INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

Bell & Howell Information and Learning
300 North Zeab Road, Ann Arbor, MI 48106-1346 USA
800-521-0800

UMI®
The Effect of Proprioceptive Neuromuscular Facilitation With Warm-Up on Ankle Joint Dynamics

Christopher Barrett

Department of Biomedical Engineering
McGill University
Montréal, Canada

June, 1998

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements of the degree of Master of Engineering.

© Christopher Barrett, 1998
The author has granted a non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of this thesis in microform, paper or electronic formats.

L’auteur a accordé une licence non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de cette thèse sous la forme de microfiche/film, de reproduction sur papier ou sur format électronique.

The author retains ownership of the copyright in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author’s permission.

L’auteur conserve la propriété du droit d’auteur qui protège cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

0-612-50590-1
Abstract

System identification techniques were used to obtain a parametric description of the mechanical stiffness of the ankle, which was then used to assess the effect of four weeks of stretching the ankle flexors and extensors. The ankle musculo-articular system was described in terms of elastic stiffness, viscosity and inertia. This description of the mechanical behaviour of the ankle joint provided an objective basis for evaluating the effects of a long-term stretching program that utilized proprioceptive neuromuscular facilitation (PNF). PNF, preceded by a warm-up, was performed daily on the soleus, tibialis anterior and gastrocnemius of six healthy subjects, for four weeks.

The question that we asked in this study was: "Does stretching have an effect on the dynamic stiffness of the ankle joint?" A significant increase in ankle range of motion (p<0.02) was observed. However, the intrinsic stiffness of the ankle joint did not change. We concluded that the gains in joint range of motion achieved by long-term stretching were due to an increase in the subjects' tolerance to stretching and not to changes in the dynamic stiffness of the joint.
Résumé

Des techniques d’identification de système ont été utilisées afin d’obtenir une description paramétrique de la raideur mécanique de la cheville. Cette description a ensuite été utilisée pour étudier les effets d’un programme d’étirement des fléchisseurs et extenseurs de la cheville, qui a duré pendant une période de quatre semaines. Ce système musculo-articulaire a été décrit en fonction de la raideur élastique, de la viscosité et de l’inertie. Cette description du comportement mécanique de la cheville donnait une base objective pour l’évaluation des effets d’un programme d’étirement basé sur la facilitation proprioceptive neuromusculaire (PNF) à long terme. Tous les jours, pendant quatre semaines, après un réchauffement, des étirements PNF ont été faits sur le soleus, le tibialis antérieur et le gastrocnémius de six sujets en bonne santé.

La question à répondre était: “Est-ce que les étirements ont un effet sur la raideur mécanique de la cheville?” Une augmentation significative (p<0.02) a été observée dans l’amplitude de mouvement. Cependant, il n’y a pas eu de changement dans la raideur intrinsèque de l’articulation de la cheville. Nous concluons donc que l’augmentation dans l’amplitude de mouvement du joint de la cheville est attribuable à une augmentation de la tolérance des sujets aux étirements et non à des changements dans la raideur dynamique du joint.
Acknowledgements

There are many individuals and groups of people to whom I would like to express my appreciation, for their help in the completion of this project. First of all I would like to thank my supervisor, Dr. Robert Kearney, for challenging me to ask good questions and to be thorough in trying to answer them. I am grateful to my lab companions, Andrzej Kozakiewicz, Mireille Lortie, Mehdi Mirbagheri, Sunil Kukreja and Luckshman Parameswaran, for their help throughout the experimentation, data analysis and writing stages of this thesis.

The subjects who participated in my experiments (the “Super Stretchers of ‘97”) responded above my expectations – thanks. Thank you, too, to Dr. Joyce Fung and Dr. Diane St. Pierre who provided valuable feedback on my initial proposal. As well, thanks to Shannon Larkins, from the McGill Sports Medicine Clinic, for her advice on the structure of the PNF protocol used in this study. I would also like to thank Dr. Michael Slawnych for being a helpful resource with respect to physiology-related concerns.

I appreciate the help of NSERC and the MRC, in providing the financial resources for this project, and of Pina Sorrini, in things administrative.

Finally I’m very thankful for the help of my wife, Heather, who throughout this project encouraged me in ways seen and unseen. Not only were we able to birth this thesis over last several months but our beautiful daughter, Emma, as well.
## Table Of Contents

1 Introduction .................................................................................................................. 1  
2 Background ................................................................................................................. 3  
   2.1 Stretching .............................................................................................................. 3  
      2.1.1 What is stretching? ....................................................................................... 3  
      2.1.2 Why and where is stretching used? ............................................................... 4  
      2.1.3 What are the different types of stretching? .................................................... 4  
      2.1.4 The History of Proprioceptive Neuromuscular Facilitation (PNF) .............. 5  
2.2 The Physiology of Stretching ................................................................................... 5  
   2.2.1 Connective Tissue ............................................................................................ 6  
   2.2.2 Muscle Tissue .................................................................................................. 7  
   2.2.3 Neurophysiology ............................................................................................. 9  
      Muscle Proprioceptors ......................................................................................... 10  
      The Stretch Reflex ............................................................................................... 13  
      Reciprocal Innervation .......................................................................................... 13  
      The Inverse Stretch Reflex .................................................................................. 14  
2.3 Stretching Strategies: Effectiveness and Comparisons ............................................ 15  
   2.3.1 The Effects of Stretching ................................................................................. 15  
   2.3.2 Comparison of Stretching Techniques ............................................................ 16  
   2.3.3 Contract Relax Antagonist-Contract (CRAC) ................................................ 17  
2.4 Warm-Up ................................................................................................................ 17  
2.5 Rationale ................................................................................................................ 18  
2.6 System Identification of Joint Dynamics: Quasi-Linear Model .............................. 19  
   2.6.1 System Identification ..................................................................................... 19  
   2.6.2 Quasi-Linear Models of Joint Dynamics ......................................................... 20  
   2.6.3 Previous Work on System Identification of Joint Dynamics .......................... 20  
2.7 The Ankle Joint ...................................................................................................... 21  
3 Methods ..................................................................................................................... 25  
   3.1 Apparatus ........................................................................................................... 25
3.1.1 Electro-Hydraulic Actuator and Control Strategy ......................................................... 26
3.1.2 Rigid Limb Fixation and Ankle Axis of Rotation ......................................................... 27
3.1.3 Signal Transduction and Processing ............................................................................. 27
3.2 Experimental Methods ...................................................................................................... 29
3.2.1 Experimental Schedule ................................................................................................. 29
3.2.2 Subjects ........................................................................................................................ 29
3.2.3 Testing Protocol ............................................................................................................ 30
3.2.4 Training Protocol ......................................................................................................... 33
  Warm-Up ............................................................................................................................... 33
  Stretching Protocol .............................................................................................................. 33
  Self-Stretching Protocol ....................................................................................................... 37
3.3 Analytical Methods .......................................................................................................... 37
3.3.1 Non-Parametric Model ............................................................................................... 37
  Parallel Cascade Model .......................................................................................................... 38
3.3.2 Parametric Model .......................................................................................................... 39
3.3.3 Assessing Changes in Intrinsic Stiffness .................................................................... 40
3.3.4 Statistical Analysis ........................................................................................................ 41
4 Results ................................................................................................................................. 42
4.1 Introduction ....................................................................................................................... 42
4.2 Pilot Studies ....................................................................................................................... 42
  4.2.1 Pilot Study No.1: The Effects of a Single PNF Session Without Warm-Up on Ankle Joint Dynamics ............................................................... 42
  4.2.2 Pilot Study No.2: The Effects of a Single PNF Session With Warm-Up on Ankle Joint Dynamics ............................................................... 44
4.3 Long-Term PNF Study ..................................................................................................... 46
  4.3.1 Subjective Changes ...................................................................................................... 46
  4.3.2 Range of Motion ......................................................................................................... 47
  4.3.3 Passive Tension .......................................................................................................... 48
  4.3.4 Non-Parametric and Parametric Fits ....................................................................... 49
  4.3.5 Dynamic Stiffness Parameters .................................................................................. 50
  4.3.6 Passive Elastic Stiffness: $K_{min}$ and $\theta_{low}$ .......................................................... 53
5 Discussion .................................................................................................................. 56

5.1 Summary of Study ................................................................................................. 56

5.2 Feasibility and Sensitivity of System Identification Approach .......................... 56

5.3 Summary of Results .............................................................................................. 57

5.4 Possible Explanations of Results ......................................................................... 57

5.4.1 Avoidance of Extreme Positions ..................................................................... 58

5.4.2 Changes in Connective Tissue ......................................................................... 59

5.4.3 Endo- and Exo-Sarcomere Skeleton ............................................................... 59

5.4.4 Sarcomere Remodeling .................................................................................... 60

5.4.5 Perception and Proprioception – Increased Tolerance to Stretching ............. 61

5.5 Significance of Results .......................................................................................... 63

5.5.1 The Effects of Stretching ................................................................................ 63

5.5.2 Flexibility and Stiffness ................................................................................... 64

5.5.3 Perceived Stiffness Versus Mechanical Stiffness ........................................... 64

5.6 Potential Implications ............................................................................................ 65

5.6.1 Stretching and Injury Prevention .................................................................... 65

5.6.2 Stretching and Performance ........................................................................... 66

5.6.3 Experimental Design and Interpretation of Results ........................................ 66

5.7 Recommendations for Future Work ..................................................................... 67

5.7.1 Looking Into ROM Extremes ......................................................................... 67

5.7.2 Ramp in Pilot Study ........................................................................................ 67

5.7.3 Effect of Stretching on Reflexes ..................................................................... 67

5.7.4 Stretching, Lower Leg Pathologies and Ankle Joint Dynamics ...................... 68

5.7.5 Strengthening and Ankle Joint Dynamics ....................................................... 68

6 Conclusions ............................................................................................................... 69

7 Appendix – Analytical Tools ...................................................................................... 70

Impulse Response Functions ..................................................................................... 70

Chebyshev Polynomials ............................................................................................ 70

Mean and Variance .................................................................................................... 71

Variance Accounted For (VAF) ................................................................................. 71
## Table of Figures

**Figure 2.1** Stretching the calf muscle (triceps surae). (from Alter [13]) .................................................. 3

**Figure 2.2** Connective tissue of a muscle. (a) Entire muscle, with the belly sectioned. (b) Enlargement of a cross section of the belly. (from Alter [13]) ................................................................. 6

**Figure 2.3** Organization of skeletal muscle tissue from the gross to the molecular level. (from Alter [18]) .................................................................................................................. 7

**Figure 2.4** Titin and nebulin. (from Keller [30]) ................................................................................................. 8

**Figure 2.5** Muscle spindles and Golgi tendon organs. (from Kandel [39]) .......................................................... 10

**Figure 2.6** Muscle spindles and Golgi tendon organs. Mammalian muscle proprioceptors and their reflex pathways. (from McMahon [40]) .......................................................... 11

**Figure 2.7** Comparison of the sensitivity to sinusoidal stretching within the linear range of a primary and secondary muscle spindle ending at various frequencies. The continuous lines represent the vector sums of a length component (horizontal segment) and a velocity component (diagonal segment). (from Stein [35]) .................................................................................. 12

**Figure 2.8** The stretch reflex. (from Kandel [39]) ............................................................................................... 13

**Figure 2.9** The neural connections that facilitate the inverse stretch reflex. (from Kandel [39]) .. 14

**Figure 2.10** The ankle joint: bones and muscles. (from Yunan [88]) ................................................................. 22

**Figure 2.11** Movement of the ankle in the sagittal plane: dorsiflexion and plantarflexion. (from Parameswaran [90]) ........................................................................................................... 23

**Figure 2.12** A more detailed view of the ankle joint. (from Moore [89]) .............................................................. 24

**Figure 3.1** Experimental set-up. (drawing created by Robert Kirsch) ................................................................. 25

**Figure 3.2** The booted foot of a subject attached to the actuator assembly. ......................................................... 26

**Figure 3.3** Angular position ramp input, with subject relaxed, to evaluate passive tension in the ankle. Angular position, torque and TA and GS EMGs, are shown. (Subject PG) Note: plantarflexion torques and EMG are negative by lab convention .......................................................... 31

**Figure 3.4** Five seconds of data recorded from the four channels during a PRBS of angular position input trial. .......................................................................................................................... 32

**Figure 3.5** Applying the CRAC PNF technique to the soleus. ............................................................................. 34

**Figure 3.6** Applying CRAC PNF to the tibialis anterior. .................................................................................... 35
Figure 3.7 Applying the CRAC PNF technique to the gastrocnemius.......................................................... 36
Figure 3.8 The parallel cascade model...................................................................................................... 38
Figure 3.9 Compliance impulse response functions: non-parametric and best fit 2nd order IRFs.
Taken from Subject MD, Test #4. ............................................................................................................. 40
Figure 3.10 Assessment of elastic stiffness. Parametric model data and 3rd order Chebyshev fit.
Taken from Subject MD. .......................................................................................................................... 41
Figure 4.1 Dynamic stiffness parameters vs. mean angular position before and after a single PNF session without warm-up: passive conditions, Subject HB relaxed. ......................................................... 43
Figure 4.2 Dynamic stiffness parameters vs. mean angular position before and after a single PNF session without warm-up: active conditions, Subject HB contracting ankle extensors at 5% MVC.
.............................................................................................................................................................. 44
Figure 4.3 Dynamic stiffness parameters vs. mean angular position before and after a single PNF session with warm-up: passive conditions, Subject HB relaxed................................................................. 45
Figure 4.4 Dynamic stiffness parameters vs. mean angular position before and after a single PNF session with warm-up: active conditions, Subject HB contracting ankle extensors at 5% MVC. ......................... 45
Figure 4.5 Range of motion of three subjects plotted over the six weeks. ................................................. 47
Figure 4.6 Superimposed torque records from ramp input for Subject PG................................................ 48
Figure 4.7 Torque range from passive tension test .................................................................................... 49
Figure 4.8 Quality of non-parametric and parametric fits. Taken from Test #3 of Subject MD. ................. 50
Figure 4.9 Dynamic stiffness parameters: passive conditions, Subject PG relaxed. ................................ 51
Figure 4.10 Dynamic stiffness parameters: active conditions, 5% MVC of ankle extensors; Subject PG. .................................................................................................................................................. 52
Figure 4.11 Minimum elastic stiffness, $K_{min}$. .......................................................................................... 53
Figure 4.12 Spread of K curves, $\theta_{low}$. ................................................................................................. 54
Table of Tables

Table 3.1 The experimental timeline................................................................................................................. 29
Table 3.2 Physical characteristics of the subjects. ................................................................................................. 30
Table 4.1 Subjective changes noted by subjects. .................................................................................................... 46
Table 4.2 Results of two-tailed Student’s t Tests comparing pre- and post stretching program (i.e. between Control #2 and Test #4) parameters. ........................................................................................................ 55
1 INTRODUCTION

Stretching following warm-up, prior to strenuous exercise, is endorsed widely by athletic therapists, trainers, coaches and athletes as a means of injury prevention and performance optimization [1-4]. Stretching techniques are also an integral part of many physiotherapeutic and rehabilitation programs [5-7]. Stretching is thought to increase the range of motion (ROM) of a joint, to reduce joint stiffness and even to reduce muscle soreness [1, 8, 9]. Warming-up is believed to enhance the effects of stretching the muscle-tendon unit by increasing blood flow through the active muscles and increasing body and tissue temperatures [10].

The effects of stretching on muscle and joint properties, however, are not well understood [1, 8]. Some feel that stretching has a significant effect while others feel that the effect is only transient or non-existent. Most strategies that are used to evaluate the effects of stretching involve goniometry, that is, determining the ROM of the joint. This limits the investigation of the effects of stretching on tendon and muscle to the extreme positions of the joint. Furthermore, this approach is somewhat subjective in nature; it depends on how the subject feels at that particular time and the degree of discomfort he or she is willing to endure.

What if one were able to determine the mechanical properties of a joint throughout its ROM in an objective, quantitative manner? Would assessing changes in those properties not shed more light on how stretching influences joint function? This would be particularly instructive since the functional range of movement of a joint generally excludes the extreme positions. In this study just such an approach was taken. We sought to answer the question: “Does stretching have an effect on ankle joint dynamics?” In order to answer this question subjects undertook a four week long stretching program that was designed to yield optimal results (i.e. a maximal increase in flexibility and a maximal decrease in joint stiffness). The effects of the stretching were evaluated in terms of the mechanical behaviour of the ankle joint. Ankle joint dynamics were determined by using established system identification techniques [11].
To put this study into an appropriate framework, relevant background information concerning stretching and warm-up techniques, the rationale for the study, system identification of ankle joint dynamics and ankle joint anatomy is presented in Chapter 2. Chapter 3 describes the apparatus and outlines the experimental and analytical methods that were used. The results from two pilot studies and from the six weeks of experiments performed on each of six subjects are documented in Chapter 4. Several possible explanations for the results and the significance of these findings, as well as recommendations for future investigation and research, are addressed in Chapter 5. Chapter 6 contains the overall conclusions drawn from this work.
2 BACKGROUND

2.1 Stretching

2.1.1 What is stretching?

Collins defines the verb to stretch: “to extend or be extended to an undue degree, esp. so as to distort or lengthen permanently” [12]. Stretching is the exercise of contorting one’s limbs in such a fashion so as to place a particular muscle or group of muscles in the body under tension. This may be done on one’s own or with the help of a wall, bench or another person. Figure 2.1 below illustrates a very familiar stretch to athletes and recreational exercisers. Since muscles and their tendon attachments serve to move the joints across which they attach, it is thought that lengthening a muscle-tendon unit could affect the behaviour of a joint.

Figure 2.1 Stretching the calf muscle (triceps surae). (from Alter [13])
2.1.2 Why and where is stretching used?

Flexibility is specific to each joint in the body and is understood to be the possible range of motion or movement of a particular joint or group of joints [2, 3]. The ancient Greeks incorporated flexibility into three types of gymnastic activities: (i) medical, made up of prophylactic (preventing disease and maintaining health) and therapeutic (curing disease) exercises, (ii) military and (iii) athletic [13]. Today stretching exercises can still be encountered in those spheres.

Stretching is used because it is thought to increase the flexibility of a joint and to reduce joint stiffness. These two factors are believed to optimize performance and prevent injury in all types of sports and manual labour contexts. Stretching is also held to be crucial in neuromuscular rehabilitation and physiotherapy practices [5, 6, 14]. Furthermore stretching is regarded by many to promote spiritual well-being, relieve stress, provide muscular relaxation and relieve lower back pain as well as muscular cramps [13].

2.1.3 What are the different types of stretching?

Several stretching strategies have been implemented over time. The most common stretching approaches are ballistic, static, passive, active and proprioceptive neuromuscular facilitation (PNF). All of these techniques have been shown to be effective for increasing ROM, presumably by increasing the resting length of muscles and possibly connective tissue around the joint [8].

- **Ballistic stretching**, also known as bounce or rebound stretching, is probably the oldest technique. It uses bouncy or jerky movements to increase flexibility. It has virtually been abandoned due to its potential for causing acute injuries when the force generated during a jerk is greater than that which the muscle-tendon unit can withstand. [1-3, 14]

- **Static stretching** is also known as hold or slow stretching. With this approach a muscle is slowly stretched to the point of discomfort, without hurting the muscle, and held there for an extended period (anywhere from 3 to 60 s has been recommended; 3,4 repetitions of 30 s is most frequently suggested). This is probably the most popular form of stretching. [1-3, 15-17]

- **Passive stretching** requires the use of an external force, usually another person. Additional pressure is applied to ballistic or, more commonly, static stretching exercises. [3]
• **Active stretching** is achieved by an individual contracting their own muscles without any help from an external force. Often it is used in combination with static stretching. [18]

• **Proprioceptive Neuromuscular Facilitation (PNF)** is, comparatively, a more recent approach involving alternating contractions and stretching. The subject moves the muscle into a stretched position and then, with the aid of another person, isometrically contracts the muscle for 5 to 10 s. The subject relaxes and then goes into a static stretch position either on his own or passively with the force exerted by another person. [1-3, 7, 19]

### 2.1.4 The History of Proprioceptive Neuromuscular Facilitation (PNF)

PNF was developed in the late 1940’s and early 1950’s by Herman Kabat, an American medical doctor, with help from physiotherapists Margaret Knott and Dorothy Voss [7, 19, 20]. Initially PNF was used mainly to treat and rehabilitate polio patients suffering from neuromuscular paralysis [7, 19, 20]. More recently it has been used for increasing flexibility [2]. In a general sense, techniques of proprioceptive neuromuscular facilitation may be defined as “methods of promoting or hastening the response of the neuromuscular mechanism through stimulation of proprioceptors” [7].

PNF is a form of stretching that uses an isometric contraction prior to the stretch to achieve greater gains than from stretching alone. It has been shown to increase joint ROM [8]. PNF, as its name implies, is a stretching technique based on the behaviour of muscle proprioceptors, specifically muscle spindles and Golgi tendon organs. By exploiting the operating characteristics of these muscle sensors, PNF is believed to be able to bring the muscle to a greater point of inhibition, thereby allowing it to be stretched more fully.

### 2.2 The Physiology of Stretching

Aside from the voluntary contraction of a muscle, the three main mechanisms that offer resistance to joint movement are: (i) connective tissue, (ii) muscle tissue and (iii) the reflex connections to the muscle [1, 21, 22].
2.2.1 Connective Tissue

Connective tissue forms the matrix that holds muscles and bones together. Among the various types of connective tissue are tendons (which fasten muscle to bone), ligaments (which fasten bone to bone), joint capsules and fascia (all fibrous connective structures not otherwise classified) [13, 22]. Connective tissue is made up of various densities and spatial arrangements of collagen fibers embedded in a protein-polysaccharide matrix, commonly referred to as ground substance. Collagen, the most abundant protein in the body and the primary structural component of living tissue, is a fibrous protein with great tensile strength that behaves in a visco-elastic manner under tensile stress [22-24].

Figure 2.2 shows tendons and fascial sheathing, encasing the muscle fibres, both comprised of collagen. Some believe that the various forms of connective tissue offer the greatest sources of passive resistance at the normal extremes of joint motion [13, 21, 22] and that they deform to allow the muscle-tendon unit to lengthen [9]. Tendon has been shown to play a greater role in restraining joint movement at the extremes [21]. Changes in connective tissue due to stretching are thought by many to be strictly transient and to arise from the acute visco-elastic accommodation of the collagen fibres [16, 25-28].

Figure 2.2 Connective tissue of a muscle. (a) Entire muscle, with the belly sectioned. (b) Enlargement of a cross section of the belly. (from Alter [13])
2.2.2 Muscle Tissue

Muscles are made up of complex biological machinery. Figure 2.3 illustrates the organization of skeletal muscle tissue. A whole muscle (A) is made up of many muscle fasciculae (B), bound together by connective tissue, which are in turn made up of muscle fibres (C). The striated patterns seen in skeletal muscle arise from the thick myosin (L) and thin actin (K) filaments that are organized into cylindrical bundles known as myofibrils (D). One unit of the repeating pattern in a

Figure 2.3 Organization of skeletal muscle tissue from the gross to the molecular level. (from Alter [18])
myofibril is known as a sarcomere. Projections from the myosin molecules (M) called crossbridges, extend from the surface of the thick filaments to the thin filaments. These crossbridges "cycle" to create the force generating mechanism in a muscular contraction [29]. Figures 2.3 and 2.4 show the characteristic lines and bands within a given sarcomere. The thin actin filaments, along with a large protein, nebulin, attach directly to the Z lines. The myosin filaments are linked in the centre of the sarcomere at the M line (H in Figure 2.3) and to the Z line by another protein of even larger molecular weight, called titin [30].

![Figure 2.4 Titin and nebulin. (from Keller [30])]  

As Figure 2.4 shows, titin (also known as connectin) molecules span half of each muscle sarcomere from the M-line to the Z-disc, centering the thick filaments during contraction and generating passive tension through elastic extension when sarcomeres are stretched [30, 31]. Within the A-band segment, titin is bound along the thick filament and so is stiff under physiologic conditions, whereas the I-band segment is compliant [31]. When sarcomeres are stretched, titin behaves as a dual stage spring with two distinct degrees of reversible extensibility (a passive tension stage followed by a structural fail-safe stage that prevents the destruction of the sarcomere) [30, 32]. Nebulin molecules interact closely with the thin filaments, extending with them as they are stretched and provide a molecular "ruler" for the regulation of thin filament length [30].

Like most biological materials, muscles exhibit visco-elastic behaviour; a combination of elastic (force $\propto$ amount of stretch) and viscous (force $\propto$ rate of stretch) properties, so that when stretched
the response of the muscle tissue is time and history dependent [13, 14, 16, 33, 34]. Some purport that this behaviour is due largely to the extracellular connective tissue. Previously it was believed that this response to stretching passive muscle fibres was related to the cycling of crossbridges (the interaction of the actin and myosin proteins, see Figure 2.3) [1, 34, 35]. Bartoo et al feel that, for a truly relaxed myofibril, both passive force and dynamic stiffness principally reflect the intrinsic visco-elastic properties of the titin filaments [36].

Recent studies indicate that within the physiological range of muscle length changes, myofibrillar structures are the major source of elasticity and that extracellular connective tissues contribute significantly only in highly extended skeletal muscles [30-32, 37]. These myofibrillar structures are comprised of endo- and exo-sarcomere lattices. The endo-sarcomere structure is the titin-nebulin complex described above and the exo-sarcomeric network is made up of intermediate filaments that envelop and interconnect the sarcomere both radially to parallel myofibrils and longitudinally to adjacent sarcomeres. Wang et al, from their investigation of the tension response to rabbit psoas sarcomere stretch, conclude that the interlinked endo- and exo-sarcomeric lattices are both visco-elastic, force bearing elements. These distinct cytoskeletal lattices enable myofibrils to respond visco-elasticity over a broad range of sarcomere and fibre lengths [32].

Clearly, characterizing the biomechanical properties of muscle is difficult because of the overlapping influence of connective tissue, the contractile machinery of the sarcomere and the structures that support the sarcomere and give it form [33].

2.2.3 Neurophysiology

Not only do connective tissue and muscle tissue limit joint ROM, but the interaction of the central and peripheral nervous systems with muscle does as well [10]. The central nervous system allows one to consciously contract their muscles, thereby influencing muscle length directly. Muscle behaviour is also influenced by the reflex connections that comprise the peripheral nervous system. Saepga et al believe that optimal passive stretching is attained only when all voluntary and reflex muscular resistance is eliminated [22, 25].
**Muscle Proprioceptors**

Information about the length of a muscle and the tension within it are transmitted to the central nervous system by two types of muscle receptors: Golgi tendon organs and muscle spindles, illustrated in Figures 2.5 and 2.6. Golgi tendon organs are located in the tendon where muscle and tendon merge. Each is innervated by a single Ib afferent axon. (Afferent nerves carry information to the spinal cord; efferent nerves carry information away from it.) Golgi tendon organs signal changes in tension in the muscle and so function as force transducers. They are particularly sensitive to active contractions of the muscle. This sensitivity is due to the structure of the Golgi tendon organ: each individual organ attaches to a small number of muscle fibres, hence each one “sees” all of the force produced by the contraction of one of these fibres [38]. [1, 2, 39, 40]

![Figure 2.5 Muscle spindles and Golgi tendon organs. (from Kandel [39])](image-url)
Muscle spindles are the most common and the most complex of the peripheral sensory receptors [38]. As their name suggests these sensors are spindle shaped and are composed of two different types of intrafusal fibres: nuclear bag and nuclear chain fibres. These fibres are buried parallel to and among the rest of the main muscle (extrafusal fibres). Muscle spindles receive both afferent and efferent innervation. There are two different types of afferent nerve endings: primary (Group Ia) endings which form helical coils around the intrafusal fibres and secondary (Group II) endings which attach to the intrafusal fibres to form flower spray endings (see Figure 2.6). The efferent innervation of the muscle spindles is achieved by nerve fibres called gamma or fusimotor fibres [38]. [1, 2, 39, 40]

Originally it was thought that spindle primary endings were velocity detectors and that secondary endings sensed changes in position. Although this is true to some extent, it is not a complete picture, as illustrated in the frequency response of the sensitivity of primary and secondary spindle
endings to sinusoidal stretching in Figure 2.7. The two responses indicate that both sensors are high pass in nature with a break at roughly 1 Hz. The curves demonstrate a greater sensitivity among primary endings across all frequencies as compared with secondary endings. Also, as is shown in Figure 2.7, the primary muscle spindle endings have a sensitivity to acceleration at higher frequencies that secondary endings do not possess. [35, 38]

Figure 2.7 Comparison of the sensitivity to sinusoidal stretching within the linear range of a primary and secondary muscle spindle ending at various frequencies. The continuous lines represent the vector sums of a length component (horizontal segment) and a velocity component (diagonal segment). (from Stein [35])
The Stretch Reflex

Most stretching techniques are thought to be based on the stretch reflex, illustrated in Figure 2.8. When a muscle is stretched, as in Figure 2.8 B, the muscle spindles are stretched by the same amount since the intrafusal fibres are oriented in parallel with the extrafusal ones. This stretch mechanically deforms the spindles and a signal is sent along the la afferent nerve fibres to the spinal cord and then back through an α motoneuron to the muscle being stretched, causing it to contract and counteract the stretch, shown in Figure 2.8 C. This is the stretch reflex: a fast single synapse reflex arc. [1, 2, 39, 40]

![Figure 2.8 The stretch reflex. (from Kandel [39])]({})

Reciprocal Innervation

Generally skeletal muscles act in pairs so that when one set of muscles, the agonists (homonymous and synergist in Figure 2.8 A), are contracting, the opposing set of muscles, the antagonists, are
relaxing. As shown in Figure 2.8 C, the antagonistic muscles are inhibited from contracting when the agonists are contracting. The Ia afferent nerves from the muscle spindles connect with inhibitory interneurons to prevent the opposing muscle from contracting. At times this reflex loop, shown in Figure 2.8, is overridden by descending signals from higher centers and co-contraction occurs. Some PNF techniques take advantage of reciprocal innervation to place the muscle to be stretched in a greater state of inhibition. [10, 39, 40]

The Inverse Stretch Reflex

Owing to the manner in which Golgi tendon organs attach to tendons, they are much more sensitive to forces produced by active contractions than by those imposed passively. Thus when the Golgi tendon organs sense force generated by active muscle contractions, they send a signal via the Ib afferent neuron to inhibit the α motoneuron and cause the flexor muscle to relax (see Figure 2.9). This phenomenon is called the inverse stretch reflex or autogenic inhibition. It is on the basis of this principle that some forms of PNF use the isometric contraction of a muscle to subsequently place the muscle in a greater state of relaxation. Experimentally it has been shown that following voluntary contractions there is a strong but brief neuromuscular inhibition useful for applying a stretch [41]. In this state the muscle exhibits more compliance to stretching. [10, 20, 38]

Figure 2.9 The neural connections that facilitate the inverse stretch reflex. (from Kandel [39])
2.3 Stretching Strategies: Effectiveness and Comparisons

2.3.1 The Effects of Stretching

The majority of the scientific literature investigating stretching supports the idea that warm-up and stretching prior to strenuous exercise enhance performance and prevent injury [2, 42]. Despite this widespread belief of the effectiveness of stretching, most researchers would agree that the actual effects of stretching on the behavior of muscles and joints are not well understood [43].

Researchers have implemented many different stretching approaches on many different joints and combinations of joints in the body to study the effects of stretching. The most common observation is that stretching appears to increase muscle length [44-52] and joint ROM [1, 17, 44-54] and to reduce joint stiffness [2, 10, 44-52, 55]. These apparent primary mechanical and physiological changes have contributed to several conjectures regarding the effects of stretching, namely that stretching prevents injuries [1, 9, 42, 56, 57], reduces delayed onset muscle soreness (DOMS) [1, 9], improves running economy [1, 48, 58], enhances athletic performance [1, 55] and diminishes muscle spasms and pain after exercise [1].

In direct contradiction to these studies there are those experimental findings that suggest that the effect of stretching a muscle [25-27], or of performing some sort of therapy on it [59], is purely transient. That is, the effect of stretching has no long-term effect on the range of motion or stiffness of the joint. Specifically, Halbertsma and Goeken [26] and Magnusson et al [25] feel that stretching exercises do not make a muscle longer or less stiff, but simply influence the tolerance of the muscle to be stretched. DeVries showed that increased flexibility due to stretching had little or no effect on economy of exercise [1]. Ironically, many purport that stretching can actually do more harm than good [1, 42].

Generally a chronic increase in ROM is regarded as synonymous with decreased tissue stiffness. There are, however, relatively few studies that have examined changes in passive stiffness following a stretching program. Toft et al [9] found up to a 36% reduction in the passive tension of the plantar flexors following a three week stretching program. In contrast, Halbertsma and Goeken [26] and Magnusson et al [25] showed that hamstring stiffness was invariant following even longer training
periods despite increases in joint ROM. These researchers attributed increases in ROM to increased
tolerance to stretching. An increase in ROM, thus, does not necessarily lead to a decrease in passive
stiffness in a joint.

A number of different strategies have been implemented to investigate the effects of stretching.
These have included: measuring the Hoffman reflex (under the assumption that greater motor pool
inhibition reduces muscle contractibility and therefore allows more muscle compliance) [60, 61],
using mechanical models of muscle behaviour [62], testing the passive stiffness of a joint [9, 25,
26], measuring the reflex response to Achilles tendon tap [63], examining electromyographic
responsiveness [64] and simply recording subjective impressions [1]. By far, though, the most
popular strategy has been to use some form of a goniometric technique to measure changes in the
range of motion of a joint [8, 26, 27, 44-46, 48-52, 65-67]. As is evidenced by the prevalence of the
use of goniometry, researchers have traditionally regarded changes in the range of movement of a
joint as a measure of the effect of stretching.

2.3.2 Comparison of Stretching Techniques
Besides investigating the effects of stretching, a number of studies have been conducted to compare
the effectiveness of different stretching strategies. The results of these initiatives have been
confusing and contradictory. However the majority of the information on this topic tends to support
the use of proprioceptive neuromuscular facilitation (PNF) techniques for providing the best
improvement in flexibility [2]. Specifically, the results of several experiments have demonstrated
that PNF is more effective in increasing flexibility and decreasing joint stiffness than static
stretching [2, 8, 15, 61, 68, 69]. Others have found that PNF and static stretching are equally
effective [8, 60, 65, 70].

A variety of studies have compared different PNF techniques: stretch-relax (SR), contract-relax
(CR), agonist contract-relax (ACR) and contract-relax-antagonist-contract (CRAC) [8, 60, 61, 66,
67]. Some of the studies have found that the ACR form of PNF is most effective [66, 67] (the
CRAC form of PNF was not included in these studies). Others found the CRAC form of PNF, a
reciprocal activation approach, to exhibit the best results [7, 8, 19, 61, 64]. Still others found that there was no significant difference between PNF approaches [60].

Since PNF techniques exploit autogenic inhibition and reciprocal inhibition, it is thought that they should allow muscles to be stretched to a greater degree than ballistic, static or other forms of stretching. Experimentally this certainly has proven to be the case. It would seem then, after surveying the whole of the literature, that when performed correctly, PNF is the most effective method of stretching, in terms of ROM improvement. Furthermore, following close examination of the various PNF techniques, our conclusion is that contract-relax antagonist contract (CRAC) is the most effective form of PNF. Here lies the motivation to use this specific form of stretching in this study.

2.3.3 Contract Relax Antagonist-Contract (CRAC)

As the studies referred to in the previous section indicated, contract-relax antagonist contract (CRAC) is one of the most, if not the most, effective form of PNF stretching [7, 8, 19, 61]. In this type of PNF the muscle to be stretched is contracted and then relaxed. It is thought that this initiates the inverse stretch reflex (autogenic inhibition) and allows the muscle to enter a greater state of inhibition. The agonist is thus contracted (C) and then relaxed (R) at the beginning of the CRAC sequence. Furthermore, a muscle is presumably inhibited from contracting when the antagonist or opposing muscle contracts (reciprocal innervation). This is the motivation behind the contraction of the antagonist (AC) in the final steps of the CRAC sequence. [2, 8, 20]

2.4 Warm-Up

There is an unwritten law among elite athletes and recreational exercisers that warming-up prior to exercise is important, even necessary, for preventing injury [71] and optimizing performance [1, 3]. Studies have demonstrated that warming-up enhances the effects of stretching [1, 53]. This warm-up is best performed prior to stretching [3].

There are three broad categories of regimens used for warming-up: passive warm-up, general warm-up and specific warm-up [2]. A passive warm-up involves raising the body temperature externally
by warm baths, heating pads or a sauna. Jogging, riding a stationary bicycle or calisthenics are used for general warm-up and have the advantage of elevating the deep muscle temperature more effectively. A specific warm-up focuses on the particular neuromuscular apparatus that will be used in the subsequent, more strenuous activity.

From a physiological perspective, active warm-up is thought to increase body and tissue temperatures, as well as the flow of blood through the active muscles [2, 10]. This increase in tissue temperature and blood flow is felt to augment the effects of stretching for a number of reasons:

- The extensibility of tendons, ligaments and other connective tissues supposedly increases at higher temperatures [22, 71].
- Muscle elasticity is thought to depend on blood saturation [10].
- The ability to do physical work improves at elevated temperatures [72, 73].
- Many physiological mechanisms related to O₂ transport and the vascular and nervous systems are thought to be enhanced by an increase in temperature [2, 72, 74].

An increase of 1 to 2 °C in tissue temperature is needed to attain the physiological benefits associated with warming-up [75]. This temperature rise is associated with light to mild sweating at ambient temperatures. In spite of the physiological effects of warming-up, some researchers still maintain that its benefits are more psychological in nature [2].

### 2.5 Rationale

In the past, the evaluation of the effects of stretching has almost exclusively focused on changes in joint ROM. This limits the investigation to the effects on muscles and tendons at the extreme positions of the joints. But what is happening to the properties of the joint in the more central, functional range of motion (i.e. those parts of the ROM used during normal walking and running conditions)? Why not investigate joint properties throughout the ROM of the joint?

Evaluating the ROM alone not only ignores joint property information away from extreme positions but it is also a subjective approach. The degree to which an individual allows one of his or her joints to be extended depends on a number of factors. Measurements taken of a subject’s ROM in a joint at a given point in time may be influenced by their pain threshold, level of anxiety, desire to
show improvement in flexibility and other psychological factors. It would be beneficial to have access to more quantitative measures so that these factors did not bias the measurements. Passive tension measurements demonstrate the effect of stretching over a wide range of motion of the joint [9, 25, 26] but they miss the time and rate dependent joint properties. What if a system identification approach was implemented to discover even more about the mechanical properties of the joint throughout its range of motion?

In McGill's Neuromuscular Control Lab, system identification procedures have been used to investigate posture, locomotion, reflexes, joint dynamics and neuromuscular disease as well as other aspects of neuromuscular control [11]. The methods that have been developed provide an objective, quantitative description of the mechanical behaviour of a joint. This description is useful for the clinical assessment of joint function. It has been found in the past that there is little intra-subject variability in the joint model parameters obtained [11, 76]. This approach is strategic for assessing the effects of stretching and warming-up on joint dynamics. These established system identification techniques were used in this study.

To now, a system identification approach has not been used to assess the effect of a long-term stretching program on a human joint. The goal of this study was to investigate the effect of a four week PNF with warm-up program on ankle joint dynamics. A stretching program, designed to yield optimal results (i.e. daily CRAC PNF sessions preceded by a warm-up) was employed. Quantitative measures describing various properties of the ankle joint were acquired using system identification. These joint properties, which are influenced by the passive characteristics of tissue, the mechanical behaviour of muscle and stretch reflexes [11, 77], were used to evaluate the effects of stretching. Essentially then, the purpose of this study was to answer the question: "Does stretching have an effect on the dynamic stiffness of the ankle joint?"

2.6 System Identification of Joint Dynamics: Quasi-Linear Model

2.6.1 System Identification

System identification methods have been used to successfully describe the overall dynamic stiffness of joints [11]. System identification is the process of determining a mathematical model of a system
from the analysis of the relationship between its inputs and outputs. This process then is directly based on experimentation. A model is inferred from the analysis of collected input and output signals. The system identification procedure involves three broad stages: collecting the data, choosing a type of model and then selecting the best model based on some criterion [78, 79]. In this study, linear system identification was used to evaluate the changes in the mechanical behaviour of the ankle joint due to stretching.

2.6.2 Quasi-Linear Models of Joint Dynamics

The manner in which the ankle joint, or any joint for that matter, interacts with its environment may be understood in terms of its dynamic stiffness. Dynamic joint stiffness is the dynamic relationship between the angular position of a joint and the torque acting about it. It is important because it provides a quantitative, objective measure of the mechanical behaviour of the joint throughout its ROM.

Linear models have been found to describe joint dynamics well, provided that the operating conditions (e.g. activation level or mean position) are kept relatively constant throughout the experiment [11]. The non-parametric models of joint dynamics achieved using this strategy bear features that correspond to a second order system. Consequently joint dynamics have frequently been modeled using a parametric model formulated in the Laplace domain as:

\[
\frac{TQ(s)}{\theta(s)} = Is^2 + Bs + K
\]

(2.1)

where \(TQ, \theta, I, B\) and \(K\) respectively represent torque, angular position, inertia, viscosity and elastic stiffness. For given operating conditions, this parametric model provides a good description of experimental data.

2.6.3 Previous Work on System Identification of Joint Dynamics

These linear system identification methods to determine quasi-linear models of joint dynamics have been used to investigate a plethora of peripheral neuromuscular control related issues. These issues
have been explored through the use of a number of different joints including the ankle, wrist, elbow, knee, jaw, and neck [11]. Among the joint behaviour issues that have been considered are: the variation of ankle joint stiffness with torque [80], displacement amplitude [81], fatiguing contractions [82] and angular position [83, 84] and the system identification of stretch reflex dynamics [77, 85]. Furthermore, the inter-subject variability and intra-subject reliability of the system identification of ankle joint dynamics has been studied [86]. Recently, as well, these methods have been employed to develop a parallel cascade model describing joint dynamics taking into account both intrinsic and reflex contributions to the total torque seen at the ankle joint [87].

2.7 The Ankle Joint

The ankle joint was selected for this study in light of two main considerations: (i) an electro-hydraulic actuator set up to investigate ankle joint behaviour was readily available [80] and (ii) the muscles chiefly responsible for manipulating the ankle joint are relatively easy to stretch.

Initially the electro-hydraulic actuator in the Neuromuscular Control Lab was designed to investigate the ankle joint in particular for a number of reasons:

- the ankle joint exhibits comparatively simple movements and can easily be restricted to move freely in the sagittal (antero-posterior) plane only
- the foot has low inertia
- all ground reactions are translated through the ankle
- the ankle plays a critical role in locomotion
- the ankle has strong postural responsibilities
- the muscles that manipulate the ankle joint reside close to the skin for easy electromyographic measurement.

The ankle joint, illustrated in Figures 2.10 and 2.12, is located between the superior surface of the talus and the inferior ends of the tibia and fibula. Another term for the ankle is the talocrural joint. It is a hinge-like synovial joint that is comprised of three articulations of bone interfaces. The pulley shaped talus articulates with: (i) the inferior surface of the tibia (ii) the medial surface of the lateral
malleolus formed by the fibula and (iii) the lateral surface of the medial malleolus also formed by the tibia.

As Figure 2.11 illustrates, the talocrural joint allows for movement in the sagittal plane: dorsiflexion and plantar-flexion. In dorsiflexion the foot is brought closer to the anterior side of the lower leg and plantar-flexion moves the foot away from the anterior surface of the lower leg. The talocrural joint tends to be very stable during dorsiflexion owing to the support of stronger ligaments, crossing tendons and bands of thickening fascia called retinacula. As well, the talus fits snugly with the malleoli. During plantar-flexion the joint is relatively unstable, and some rotation, abduction and adduction of the ankle are possible [89].

The muscles that are chiefly involved in dorsiflexion are the tibialis anterior and the extensor digitorum longus. The extensor hallucis longus and the fibularis (peroneus) tertius are also involved. The triceps surae, comprised of the soleus and the two heads of the gastrocnemius, is the main plantar-flexor of the ankle. The plantaris, tibialis posterior, flexor hallucis longus and flexor digitorum longus are also involved in plantar-flexion.
The lateral and medial heads of the gastrocnemius originate from the lateral and medial condyles of the femur respectively. They attach to the posterior surface of the calcaneus via the Achilles tendon. Thus the gastrocnemius is a muscle that spans two joints: the knee and ankle. The soleus originates from posterior aspects of both the fibula and tibia. Like the gastrocnemius, the soleus inserts at the calcaneus by way of the Achilles tendon. The tibialis anterior originates from the lateral condyle of the tibia and inserts on the medial and inferior surfaces of the medial cuneiform as well as the base of the first metatarsal. Both the soleus and tibialis anterior are one joint muscles. Refer to Figure 2.10. [89]

The fibrous capsule surrounding this synovial joint is supported on each side by strong collateral ligaments, shown in Figure 2.12. The strong medial ligament attaches the medial malleolus to the three tarsal bones (talus, navicular, and calcaneus). Laterally the talocrural joint is supported by
three ligaments: the anterior and posterior talofibular ligaments and the calcaneofibular ligament. [89]

Figure 2.12 A more detailed view of the ankle joint. (from Moore [89])
3 METHODS

3.1 Apparatus

The experimental apparatus that was used in this study is shown in Figure 3.1. Subjects lay supine on a table that also supported the actuator assembly. The subject’s left foot was attached to the pedal of a stiff, position controlled electro-hydraulic rotary actuator by means of a custom-fitted fibre glass boot. A strap was attached over the lower part of the femur to eliminate any lower leg movement. The angular position of the ankle, as well as the torque acting about the ankle, were measured by transducers attached to the actuator. Electromyograms (EMGs) from the tibialis anterior and gastrocnemius were recorded using bipolar surface electrodes. A target torque signal, as well as a low pass filtered version of the recorded torque, were displayed on an oscilloscope suspended above the subject to aid the subject in maintaining a specified torque level.

Figure 3.1 Experimental set-up. (drawing created by Robert Kirsch)
3.1.1 Electro-Hydraulic Actuator and Control Strategy

The electro-hydraulic actuator, experimental platform, control system and data acquisition set-up used in this study are described in detail elsewhere [90-92]. Briefly, a rotary hydraulic actuator was used to perturb the angular position of the ankle. A two stage servo-valve controlled the flow to the actuator.

The rotary actuator was controlled using proportional position feedback techniques. The proportional control law was implemented digitally using a two-processor transputer system configured for servo-hydraulic systems. The rotary actuator allowed movement of the foot in the sagittal (antero-posterior) plane only. Figure 3.2 shows the foot of a subject attached to the actuator pedal by a fibre-glass cast.

![Figure 3.2 The booted foot of a subject attached to the actuator assembly.](image)

Four independent safety mechanisms protected the ankle joint from injury: a mechanical stop, a hydraulic stop, pre-set servo control parameters and a panic button held by the subject.
3.1.2 Rigid Limb Fixation and Ankle Axis of Rotation

The light weight, rigid fixation of the foot to the actuator pedal was achieved by means of fibreglass cast boots that were constructed for each subject individually. The details of boot construction are outlined elsewhere [90, 93]. The boot did not restrict the freedom of ankle rotation.

The axis of rotation (AOR) of the ankle joint was initially estimated using anthropometric data [94]. The AOR was “fine tuned” by having the subject plantar- and dorsi-flex their foot while two pointers were applied to the points on the boot corresponding to the ankle’s approximate AOR. The points were updated until there was little movement of the pointers in any plane. General observations regarding the degree of supination/pronation and inversion/eversion were also taken into account in the final determination of the AOR of the ankle.

After the AOR was determined, pre-threaded aluminum posts were attached to the sides of the boot with plastic epoxy cement. These posts were used to firmly bolt the subject’s boot to the pedal of the actuator. The actuator axis of rotation was coincident with the plantarflexion-dorsiflexion axis of the ankle.

3.1.3 Signal Transduction and Processing

Four signals were recorded during each experimental session: angular position, torque and electromyograms from the tibialis anterior and gastrocnemius.

The angular position of the ankle was measured by a Beckman precision plastic film rotary potentiometer (Model 6273-R5K), located on the axis of the actuator. The potentiometer had a maximum non-linearity of ±0.2 % and a maximum resistance range of 0 to 5kΩ. Ankle angles were taken with reference to the mid-position, i.e. the position equidistant from the extremes of the ankle ROM.

The torque generated about the ankle joint was measured by a Lebow 2110-5K general purpose reaction torque sensor. This four arm bonded strain gauge was characterized by a high torsional
stiffness (10^5 Nm/rad), a large capacity (565 Nm) and a maximum non-linearity of ± 0.2 %. The strain gauge was mounted directly on the shaft of the actuator.

Surface electromyograms (EMGs) were acquired from the gastrocnemius (GS) and tibialis anterior (TA) using disposable, self adhesive Ag-AgCl electrodes (Hewlett Packard, 10 mm diameter, Model 13951C). Pairs of electrodes, their centres separated by 10 mm, were placed parallel to the orientation of the muscle fibres over the lateral head of the GS and over the belly of the TA (roughly one-third of the distance between the patella and the ankle). A reference electrode was placed over the patella. Prior to electrode attachment the surface of the skin was shaved and cleaned with rubbing alcohol.

The EMGs detected by the electrodes had amplitudes ranging from 0.1 to 10 μV. These raw EMG signals were amplified using a custom-made, three stage differential pre-amplifier consisting of an instrumentation amplifier, a passive single-pole RC high pass filter with a 1 Hz cut-off and an isolation amplifier. The high pass filter removed low frequency artifacts due to electrode polarization and cable and electrode motion. The overall gain of the pre-amplifier could be switched from 1000 to 10000.

The pre-amplified EMG signals were then passed through a fourth order Butterworth high-pass filter with a 5 Hz cut-off. By lab convention, the TA EMG signal was positively full-wave rectified and the GS EMG signal was negatively full-wave rectified.

The conditioned and amplified angular position, torque and EMG signals were anti-alias filtered at 200 Hz and sampled at 1000 Hz using an, 16 bit analog to digital converter. Measurement resolution of the torque (0.006 Nm) and angular position (0.002 rad) was limited by the quantization of the A/D converter.
3.2 Experimental Methods

System identification methods were used to assess the effect of a four week stretching program on ankle joint dynamics.

3.2.1 Experimental Schedule

Six reasonably physically active, healthy subjects were placed on a stretching program for muscles directly involved in the movement of the ankle. Prior to the study, the subjects were not following a regular stretching routine for their lower legs. A system identification approach developed in this lab was used to assess the effect of the training program. The timeline of the study, which in total lasted six weeks, is shown in Table 3.1. For the four weeks following the second control test, subjects were required to attend a training session, consisting of a 15 minute stationary cycling warm-up session followed by a 12 minute lower leg PNF routine, five days a week. The subjects were also trained to perform a self-stretching variation of the PNF stretches on their own daily and twice daily on weekends. Subjects were tested once a week; the first two tests were control tests and the last four were evaluative.

<table>
<thead>
<tr>
<th>Week</th>
<th>Session</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>none</td>
<td>Construction of custom made fibre-glass boot.</td>
</tr>
<tr>
<td>1</td>
<td>Control #1</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Control #2</td>
<td>Commencement of PNF (both partner assisted and self-stretching protocols).</td>
</tr>
<tr>
<td>3</td>
<td>Test #1</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Test #2</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Test #3</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Test #4</td>
<td>Final test.</td>
</tr>
</tbody>
</table>

Table 3.1 The experimental timeline.

3.2.2 Subjects

Table 3.2 summarizes the characteristics of the subjects participating in this study. Their mean ± standard deviation physical characteristics were as follows: age, 24.3 ± 4.5 years; height, 174.8 ± 9.4 cm; body mass, 69.2 ± 7.3 kg. Subjects gave informed consent.
3.2.3 Testing Protocol

No testing was performed within six hours following a stretching session in order to avoid any transient effects due to the visco-elastic nature of the tissues involved. The testing protocol during each of the six testing sessions consisted of the following:

1) The ankle range of motion (ROM) of the subject was evaluated with the subject's foot attached to the pedal but with the hydraulic fluid flow off. The foot, in the cast, was rotated manually from a mid-position toward maximum dorsi-flexion and then toward maximum plantar-flexion. Angular displacement was continued in each direction until the subject could tolerate no further movement. This process was performed three times and the maximum values were recorded. The range of motion was deemed to be the angle between these two extreme values.

2) In first control session the maximum voluntary contractions (MVC) of the ankle extensors and flexors were determined. The subjects were required to generate a MVC in both dorsi-flexion and plantar-flexion in response to changes in a tracking stimulus displayed on the oscilloscope above them. A level equivalent to 5% of the triceps surae MVC was used subsequently as a target torque to be maintained during the “active” portion of the tests at each position.

3) To evaluate the passive tension in the ankle, a position ramp was used as an input. With the subject relaxed, the subject’s foot was slowly rotated (0.05 rad/s) from the mid-position to 0.35 rad dorsi-flexion to 0.35 rad plantar-flexion and back to the mid-position. A sample trial is shown in Figure 3.3.
Figure 3.3 Angular position ramp input, with subject relaxed, to evaluate passive tension in the ankle. Angular position, torque and TA and GS EMGs, are shown. (Subject PG) Note: plantar-flexion torques and EMG are negative by lab convention.

4) PRBS inputs were applied.
   a) For the purposes of system identification, pseudo random binary sequences (PRBS) of angular position were applied at 15 different mean positions throughout the ankle's range of motion. The subject's foot was randomly moved to the different positions and for each of the six testing sessions the same positions were used and in the same order. At each mean position, the PRBS inputs moved the joint quickly over small angles between two values, at random times determined by the switching rate (see Figure 3.4).
   b) At each position two PRBS inputs were applied to the subject's foot. The first sequence was applied with the subject relaxed and the second applied with the subject contracting the ankle extensors (triceps surae) at 5% of their MVC. Upon being moved to a new position, the subject's foot was left for one minute or until the passive torque level reached a steady
state [87]. In this manner the passive-position and active-position operating points of the system in question were investigated. Subjects were trained during the first control test.

(c) The angular position inputs were 20 s in duration with an amplitude of 0.035 radians and a mean switching interval of 150 ms. This type of input was selected because it has enough power to identify a system with a bandwidth of up to 40 Hz without attenuating reflexes [87] (it is known that broadband inputs suppress the reflexes [95]).

![Graphs showing angular position, torque, TA EMG, and GS EMG](image)

**Figure 3.4** Five seconds of data recorded from the four channels during a PRBS of angular position input trial.

5) At the end of each experimental session, after the subject was released from the fixation apparatus, a calibration trial was performed in order to correct for the inertial influence of the fibre-glass boot and the actuator itself on the torque signal that was sampled. The same PRBS of angular position as that used in the experiment was applied with the boot at the neutral position.
3.2.4 Training Protocol

The training regimen, which began following the two control sessions, had three main components: warm-up, partner assisted stretching sessions and self-stretching sessions. These are outlined below.

Warm-Up

The subjects cycled on a stationary bicycle for 15 minutes at 60 to 70 rpm, with low to medium tension, prior to each partner assisted PNF session. An adequate warm-up was indicated by a freely sweating subject.

Stretching Protocol

A contract-relax antagonist contract (CRAC) PNF protocol was applied to the soleus, tibialis anterior and gastrocnemius muscles. These muscles were of concern because of their importance in manipulating the ankle joint. The muscles were stretched in the same order in each stretching session. The stretching routine was performed five times weekly at the same time of day.

To briefly review the principles behind CRAC PNF, first the muscle to be stretched (agonist) is contracted and then relaxed (this is thought to further relax or inhibit the muscle). Second the antagonist or opposing muscle is contracted in order to bring the agonist into a stretch and presumably to inhibit it from contracting. Throughout the alternating contracting and relaxing process, a partner helps the stretcher by providing resistance and maintaining the position of the joint. The stretching technique employed for each muscle is outlined below.
CRAC PNF for Soleus (see Figure 3.5):

1) The subject lay prone on the therapy table with knee bent to isolate the soleus and avoid stretching the gastrocnemius.

2) The subject contracted the tibialis anterior to stretch the soleus.

3) The position of the ankle that was achieved was maintained by the assisting partner. The soleus was contracted (25 to 50% of MVC) for 8 s in this stretched position while the partner resisted any movement, creating an isometric contraction.

4) The soleus was relaxed for 2 s, held in the stretched position by the partner.

5) The tibialis anterior was contracted (25 to 50% MVC) for 8 s.

6) Steps 3) to 5) were repeated 5 times.

Figure 3.5 Applying the CRAC PNF technique to the soleus.
CRAC PNF for Tibialis Anterior (see Figure 3.6):

1) The subject lay supine on the therapy table with leg straight.
2) The subject contracted the triceps surae to stretch the tibialis anterior.
3) The position of the ankle that was achieved was maintained by the assisting partner. The tibialis anterior was contracted (25 to 50% of MVC) for 8 s in this stretched position while the partner resisted any movement, creating an isometric contraction.
4) The tibialis anterior was relaxed for 2 s, held in the stretched position by the partner.
5) The triceps surae was contracted (25 to 50% MVC) for 8 s.
6) Steps 3) to 5) were repeated 5 times.

Figure 3.6 Applying CRAC PNF to the tibialis anterior.
CRAC PNF for Gastrocnemius (see Figure 3.7):

1) The subject lay supine with knee straight and heel resting on the shoulder of the assisting partner.
2) The subject contracted the tibialis anterior to stretch the gastrocnemius.
3) The position of the ankle that was achieved was maintained by the assisting partner. The gastrocnemius was contracted (25 to 50% of MVC) for 8 s in this stretched position while the partner resisted any movement, creating an isometric contraction.
4) The gastrocnemius was relaxed for 2 s, held in this stretched position by the partner.
5) The tibialis anterior was contracted (25 to 50% MVC) for 8 s.
6) Steps 3) to 5) were repeated 5 times.

Figure 3.7 Applying the CRAC PNF technique to the gastrocnemius.
Self-Stretching Protocol

The self-stretching protocol was the same as outlined above except that instead of using a partner to help stretch, the subject used a towel when stretching the gastrocnemius and soleus, and a bed, sofa or a set of shelves to help when stretching the tibialis anterior. Subjects performed the self-stretching exercises daily, in addition to the partner assisted stretching, and twice daily on weekends.

3.3 Analytical Methods

The dynamics of the ankle joint were modeled in a quasi-linear fashion. That is, the system was assumed to be linear for small perturbations about each discrete position in the ankle’s range of motion (ROM). At each position, a non-parametric model of the ankle joint intrinsic dynamics was first obtained by determining the compliance impulse response function (IRF) relating the torque to the angular position. Non-linear minimization techniques were then used to fit a parametric model to this IRF. The compliance IRFs were assessed in terms of the percentage of torque variance that they accounted for. The validity of the parametric fit was assessed in terms of the percentage variance of the IRF that was accounted for by the parametric model. The second order system parameters, were evaluated throughout each subject’s ROM and over all six experimental trials.

Before engaging in any analysis of the collected data, its stationarity was ensured. The signals were de-trended and the mean was subtracted. As well, the inertial effects of the fibre-glass boot were corrected for. This was accomplished by (i) calculating the impulse response function (IRF) relating the angular position of the actuator/cast to the torque records obtained during the calibration trial, (ii) convolving the actuator/cast stiffness IRF with the angular position, and (iii) subtracting the resulting predicted torque from the sampled torque. The residual torque representing the contribution from the ankle joint alone was subsequently referred to simply as the torque.

3.3.1 Non-Parametric Model

Intrinsic stiffness dynamics were estimated in terms of linear, dynamic impulse response functions (IRFs) relating position and torque. Dynamic compliance IRFs were computed for analysis purposes. The response, \( y(t) \), of a system to a particular input may be obtained by convolving the
IRF or filter describing the system, $h(t)$, with the input signal, $x(t)$. This is described mathematically by in Equation 3.1:

$$y(t) = \int_{-\infty}^{\infty} h(\tau) \cdot x(t - \tau) d\tau$$  \hspace{1cm} (3.1)

**Parallel Cascade Model**

Intrinsic and reflex contributions to the stiffness dynamics were separated using a parallel-cascade identification method [87]. This parallel pathway model is illustrated in Figure 3.8. The intrinsic

![Parallel Cascade Model Diagram](image)

**Figure 3.8** The parallel cascade model.
stiffness dynamics were estimated in terms of linear, dynamic impulse response functions (IRFs) relating angular position to torque. These IRFs were convolved with the position signal to predict the intrinsic torque, which was subtracted from the observed total torque to get the reflex torque. The dynamic compliance IRF was computed for analysis purposes. These compliance IRFs exhibited the characteristic features of a second order low pass system. Reflex stiffness dynamics were estimated by determining the IRF between half-wave rectified velocity and the reflex-torque. The IRFs were assessed in terms of the percentage of the torque variance that they accounted for.

3.3.2 Parametric Model

Non-linear least squares methods were used to fit the parametric models to the IRFs. A second order low pass system was used to describe the linear dynamics of the intrinsic contribution to stiffness. Analysis was limited to the intrinsic contribution since it was found that over 90% of the variance in torque was due to the intrinsic pathway of the model. Equations 3.2 and 3.3 show the parametric model describing the linear dynamics of the intrinsic contribution to ankle joint stiffness:

\[
\frac{TQ(s)}{\theta(s)} = Is^2 + Bs + K \tag{3.2}
\]

\[
\frac{TQ(s)}{\theta(s)} = \frac{s^2 + 2\zeta\omega_n s + \omega_n^2}{G\omega_n^2} \tag{3.3}
\]

Here the second order model is represented in two different forms of its Laplace transform [96]. In both Equations 3.2 and 3.3 the angular position, \( \theta(s) \), is the input and the torque, \( TQ(s) \), is the output to the system. Inertia, viscosity and elastic stiffness are represented respectively by \( I \), \( B \) and \( K \) in Equation 3.2. In Equation 3.3 damping, natural frequency and gain are represented by \( \zeta \), \( \omega_n \) and \( G \) respectively.

The parametric representation of the dynamic relation between torque and position provided a concise description of the ankle joint system which accounted for more than 90% of the variance of...
the original compliance function. Figure 3.9 shows the plots of both a non-parametric compliance IRF and the 2nd order parametric model that best fits it. The variance accounted for (VAF) by the parametric model in this case was very good (96.17%).

The quasi-linear model is only valid for a particular position or operating point. The parameter values, which describe the passive contribution to ankle dynamics of the ankle joint, were a good gauge for any changes in ankle behaviour due to the training program. The validity of the parametric fit was assessed in terms of the percentage of the variance of the IRF accounted for.

![Figure 3.9 Compliance impulse response functions: non-parametric and best fit 2nd order IRFs. Taken from Subject MD, Test #4.](image)

3.3.3 Assessing Changes in Intrinsic Stiffness

Position dependent changes in the intrinsic stiffness of the ankle joint were investigated in terms of the three parameters describing the non-reflex contribution to dynamic stiffness: inertia, $I$, viscosity, $B$, and elastic stiffness, $K$. Figure 3.10 illustrates how the elastic stiffness for passive conditions was evaluated in more detail, providing a greater basis for comparison and analysis. Values of the
intrinsic elastic stiffness parameter (dashed line) were plotted against mean angular position. A third order Chebyshev polynomial was fitted to the experimental data (solid line) to interpolate the values of $K$. (This particular fit had a VAF of 89.05%. These fits were generally well above 90% VAF.) These interpolated curves were characterized by two parameters for comparison purposes. The minimum value of the elastic parameter, $K_{\text{min}}$, was used as a measure of the resting joint stiffness. A second parameter, $\theta_{\text{low}}$ (dotted line), the range of positions for which the stiffness was less than $K_{\text{min}} + 20 \text{ Nm}/\text{rad}$, was used as a measure of the range of motion over which the elastic parameter showed low sensitivity to changes in position.

![Graph](image)

**Figure 3.10** Assessment of elastic stiffness. Parametric model data and 3rd order Chebyshev fit. Taken from Subject MD.

### 3.3.4 Statistical Analysis

The variance accounted for (VAF) was used to assess the accuracy of the non-parametric and parametric models. The Student's $t$ test, a test of hypothesis and significance, was used to determine whether the PNF with warm-up program had a significant effect on the ankle range of motion, passive tension and stiffness parameters.
4 RESULTS

4.1 Introduction

This section documents the results of the study. The pilot studies were single session tests performed on the same subject. The experiments were performed on two groups of three subjects, over a six week period for each subject. Initially we were primarily concerned with the dynamic stiffness of the ankle and so range of motion and passive stiffness were not closely investigated. However, with the second group of three subjects, these properties were carefully followed as well. The subjects completed 90.4% of the stretch sessions and none of the subjects undertook any new forms of training during the experiment.

4.2 Pilot Studies

Two pilot studies were run to evaluate the effects of a single PNF session on ankle joint dynamics. The primary motivation for these was to investigate the short-term effects of stretching on the mechanical behaviour of the ankle joint before exploring longer term effects. A secondary motivation was to assess the influence of an active warm-up on the effectiveness of a single PNF session. Hence, the first pilot study was performed without a warm-up session prior to the stretching and the second was performed with a warm-up session prior to stretching. Subject HB (28 years, 166 cm and 60.5 kg) was used for both pilot studies.

4.2.1 Pilot Study No.1: The Effects of a Single PNF Session Without Warm-Up on Ankle Joint Dynamics

Figure 4.1 shows the parameters describing the dynamic stiffness of the ankle joint, namely elastic stiffness (K), viscosity (B) and inertia (I), prior to and following a single PNF session, plotted against mean ankle position, with the subject relaxed. There was little difference between the pre- and post-PNF parameter curves.
The elastic parameter, $K$, (top plot of Figure 4.1) was minimum near the middle of the range of motion and increased by a factor of four as the ankle was plantar-flexed and by a factor of eight when it was dorsi-flexed. The viscous, $B$, and inertial, $I$, parameters (middle and bottom plots of Figure 4.1) were smallest near the middle of the range of movement and increased to toward the extremes of the range of motion but not as much as the elastic parameter. The changes in $I$ with mean joint position are puzzling and were perhaps due to estimation errors related to the inputs that were used.

Figure 4.1 Dynamic stiffness parameters vs. mean angular position before and after a single PNF session without warm-up: passive conditions, Subject HB relaxed.

Figure 4.2 shows the results of performing the same test while the subject contracted the ankle extensors at 5% MVC. The parameters varied with mean joint position in a fashion similar to that in the passive case but exhibited an increase in magnitude throughout the range of positions. This increase was more noticeable in $K$, less so in $B$ and even less in $I$. Again, as under passive conditions, there was little no difference between the parameters describing ankle dynamic stiffness before the PNF session as compared with those describing the stiffness after.
4.2.2 Pilot Study No.2: The Effects of a Single PNF Session With Warm-Up on Ankle Joint Dynamics

Figures 4.3 and 4.4 show the dynamic stiffness parameters, plotted against mean joint position, for the passive and active cases respectively, before and after a single PNF session preceded by 15 minutes of stationary cycling at a moderate intensity. As with Pilot Study No.1, with the exception of some variation in $B$ and $I$ for some mid-range and plantar-flexed positions under passive conditions, there was little effect on the parameters across the range of motion of the ankle due to a single PNF session preceded by warm-up.

The changes in $I$ that were observed in the passive trials of both pilot studies are difficult to explain and are most likely related to errors in estimation. One would certainly have expected $I$ to remain more or less invariant with respect to both ankle position and torque level, as was observed in both active trials in the pilot studies and in the long-term PNF study.
Figure 4.3 Dynamic stiffness parameters vs. mean angular position before and after a single PNF session with warm-up: passive conditions, Subject HB relaxed.

Figure 4.4 Dynamic stiffness parameters vs. mean angular position before and after a single PNF session with warm-up: active conditions, Subject HB contracting ankle extensors at 5% MVC.
4.3 Long-Term PNF Study

4.3.1 Subjective Changes

All six subjects perceived changes in their lower legs and ankles during and following the four weeks of PNF and warm-up. They used words and phrases such as “easier to stretch, feels better, more loose, flexible and strong” during and following the 4 weeks of daily stretching. Table 4.1 summarizes the comments that subjects made in interviews conducted toward the end of the stretching program.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Subjective Impressions</th>
</tr>
</thead>
<tbody>
<tr>
<td>PG</td>
<td>He noted that his lower left leg felt “more stretchable, loose, agile and strong”. He even noticed a difference in how his leg felt walking.</td>
</tr>
<tr>
<td>DD</td>
<td>“… left leg feels generally better overall… stronger.”</td>
</tr>
<tr>
<td>MD</td>
<td>During stretching he noticed that his left leg was more flexible and easier to stretch and that his ankle had a greater range of motion than before. He also noticed that when playing soccer he seemed to have “a much longer reach with his lower leg and foot”.</td>
</tr>
<tr>
<td>DH</td>
<td>Her left leg felt “more flexible, less tight during normal everyday activities”. She found that her left leg was easier to stretch following several weeks of stretching, especially as compared with her right leg which was not stretched as often.</td>
</tr>
<tr>
<td>LN</td>
<td>He remarked a noticeable change. During daily movements his left leg and ankle felt less stiff and more fluid.</td>
</tr>
<tr>
<td>PT</td>
<td>He noted that his lower legs felt more loose: “if I had to play a sport, I feel more able to do it well without even a warm-up”.</td>
</tr>
</tbody>
</table>

Table 4.1 Subjective changes noted by subjects.
4.3.2 Range of Motion

Three of the six subjects were followed carefully with respect to changes in their ankle joint range of motion over the six week experiment. The results are shown below in Figure 4.5. The first two trials were control tests and the last four were evaluative; i.e. the PNF program began after Trial #2. Ankle joint ROM increased in all three subjects by about 27% or 16° over the four weeks of stretching. This test was subjective since the subject’s foot was moved until their discomfort was too great. The trend in control tests was perhaps due to a decrease in subject anxiety or boot manipulations performed following the first control test.

![Figure 4.5](image)

**Figure 4.5** Range of motion of three subjects plotted over the six weeks.

The mean ± standard deviation increase in ROM in the training group over the four week PNF program was 0.29 ± 0.03 rad. A two tailed Student’s *t* Test between pre- (Control #2) and post-treatment (Test #4) range of motion measurements indicated significant changes (*t*=-3.94, *p*=0.02). In other words, *H₀* (the null hypothesis that the treatment had no effect on the parameter in question) could be rejected with more than 98% confidence. Control #2 was used for the pre-treatment measurement to account for the fact that the Control #1 values may have been influenced by testing procedure familiarization, subject anxiety or alterations made to the boot.
4.3.3 Passive Tension

As a first measure of the mechanical effects we superimposed torque records. Figure 4.6 shows the superimposed torque records from the position ramp trial in the six tests with Subject PG relaxed. The control tests are represented by dotted lines and the evaluative tests are shown as dashed and solid lines. This test, performed on three subjects, was used to investigate the passive tension in the ankle joint over most of the ROM.

![Figure 4.6 Superimposed torque records from ramp input for Subject PG.](image)

The torque increased in an exponential fashion as the ankle was plantar-flexed. The torque (passive tension in the ankle joint) curves exhibited a high degree of similarity over the six trials. The difference between the maximum and minimum torque recorded during these passive position ramp trials was calculated for each of the six trials and is shown in Figure 4.7. A Student's t Test, performed on the pre- (Control #2) and post- (Test #4) PNF program measurements, indicated that there was no significant change in the passive tension of the ankle due to the treatment ($t=0.74$, $p=0.50$).
4.3.4 Non-Parametric and Parametric Fits

Figure 4.8 demonstrates the quality of the non-parametric and parametric fits across all mean positions of the ankle joint. The solid line represents the percent variance in the total torque attributed to the intrinsic torque: the ankle torque due to non-reflex contributions which may include limb inertia, joint surfaces and capsule, fascia, ligaments, tendons and active and passive muscle tissue. Over 90% of the total torque variance was accounted for by the intrinsic mechanics. This was the case for all subjects for both passive and active conditions. Thus the reflex contributions were minimal.

The dashed line in figure 4.8 represents the quality of fit of the second order stiffness parameters. It shows the variance of the non-parametric compliance IRF accounted for by these parameters. All fits were very good, accounting for over 90% of the variance of the compliance IRF. This was the case for each of the tests for all of the subjects.

Figure 4.7 Torque range from passive tension test.
4.3.5 Dynamic Stiffness Parameters

The second order system parameters acquired from the system identification process, plotted as functions of mean joint position, are shown in Figure 4.9 for the passive case. These parameters (elastic stiffness, $K$, viscosity, $B$, and inertia, $I$) characterize the dynamic stiffness of the ankle joint. All six trials for a relaxed Subject PG are plotted here. Each of the three parameter vs. mean ankle angular position curves varied little over the six trials. The $K$/mean angle curves (top plot in Figure 4.9) were smallest at the mid range and moderate plantar-flexion positions, increasing by a factor of three at the plantar-flexion limit and by a factor of six as the ankle was dorsi-flexed. The $B$/mean joint position curves (middle plot in Figure 4.9) were smallest at moderate plantar-flexion, exhibiting a two fold increase in plantar-flexion and a four fold increase as the ankle was dorsi-flexed. The $I$/mean angle curves were generally constant throughout all positions. These stiffness
parameters varied with mean joint position in ways that have been seen before [8]. This general behaviour of the curves was observed in all subjects.

![Graph showing dynamic stiffness parameters](image)

**Figure 4.9** Dynamic stiffness parameters: passive conditions, Subject PG relaxed.

The same invariance in system parameters vs. mean joint position curves that was seen under passive conditions over all six trials (Figure 4.9) for all subjects was also observed under active conditions (5% MVC of ankle extensions) for all trials (Figure 4.10) for all subjects. The PNF program did not seem to influence the intrinsic stiffness of the ankle joint to any appreciable degree.
Each of the sets of curves for the various parameters for active conditions were similar in shape to those for passive conditions, except for the B vs. mean joint position curves (middle plot in Figure 4.10), whose minimum moved closer to the plantar-flexion limit. The magnitudes of the curves, however, with the exception of the I vs. mean position curves whose magnitudes remained somewhat constant, changed with the addition of the active contractions. The minimum of the K vs. mean ankle position curves (top plot of figure 4.10) increased five fold and the minimum of the B vs. mean ankle position curves doubled during ankle extensor contraction as compared with relaxed conditions.

Figure 4.10 Dynamic stiffness parameters: active conditions, 5% MVC of ankle extensors; Subject PG.
4.3.6 Passive Elastic Stiffness: $K_{\text{min}}$ and $\theta_{\text{low}}$

The minimum value of the interpolated elastic stiffness vs. mean ankle position curve (third order Chebyshev polynomial), $K_{\text{min}}$, a measure of the resting ankle joint stiffness, is shown for all six subjects across all six trials under passive conditions in Figure 4.11. In Figure 4.11, weeks one and two of the trials correspond to Controls #1 and #2.

![Figure 4.11 Minimum elastic stiffness, $K_{\text{min}}$.](image)

There was little variation in this minimum stiffness parameter for all six subjects as a result of the PNF program. A Student's $t$ Test comparing pre- (Control #2) and post- (Test #4) treatment data revealed that there was no significant change in $K_{\text{min}}$ due to the four weeks of warm-up and PNF sessions ($t=1.02$, $p=0.33$).

A second parameter that was used to characterize the passive elastic stiffness vs. mean ankle joint position curves was $\theta_{\text{low}}$. $\theta_{\text{low}}$, arrived at by adding 20 Nm/rad to $K_{\text{min}}$ and taking the angular
position spanned by the fitted elastic stiffness curve, is a measure of the "spread" of the interpolated elastic stiffness curves. It indicates the range of motion over which the elastic stiffness parameter showed low sensitivity to changes in position. Figure 4.12 shows the $\theta_{low}$ values for all six subjects over all six trials, the first two of which were the control sessions.

![Graph showing spread of $K$ curves, $\theta_{low}$](image)

**Figure 4.12** Spread of $K$ curves, $\theta_{low}$.

As Figure 4.12 demonstrates, there was very little change in $\theta_{low}$ for all subjects over the duration of the experiment. The results of a Student’s $t$ test on pre- (Control #2) and post- (Test #4) PNF program $\theta_{low}$ data for all six subjects showed an absence of any significant variation due to the treatment ($t=0.92$, $p=0.38$). The warm-up and PNF program had no effect on the ankle ROM over which $K$ demonstrated low sensitivity to changes in mean joint position.
4.3.7 Summary

The results showed that there was a significant increase in ankle ROM due to the PNF treatment. All of the investigative strategies implemented, however, failed to demonstrate a significant change in the mechanical properties of the ankle. Ankle dynamics remained invariant even after four weeks of twice daily PNF stretching. Table 4.9 tabulates the results of mean ± standard deviation, before and after the stretching intervention, of the parameters investigated as well as the results of the two-tailed Student’s t Tests performed on them.

<table>
<thead>
<tr>
<th>variable</th>
<th>Pre PNF Program</th>
<th>Post PNF Program</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>ROM [rad]</td>
<td>1.04 ± 0.06</td>
<td>1.32 ± 0.09</td>
<td>-3.94</td>
<td>0.02</td>
</tr>
<tr>
<td>ΔT [Nm]</td>
<td>16.07 ± 1.52</td>
<td>15.12 ± 1.05</td>
<td>0.74</td>
<td>0.50</td>
</tr>
<tr>
<td>K_{min} [Nm/rad]</td>
<td>23.55 ± 5.26</td>
<td>20.07 ± 5.56</td>
<td>1.02</td>
<td>0.33</td>
</tr>
<tr>
<td>θ_{low} [rad]</td>
<td>0.39 ± 0.06</td>
<td>0.35 ± 0.04</td>
<td>0.92</td>
<td>0.38</td>
</tr>
</tbody>
</table>

Table 4.2 Results of two-tailed Student’s t Tests comparing pre- and post stretching program (i.e. between Control #2 and Test #4) parameters.
5 DISCUSSION

5.1 Summary of Study

System identification techniques were used to obtain a parametric description of the mechanical stiffness of the ankle musculo-articular system in terms of elastic stiffness, viscosity and inertia. This description of the mechanical behaviour of the ankle joint provided an objective basis for evaluating the effects of a long-term stretching with warm-up program. Proprioceptive neuromuscular facilitation (PNF) was performed twice daily on the ankle flexors and extensors of six healthy subjects for four weeks. The question that we asked in this study was: "Does stretching have an effect on the dynamic stiffness of the ankle joint?"

5.2 Feasibility and Sensitivity of System Identification Approach

Kearney et al examined the inter-subject variability and intra-subject reliability of quantitative measures, arrived at through system identification, describing normal human ankle mechanics in fifteen young adults [86]. They found that the measures of passive and active joint dynamics generally had an intra-subject reliability which was as good as or better than most clinical measures. As was expected and true for clinical measures, the inter-subject variability was larger. It was concluded that measures of joint dynamics acquired using system identification are suitable for use in the clinical assessment of joint function, especially for longitudinal studies.

This system identification approach is sensitive enough to detect changes in joint stiffness with minor contractions of ankle extensors. Weiss et al found that with only 1 Nm of torque created by the active contraction of the triceps surae that the elastic stiffness of the ankle joint increased by 15 to 30 Nm/rad depending on the mean joint position [84]. In our study it was observed that the ankle elastic stiffness curves were displaced by as much as 100 Nm/rad when only 5 Nm of torque was produced in a contraction. Thus, the quantitative measures describing ankle joint behaviour have sufficient intra-subject reliability and sensitivity to external influences to be used to evaluate the
effects due to stretching. If there were significant changes in ankle dynamics we believe they would have been detected.

5.3 Summary of Results
The pilot studies demonstrated that a single PNF session either with or without warming-up before did not influence ankle stiffness to any appreciable degree.

We found that the four week PNF with warm-up program resulted in:
1) a significant increase in ankle joint ROM,
2) definite changes Perceived by the subject in ankle joint behaviour, but
3) no measurable changes in ankle joint mechanics within the range of motion tested.

We observed an invariability in ankle passive tension as well as in the elastic stiffness, viscosity and inertia vs. mean ankle position curves, following four weeks of stretching. Over the six trials the passive K vs. mean position curves varied less than changes that would be expected due to a 1 Nm producing contraction of the ankle extensors. Given these observations, it is highly unlikely that any changes in the mechanical properties of the ankle occurred; there was an absence of adaptation of the musculo-articular structure responsible for moving the ankle in the sagittal plane.

Within the majority of the ROM of the ankle joint, that is excluding the extremes, stretching did not significantly affect the dynamic stiffness of the ankle joint. We found then, that stretching made the subjects more flexible (i.e. greater ROM around a specific joint) but that it did not make their joints less stiff. These results were surprising and not what the author expected at all. A change in the elastic stiffness was expected. This study demonstrates that increases in joint ROM can be achieved without an accompanying change in the mechanical properties of the joint.

5.4 Possible Explanations of Results
There are several possible mechanisms that could account for the observed increase in ankle joint range of movement and subjective changes with no change in ankle stiffness. Some explanations are more plausible than others. These include: (i) that changes may have happened at extreme
positions of the joint to allow for an increase in ROM but did not influence the joint at more moderate positions, (ii) changes in connective tissue or (iii) in sarcomere-related structures, (iv) sarcomere remodelling or, and most likely, (v) neurophysiological factors related to perception and proprioception (increase in stretch tolerance). According to Halbertsma and Goeken [26], an increase in joint ROM can be achieved by one of two ways: a change in the mechanical properties of the tissue about the joint or by an increase in pain tolerance, i.e. stretch tolerance.

5.4.1 Avoidance of Extreme Positions

The results of our study do not support those of Toft et al [9] who found that the mechanical properties of the ankle joint did change after a three week stretching program. In their experiments, they implemented a twice-daily contract-relax stretching strategy on the plantar-flexors for three weeks and found that the passive tension in the plantar-flexors was lowered by up to 36%. Although these authors purported to have seen changes due to stretching in properties of passive tissue, even at muscle lengths used during walking and running, this tension reduction was most significant at extreme positions in the ROM.

One way of reconciling this difference in findings is to remark that in our experiments the ankle joint was not moved to the very extremes of the range of motion for both safety and practical reasons. For comparison purposes, the same positions were used for all six tests. For all subjects this meant testing a ROM of between 0.80 to 0.90 radians. The achievable ROM was found to increase to ~1.30 radians, so in time the % ROM tested decreased. If the extremes were tested as well perhaps changes in joint stiffness would have been observed. Stretching may reduce stiffness at the very ends of ROM but with the experimental paradigm that we have in our lab we are not able to safely (without hurting the subject) or practically evaluate this. The practical considerations arise from the fact that the foot needs to be rigidly fixed and at the same time be allowed to freely move around the talo-crural axis of rotation.

So then, possibly there were changes in the mechanical properties of the joint that were not observed because they occurred at the untested, extreme positions. This is perhaps the simplest explanation but it still does not address the fact that there were no changes in the mechanical properties of the
ankle joint throughout the rest of the range of motion (over three-quarters of the ROM at that!). Indeed, studies performed by both Magnusson et al [25] and Halbertsma et al [26] on the hamstrings and hip joint observed similar things as our study. They found that long term stretching increased the range of motion but did not influence the mechanical properties of the joint. Are there other possible explanations for what we observed?

5.4.2 Changes in Connective Tissue

It has been suggested that the greatest long term gains in stretching are related to the permanent deformation of connective tissue structures [9, 22]. Viidik investigated the corrugated nature of collagen fibril in unstretched rabbit Achilles tendon. He found that with elongation, the waviness disappeared gradually and simultaneously with an exponential increase in passive tension [24]. It is difficult to say, though, whether or not these changes were due to loading the tendon beyond conditions experienced in physiologic conditions.

Although a permanent deformation in the connective tissue might account for the gains in ankle joint flexibility that were observed, this does not explain the lack of appreciable changes in dynamic properties of the ankle joint. If the length of the connective tissue were altered one would have expected this to influence the elastic stiffness of the joint. Moreover, it is known that the passive mechanical behaviour of isolated collagenous materials such as tendon, ligaments, fascia and skin is characterized by transient compliance following repeated and maintained extension [27, 28]. Any changes in the connective tissue, then, were most likely transient due to the visco-elastic accommodation of the collagen fibres. Connective tissue changes probably did not account for the long-term gains in ankle ROM that were observed.

5.4.3 Endo- and Exo-Sarcomere Skeleton

For another possible explanation of the results, one may look to the elastic properties of the sarcomere infrastructure as well as the exo-sarcomere lattice. In direct contradiction to Toft et al [9] and Sapega et al [22], Wang et al [32] point out that within the physiological range of muscle length changes, the myofibrillar structures are the major source of elasticity and that extracellular connective tissues (fascia, etc.) contribute only in highly extended skeletal muscles. As was
intimated in Chapter 2, when a sarcomere is stretched, titin behaves like a dual stage molecular spring with two distinct degrees of reversible extendibility (a passive tension stage followed by a structural fail-safe stage) [30, 32]. The elastic properties cause the tissue to revert back to its original length [31]. Any gains made in the increase in muscle length due to the elastic nature of the infrastructure of the sarcomere appear to be transient. It would seem that, except at extreme, non-physiologic positions, all stretching effects on titin and nebulin and extra-sarcomere structures are reversible due to the visco-elastic accommodation that they exhibit. Permanent changes to the endo- or exo-skeleton of the sarcomere do not appear to be a plausible explanation for the long-term ankle joint range of motion increases due to the PNF program.

5.4.4 Sarcomere Remodeling

Another possible explanation for the increase in ankle joint range of motion that needs to be considered is that the stretching program caused the serial increase of sarcomeres within the muscle fibres. These changes, however, would have most likely produced a change in ankle mechanics as well. Animal studies have shown that if a muscle is placed under tension for an extended period of time (hours a day up to days at a time), there is growth in the length of the tissue that is achieved by the serial addition of sarcomeres at the ends of the myofibrils [97-99]. The mechanism that accounts for this is similar in nature to that which occurs during the postnatal growth of skeletal muscle, as the muscle adapts to the lengthening of the limbs that it is attached to [98]. Thus the muscle is then able to function normally at the new limb length [99].

Williams performed an interesting study in which he sought to determine whether short daily periods of stretch prevent sarcomere loss in immobilised ankle extensors in mice [97]. When muscle is immobilised in a shortened position there is a reduction in muscle fibre length due to a loss of serial sarcomeres and a change in intramuscular connective tissue that is believed to lead to an increase in joint stiffness and losses in joint range of motion [97]. Morier [100] and Chesworth et al [101] have performed studies involving ankle fracture patients that have shown an increase in passive elastic stiffness in the joint and reduction in ankle joint range of motion due to the immobilisation of the joints. Williams found that over a two week period, half an hour of sustained
stretch daily was required to prevent sarcomere loss and two hours of daily stretching was needed to increase the number of serial sarcomeres in a given muscle fibre by 10%.

Given the fact that in our study the lower leg muscles of the subjects were not immobilised between stretching sessions and that daily a given target muscle was in an extended position for at most 90 seconds, it seems highly unlikely that any appreciable amount of sarcomere remodeling took place in the subjects due to the PNF program. This is therefore not a plausible explanation for the experimental results that were observed.

5.4.5 Perception and Proprioception – Increased Tolerance to Stretching

This brings us to our fifth, and perhaps most likely, conjecture to describe the mechanism behind what we observed. Despite the fact that human studies have shown that habitual stretching produces chronic increases in joint ROM, it has never been confirmed that tissue properties are affected by stretch training [25]. Tissue properties may be affected by repeated stretches but the change is transient in nature and the visco-elastic properties return to baseline values within one hour. It can not be excluded, however, that rigorous forms of stretching regimens, such as in ballet or gymnastics, over several years is a sufficient stimulus to yield a lasting change in the tissue properties [25]. Another possible explanation for the results is that the stretching program served to increase the tolerance to stretch of the subjects. The mechanism for an altered stretch tolerance is unknown [25] but it may be explained by neurophysiological mechanisms related to perception, proprioception and other psychophysical aspects.

According to psychophysics, that area of neurophysiology that involves the relationship between the physical characteristics of a stimulus and the attributes of the sensory experience, what we perceive differs qualitatively from the physical properties of stimuli. This is because the nervous system only extracts certain information from a stimulus and then interprets this information in the context of previous experience [39].

It has been demonstrated that although people are able to accurately perceive the position of a limb [39], they are less capable when it comes to perceiving changes in tension, and consequently
stiffness, in a muscle [102]. Furthermore experiments have shown that even though the human proprioceptive system has the capacity to discriminate changes in the stiffness and viscosity of an external mechanical system, there is considerable intra-subject variability in judging both stiffness [103] and viscosity [104].

The afferent nerves that innervate joint receptors, mechanoreceptors located in joint capsules, that, along with muscle spindles and Golgi tendon organs, contribute to sensing the position and movement of limbs (limb proprioception) are known to be especially sensitive to extremes of joint angle. Afferent nerve fibres also transmit sensations of pain, elicited by noxious or tissue damaging stimuli and mediated by mechanical nociceptors, to the central nervous system [39].

Perhaps the subjects experienced a training effect during the four weeks of PNF. The repeated conditioning of joint receptors and mechanical nociceptors, through frequent stretching may have stimulated a greater tolerance to extreme positions, which in turn accounted for the observed increase in ankle joint range of movement. So then, the subjects grew in their tolerance of stretching; they became more comfortable with moving further into their ankle ROM. This was perhaps influenced by a decreasing sense of pain, familiar afferent signals from proprioceptors and other psychophysical factors. This explanation is compatible with those of Halbertsma and Goeken [26] and Magnusson et al [25] who obtained findings similar to ours. They explained their results (increase in ROM and invariance of stiffness) as an increased tolerance to stretching by the subjects and not due to a change in the mechanical or visco-elastic properties of muscle. Specifically, Magnusson felt that this stretch tolerance could be explained with respect to nociceptive nerve endings in joint or muscle [25].

These perception issues address the discrepancy between subjective impressions and invariability of ankle joint stiffness as well as the increased stretch tolerance that allowed for an increase in ROM. Perceived stiffness is different than mechanical stiffness. The increase in ROM observed may be reconciled with the invariability of ankle stiffness due to an increased tolerance to stretching mediated by joint and pain receptor involvement.
5.5 Significance of Results

The results of this work are significant from a number of standpoints. They speak to the nature of the effects of stretching, how we define flexibility and stiffness and the marked difference between perceived stiffness and mechanical stiffness.

5.5.1 The Effects of Stretching

Traditionally, most studies evaluating the effect of stretching are based on changes in the range of motion of a joint. This approach limits the investigation to the effects of stretching exercises on muscles and tendons at the extreme positions of the joints. As well, this approach is somewhat subjective and makes it very difficult to distinguish between changes in stretch tolerance as opposed to changes in muscle stiffness.

The study performed for this thesis used more objective measures of ankle joint behaviour to investigate changes in the joint due to stretching. The results of this study suggest that stretching to increase flexibility has more to do with increasing the comfort or ease of moving more deeply into one’s ROM than it does with changing the mechanical properties of the joint. With confidence this can be said to be true within the majority of the positions in the ROM, excluding the extremes. In other words, engaging in long term, habitual stretching serves to increase one’s tolerance for stretching. This manifests itself in an increase in joint range of motion. How the joint interacts with its environment vis à vis its mechanical properties appears to be invariant after long term stretching. These results support the findings of Magnusson et al [25] and Halbertsma and Goeken [26].

The results of this study do not support the hypothesis that stretching makes joints more compliant in the long term. Certainly there are acute changes in joint compliance that appear to be due the visco-elastic accommodation of muscle and/or connective tissue, however chronic changes in ROM are due to an increased stretch tolerance.

In light of these results it is possible that stretching may simply affect the way muscles “feel” without influencing the way they actuate joints to interact with their environment. Is it possible that because of the complexity of the human animal that stretching may even improve performance and
help to prevent injury without affecting ankle stiffness? Could an increased tolerance to stretching and the subjective changes that stretching effects be that important and account for the widely accepted perceived benefits of stretching?

5.5.2 Flexibility and Stiffness

This study begs the question as to how to define flexibility. Magnusson feels that flexibility is better regarded as joint range of motion rather than compliance [25]. Flexibility is directly related to the range of movement attainable in joint. Stiffness is a property of the joint system that is characterized by the way angular position and torque are related by the system. The two are not as related as one might first think. Lack of flexibility in a joint does not necessarily correspond to a stiff joint and vice versa.

Confusion arises if the popular definitions of flexibility and stiffness are adhered to. Collins offers the following definitions: flexible = able to be bent easily without breaking, pliable; and stiff = not easily bent; rigid; inflexible [12]. If we understand flexibility as an increase in ROM then yes, the ankle joints of the subjects in this study became more flexible. If, however, we understand flexibility to be synonymous with compliance (the inverse of stiffness) then, no, the subjects' ankles did not become more flexible. Technically speaking it is not correct to say that a flexible joint is more compliant or less stiff.

5.5.3 Perceived Stiffness Versus Mechanical Stiffness

Another issue that this study raises is that of perception. That which we perceive may not actually be happening. For instance with respect to stiffness, is it a property of a joint that is position and rate dependent or is it a sensation related to other factors?

Perceived stiffness is different than mechanical stiffness. Subjects sensed a change in flexibility and not in stiffness. This is related to a greater tolerance to discomfort and deeper stretching. In light of the observations in this study the joint stiffness perceived by the individual does not correlate with actual measurable changes in the joint mechanics. Stretching allowed the subjects to more comfortably move into their full ROM. This was perceived by the individual as a decrease in joint.
stiffness. In reality, however, the mechanical properties of the joint, within the most of the ROM, were not significantly influenced by stretching.

5.6 Potential Implications

The findings of this study have a number of implications for the physical therapy, athletic and scientific communities. How does stretching influence injuries? How does stretching influence athletic performance? How is the conduction of experiments and interpretation of their results implicated by this study?

5.6.1 Stretching and Injury Prevention

If the main effect of stretching is simply an increased tolerance to stretching, is it important for the prevention of injuries? What are the implications here? The sports medicine community generally believes that muscle tightness increases the risk of muscle sprains, muscle ruptures and tendinitis [9]. Toft et al found that muscle tightness decreases in a wide range of motion after stretching, emphasizing the possible beneficial effect of stretching on the incidence of muscle injuries in sports [9]. This was not found to be true in this study and by others [25, 26]. There were no functional changes in the joint in the majority of the ROM due to stretching.

These results indicate that stretching does not really do anything to make the joint behave differently within its functional range of movement. If stretching does not influence long-term muscle joint stiffness, could it prevent injuries? Although the widely held notion is that is does, the literature is inconclusive on this. Gleim and McHugh point out that no conclusive statements can be made about the relationship between flexibility and athletic injury and that divergent views on the importance of injury prevention, as well as athletic performance, are the rule [43]. Van Michelen et al [105] found that when they evaluated the effect of a health education intervention (that involved warm-up, cool-down and stretching exercises) on running injuries in a 16 week study in Amsterdam, with hundreds of subjects (n=326), the incidence of running injuries was not reduced.

Another interesting question raised by the results of our study is this: If a perception of stiffness limits or protects our joints by helping us to keep them within reasonable functional limits, is it possible to do more damage by stretching by removing the body’s protective mechanisms? Thus
through increasing flexibility we may not “hear” the body’s warning signals and put our bodies in a greater risk of injury. These thoughts are congruent with those who believe that stretching may even increase the risk of injury [1, 42].

5.6.2 Stretching and Performance

Another issue implicated by this study is the impact of improved flexibility on athletic performance. If stretching merely increasing one’s tolerance to stretching, is it important for athletes to stretch? Is it possible for an athlete to improve their performance through increasing the flexibility of their joints even if the joint dynamics remain invariant? How critical are the psychophysical factors that are influenced by stretching?

Acute stretching exercises were found to have no effect on the oxygen cost or speed of a 100 yard (91.4m) dash [43] and DeVries showed that increased flexibility due to stretching had little or no effect on economy of exercise [1]. In fact studies have shown that “tight” runners were 12% more economical than their “loose” counterparts [43]. While increased flexibility is important for performance in some sports that rely on extremes of motion for movement (gymnastics, swimming, goalkeeping in soccer), decreased flexibility may actually increase economy of movement in sports which use only the mid portion of ROM (running, linemen in American football, field players in soccer) [43]. It is quite possible that warming up itself may have a greater impact on athletic performance than does stretching.

5.6.3 Experimental Design and Interpretation of Results

Clearly it is not sensible to conclude that an increase in ROM is due to a change in the mechanical properties of the joint in question. Frequently, however, researchers have jumped to this conclusion. The findings of this study challenge the protocol of experiments that use subjective ROM measures as a basis of investigation as well as the interpretation of those results.
5.7 Recommendations for Future Work

There are a number of different initiatives that could be taken in the future to further explore issues raised by, and related to, this study.

5.7.1 Looking Into ROM Extremes

We examined roughly the middle three-quarters of each subject's maximum range of motion, but it is possible that there were changes occurring at the extremes of the ROM. It would be instructive to devise a way to look further into the ankle ROM using this system identification approach. This would require redefining the safety measures and the method of limb fixation currently in place or, at the very least, enlisting very brave subjects with a high pain tolerance.

5.7.2 Ramp in Pilot Study

Stretching produces transient changes in joint stiffness. These changes disappear given enough time due to the visco-elastic accommodation of the tissue involved. Unlike the experiments in the main portion of the thesis, the single PNF session pilot studies that we performed did not examine the passive tension in the ankle with an input ramp of angular position. The pilot studies we implemented looked only at the system identified parameters describing ankle stiffness. Since it took at least 30 minutes to move through all of the positions, it is possible that the transient effects of the stretching dissipated before adequate measurements were taken. (Kirsch et al found that the transient effects following a single 60 s maintained stretch disappeared after only 300 s of rest [27].) By using the ramp of angular position as an input, information could be gathered within minutes of the single stretch session increasing the likelihood for observing any short term transient changes in passive tension and thus elastic stiffness.

5.7.3 Effect of Stretching on Reflexes

It would be interesting to explore the effect of long term PNF stretching on the reflexes that influence the ankle. Using the parallel cascade model it is possible to look at the reflex contribution to the total torque seen at the ankle. This information was neglected in the study given that the reflex torque contributions were minimal (5 to 10%) compared to the intrinsic contributions (90 to
95%). Reflex behaviour could also be examined through the use of impulses in ankle angular position.

5.7.4 Stretching, Lower Leg Pathologies and Ankle Joint Dynamics

The present work could easily lead to future investigations involving subjects with musculo-skeletal injuries. Among recreational athletes, the presence of muscle fibre adhesions, scar tissue and collagen misalignment in lower leg tissues manifest themselves in chronic injuries that include shin-splints, Achilles tendinitis, plantar fasciitis and calf tightness. The impact of these lower leg pathologies on ankle joint dynamics as well as the degree to which these pathologies are influenced by stretching or other forms of therapy could be investigated.

5.7.5 Strengthening and Ankle Joint Dynamics

In the future this work could also be extended to look at the effects of strengthening muscles on joint dynamics. A study performed by Klinge et al indicated that an increase in isometric strength is accompanied by changes in the material properties of the muscle that are unaffected by flexibility exercises [106]. This could be verified through an experiment with a similar design to the one implemented in this thesis, but targeting the triceps surae and tibialis anterior muscles in a strength training regimen.
6 CONCLUSIONS

The question that was posed at the beginning of this study was: "Does stretching have an effect on ankle joint dynamics?" The short answer to this question is no. The results showed that although there was on average a 27% increase in ankle joint range of motion (ROM) due to four weeks of twice daily stretching, the parameters describing the ankle stiffness remained invariant. In other words, long-term proprioceptive neuromuscular facilitation (PNF) preceded by a warm-up did not have a noteworthy effect on ankle joint stiffness. This supports the findings of other investigators [25, 26]. It would appear that long-term stretching improves joint ROM due to increases in the stretch tolerance of a joint and not changes in the mechanical properties of the joint.
Impulse Response Functions

There are a number of different ways of characterizing a linear time-invariant system. One way is by evaluating its response to an impulse. An impulse response function can be used to predict the response of a linear system to any input and so it can be used to represent that system. Convolution is a key component of assessing the output of a system as super-positioned, scaled, time delayed series of impulse inputs [79, 96, 107]. An impulse response function may be represented by a curve (parametric representation) or by an equation (non-parametric representation). The structure of the equation defines the class of systems that it represents. Equation A.1 describes an impulse response function or filter, \( h(t) \), relating the input, \( x(t) \), to the output, \( y(t) \).

\[
y(t) = \int_{-\infty}^{\infty} h(\tau) \cdot x(t - \tau) d\tau \tag{A.1}
\]

Chebyshev Polynomials

The general form of the Chebyshev polynomial is shown in Equation A.2 below. Chebyshev polynomials were used for curve fitting in favour of regular polynomials for 2 reasons:

1. Numerically they are better behaved; they fit higher order expressions better.
2. All Chebyshev polynomials have a peak amplitude of 1. At all of the maxima \( T_n(x) = 1 \), while at all of the minima \( T_n(x) = -1 \); it is precisely this property that makes the Chebyshev polynomials so useful in polynomial approximation of functions

\[
T_n(x) = \cos(n \cdot \arccos(x)) \tag{A.2}
\]

Chebyshev polynomials of the third order were fitted to the intrinsic elastic stiffness vs. mean joint position curves. This was done in order to have a stronger basis for comparing position-dependent \( K \) parameter changes for each subject over the six week experiment.
Mean and Variance

The mean and variance of a given variable under investigation formed the basis for all statistical work employed. The mean value of a signal is equivalent to the expected value of a signal \[108\], and is defined as

\[
\mu_x = E[x] = \lim_{T \to \infty} \left[ \frac{1}{T} \int_0^T x(t) dt \right]
\]

(A.3)

The variance of a signal is a measure of the degree to which a signal deviates from its mean value \[108\]. Equation A.4 gives the mathematical definition of the variance of a signal:

\[
\sigma_x^2 = E[(x - \mu_x)^2]
\]

(A.4)

Variance Accounted For (VAF)

The non-parametric and parametric models were assessed in terms of their accuracy through the use of the test statistic, variance accounted for (\%VAF). If \(\hat{y}\) is an estimate of the variable, \(y\), then \%VAF is defined as:

\[
\%\text{VAF} = 100 \times \left[ 1 - \left( \frac{\text{var}(y - \hat{y})}{\text{var}(y)} \right) \right]
\]

(A.5)

Note that if the residuals are larger than the signal itself, \%VAF will be negative \[109\]. The variance accounted for evaluated how well an impulse response function described a system by comparing the output depicted by the IRF with the output observed experimentally. The compliance IRFs were assessed in terms of the torque variance that they accounted for. The validity of the parametric fit was assessed in terms of the percentage of the variance of the IRF accounted for by the parametric model. In all cases, the VAF by both the non-parametric and parametric models was greater than 90%.
**Student's t Test**

The Student's t Test is a test of hypothesis and significance, designed to compare individual values within a set of data [110]. It was used to ascertain if there were any differences between the population of data prior to and after stretching. The test was set up in the following way:

If \( \mu_{\text{pre}} \) and \( \mu_{\text{post}} \) denote the population means for pre- and post-treatment data, a decision needs to be made between the following null (\( H_0 \)) and alternative (\( H_1 \)) hypothesis:

- \( H_0: \mu_{\text{pre}} = \mu_{\text{post}} \) there is essentially no difference between the groups; the PNF program had no effect.
- \( H_1: \mu_{\text{pre}} \neq \mu_{\text{post}} \) there is a significant difference between the groups; the PNF program had an effect.

Thus, initially it was assumed that there was essentially no difference between the statistical properties of the pre- and post-stretching values of a certain parameter. If this hypothesis was rejected, based on examination of a derived \( t \) distribution of the random variables, then it was concluded that there was a significant difference between the pre- and post-stretching values of the parameter [110].
8 REFERENCES


100. Morier, R., *Dynamic joint mechanics as an objective clinical measure of ankle function*, in *School of Physical and Occupational Therapy*. 1988, McGill University: Montreal, Canada.


