of the \( a_{PNS} \), the muscle length is left constant, \( X_m = 1 \) to generate a \( a_{PNS} \) that is independent of muscle mechanics. The resulting spinal feedback is seen in the equation 38.1 below (where \( \tau_{sr} \) is the spinal reflex delay and \( \tau_{ag} \) is the spinal alpha gamma delay)

\[
a_{SR} = e^{-\tau_{sr}} K_{SR} \left( \frac{\tau T s + 1}{T s + 1} \right) + e^{-\tau_{ag}} K_{AG} \frac{1}{T s + 1}
\]

(38.1)

where

\[
e^{-\tau_{sr}} = e^{-a(\tau_s + \tau_e)}
\]

\[
e^{-\tau_{ag}} = e^{-a(\tau_s + \tau_e + \tau_G)}
\]

(39.1-39.4)

\[
K_{SR} = A \cdot E \cdot K_b \cdot \lambda
\]

\[
K_{AG} = F_y \cdot G \cdot A \cdot E \cdot K_b
\]

The values for the parameters above are obtained from Lin and Crago’s [79] study of the structural model of muscle spindle and shown in Table 4.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>H</td>
<td>28.9</td>
<td>N</td>
</tr>
<tr>
<td>T</td>
<td>0.00133</td>
<td>N</td>
</tr>
<tr>
<td>( F_{\gamma} )</td>
<td>0.35</td>
<td></td>
</tr>
<tr>
<td>( K_b )</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>G</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>1.10E-03</td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>( \Lambda )</td>
<td>0.02</td>
<td>imps(^{-1})N(^{-1})</td>
</tr>
<tr>
<td>( \tau_E )</td>
<td>1.00E-02</td>
<td>S</td>
</tr>
<tr>
<td>( \tau_G )</td>
<td>1.80E-02</td>
<td>S</td>
</tr>
<tr>
<td>( \tau_A )</td>
<td>4.00E-03</td>
<td>S</td>
</tr>
</tbody>
</table>

The values of the parameters are substituted into equation 38.1 and 39.1-39.4 and inputted the equation into Mathworks MATLAB to obtain the following frequency table (ref. Table 5):
Table 5 Frequency response of the spinal feedback system

<table>
<thead>
<tr>
<th>ROOTS</th>
<th>DAMPING, $\zeta$</th>
<th>FREQUENCY, $\omega$</th>
<th>FREQUENCY, $f$</th>
</tr>
</thead>
<tbody>
<tr>
<td>-1.45E+02</td>
<td>1.00E+00</td>
<td>1.45E+02</td>
<td>23.07</td>
</tr>
<tr>
<td>-1.15e+002 + 1.10e+002i</td>
<td>7.24E-01</td>
<td>1.59E+02</td>
<td>25.30</td>
</tr>
<tr>
<td>-1.15e+002 - 1.10e+002i</td>
<td>7.24E-01</td>
<td>1.59E+02</td>
<td>25.30</td>
</tr>
<tr>
<td>-3.32E+02</td>
<td>1.00E+00</td>
<td>3.32E+02</td>
<td>52.83</td>
</tr>
<tr>
<td>-2.63e+002 + 2.51e+002i</td>
<td>7.24E-01</td>
<td>3.63E+02</td>
<td>57.77</td>
</tr>
<tr>
<td>-2.63e+002 - 2.51e+002i</td>
<td>7.24E-01</td>
<td>3.63E+02</td>
<td>57.77</td>
</tr>
<tr>
<td>-7.52e+002 + 7.30e-005i</td>
<td>1.00E+00</td>
<td>7.52E+02</td>
<td>119.67</td>
</tr>
<tr>
<td>-7.52e+002 - 7.30e-005i</td>
<td>1.00E+00</td>
<td>7.52E+02</td>
<td>119.67</td>
</tr>
</tbody>
</table>

When an input is introduced to a dynamic system, the response of the system is generally due to the contributions of all the roots of the system and the sum of these contributions. Table 5 above shows the roots that contribute to the response of the spinal feedback system. However, the system may exhibit a dominant root behaviour largely attributed to roots near
the origin of the s-plane (Imaginary axis as seen in Figure 20). Roots near the origin give rise to the longest lasting transient response whereas roots further from the origin have a small duration and magnitude of transient response. In this case, the roots with the longer response will dominate the transient response thus causing a dominant behaviour. The rule of the thumb is if the real part of the root(s) closest to the origin is less than ten times to the next closest root(s) to the origin (root<sub>i</sub> < 10xroot<sub>i+1</sub>), the former is considered dominant [80]. In Table 5 above, the closest root(s) to the origin is the complex root pair and the real roots (roots: -1.45e+02; -1.15e+002+1.10e+002i, -1.15e+002-1.10e+002i) which will be termed r<sub>1</sub> and the next consecutive root is the complex root pair and real roots (roots: -3.32e+02; -2.63e+002+2.51e+002i, -2.63e+002-2.51e+002i) termed r<sub>2</sub>. In this case r<sub>1</sub> is not ten times smaller than r<sub>2</sub> so the above rule of thumb cannot be applied. In order to view the dominant root, the step response of the two root(s) are plotted against the actual step response of the spinal feedback system as seen in Figure 21.

![Comparison of step response of root(s)](image)

**Figure 21** Comparison of step response of root(s) for the spinal feedback system
The comparison of the step response of root(s) of the transient system indicates \( r_2 \) acts as a dominant root. The frequency of \( r_2 \) from Table 5 for the real root is 52.83Hz and the complex conjugate pair is 57.77Hz and the resulting sum of the root(s) is 110.6Hz.

### 4.2.3 Supraspinal feedback

To consider the supraspinal feedback of the system independent of muscle mechanics and a central feedback oscillation, equation 31.1 is rewritten into the following (where \( X_m=1, f_{\text{CNS}}=0 \)):

\[
a_{\text{CNS}} = e^{-\tau_{\text{AS}}} E_S e^{-\tau_{\text{ES}}} E_K a \left[ e^{-\tau_{\text{AS}}} A_s K_s \left( \frac{\lambda(\eta T_s + 1)}{T_s + 1} \right) \left( \frac{F_{\text{act}}}{MB} \frac{1}{s^2 + Ms + B} \right) + f_{\text{CNS}} \right] \tag{40.1-40.2}
\]

\[
a_{\text{SSR}} = e^{-\tau_{\text{SSR}}} K_{\text{SSR}} \frac{(\eta T_s + 1)}{T_s + 1} \tag{41.1-41.2}
\]

where:

\[
e^{-\tau_{\text{SSR}}} = e^{-t(\tau_{\text{AS}} + \tau_{\text{ES}} + \tau_{\text{E}})}
\]

\[
K_{\text{SSR}} = A_s \cdot E \cdot K_e \cdot \lambda \cdot K_a \cdot E_s \tag{41.1-41.2}
\]

The variables \( \tau_{\text{AS}} \) and \( \tau_{\text{ES}} \) are the afferent supraspinal delay and efferent supraspinal delay respectively whilst the variable \( \tau_{\text{E}} \) represents the alpha motoneuron delay which occurs after supraspinal signal converges to alpha-motoneurone. To find the values of \( \tau_{\text{AS}} \) and \( \tau_{\text{ES}} \) can be found from the overall supraspinal feedback delay, \( \tau_{\text{AS}} + \tau_{\text{ES}} + \tau_{\text{E}} \) which is postulated to manifest itself in the latency of EMG response to perturbation, stimulus or stretch [81]. The spinal and supraspinal delay found from various literatures studying stretch latency along with supraspinal delay used in neuromuscular modelling is listed in Table 6 below:
From Table 6 above, the spinal delay which refers to the alpha-gamma reflex loop shows an average of 26-34ms. This experimental delay time is taken from the start of the stretch to the first detected response in the EMG signal and is believed to be a sum of the afferent spindle delay and the alpha and gamma efferent feedback delay, $\tau_A + \tau_E + \tau_G$. Table 4 which indicates the experimental values used in the current simulation showed the $\tau_G$: 18ms and the resulting alpha efferent and spindle afferent delay, $\tau_E + \tau_A$: 14ms giving a resulting alpha-gamma efferent loop delay of 32ms which falls within the range of monosynaptic alpha-gamma loop latency found in literature. In literature, this alpha-gamma loop latency is often known as the M1.

This suggests that the experimental long-loop latency (known in literature as M2) which shows a range of 60-68ms reflects the supraspinal loop delay which is indicated by the sum of afferent supraspinal loop delay, $\tau_{AS}$, efferent descending supraspinal delay, $\tau_{ES}$ and the $\tau_E$ experienced after converging into the alpha-motoneuron

$$\tau_{AS} + \tau_{ES} + \tau_E = \text{experimental supraspinal delay} \quad (42.1)$$

$$\tau_{AS} + \tau_{ES} + \tau_E = 68 \text{ms} \quad (43.1)$$
The value 68ms is the upper limit of the experimental supraspinal delay range as reflective of upper range used for modelling the spinal delay. The parameter values are then substituted into equation 41.1 and simulated to obtain Table 7:

<table>
<thead>
<tr>
<th>ROOTS</th>
<th>DAMPING, $\zeta$</th>
<th>FREQUENCY, $\omega$</th>
<th>FREQUENCY, $f$</th>
</tr>
</thead>
<tbody>
<tr>
<td>-6.83E+01</td>
<td>1.00E+00</td>
<td>6.83E+01</td>
<td>10.87</td>
</tr>
<tr>
<td>-5.41e+01 + 5.16e+00i</td>
<td>7.24E-01</td>
<td>7.48E+01</td>
<td>11.90</td>
</tr>
<tr>
<td>-5.41e+01 - 5.16e+00i</td>
<td>7.24E-01</td>
<td>7.48E+01</td>
<td>11.90</td>
</tr>
<tr>
<td>-7.52E+02</td>
<td>1.00E+00</td>
<td>7.52E+02</td>
<td>119.67</td>
</tr>
</tbody>
</table>

Table 7 Frequency table of supraspinal feedback

Figure 22 and Table 7 show complex and real root(s) close to the origin (-6.83E+1; -5.41e+01 + 5.16e+001i, -5.41e+001 - 5.16e+001i) termed $r_1$ and a real root (-7.52E+02) termed $r_2$. It is seen that $r_1 < 10 \times r_2$ thus the dominant root is $r_1$ with a frequency of 11.9Hz.
4.3 Interaction of Mechanisms

The final section of this chapter will look to the interaction effects between the various peripheral oscillatory mechanisms indicated in Figure 2, Chapter 2: a) muscle mechanics, b) spinal feedback c) supraspinal feedback. The control block diagram in Figure 18 is representative of the interactions shown in Figure 2 (ref Chapter 2) and the overall transfer function for the system can be derived from the following equation (where $\tau_{SS}$ and $K_{SS}$ are supraspinal loop delay and gain before the descending efferent signal converges to the alpha-motoneuron):

$$a_{MR} = e^{-\tau_{ss}K_{SR}} + e^{-\tau_{ss}K_{SS}} \left( \frac{\eta Ts + 1}{Ts + 1} \left( \frac{F_{act}(s)}{Mb + Ms + B} \right) \right) + e^{-\tau_{AG}K_{AG}} \frac{1}{Ts + 1} \quad (44.1)$$

4.3.1 Mechanical-reflex feedback

The mechanical-reflex response is a combination of the muscle mechanism and spinal stretch reflex mechanism which is given to be the equation 34.1 ($a_{PNS}$) and can be simplified to the following:

$$a_{SR} = e^{-\tau_{ss}K_{SR}} \left( \frac{\eta Ts + 1}{Ts + 1} \left( \frac{F_{act}(s)}{Mb + Ms + B} \right) \right) + e^{-\tau_{AG}K_{AG}} \frac{1}{Ts + 1} \quad (45.1)$$

In the above equation, $F_L$ was considered negligible as the experimental setup considers the arm in isometric contraction without the introduction of an external perturbation. The values of $K_{SR}$, $K_{AG}$ and their corresponding time delays remain the same. The output of the system
reflect the changes in muscle length following a step response as such the muscle mechanics is defined with $v_m$.

Figure 23 Pole zero plot of the mechanical-reflex loop

Table 8 Frequency response of the mechanical-reflex system

<table>
<thead>
<tr>
<th>Roots</th>
<th>Damping, $\zeta$</th>
<th>Frequency, $\omega$</th>
<th>Frequency, $f$</th>
</tr>
</thead>
<tbody>
<tr>
<td>-8.09 + 27.26i</td>
<td>2.84E-01</td>
<td>2.84E+01</td>
<td>4.53</td>
</tr>
<tr>
<td>-8.09 - 27.26i</td>
<td>2.84E-01</td>
<td>2.84E+01</td>
<td>4.53</td>
</tr>
<tr>
<td>-145.14</td>
<td>1.00E+00</td>
<td>1.45E+02</td>
<td>23.10</td>
</tr>
<tr>
<td>-114.93 + 109.65i</td>
<td>7.24E-01</td>
<td>1.59E+02</td>
<td>25.28</td>
</tr>
<tr>
<td>-114.93 - 109.65i</td>
<td>7.24E-01</td>
<td>1.59E+02</td>
<td>25.28</td>
</tr>
<tr>
<td>-331.74</td>
<td>1.00E+00</td>
<td>3.32E+02</td>
<td>52.79</td>
</tr>
<tr>
<td>-262.70 + 250.63i</td>
<td>7.24E-01</td>
<td>3.63E+02</td>
<td>57.78</td>
</tr>
<tr>
<td>-262.70 - 250.63i</td>
<td>7.24E-01</td>
<td>3.63E+02</td>
<td>57.78</td>
</tr>
<tr>
<td>-751.88</td>
<td>1.00E+00</td>
<td>7.52E+02</td>
<td>119.65</td>
</tr>
<tr>
<td>-751.88</td>
<td>1.00E+00</td>
<td>7.52E+02</td>
<td>119.65</td>
</tr>
</tbody>
</table>
Figure 23 and Table 8 indicate a complex root close to the origin $(-8.09 + 27.26i, -8.09 - 27.26i)$ termed $r_1$ and a complex and real root(s) $(-145.14; -114.93 + 109.65i, -114.93 - 109.65i)$ termed $r_2$. It can be seen $r_1 < 10 \times r_2$ thus the dominant root is $r_1$ with a frequency of 4.53Hz.

### 4.3.2 Interaction of mechanical reflex and supraspinal feedback

The interaction of mechanical reflex and the supraspinal feedback can be seen in Figure 18 and the resulting transfer function of the control block diagram is given by equation 44.1 where:

$$
e^{-\tau_{SS}} = e^{-(\tau_{AS} + \tau_{ES})}$$

$$K_{SS} = A_j \cdot E_s \cdot K_a \cdot K_c \cdot \lambda$$  \hspace{1cm} (46.1-46.2)

The values for the parameters in equation 46.1 and 46.2 above can be derived from the experimental values shown in Table 6 and equation 43.1. From Table 4, the $\alpha$-delay, $\tau_E$ is 0.01s, and thus the supraspinal feedback delay can then define $\tau_{AS} + \tau_{ES} = 58ms$. The values of equation 46.1 and 46.2 are shown in Table 9 below:

**Table 9 Parameter values of the supraspinal feedback**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\tau_{AS} + \tau_{ES}$</td>
<td>5.80E-02</td>
<td>S</td>
</tr>
<tr>
<td>$K_a$</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>$K_c$</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>$A_sE_s$</td>
<td>0.016</td>
<td></td>
</tr>
</tbody>
</table>

Simulating the system response leads to the following mechanical-reflex and supraspinal feedback frequency response and its pole zero plot:
Table 10 Frequency table of the interaction between the mechanical reflex and the supraspinal loop

<table>
<thead>
<tr>
<th>Roots</th>
<th>Damping, ( \zeta )</th>
<th>Frequency, ( \omega )</th>
<th>Frequency, ( f )</th>
</tr>
</thead>
<tbody>
<tr>
<td>(-8.08 + 27.26i)</td>
<td>2.84E-01</td>
<td>2.84E+01</td>
<td>4.53</td>
</tr>
<tr>
<td>(-8.08 - 27.26i)</td>
<td>2.84E-01</td>
<td>2.84E+01</td>
<td>4.53</td>
</tr>
<tr>
<td>(-80.08)</td>
<td>1.00E+00</td>
<td>8.01E+01</td>
<td>12.74</td>
</tr>
<tr>
<td>(-63.41 + 60.50i)</td>
<td>7.24E-01</td>
<td>8.76E+01</td>
<td>13.95</td>
</tr>
<tr>
<td>(-63.41 - 60.50i)</td>
<td>7.24E-01</td>
<td>8.76E+01</td>
<td>13.95</td>
</tr>
<tr>
<td>(-145.14)</td>
<td>1.00E+00</td>
<td>1.45E+02</td>
<td>23.10</td>
</tr>
<tr>
<td>(-114.93 + 109.65i)</td>
<td>7.24E-01</td>
<td>1.59E+02</td>
<td>25.28</td>
</tr>
<tr>
<td>(-114.93 - 109.65i)</td>
<td>7.24E-01</td>
<td>1.59E+02</td>
<td>25.28</td>
</tr>
<tr>
<td>(-331.74)</td>
<td>1.00E+00</td>
<td>3.32E+02</td>
<td>52.79</td>
</tr>
<tr>
<td>(-262.70 + 250.63i)</td>
<td>7.24E-01</td>
<td>3.63E+02</td>
<td>57.78</td>
</tr>
<tr>
<td>(-262.70 - 250.63i)</td>
<td>7.24E-01</td>
<td>3.63E+02</td>
<td>57.78</td>
</tr>
<tr>
<td>(-751.88 + 6.81e-05i)</td>
<td>1.00E+00</td>
<td>7.52E+02</td>
<td>119.65</td>
</tr>
<tr>
<td>(-751.88 - 6.81e-05i)</td>
<td>1.00E+00</td>
<td>7.52E+02</td>
<td>119.65</td>
</tr>
</tbody>
</table>

Figure 24 Pole zero plot of the interaction between the mechanical reflex and the supraspinal loop
Figure 24 and Table 10 indicate a complex root close to the origin \((-8.08 + 27.26i, -8.08 - 27.26i)\) termed \(r_1\) and a complex and real root(s) \((-80.08; -63.41 + 60.50i, -63.41 - 60.50i)\) termed \(r_2\). It can be seen \(r_1\) is not less than ten times of \(r_2\) thus in order to determine the dominant root, the response of the root(s) in Table 10 are simulated as seen in Figure 25.

![Comparison of step response of root(s) for mechanical reflex and supraspinal reflex](image)

**Figure 25 Comparison of step response of root(s) for mechanical reflex and supraspinal feedback interaction**

Figure 25 shows the dominant root to be \(r_1\) with a frequency of 4.53Hz.

In an effort to analyse the role of peripheral mechanism in physiological tremor, the individual and resulting oscillatory response of each mechanism proposed by McAuley (ref Figure 2) [28] has been modelled and simulated. The following chapter covers the experimental procedures and acquisition of EMG data, its signal analysis and the statistical analysis of the resulting values.
5. Experimental Analysis of surface EMG signal

It is postulated that stretch reflex loops operate over time and may oscillate at frequencies inversely proportion to the loop delay as such, stretch-reflex oscillation may be a result of the delays in peripheral loops. Consequently this research will analyse the temporal information of the oscillations found in EMG signal, hereafter known as the time constant ($T_C$) based on the hypothesis above. This chapter will look to the experimental methodology, signal analysis and statistical analysis. Following that, the next chapter will discuss the results obtain and compare its value to the theoretical values simulated.
5.1 Experimental methodology

5.1.1 Subjects

A total of 54 subjects had voluntarily participated in the experiment. Their ages range from 20-49 years old and their data was segregated into three age categories: A20, range: 20-29 years old; A30, range: 30-39 years old; and A40, range: 40-49 years old. All participants had no history of myopathy or neuropathy and were able bodied. At the start of the experiments, subjects were given a short, verbal summary of the experimental protocol and a written information sheet detailing the purpose and experimental protocol. Once subjects are in agreement to the experimental process they are given a consent form to sign. This research has been approved by the College Human Ethics Advisory Network (CHEAN).

5.1.2 EMG Signal Acquisition

The EMG signal was obtained via a surface EMG acquisition system by DELSYS. The system supports bipolar recording and has a gain of 1000, CMRR of 92 dB and bandwidth of 20-450 Hz, with 12dB/octave roll-off. The sampling rate was fixed at 1000 samples per second, and the resolution was 16 bits/sample. The EMG sensor used was the DE2.1, a single-differential parallel bar sensor and can be seen in Figure 26. The bar sensors are fixed at an inter-electrode distance of 10mm and the sensor has gain of 1000. Further information on the DE2.1 can be found on the DELSYS website [91].
Prior to placing the electrodes, the skin of the subject was cleaned with a 70% v/v alcohol swab to remove oil or any dead skin cells. If necessary, the skin of the subject was prepared by shaving and exfoliating. A thin layer of gel is spread over the surface of the electrode before placement to improve conductivity. A reference electrode is placed on the medial epicondyle of the humerus.

The EMG signal will be obtained from the biceps brachii which is active during isometric elbow flexion as in this experiment participants were asked to flex their forearm towards their body (flexion). The surface EMG electrode will be placed on the midline of the belly of the muscle.

5.1.3 Experimental protocol

During the experiments, the participants were seated on a sturdy, adjustable chair with their feet flat on the floor and their upper arm resting on the surface of an adjustable desk fixed at a level on which the upper arm can rest comfortably in a horizontal position as seen in Figure
27. The forearm was drawn towards the body such that the forearm was held vertical and the elbow was maintained at 90 degrees with the palm facing towards the subject and fingers in line with a wall mounted force sensor (S-type force sensor – INTERACE SM25). Subjects were told to loosely grip on a comfortable hand size wrist ring attached to the force sensor with a flexible steel wire henceforth known as force pulley. The output of the force sensor was recorded on one of the channel in the EMG acquisition system and displayed in real time. The experimental setup can be seen in Figure 27.

![Diagrammatic representation of the experimental setup](image)

**Figure 27 Diagrammatic representation of the experimental setup conduction to obtain EMG signal of subject maintaining isometric contraction**

On instruction, subjects are told to pull the force pulley towards their body. Four levels of contractions were measured through the experiment. The force levels were: 1) 25% of maximal voluntary contraction, 2) 50% of maximal voluntary contraction, 3) 75% of maximal voluntary contraction and 4) 100% of maximal voluntary contraction. The
percentages were calculated from the maximal voluntary contraction (MVC) value obtained from an average of three MVC trials of 5 seconds with 120 seconds rest time between each effort prior to the start of the experiment. Subjects were told to maintain contraction until either the end of trial or feeling fatigue, depending on whichever came first.

Following that, subjects performed the experiments at four levels of force values, referred as 25MVC set, 50MVC set, 75MVC set and 100MVC set. The sequence of force levels sets were given at random. The force performed was sampled and displayed at real time on a desktop monitor with a marked line on the monitor to indicate the force contraction level to maintain. Two trials were recorded for each force level set, henceforth referred to as trial 1 and trial 2. Each trial lasted for a maximum of 10s or until the subject felt fatigued. Between each trial, the subjects were given a rest period of 60 minutes or until the subject felt able to continue. In total the subject performed a total of eight trials for the experiment with an additional three trials prior to the start of the experiment for MVC. The trials were repeated if there were any outliers.

### 5.2 Signal Analysis

In the field of tremor oscillation studies, firing rate information of EMG signals are commonly observed from the peak frequency found in the power spectrum of rectified EMG signal. The power spectrum is obtained by applying zero-padding to 1024 samples and then taking the square of the magnitude of a 1024 point fast Fourier transform (FFT) of the time series. Prior to FFT, EMG signal is rectified to enhance the timing or firing rate information of EMG signals [15, 92]. It has been suggested that ‘full wave rectification of the EMG signal
provides the temporal pattern of grouped motor unit (MU) firing rates regardless of its action potential (AP) shape’ [93]. In tremor research, power spectrum is applied to enhance the firing information of EMG signal particularly for composite EMG signal where the firing frequency is harder to detect. Whilst the power spectrum method is commonly employed in tremor oscillation research, Myers [93] stated that it remained speculative if the observed peaks in the spectrum of rectified EMG signals are indicative of the firing rate information.

Another method of obtaining the timing information of the EMG signal is to study the envelope waveform of the EMG signal itself. Power spectrum analysis of the rectified EMG signal is actually an approximation of the waveform envelope which gives the firing rate information of the EMG signal. The waveform envelope can be seen in the varying amplitudes of the rapidly oscillating components in EMG signal. To obtain this waveform envelope Myers used Hilbert transform (HT). He found that there is no significant difference between the power spectrum method and the envelope extraction method for extracting firing rate information and states that these two methods can be used interchangeably.

This research intends to study the role of peripheral mechanisms in the oscillation in EMG signal based on the proposed hypothesis: that tremor oscillation in EMG signal oscillates at frequencies inversely proportional to the stretch reflex loop delay. EMG oscillation will be studied in its time domain using the envelope extraction method using HT for the envelope extraction.
5.2.1 Hilbert Transform

Hilbert transform is an original technique introduced by Huang et al [94] which adaptively decomposes a signal into its simplest intrinsic oscillatory components. It is extremely popular for nonlinear and nonstationary signals making HT an effective technique for waveform envelope extraction which is a non-linear operation. The HT is an integral transformation that allows complex signals such as the EMG signal to be analysed in the time domain. It can be used to examine the signal’s instantaneous attributes such as the frequency, phase and amplitude [95]. The envelope of the EMG signal can be obtained by applying Hilbert’s amplitude which calculates the instantaneous amplitude of the signal.

The HT function is defined by an integral transform:

\[ H[x(t)] = \tilde{x}(t) = p.v.\int_{-\infty}^{\infty} \frac{x(\tau)}{t-\tau} d\tau \]  \hspace{1cm} (47.1)

The variable \( p.v. \) denotes the Cauchy principal value of the integral. When dealing with general modulated signals, \( x(t) \), it is often convenient to define the analytic signal, \( X(t) \) which can be given to be:

\[ X(t) = x(t) + i\tilde{x}(t) \]  \hspace{1cm} (48.1)

The term ‘analytic’ is used to refer to a complex function of a complex variable whose imaginary part is the Hilbert transform, \( \tilde{x}(t) \) of the real part, \( x(t) \) as shown in equation 48.1 above. The analytic signal [95] can be represented in its trigonometric or instantaneous form to be:

\[ X(t) = |X(t)|[\cos \phi(t) + i \sin \phi(t)] = A(t)e^{i\phi(t)} \]  \hspace{1cm} (49.1)
Chapter 5: Experimental analysis of surface EMG signal

$A(t)$ represents the instantaneous amplitude and can be used to determine the envelope of the signal using:

$$A(t) = |X(t)| = \sqrt{x^2(t) + \ddot{x}^2(t)}$$

(50.1)

A plot of the resulting HT amplitude signal obtained can be seen in Figure 28 below. The x-axis for both the HT amplitude and EMG signal were normalised. Figure 28 has been enlarged.

![Figure 28 Plot of Rectified EMG signal superimposed with Rectified Hilbert transform amplitude envelope](image)

**5.2.2 Smoothing filter**

In tremor oscillation literature, moving average window is commonly applied to smooth the data for rectified EMG signal. Hahs and Stiles [15] noted that for this filter, there is a variance of frequency components at 10 and 32 Hz which is attenuated to 0.92 and 0.4 of the
unfiltered values respectively. This attenuation causes an amplitude distortion which may pose to be a problem as 10Hz is the common peak frequency found in EMG oscillation. Hahs and Stiles found that the use of power spectrum compensates for the effects of the moving average smoothing filter. An alternative method is adapted to filter the data as it is to be analysed in the time domain.

Least squared filter is a low pass filter that smooths the data directly in time domain, preserving its temporal information. In a normal moving average window filter, each data point is replaced by an average of the window sample. For least squared filter, an approximate of the underlying function of the moving window is used in the place of the mean value. The function is typically a polynomial of higher order which is commonly either quadratic or quartic and in the case of derivatives, quartic or higher [96]. The HT amplitude signal was smoothed with a least squared filter of the 5th order and a windowing size of 51ms resulting in the smoothed signals show in Figure 29 below. Figure 29 shows an enlarged plot of the smoothed HT signal against the EMG signal and Figure 30 shows a further enlarged plot of the smoothed HT amplitude without the superimposed EMG signals.
Figure 29 Plot of rectified EMG signal superimposed over the smoothed, rectified Hilbert transformed envelope

Figure 30 Plot of smoothed Hilbert transformed EMG signal
The peaks of the envelopes in the smoothed HT data are detected by obtaining the maxima points of the peaks. As seen in Figure 30 some envelopes have a number of peaks in close proximity due to superimposed MUAP of the same frequency. There is a tolerance of 10ms for each maxima point detected. If a subsequent maxima point is found within the 10ms range, the maxima points are grouped as a single envelope peak. The 10ms was based on visual and empirical tests.

5.3 Data Analysis

Time constant ($T_C$) is the time between two envelope waveforms detected. A window of 1000 samples is applied to the smoothed HT amplitude signal and the average $T_C$ per window is calculated and recorded. The first five seconds were ignored to ensure participants reaching and maintaining at the intended force level and the next five seconds were used in the analysis.

The stretch reflex delay is hypothesised to reflect in the $T_C$ between the waveform envelopes of EMG signal. This delay is the neural delay of the peripheral feedback and is suggested to be influenced by the type of contraction and the anthropomorphic features [97]. In order to assess the results to the above hypothesis, the following null hypothesis is analysed: the neural delay remains unchanging for subject variation (bearing subjects do not suffer from neuropathy and are of similar size) $X_1$, number of repeated trials, $X_2$ and different levels of force level, $X_3$. The low frequency variation which is hypothesised to reflect the neural delays can be monitored through the time constant of the EMG modulation. Thus if the $T_C$
between the chosen variables (X1, X2 and X3 respectively) show a significant difference in the analysis of variance (ANOVA), then the null hypothesis is rejected.

In order to validate the significance of the time constant obtained, the results are subjected to a statistical analysis of variance (ANOVA) test. The ANOVA test examines the variation between the mean $T_C$ of two groups or more. The statistical analysis will consider three of the following variables shown below and will utilise a multiway (n-way) ANOVA to study the interaction effects between these variables using a one-way, two-way and three-way ANOVA in Mathworks MATLAB:

- **Subject, X1**: Variation between the mean $T_C$ of 54 subjects are analysed
- **Trial, X2**: Variation between the mean $T_C$ of trial 1, T1 and trial 2, T2
- **Force level, X3**: Variation between the mean of $T_C$ of 25MVC, 50MVC, 75MVC and 100MVC

### 5.3.1 Results

Table 11 shows linear regression analysis which analyses the variance of the mean $T_C$ values from linear regression of mean $T_C$ across the subjects. There is no significant variation from the mean value for each trial-force category.

<table>
<thead>
<tr>
<th>$P$-Value</th>
<th>25MVC</th>
<th>50MVC</th>
<th>75MVC</th>
<th>100MVC</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1</td>
<td>0.050537</td>
<td>0.258374</td>
<td>0.780447</td>
<td>0.504862</td>
</tr>
<tr>
<td>T2</td>
<td>0.987326</td>
<td>0.877534</td>
<td>0.909349</td>
<td>0.075961</td>
</tr>
</tbody>
</table>

Following this, Figure 31 and 32 observes the plot of the average values of the time constant, $T_C$ of EMG signal for different levels of muscle contraction (% MVC). The mean ($\pm$std) for
trial 1 and trial 2 across the force level are 0.0782s (±3.0629e-004) and 0.0785s (±2.5140e-004) respectively. The standard deviation is very small for all the experiments (ref Fig. 28 and 29).

---

![Chart](image.png)

**Figure 31** Plot of mean average time constant from 25MVC to 100MVC for trial 1 of all subjects

![Chart](image.png)

**Figure 32** Plot of mean average time constant from 25MVC to 100MVC for trial 2 of all subjects
Linear regression analysis of the mean time constant across all levels for trial 1 and trial 2 showed no significant variation. The $p$-values indicating the goodness of fit of the regression line for trial 1 and trial two are $p$-value: 0.742>0.05 and $p$-value 0.067>0.05 respectively. In Figure 32 the $T_C$ for trial 2 showed a gradual increase across different levels of force but $p$-value showed no significant variation from the goodness of fit. Thus it is observed that the time constant corresponding to the oscillation of the EMG signal is not significantly changed for different people, and for different levels of force of muscle contraction.

Following that, the effects of variable interaction on the mean value of $T_C$ obtained was obtained using the statistical multivariate analysis of variance (MANOVA) analysis on Mathworks MATLAB. The calculations and the statistical significance obtained can be seen in Table 12:

<table>
<thead>
<tr>
<th>Variables</th>
<th>SSE $10^{-4}$</th>
<th>Mean Sq $10^{-6}$</th>
<th>F</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>X1</td>
<td>0.34</td>
<td>6.40</td>
<td>1.21</td>
<td>0.166</td>
</tr>
<tr>
<td>X2</td>
<td>0</td>
<td>3.93</td>
<td>0.74</td>
<td>0.389</td>
</tr>
<tr>
<td>X3</td>
<td>0.1</td>
<td>2.71</td>
<td>0.51</td>
<td>0.673</td>
</tr>
<tr>
<td>X1*X2</td>
<td>0.19</td>
<td>6.90</td>
<td>1.37</td>
<td>0.124</td>
</tr>
<tr>
<td>X2*X3</td>
<td>0</td>
<td>8.47</td>
<td>0.17</td>
<td>0.845</td>
</tr>
<tr>
<td>X1*X3</td>
<td>0.67</td>
<td>5.07</td>
<td>1.01</td>
<td>0.484</td>
</tr>
<tr>
<td>X1<em>X2</em>X3</td>
<td>0.9</td>
<td>5.65</td>
<td>1.08</td>
<td>0.312</td>
</tr>
</tbody>
</table>

The statistical analysis shows that $T_C$ is not affected by the difference in the participants, experiment number or the level of muscle contraction. The results also show that there is no significant interaction effect of the three factors. From Table 12, and the Figure 31 and 32, it is observed that the time constant, $T_C$, of EMG during isometric muscle contraction of the biceps is 78ms, and this remains unaffected for different levels of force of muscle contraction.
contraction, when the experiments are repeated on the same subject, or repeated on different subjects. The null hypothesis is accepted.

5.4 **Summary: Experimental Analysis**

The results are found not to vary significantly with subject variation, force levels or consequent trials following a multiway analysis of variance (MANOVA). These results are in line with the reflex latency behaviour reported in literature as discussed above. The mean value of time constant, 78ms, falls within the range of the 50-80ms value of M2 proposed in literature suggesting that the $T_C$ is a value of the M2. This M2 is suggested to reflect longer reflex loops such as the slower Group II afferents and in several works have been termed the supraspinal reflex loop but thus far there is no conclusive consensus on the origin of the M2. The next chapter will look to the origins of M2 and its relation to the supraspinal feedback and a comparison of the experimental and theoretical results. It will also further discuss the statistical results obtained in this chapter.
The aim of this research is to consider the role of peripheral feedback and muscle mechanics in EMG signal oscillation. In chapter 1, oscillations from the peripheral feedback were proposed to originate from the neural delay of the feedback loop whilst oscillations from muscle mechanics were proposed to be a result of the inertial, viscous and elastic properties of muscle. Considering the above hypothesis, the current research discusses:

- Time constant of rhythmical oscillations found in EMG signals reflect the neural delay of peripheral feedback
- Physiological tremor oscillations are a resultant of the peripheral feedback mechanism

The first section compares the statistical observations of the time constant value to observations of the peripheral neural delay published in literature. Following that, the subsequent section compares the time constant value to the simulated values of individual and resultant peripheral oscillatory mechanisms found in Chapter 4. This chapter concludes
by analysing the mechanisms of physiological tremor and proposes the mechanism generating the $T_C$ value and consequently the oscillatory activity in EMG signal.

### 6.1 Time constant reflect neural delay

Latency is the measure of the conduction time between two stimulus sites. In nerve conduction studies, the nerve conduction velocity (NCV) is obtained from the following equation 51.1 [81]:

$$NCV(m/sec) = \frac{DISTANCE\ BETWEEN\ TWO\ STIMULATION\ SITE(mm)}{LATENCY(ms)} \quad (51.1)$$

Latency is the reflex response time by from when the stimulus artefact is induced to the beginning of the EMG responses [98]. Neural conduction studies which look to the effects of age, sex, anthropometric, fatigue and contraction levels used latency time as a measure of neural delay of the reflex loop. The current hypothesis proposed that the time constant, $T_C$ reflects this neural delay. Following that, the subsequent variables: subject variations (wherein subjects do not suffer neuropathy and are relatively similar in size), repeated trials and different levels of force contractions and will thereafter be compared to the results reported in literature.

#### 6.1.1 Subject variation

Studies into neural conduction commonly reports two latency peak onsets in their recorded EMG after stimulus: the M1 at 20-50ms and the M2 at 50-80ms [89]. The reproducibility of
these values between subjects has been noted by Toft et al [88] who found, M1, M2 and M3 responses in all their subjects with surprisingly small variations. They noted that this observation coincided with Bedingham and Tatton’s experimental observations. In later works, M1 and M2 values were also reported in all subjects and within the expected range.

Age and height are observed to affect the NCV with studies by Rivner et al [99] and Stetson et al [97] reporting NCV to be inversely correlated to age and height. However, Rivner et al noted the effects of height to be more prominent than that of age and concluded that in subjects less than 60 years old, the effects of age were minimal. He also found that while height showed a strong inverse correlation to NCV, it is weakly related to distal latency. As the motor nerve distal latency includes the neuromuscular transmission delay and the propagation delay, he proposed that height had less effect on latency. He also found there is a stronger inverse correlation of height to NCV in the lower extremity than those in the upper extremity. Finally, in their studies, Rivner et al and Stetson et al have indicated that neuromuscular disorder such as carpal tunnel or diabetes is known to affect the values of NCV and latency as such they have excluded subjects with carpal tunnel or known neuropathy from their studies.

Recruitment of subjects has been restricted to subjects between the ages of 20 to 40 years old with no known history of neuropathy or myopathy and is able bodied. EMG signal is taken from the biceps brachii in an effort to minimise the effects of height on neural loop delay. Results indicate the value of time constant, $T_C$ does not vary significantly for subject variation.
6.1.2 Repeated trials

Amplitude, firing rate and frequency properties of surface EMG signals are not dependent on contraction information and muscle properties alone but a variety of other non-muscular factors including electrode distance to the active muscle area, skin impedance, electrode position and fixation on skin, and electrode and amplifier properties. These factors contribute to the variability in EMG records, making interpretation of the results more difficult. A number of methods can be employed to minimise the impact of non-muscular factors on the properties of EMG signal. These methods can be found in Hermens et al.’s [100] published multi-national consensus initiative called SENIAM (surface EMG for the Non-Invasive Assessment of Muscles). Their review largely impressed on the importance of sensor properties, placements, acquisition, signal processing and modelling.

From this, Day [101] suggested that a large number of these non-muscular factors can be reduced by a) using the same electrodes and amplifiers for all experiments and b) ensuring consistency in quality of contact between the electrodes and skin. Additionally, Day stated for experiments performed within subjects, the variability can be reduced with consecutive recording sessions placed at the same skin location.

The experimental procedure maintained the use of the same electrode and acquisition equipment throughout all the experiments. Proper skin preparation is conducted before the start of experimental procedures and SENIAM’s guidelines are utilised during the procedural placement of electrodes to reduce non-muscular variability. The experiment conducts two consecutive recording sessions using the aforementioned precautions. Statistical analysis of
results found no significant variability between the two trials indicating there is no significant non-muscular variability in the surface EMG signal recorded and analysed.

6.1.3 Force level

Studies into reflex latency noted a chance in peak amplitude in the face of fatigue and changing contraction levels. However these changes were noted to have no effect on the latency time. Toft et al [88] studied the EMG response in the human anterior tibial muscle to stretch ranging from 0% to 80% MVC. He found that the peak latency for M1 and M2 to be 57-62ms and 79-93ms respectively with a decrease of about 1.3ms; and M3 with a latency of 110-130ms with a decrease by about 8.5ms. The 1.3ms decline in M1 and M2 latency values were found to be not statistically significant. In addition, Nakazawa and Yamamoto [89] also compared latency values of different types of contractions (isometric, stretching and lengthening) in the following three muscles: barchioradialis, biceps brachii and triceps brachii. They reported the overall largest magnitude change for M1 and M2 during isometric contraction and M3 during shortening activity. However, the latency components of the M1: 20-50ms, M2: 50-80ms and M3: 80-100ms showed no significant change. Finally, Duchateau et al [90] performed two fatiguing isometric contractions at 25% and 50% MVC and one intermittent contraction at 25% MVC (henceforth known as ISO25, ISO50 and INT25 respectively). They reported a latency change for M1 (26.5ms to 27.1ms) and M2 (49.2ms to 49.8ms) for ISO50; and a M1 (27.6ms to 29.8ms) and M2 (49.6ms to 51.3ms) for ISO25; and a M1 (28.2ms to 29.1ms) and M2 (50.4 to 51.4ms) for INT25. The changes in latency time in the face of fatigue and between different forces were not significant.
The current experiment considers the EMG signal from a sustained contraction of 10s or until fatigue of the biceps brachii at four contraction levels of 25%, 50%, 75% and 100%. The statistical analysis reports there is no significant variation of time constant between force levels. Fatigue has not been considered in current experimental setup.

### 6.1.4 Summary: Time constant reflect neural delay

Statistical analysis indicates the results do not vary significantly with subject variation, force levels or consequent trials. These results are in line with the reflex latency behaviour reported in literature as discussed above. The mean value of time constant, 78ms, falls within the range of the 50-80ms value of M2 proposed in literature. The next section will examine the origins of M2 which is believed to represent the long-loop latency response to stimulus and its possible relation to the supraspinal feedback. Following that, the role of peripheral mechanism in the tremor oscillations found in EMG signals is addressed. $T_C$ values will be compared to the simulated tremor oscillations of various peripheral mechanisms, including the supraspinal feedback mechanism.

### 6.2 Origins of M2 in upper limb

Whilst the origins of M1 have always been correlated to the monosynaptic alpha-gamma loop latency, the origins of M2 and M3 still remain a debate [102]. Hammond first noted a second activation, M2 in addition to the monosynaptic (M1) latency in the human biceps brachii following a voluntary stretch response of the muscle [83, 103]. This observation was found in
proceeding works and eventually it became clear that there exist three responses in EMG to perturbation of the limb which are the M1, M2 and less documented M3. The M1 component has been long attributed to the result of alpha-gamma response which forms direct connections between muscle spindle Ia afferents and motoneurons of the same muscle [73] whereas the origins of M2 and M3 components remain inconclusive. Hammond had initially related the M2 to the supraspinal pathways, suggesting that the later appearance of the M2 could be attributed to the longer delay loop. Following that, studies into the stretch reflex latency have put forward various origins to the M2 and of these, the most notable were: 1) M2 response is mediated by slower conducting afferents specifically the Group II afferents of muscle spindle or cutaneous afferents and 2) transmission of Ia afferent input over polysynaptic pathways [104].

These various origins have been the subject of many debates following conflicting results and studies. Marsden et al have proposed the possibility of a mixture of mechanisms producing the long-latency response [102]. In arm movement, the supraspinal loop may contribute to the long latency response as the upper limb such as the hand is predominantly under direct cortical control whereas more automatic movements such as stance and gait would invoke a periphery reflex control (e.g. slower afferents). In experiments involving the upper arm, studies have found that the long-latency response is modified substantially by the subject’s intent (following verbal instructions or repetitive exercise of movement over time) suggesting supraspinal involvement. Furthermore, the value of the long-latency reflex has been shown to be absent in the upper arm EMG of patients suffering from Huntington’s Disease (CNS damage in HD patients are known to be supraspinal nature) suggesting that the long-latency in arm movements involve the supraspinal loop [104].
6.3 Comparison of experimental to theoretical values of oscillation frequency

In neuromuscular modelling for tremor oscillations, the M2 response is often considered to travel the supraspinal loop where it interacts with the central oscillations [105] from the CNS (ref Figure 2). This interaction can be seen in the neuromuscular model above (from Figure 18 of Chapter 4) where the neural signal from the CNS is summed with the supraspinal feedback at the central motoneuron. In the model, supraspinal feedback is represented by a longer time delay. Consequently, the $T_c$ values obtained can now be compared to the supraspinal feedback mechanism simulated from Chapter 4 and in turn compare the individual and resulting mechanisms of oscillations found in Chapter 4 to $T_c$.

The simulated frequency of oscillations from individual and interacting mechanisms can be found from the dominant roots noted in section 4.2.1, 4.2.2, 4.2.3, 4.3.1 and 4.3.2 in Chapter
4. The values have been compiled into the following Table 13. The resulting range of simulated frequencies in Table 13 were obtained by using the lowest and highest latency values found in Table 6, Chapter 4.

**Table 13 Frequency oscillation of individual and interaction of peripheral mechanism between the lowest average and highest average values in Table 6**

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Frequency Range (Hz)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle</td>
<td>4.53</td>
</tr>
<tr>
<td>Spinal</td>
<td>50.56</td>
</tr>
<tr>
<td></td>
<td>101.11</td>
</tr>
<tr>
<td>Supraspinal</td>
<td>11.9</td>
</tr>
<tr>
<td></td>
<td>13.8</td>
</tr>
<tr>
<td>Mechanical-reflex (MR)</td>
<td>4.25</td>
</tr>
<tr>
<td></td>
<td>4.44</td>
</tr>
<tr>
<td>MR+Supraspinal</td>
<td>4.25</td>
</tr>
<tr>
<td></td>
<td>4.34</td>
</tr>
</tbody>
</table>

**Figure 34 Plot of frequency of oscillation found experimentally and theoretically for peripheral mechanisms**

Figure 33 shows the comparison of the experimental value of $T_C$ to the theoretical values simulated in Chapter 4. The frequency representation of $T_C$ is obtained from the inverse of the time constant based on the hypothesis: neural delay is inversely proportional to frequency...
of oscillation [14] proposed in literature. Table 14 shows the mean absolute error of the simulated values and experimental values plotted in Figure 34.

Table 14 Mean absolute error of simulated values to experimental values for the individual and interacting mechanisms of oscillation

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Mean Absolute Error</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle</td>
<td>8.27</td>
</tr>
<tr>
<td>Spinal</td>
<td>67.91</td>
</tr>
<tr>
<td>Supraspinal</td>
<td>0.95</td>
</tr>
<tr>
<td>Mechanical-Reflex (MR)</td>
<td>8.46</td>
</tr>
<tr>
<td>MR+Supraspinal</td>
<td>8.51</td>
</tr>
</tbody>
</table>

It can be seen that the supraspinal mechanism shows the lowest mean absolute error whilst the spinal mechanism shows the highest mean absolute error. As the supraspinal mechanism has the smallest error between the simulated values and its real values, this suggests that the low frequency oscillation found in this experiment originates from the supraspinal mechanism.

This observation is in line with the previous statement where $T_C$ represents supraspinal loop delay.

In the experimental procedures, subjects maintained an isometric contraction. Muscle length is held constant, suggesting a lack of involvement from the spinal stretch reflex (which modulates the length of muscle) and the muscle mechanics. Consequently, the resulting $T_C$ value obtain from the EMG signal of the muscle in isometric contraction showed a prominent oscillation that is strongly correlated to the supraspinal feedback delay. Comparison to theoretically obtained values of oscillation showed a strong correlation to the supraspinal feedback mechanism devoid of mechanical-reflex influences. The 12.8Hz $T_C$ value is believed to correlate to the 8-12Hz component in physiological tremor.
6.4 Mechanisms of physiological tremor

In the study of physiological tremor, research have commonly found, existing 8-12Hz oscillatory component in the EMG signal of healthy subjects [24, 28, 106]. In 1990, Elble conducted a review on tremor and concluded that the physiological tremor has two distinct oscillations, the 8-12Hz and the mechanical-reflex oscillation [107].

6.4.1 Mechanical-reflex

The mechanical-reflex oscillation originates from the interaction of spinal feedback and the mechanical properties of muscle. Under certain circumstances, the frequency of the muscle mechanics and the spinal feedback are similar, causing the two frequencies to entrain into a single frequency that predominantly behaves like a mechanical system [14]. For example, inertial loading has been found to lower the frequency of mechanical-reflex oscillation whilst a spring loading has been found to increase the frequency [20, 38]. Experimental and theoretical results by Takanokura et al [37] has shown the mechanical-reflex oscillation varies with mass loading or increased displacement amplitude [20]. Takanokura et al studied the effects of inertial loading by measuring the EMG signal of a mass held in a position against gravity using four different upper limb segments. The same inertial loading was applied to his neuromuscular model by varying the input of mass in accordance to his various weight of upper limb. His theoretical results showed that found that the frequency of oscillations of spinal origin to decrease whilst the frequency of supraspinal origin to remain
unchanged at the range of 8-12Hz when varying the mass parameter of the model. A further comparison of this theoretical results to his experimental values were found to be agreeable leading to the conclusion the supraspinal feedback is responsible for the 8-12Hz oscillation and the spinal component is responsible for the mechanical-reflex. The frequency of the spinal component is dependent on the mass of the limb.

Theoretical results in Figure 33 shows the frequency of mechanical-reflex and mechanical-reflex+supraspinale to reflect the frequency of the muscle mechanics. Based on the above observations and previous observations found in literature, it is concluded the mechanical-reflex oscillation is generated from the interaction of the spinal feedback and the muscle mechanics. The two oscillations are entrained and the resulting oscillation is dominated by the muscle mechanics causing the oscillatory component to vary according to mass of loading or amplitude of displacement. This observation can be used to account for the conflicting oscillation frequencies and arguments and counter-arguments regarding oscillation origins often found in literature.

### 6.4.2 8-12Hz oscillation

The unchanging frequency of the 8-12Hz oscillation in the face of inertial or spring loading has also been observed in various literature [32, 37, 106]. In Elble’s [107]1990 review, he noted the consistency of this observation in several literature along with his own observation that tremor is independent of stretch reflex loop time and muscle twitch properties leading to postulate that the 8-12Hz oscillation emerges from networks within the central nervous system [29]. However, in the light of new information, Elble’s most recent publication of physiologic tremor stated that there was increasing evidence that the 8-12Hz component
arises from the supraspinal transcorital pathways [108]. One of these new works is Koster’s experimental studies into subjects with persistent mirror movements (PMM). Koster [109] hypothesised that if a supraspinal mechanism is involved in physiological tremor, this tremor activity should show left-right-coherence for subjects with PMM syndrome. He found that while there is no significant right-left-coherence of tremor activity in control subjects, subjects with PMM syndrome displayed a significant right-left-coherence between the frequency 8 to 12Hz. He also found mechanical loading did not affect the 8-12Hz on both sides. Consequently, he postulated that the 8-12Hz component of physiological tremor is transmitted from the supraspinal.

Comparison of the experimental $T_C$ value to the theoretical frequencies of oscillation show a correlation to the supraspinal component which is believed to be responsible for the 8-12Hz component. This is in line with the experimental procedure conducted in the current research. Isometric contraction causes the muscle to be held in constant length which eliminates the mechanical-reflex oscillation leaving only the 8-12Hz oscillatory component to be present in the EMG signal. This 8-12Hz component has been attributed to the central oscillators and supraspinal feedback however it is unclear if the oscillation is a product of a dominant oscillation from one of the two or a result of the interaction between them.
This research has examined the role of neuromuscular activity and physiological properties in EMG signal particularly the contributions of the neuromuscular system in the rhythmical grouping found in EMG signals. This rhythmical grouping is believed to represent tremor oscillations and in healthy subjects this is known as physiological tremor.

The observed experimental time between waveform envelopes in EMG signal, $T_C$ is found to be 78ms. This value reflects the long-loop neural delay, $M2$ which is reported to be between the ranges of 50ms to 80ms. Both $T_C$ and $M2$ values found in literature show no significant variation in a) subject variation b) repeated trials c) contraction force level. Literature shows $M2$ represents the supraspinal loop.

The peripheral oscillatory mechanisms observed in tremor oscillation are: a) mechanical resonance of muscle b) spinal feedback c) supraspinal feedback. Its respective frequency
oscillations generated from the neuromuscular model are: Muscle: 4.53Hz; Spinal: 50.56-101.11Hz; Supraspinal: 11.9-13.8Hz. The interaction of these individual mechanisms generates two resulting frequency oscillations: Mechanical-reflex: 4.25-4.44Hz. Mechanical-reflex + Supraspinal: 4.25-4.34Hz.

The simulation of the interaction between muscle mechanics and spinal reflex (mechanical-reflex) and between mechanical-reflex+supraspinal showed a dominant oscillation originating from the mechanical resonance of muscle. This observation coincides with previous observations found experimentally in literature. This research concludes the lower sensory loop oscillation of spinal feedback is dominated by the mechanical oscillation of muscle. This mechanical-reflex oscillation is one of the two main oscillations found in physiological tremor. Its values vary according to mass of loading and amplitude of displacement. It is postulated the conflicting results in literature on the origins of tremor can be attributed to varying frequency.

The range of the supraspinal feedback simulated from the neuromuscular, 11.9Hz-13.8Hz showed the closest correlation to the frequency representation of $T_C$, 12.8Hz. This research concludes the $T_C$ represents the neural delay of the supraspinal loop. This frequency oscillation is concluded to relate to the common 8-12Hz tremor oscillation found in EMG signals. This is in line with recent literature which suggests the involvement of the supraspinal feedback loop in the generation of the 8-12Hz. However it remains inconclusive if the 8-12Hz is a result of a prominent oscillation either from the supraspinal CNS commands or the supraspinal feedback or a resulting oscillation from the interaction of both.
7.1 Recommendations for further research

Literature on neural conduction and latency has shown that neural conduction varies with height, age (> 60) and patients with neuropathy or myopathy. A large number of researches are dedicated to studying the effects of neuromuscular disorder on the EMG signal between healthy subjects and subjects with neuropathy or myopathy. Study into the $T_C$ of the EMG signal for subjects of various height, a higher age range and subjects suffering from neuromuscular disorder can further analyse the role of peripheral feedback in EMG signals.

7.1.1 Height

Neural conduction velocity studies suggest an inverse relationship between height and NCV. For example, Hennessey WJ et al [110] and Stetson DS et al [97] have positively associated height to sensory latencies whilst A. Nardone et al noted that latency increases with a subject’s height in all responses tested. However, Wagman IH et al [111], Soudman R et al [112] and a few others did not find a conclusive correlation between height and the neural conduction velocity of the ulnar nerve of the wrist. This discrepancy can be attributed to the variations in nerves studied and the differing experimental technique. The application of $T_C$ to EMG data can be used as an alternative method to study the correlation between height and NCV. $T_C$ of EMG data from the tibialis anterior which is located at the lower half of the body can provide a measure of nerve latency. Consequently, nerve latency of subjects of differing heights can be used to study the relationship of height to NCV.

7.1.2 Age
Muscle strength has been known to decline with aging. This is attributed to the changes in neuromuscular properties which are responsible for the production of the maximal voluntary contraction in a muscle. These factors and the subsequent affects of aging on them have been comprehensively reviewed by Clark et al [113]. In his review, he had found that each of the elements in the pathway used by the CNS to elicit a muscular contraction is affected by aging. These elements include a decrease in cortical excitability, decrease in alpha-motoneuron excitability, increase in antagonistic muscle coactivity, decrease in motor firing rate, altered neuromuscular transmission, decrease in muscle mass, the excitation-contraction uncoupling process and altered muscle architecture. The current experimental results of $T_C$ can be applied to isolate and study the role of the neuromuscular transmission delay in muscle strength and aging. Furthermore, the theoretical model in Chapter 3 can be expanded to study the changes of the neuromuscular properties involved in the production of muscle contraction for aged subjects.

### 7.1.3 Neuropathy or myopathy

The study of age-related muscle weakness in plantar- and dorsi- flexor muscles is important for understanding the risk of fall in the aged [114]. It has been largely debated whether the weakness in lower limbs are associated with loss in muscle mass or loss of muscle strength [115]. Though the loss in muscle strength has been largely believed to attributed to the loss in muscle mass, it has been found that muscle strength declines at a greater rate than muscle mass [116]. This indicates that remaining neuromuscular properties mentioned in Section 7.1.1 play a greater role in the weakness of lower limbs. The understanding of the contributions of neural and muscular factors to age related risk of falling can be used to develop better preventative measures against strength decline with age. The current model
can be expanded to include studies on the gastrocnemius, soleus and tibialis anterior muscles and its age-related changes.
References


Appendix

A.1 Step response plot for individual peripheral oscillatory mechanism

The step response of the individual peripheral mechanisms noted in Chapter 4 are plotted to give a diagrammatic view of the response of each system.

Figure A 1 Step response of the mechanical muscle system
Figure A 2 Step response of the spinal feedback system

Figure A 3 Step response of the supraspinal feedback system
A.2 Step response plot of the interaction between peripheral oscillatory mechanism

The step response of the interaction between the peripheral mechanisms is plotted to give a diagrammatic view of the dominant oscillatory mechanism. Both mechanical reflex and the interaction between mechanical reflex and supraspinal feedback shows dominant oscillation from the muscle mechanics.

Figure A 4 Step response of the mechanical reflex system
Figure A 5 Step response of the interaction between the mechanical reflex system and the supraspinal feedback system