to my mum and dad

in memory of my eternal grandmothers (Umbelina and Antónia)

Technical University of Lisbon
Faculty of Human Kinetics

Effect of head, trunk and foot position on knee passive extension torque-angle response

Thesis submitted in the fulfillment of the requirements for the Degree of Master of Science (MSc) in High Performance Training

PROVISORY DOCUMENT

Advisor: Doutor Pedro Pezarat-Correia

Co-advisor: Professor Sandro R. Freitas

RICARDO JORGE NÓBREGA ANDRADE

2013

Acknowledgments

The reality of completing a MSc is that my efforts would not have been possible without the help of several people, involved at different stages throughout the process. I am very grateful for all those who have contributed to this MSc and would like to take this opportunity to thank them. In particular I would like to thank you very much:

Professor Sandro Freitas, PhD candidate, for your never-ending patience and generous giving of your time and skills. I am very grateful for you guiding me through this work; rigorous and motivator Professor Pedro Pezarat-Correia, PhD; always available João Vaz, PhD candidate; Professor Paula Bruno, PhD, for statistical support; Professor Pedro Mil-Homens, PhD; Tiago Neto, MSc; Professor Maria João Valamatos, MSc; Professor Raúl Oliveira, PhD; my MSc journey new friends Daniel Vilarinho, André Lourenço, David Sousa, Luís Barnabé, and Coach Paulo Reis (double thanks for you); old and friend Professor Joaquim Sousa; my supportive and friendly family for times not spent with them (special gratitude to godfather Maurílio Gouveia; Cirilo Vieira; Carlos Gouveia; Mónica Gouveia; Fátima Vieira; Miguel Andrade, Alexandra David and their young Martim, Carolina and Madalena); engineer Diogo Carvalho (articulate device mentor) and his future wife Catarina Marques; Tomé Marques and Joana Silva; my fantastic sister Ana Raquel Nóbrega Andrade; my parents (Jaime Andrade e Teresa Nóbrega) for your never-ending encouragement to fully explore my curiosity. My gratitude and my admiration see no limitations; my more than ever Inês Dos Santos Marques (almost coworker).

Abbreviations and symbols

%	Percent
αAnkle	Ankle angle
αCervical	Cervical angle
αHip	Hip angle
αKnee	Knee angle
αThoracic	Thoracic angle
Cm	Centimeters
EMG	Electromyography
F	Force
F_p	Passive resistance to knee extension
Hz	Hertz
Kg	Kilograms
L	Length
L_{leg}	Leg length
MAC	Musculoarticular complex
MSS	Musculoskeletal system
MTC	Muscle-tendinous complex
MVIC	Maximal voluntary isometric contraction
N m	Newton meter
^{o}	Degree
PEC	Parallel elastic component
PKE	Passive knee extension
PNS	Peripheral nervous system
PNS	Peripheral nervous system
PT	Knee passive torque
RMS	Root mean square

ROM	Range of movement
SEC	Series elastic component
SEC’s	Series elastic components
sEMG	Surface electromyography
SI	International System of Units
SREC	Short Range Elastic Component
SSI	Supersonic shear imaging
ST_{EMG}	Semintendinousus electromyography
T- θ	Torque-angle
VAS	Visual analogic scale
VM_{EMG}	Quadriceps vastus medialis electromyography

Table of contents

List of figures

List of tables

Abstract

Cadavers and *in vivo* studies have shown that cervical, thoracic and ankle movements cause changes in position and tension of peripheral nervous system (PNS). However, it is not clear if the head, trunk, and foot position affects torque-angle response of knee joint. The aim of this study was to compare the knee passive extension torque-angle response at different combinations of foot, trunk, and head testing position. Ten male subjects (with maximum passive knee extension (PKE) test deficit) performed one repetition of a PKE with a velocity of 2º.s^{-1} in six randomized experimental conditions: neutral position (N); maximal active ankle dorsiflexion (AD); maximal active cervical and thoracic flexion (CTF); and cervical and thoracic maximal flexion with ankle on maximal active dorsiflexion (CTFAD); maximal cervical extension with thoracic flexion (CETF); and maximal active cervical extension with thoracic flexion and ankle on maximal active dorsiflexion (CETFAD). Subjects were instructed to perform the maximum range of motion (ROM) without feeling pain or discomfort. The visual analog scale (VAS) was used to access subject's stretching intensity perception. Cervical and thoracic positions were performed with an articulate device specifically designed to this study. Ankle taping was used to hold maximal active dorsiflexion. ROM was assessed by kinematic data 2D analysis. Passive torque (PT) was measured with a specific apparatus designed to move the leg attached to the dynamometer (Biodex®). Electromyography (EMG) was measure in order to assure complete passive condition during the stretching maneuvers. Data was synchronized and processed by a MATLAB® routine. From our results foot position may affect stretch tolerance since it affects ROM and PT at maximum values accepted by subjects. Head and trunk positions affect PT at submaximal common ROMs. Stretch discomfort and EMG response were unaffected by head, trunk and foot positions. No significant differences were seen between interaction of ankle and upper body positions for ROM, PT, EMG and VAS. Knee passive torque-angle response is influenced by head, trunk and foot positions. Cervical and thoracic flexion increases PT during PKE movement. This matter should be considered at knee torque-angle measures and in clinical tests of PNS mobilization and tension.

Key words: Torque-angle, neurodynamics, knee, stretching, slump test, range of motion, nerve tissue, muscle extensibility, viscoelasticity, flexibility

Resumo

Estudos realizados em cadáveres e *in vivo* têm demonstrado que os movimentos da coluna cervical, torácica e tibiotársica provocam variações de posição e de tensão ao nível do sistema nervoso periférico (SNP). Contudo, não é claro se o posicionamento da cabeça, tronco e pé afecta a relação 'momento-ângulo' do joelho. O objectivo deste estudo foi comparar a relação 'momento-ângulo' durante a extensão passiva do joelho (EPJ) em testes com diferentes combinações do pé, tronco e cabeça. Dez sujeitos (com défice no teste EPJ) efectuaram uma repetição de EPJ, a uma velocidade de $2^{o}.s^{-1}$, em seis condições experimentais aplicadas segundo uma ordem aleatória balançada: posição neutra (N); máxima dorsiflexão activa da tibiotársica (P); máxima flexão activa da coluna cervical e torácica (FCT); máxima flexão activa da coluna cervical e torácica e máxima dorsiflexão activa da tibiotársica (FCTP); máxima extensão activa da coluna cervical e máxima flexão activa da coluna torácica (ECFT); e máxima extensão activa da coluna cervical e máxima flexão activa da coluna torácica, e máxima dorsiflexão activa da tibiotársica (ECFTP). Os sujeitos foram instruídos a efectuar máxima amplitude de movimento (ADM) sem sentirem dor ou desconforto. A escala visual analógica (EVA) foi aplicada para avaliar a intensidade da percepção ao alongamento. Os posicionamentos da coluna cervical e torácica foram realizados com a ajuda de um dispositivo articulado concebido especialmente para este estudo. A máxima dorsiflexão activa da tibiotársica foi assegurada com uma ligadura. A ADM foi avaliada com recurso a análise cinemática 2D. O momento de força articular do joelho (MF) foi medido com um dispositivo desenhado para encaixar num dinamómetro (Biodex®) de forma a medir a resistência durante a EPJ. A actividade electromiográfica (EMG) dos músculos em alongamento foi medida para assegurar uma condição passiva no deslocamento da perna. Os dados recolhidos foram processados e sincronizados através de uma rotina MatLab® especificamente desenhada para o efeito. Os resultados patenteiam que a posição do pé pode afectar a tolerância ao alongamento uma vez que ela afecta os valores máximos de ADM e MF tolerados pelos sujeitos. As posições da cabeça e do tronco afectam o MF em AMD's comuns submáximas. O desconforto ao alongamento e a resposta EMG não foram afectados pelos posicionamentos da cabeça, tronco e pé. Não foram identificadas diferenças significativas entre a interacção dos factores posição da tibiotársica e posição do tronco superior para a ADM, MF, EMG e EVA. A relação 'momento-ângulo' passiva do joelho é influenciada pela posição da cabeça, tronco e pé. A flexão da coluna cervical e torácica aumenta o MF durante o movimento de EPJ. Este resultado deverá ser tido em consideração em medidas de 'momento-ângulo' e em testes clínicos de mobilização e tensão do SNP.

Palavras-chave: Momento-ângulo, neurodinâmica, joelho, alongamento, slump test, amplitude de movimento, tecido neural, extensibilidade muscular, viscoelasticidade, flexibilidade

I. Introduction

The production of muscle strength allows motion of members and maintenance of posture. Given the importance of these functions, it is conceivable that the study of all phenomena that regulates the musculoskeletal system (MSS) is a major challenge to science of human kinetics. In this context, the characterization of the mechanical properties of muscles has been helped researchers to understand better its behavior and some of its adaptive mechanisms in response to different stress (e.g. strength training protocols, aging, and neuromuscular disease). The mechanical behavior of muscle during contraction can be studied through some models which have the advantage of considering isolated muscle as well as that of an *in vivo* muscle group (e.g. Hill, 1938, 1951).

Other methods of mechanical behavior of isolated muscle experiments have used conventional resources to study the muscle and its passive properties (e.g. can be made cycles of charge/discharge at different rates, relaxation and creep tests (Fung, 1983)). These tests consist in exposing the muscle to different stretching procedures and measure the tension developed in resistance to each stretch. Several studies have adjusted the model of Hill (1938, 1951) to make it compatible with experimental data obtained from muscles under passive conditions. These models describe passive mechanical properties of muscles: for example, they analyzed the incidence of genetic diseases (Anderson, Li, & Goubel, 2002; Wolff et al., 2006), various stretching protocols (Taylor, Dalton, Seaber, & Garrett, 1990) or simulate the response of muscle exposed to various stretch protocols (Speich et al., 2006). *In vivo*, it has been shown that it is possible to implement characterization protocols similar to those used on the isolated muscle. Passive mechanical properties of the musculoarticular complex (MAC) can be determined (Magnusson, 1998). However, this kind of methodology has mainly been used in clinical research studies in order to describe flexibility between different populations or changes in joint maximum range of motion (ROM) (Goubel & Lensel-Corbeil, 2003). Thus, the understanding of the passive properties behavior of MAC still unclear (i.e. very few models of the mechanical behavior of this system have been implemented in the literature among last years) and should continue to be studied. Furthermore, the properties of the MAC seem to be modified by external stress (e.g. as a result of stretching protocols in sport or in functional rehabilitation). However, the mechanical outcomes of stretching (i.e. acute and chronic effects) are somewhat inconsistent regarding literature and its potential mechanisms that could explain adaptations are yet poorly understand. Indeed, there is substantial evidence in the literature that the assessment of muscle mechanical properties allows a better understand of muscle function and the mechanisms responsible for muscle adaptations following acute or chronic intervention (Caiozzo, 2002; Goubel & Lensel-Corbeil, 2003).

Also, there has been an emergence in physical therapy of evaluation and intervention based on neurodynamics, the relationship between nerve physiology and nerve mechanics. To advance the clinical care of people with nerve injuries or mechanical impairment, first it is essential to understand peripheral nerve structure and plasticity according with its relationship and influence on the muscle-articular complex.

Flexibility is believed to be an important element of fitness. Consequently, the use of stretching exercises/protocols to improve flexibility is a widespread practice among athletes, both elite and recreational (Alter, 2004). They are widely used with the purpose to restore, maintain and increase flexibility (Alter, 2004; Gajdosik, 2001; Magnusson, 1998; Weppler & Magnusson, 2010). Stretching exercises to improve flexibility have often been associated with increased ROM, improved performance, and decreased muscle soreness. The acute effects of stretching on biomechanical properties and force production capacities of a muscle-tendinous complex (MTC) are a topic of continued interest to researchers. The literature contains controversial findings regarding the association between hamstring flexibility and the risk of injury. However, poor hamstring muscle flexibility have been suggested to predispose to hamstring strain (Jönhagen, Németh, & Eriksson, 1994; Witvrouw, Danneels, Asselman, D'Have, & Cambier, 2003).

Passive stretching exercises are commonly performed in sports and rehabilitation (Alter, 2004; Gleim & McHugh, 1997). In humans the biomechanical properties of a MTC, including structures spanning the joint structures (i.e. skin, connective tissue, muscles and neurovascular structures) (Alter, 2004; Riemann, Demont, Ryu, & Lephart, 2001; Weppler & Magnusson, 2010), can be determined by the relationship between passive torque (T)–articular angle (θ) (i.e. usually torque-angle) developed in resistance to motion. Torque-angle curves are classically characterized regarding literature (Gajdosik, 2001; Magnusson, 1998; P. McNair, Hewson, Dombroski, & Stanley, 2002; A Nordez, Casari, & Cornu, 2008; A Nordez, Gennisson, Casari, Catheline, & Cornu, 2008; Antoine Nordez, Casari, Mariot, & Cornu, 2009; Ryan et al., 2008; Weppler & Magnusson, 2010). Until lately, the only methods available for measuring passive properties of muscle were invasive and they used muscles through an individual approach (Heslinga & Huijing, 1990; Tabary, Tabary, Tardieu, Tardieu, & Goldspink, 1972). Although there are new methods to quantify *in vivo* passive muscle mechanical properties (e.g. elastography using supersonic shear imaging (SSI) (Maïsetti, Hug, Bouillard, & Nordez, 2012; A Nordez, Gennisson, et al., 2008)), torque-angle is the most often assessment used to quantify these properties. From this torque-angle measurements, it is possible conclude about passive force-length relationship (from tissues around tested joints) and thus infer about MTC passive stiffness. Muscle stiffness provides an estimate of the resistive force or tension that a muscle exerts in response to a given length change (Blackburn, Padua, Riemann, & Guskiewicz, 2004; Gleim & McHugh, 1997). The passive mechanical properties are an important component of muscle function because they are related to the muscle extensibility (Gajdosik, 2001).

The nervous system, in a global approach, is a mechanically and physiologically continuous structure from the brain to the end terminals in the periphery (Shacklock, 1995). Cadaver studies have demonstrated that the neural tissue is mechanically mobilized and strained by adding movements of the limbs and the spinal column. These facts have been measured both in fresh (Byl, Puttlitz, Byl, Lotz, & Topp, 2002; Coppieters & Butler, 2008; Wright, Glowczewskie, Cowin, & Wheeler, 2001; Wright, Glowczewskie, Wheeler, Miller, & Cowin, 1996) and embalmed cadavers (Kleinrensink, Stoeckart, Vleeming, Snijders, & Mulder, 1995), and *in vivo* through via real time diagnostic ultrasound (Dilley, Lynn, Greening, & DeLeon, 2003; Dilley, Summerhayes, & Lynn, 2007; Ellis, Hing, & McNair, 2012; Ellis, Hing, Dilley, & McNair, 2008; Hough, Moore, & Jones, 2007; Ridehalgh, Moore, & Hough, 2012).

Clinically the influence of additional neural strain and tension can be seen during neurodynamic testing where joint ROM decreases as tension is progressively added to the peripheral nervous system (PNS). This is a key premise of neurodynamic testing. For example, studies which have examined knee ROM during a slump test have concluded that significantly less knee extension occurred with the addition of cervical flexion (Johnson & Chiarello, 1997; Tucker, Reid, & McNair, 2007). The explanation given for the reduction in knee extension was that cervical flexion imposed additional tension upon the neuromeningeal structures at the spinal cord and nerve roots which led to a reciprocal increase in tension further down to the sciatic nerve tract (Herrington, Bendix, Cornwell, Fielden, & Hankey, 2008; Johnson & Chiarello, 1997; Yeung, Jones, & Hall, 1997). Therefore, we can conclude that multiple joint movements and body position significantly influence the relationship between nerve excursion and strain (Coppieters & Butler, 2008). Only two studies (Laessøe & Voigt, 2004; M. P. McHugh, Johnson, & Morrison, 2012) have examined the influence of addition of neural tension in torque-angle response. Although both had identical experimental protocols and also similar torque-angle assessment, they used different ROM recording data (**i.** only maximal ROM (Laessøe & Voigt, 2004); and **ii.** both submaximal and maximal ROM (McHugh et al., 2012)) to infer about resistance to stretch in their analysis. Moreover, torque-angle response from data collection for submaximal ROM (McHugh et al., 2012) was normalized to resistance to stretch as a percentage of the lowest maximum ROM achieved on experimental maneuvers. Therefore, the torque-angle response was not extensively examined and normalized for a given submaximal passive resistance to stretch. Moreover, there is evidence that adding neural strain maneuvers on stretched muscle do not cause additional muscle tension, and do not increase EMG response (Lew & Briggs, 1997).

Furthermore, passive torque and joint angle are often measured through dynamometer (Laessøe & Voigt, 2004; Magnusson, 1998; McHugh et al., 2012). Recently, it was shown that this torque-angle methodological assessment is less reliable and less specific than measuring whit a specific device to directly measure the resistance to passive knee extension and 2D kinematic analysis with video recording to access body segments position (Freitas, Vaz, Bruno, Valamatos, & Mil-Homens, 2012). Other previous studies have recorded hamstring passive resistance to stretch data in testing positions similar to clinical neural tension tests. Accordingly, similarities can be found between these assessments and both slump test (Magnusson, 1998) position and straight leg raising test or Lasegue's test (e.g. Halbertsma, Van Bolhuis, & Göeken, 1996). Nevertheless the effects of addition of ankle, thoracic and cervical spine movements on neural tissues, i.e. sciatic nerve tract, are well known regarding literature. However, hamstring passive resistance to stretch behavior (from torque-angle assessment) associated with neural tension tests was yet not extensively studied and information about this subject needs to be obtained. Moreover, we can hypothesize that a manipulation of the nerve tissue complex in a part of the body, which is not directly mechanically related to the joint involved, would have an influence on the resistance to stretch. Thus, the purpose of this study was design to assess hamstring direct passive torque-angle response to stretch at six different neural tension tests conditions during passive knee extension (PKE) test.

I.1. Aims and objectives of this Master research

i. To investigate whether different types of addition of neural tension results in different amounts of maximal and submaximal torque-angle measures during PKE test. Specific analysis of the amount of torque-angle was conducted among different types of neural tension tests, according with manipulation of the following body segments: ankle, thoracic spine and cervical spine.
ii. To investigate stretch tolerance (maximum ROM and resistance to stretch) and EMG response during modified PKE tests, i.e. with manipulation of body segments to lead tension on neural tissues.

Thereafter, a summary of the overall thesis is provided, conclusions are drawn and areas for future research are presented.

II. Review of literature

II.1. Introduction

In vivo it is possible to change muscles length by the mobilization of joints, and from a certain articular angle (θ_0) muscles crossing joint can start generating therefore resistance to stretch. Passive stretching is therefore characterized to place the articular angle beyond the angle θ_0 by an external action. This assumption requires verifying that the muscle electrical activity is negligible (Gajdosik, 2001; Magnusson, 1998). We will ensure this condition throughout this study, so the nervous factors (i.e. active mechanical properties) and reflex mechanisms that may are present at *in vivo* muscles stretching protocols are not detailed in this paper. Thus, it is possible characterize passive mechanical properties of joints by measuring the MAC response in different passive stretching protocols. Also, under normal physiological conditions imposed by posture and movement, nerves are exposed to various mechanical stresses. When joint motion causes elongation of the nerve bed, the nerve is inherently placed under tensile stress and accommodates the stress by both elongating and gliding (Millesi, Zöch, & Reihsner, 1995).

This chapter is focused on a brief review of main physiologic structures involved on dynamic passive stretching and muscle extensibility. Accordingly, the main goals are: **i)** characterize passive musculoskeletal biomechanical properties observed during knee passive extension test (from torque-angle assessment); and **ii)** describe neurodynamic mechanisms and their response to joint and body segment movements, and stretching maneuvers.

II.2. Torque-angle factors and concepts

II.2.1. Passive properties and concepts of skeletal muscles and joints

There are many terms used by clinicians and researchers to describe passive muscle length of skeletal muscles and contiguous tissues. So, there usually are different terms to define similar phenomena (Gajdosik, 2001). Throughout the rehabilitation literature regarding the effects of stretching, confusion arises due to inconsistent use of terminology among studies. Measures of flexibility, which can be both static and dynamic, are performed to access the ability of skeletal muscle and tendon to lengthen (Gleim & McHugh, 1997). According to the science of biomechanics, muscle length is multidimensional and length measurements are only one dimension of muscle length. When more than one dimension is included in muscle length assessment (tension, cross-sectional area, and time), important biomechanical properties of the muscle can be determined. From these dimensions, additional biomechanical properties – stiffness, compliance, energy, hysteresis, viscoelastic stress relaxation, creep and stress – can be inferred (Enoka, 2002; Özkaya & Nordin, 1999; Weppler & Magnusson, 2010).

Skeletal muscle contains deformable material. In this way, measurement at a given moment in time is always dependent upon the amount of tensile force (that pulls the specimen in the direction of elongation) applied (Özkaya & Nordin, 1999). Engineers use many terms to describe how loads tend to change the shape of a material. These include the principal or axial loadings of compression, tension, and shear. Shear is a right-angle loading acting in opposite directions, and compression is when an external force tends to squeeze the molecules of a material together. Lastly, tension is when the load acts to stretch or pull apart the material. In the present study, we considered tension as passive resistance of the muscle being stretched and is equal to the applied tensile force. The relationship between length and tension can be described by a passive length-tension curve which multiple individual length measurements are plotted according to the amount of passive tension required to reach each measurement (Knudson, 2007; Özkaya & Nordin, 1999).

Static flexibility can be defined as the ROM available to a joint or series of joints, and dynamic flexibility refers to the ease of movement within obtainable ROM. The important measurement in dynamic flexibility is stiffness (Gleim & McHugh, 1997). The stiffness of a material is the relationship between stress and strain (expressed as the slope of the line). The stress (σ; $N.m^{-2}$ in SI system), which can be described as the internal force divided by cross-sectional area ($\sigma = \mathrm{F} / \mathrm{A}$), causes the body change in shape, or deform, which is called strain. However, because the cross-sectional is at times not possible to measure or calculate, particularly in human studies, it is occasionally substituted by force. Tensile strain (ϵ) can be defined as the change in length divided by original length $[(L - L_0) / L_0]$, which is a dimensionless unit, but is often expressed as a percentage of the original length (Knudson, 2007; Magnusson, 1998; Özkaya & Nordin, 1999). In other words, the increase in strain is proportional to the applied stress (Hooke's law), such that a stiffer material will deform less for a given applied external load.

Passive stiffness, passive elastic stiffness and passive viscoelastic stiffness are synonyms terms frequently used to describe muscle's physiologic response during dynamic phase of the stretch. Passive stiffness (k) is well-defined as the ratio of the change in the passive resistance or passive force (ΔF) to the change in the length displacement(ΔL); ($k = \Delta F / \Delta L$). It can also be assessed through calculating the slope of passive moment or torque-angle curve ($k = \Delta passive\ torque / \Delta angular\ position$), and it is usually measured at a slow constant rate of applied dynamic stretch in order to avoid stretch-reflex activations. As the velocity of stretch is increased, the viscous behavior of muscles contribute to increased passive resistance and increased passive elastic stiffness – both human and animal muscles in the absence of stretch induced muscle activations (Blackburn et al., 2004; Gajdosik, 2001; Knudson, 2007; Özkaya & Nordin, 1999). Other biomechanical property that can be derived from tension, which is reciprocal of passive stiffness, is passive compliance – change in length *per unit* change in tension ($\Delta L/\Delta F$). So, these terms (i.e. passive stiffness and passive compliance) represents the same physiological response to stretch viewed from reciprocal perspectives (Gajdosik, 2001; Weppler & Magnusson, 2010). Summarizing, muscle with a passive curve that has a shallow curve is less stiff or more compliant than a muscle with a passive curve that has a steep rise. Otherwise, muscle with a passive curve that has a steep rise in the passive curve is stiffer or less compliant than a muscle with a shallow rise in the passive curve (Figure 1) (Gajdosik, 2001).

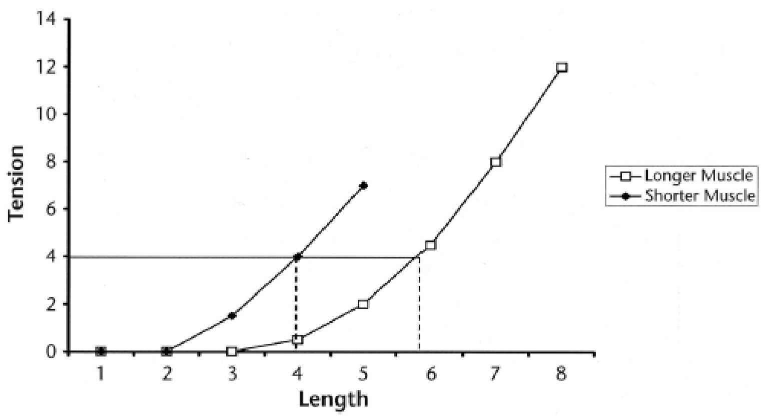

Figure 1. Model of shifting length/tension curve.
When a change in muscle length occurs, there is a shift in the entire passive length/tension curve. When "shortening" occurs, the curve shifts to the left, reflecting shorter muscle length measurements at a given passive tensile force. When lengthening occurs, the curve shifts to the right, reflecting a longer muscle length measurement at a given tensile force. Note: Number values are absolute; curve is a theoretical illustration (adapted from Weppler & Magnusson, 2010).

Human muscle length measurements are, with recent few exceptions, measurements of joint angles, and the tensile force is applied in a rotation manner (i.e. torque, or moment of force, is the product of the force and the perpendicular distance between the line of action of the force and the axis of rotation; $T = F \times d_{\perp}$). For this reason, length-tension curves are commonly presented as torque-angle curves in human studies (Figure 2). Energy (*Joule in SI*) is defined and expressed as the area under the torque-angle curve (stored viscoelastic energy) (Magnusson, 1998; Weppler & Magnusson, 2010).

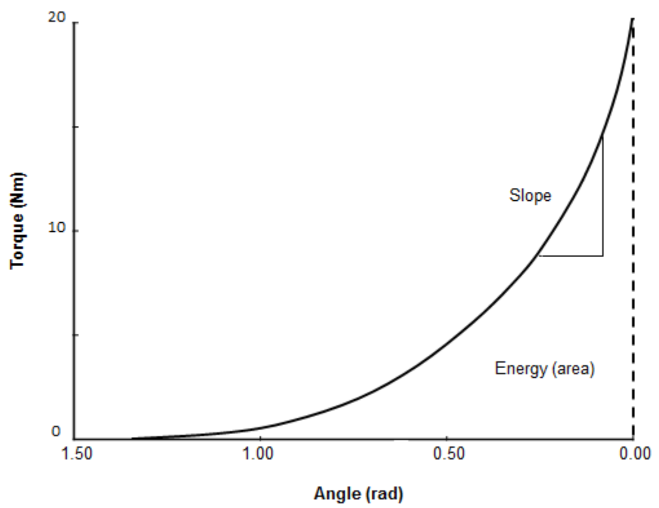

Figure 2. A typical non-linear viscoelastic response during a stretch maneuver using torque-angle assessment.
The torque-angle data for one subject from the dynamic phase of the constant angle protocol. Stiffness was defined as the change in torque (Nm) divided by change in position (radians) and was expressed as the slope of the torque-angle curve in the final third of the joint range of motion. Energy was defined and expressed as the area under the curve (joule) (adapted from Magnusson, 1998).

The sum of passive forces with active forces, produced by the interactions of actin and myosin proteins, corresponds to the total force produced by skeletal muscles. Both passive and active forces are influenced by the length of the muscles. Furthermore, passive forces increase exponentially (i.e. showing curvilinear mode) as the muscle is stretched to its maximal length (Brodie, 1895; Gajdosik, 2001; Haycraft, 1904; Stolov & Weilepp, 1966). The active length-tension curve is a theoretical model. Therefore, we cannot measure active forces directly. Accordingly, they are calculated through by subtracting passive force from the total forces throughout the full length of the muscle (Figure 3) (Gajdosik, 2001; Lieber & Bodine-Fowler, 1993). The results play an import role as a framework for how the change in passive forces contributes to total muscle function when a muscle is stretched through its available length extensibility (ability of a muscle to extend to a predetermined endpoint (Weppler & Magnusson, 2010), which in human studies assumes an endpoint of subject sensation unless otherwise noted).

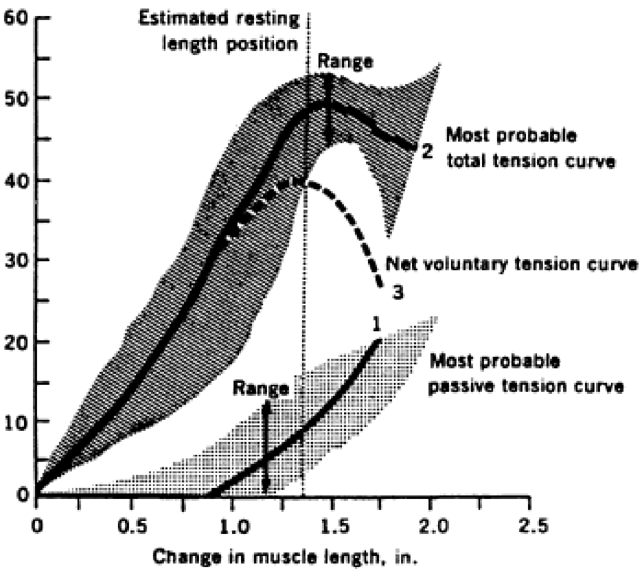

Figure 3. Classic length-tension curves for skeletal muscle.
Net voluntary active tension is predicted by subtracting passive tension from total tension (adapted from Gajdosik, 2001).

The muscle-tendon unit is the main anatomic and physiologic structure responsible for voluntary human movement. Tendons, which consist of dense regular connective tissues and considered a part of the series elastic component of the muscle-tendon unit, exhibit minimal length extensibility characteristics (Kubo, Kanehisa, & Fukunaga, 2001; Stolov & Weilepp, 1966; Tardieu, Tabary, Tabary, & Tardieu, 1982). Although there is some slight straightening of the connective tissues within tendons, for practical purposes the length of tendons can be considered constant (Gajdosik, 2001). Therefore, several researchers have suggested that the major factor contributing to passive tension in muscle is the extensibility of the connective tissue elements in parallel with the muscle fibers – parallel elastic component (PEC) (Jewell & Wilkie, 1958; Purslow, 2010; Stolov & Weilepp, 1966; Tardieu et al., 1982). Consequently, muscle passive tension is independent of the elasticity of tendon structures (Kubo et al., 2001). Thus, muscle belly is the primary part of the muscle-tension relationships of the stretched muscle-tendon unit.

Muscle extensibility can be observed during a passive stretch maneuver. It is possible that this increase is owing to a simple decrease in muscle stiffness or an increase in muscle length. A simple decrease in muscle stiffness is evidenced by a decrease in the slope of the torque-angle curve. Increases in muscle length are reflected on torque-angle curve by a shift to the right of the entire curve. This change results in decreased stiffness and an increased length measurement (joint angle) for any given tension (Figure 1). However, muscle extensibility can also increase without this change in muscle length or stiffness: it is possible through a simple increase in applied tension, which causes the muscle to stretch further. Nevertheless, without information about applied tension, there is no way to differentiate between these possibilities (Halbertsma & Göeken, 1994; Magnusson, 1998).

When a muscle is gradually and passively lengthen from maximal short position it achieves a point, initial passive resistance, where passive resistance to stretch can start to be measure. This point defines an initial length which is not identical to the resting length of the muscle. As the muscle is lengthened beyond this initial length, greater passive resistance is recorded until a maximal passive length. Stretch beyond this point results in rupture at the ends of the muscle fibers associated with the musculotendinous junction, which documented in animal studies, and avoided in human studies in order to ensure ethical guidelines (Gajdosik, 2001; Garrett, Nikolaou, Ribbeck, Glisson, & Seaber, n.d.; Garrett, Safran, Seaber, Glisson, & Ribbeck, n.d.; Knudson, 2007)

II.2.2. Joint passive factors

Various theories have been proposed to explain increases in muscle extensibility observed after stretching protocols. These mechanical theories include viscoelastic deformation, plastic deformation of connective tissues, increased sarcomeres in series, and neuromuscular relaxation (Weppler & Magnusson, 2010). When an increase in muscle extensibility is observed, it is possible that increase is due to a simple decrease in muscle stiffness or an increase in muscle length. Resistance to passive stretch of skeletal muscle in healthy human subjects has been attributed to the extensibility of the non-contractile connective tissue components of muscle tendon units, the stretch-induced contractile response to stretch or resting tension in myofibrils. However, studies that have simultaneously examined range of motion, resistance to stretch and electromyographic responses from the stretched muscle indicate that resistance to stretch is fundamentally due to the viscoelasticity of non-contractile component (Gajdosik, 2001). Thus, it is clear that passive properties of skeletal muscles are influenced by several mechanisms and structures during passive stretch of a resting muscle, considering that for practical purposes the length of tendons can be considered relatively constant and non-contributory to the measurable passive length-tension relationships of a stretched muscle (Gajdosik, 2001; Kubo et al., 2011; Tardieu et al., 1982). These include: filamentary resting tension (series elastic components – SECs); sarcomere cytoskeletons (SECs); and connective tissues (PEC).

During an *in vivo* passive stretch, different structures of MAC produce resistance to stretch whose relative contribution is unknown. Different muscle-tendon units crossing the joint develop an important part of this resistance (Gajdosik, 2001; Magnusson, 1998). The structures of muscle-tendon unit involved during stretching are shown in Figure 4.

Connective tissue, organized into different layers, ensures the cohesion of the muscle and part of transmission of the muscular strength into the bones (Kjaer, 2004). A substantial portion of the muscle passive force developed in resistance to stretch is also produced by the connective tissue (Gajdosik, 2001; Purslow, 1989). Connective tissue is made up of networks of collagen fibers (20% of total volume) and a very low percentage of elastin that provides much of their elasticity. The remaining 80% of the volume forms a gel mainly composed of water (70%).

The tendon allows the mechanical connection and force transmission between muscle and bones. Tendons are composed of dense connective tissue with collagen fibers which represents almost 80% of the tendon dry weight. They are connected to a less dense network of connective tissue surrounding the muscle (epimysium). For pennate muscles[1], aponeurosis[2] which also consists of dense connective tissue is inserted between the tendon and epimysium. The epimysium is connected to a slacker connective tissue (perimysium) that divides the muscle fascicles comprising one hundred of muscle fibers. All fibers are surrounded by slight connective tissue: the endomysium. The muscle fibers are composed of more than a thousand myofibrils which are surrounded by the sarcolemma (cell membrane). Myofibrils are themselves made up of associations of sarcomeres in series, which contain contractile proteins involved in muscle contraction.

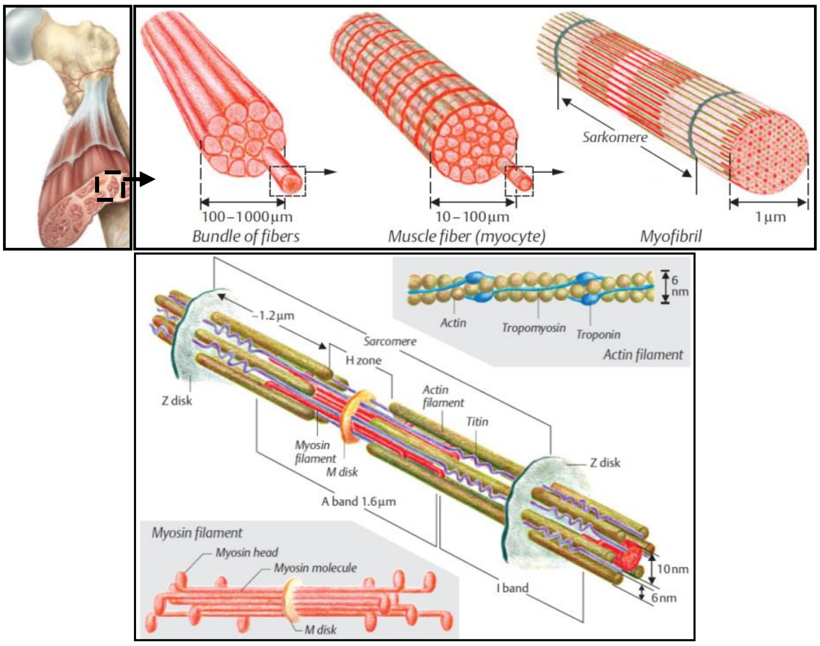

Figure 4. Different levels of organization of the muscle-tendon.
The tendon allows the mechanical link between the muscle and bone. The muscle is covered by epimysium. The epimysium which is attached to perimysium envelope fascicles (groups of muscle fibers). The perimysium is itself connected to the endomysium which cover the muscle fibers.

[1] Pennate muscles have fibers aligned at a small angle (usually less than 15º) to a tendon or aponeurosis running along the long axis of the muscle.
[2] It is a distinct connective tissue band within a muscle.

Conventionally, a rheological model of the muscle is used to describe the elastic behavior of the different structures of the muscle-tendon compared to muscle actin-myosin bridges, which are responsible for muscle force generator.

Originally, Hill (1938) developed a rheological model of the muscle to take account of their contractile properties. This model was then modified and improved by many authors. For example, the model presented in Figure 5, proposed by Shorten (2001), place the elastic structures of the muscle in series (SEC) or parallel (PEC) relatively to actin-myosin bridges. SEC is itself composed by both passive and active fraction. Different elastic structures of the muscle-tendon have, like most biological tissues, viscoelastic properties. Some authors consider that PEC produces an important part of the resistance to passive stretching produced by the muscle (Gajdosik, 2001).

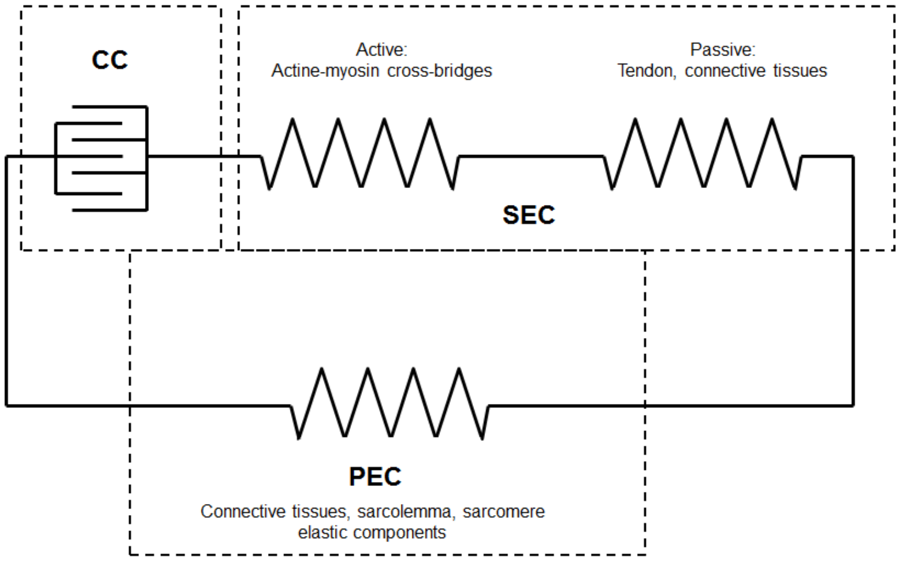

Figure 5. Model of muscle-tendon complex, with three components.
Adapted from (Goubel & Lensel-Corbeil, 2003). CC: contractile component, the force generator is located at the level of actin-myosin bridges. SEC: series elastic component. Passive fraction lies in the tendinous structures and fascia, while the active fraction corresponding to the elasticity of actin myosin bridges. PEC: parallel elastic component, localized in connective tissue of the sarcolemma of titin and intermediate filaments.

II.2.3. Contribution of contractile tissue

D. Hill (1968) showed that during a passive stretching (i.e. low ROM) of a muscle *ex vivo*, increased muscle tension is biphasic (Hill, 1968). Tension increases sharply at the beginning of stretching, and then more slowly in a second phase. The initial stiffness is higher than the stiffness calculated in a second phase. This phenomenon has been attributed by Hill to the presence, in first phase of stretching, of an elastic component specific: the Short Range Elastic Component (SREC). Thus, Axelson & Hagbarth (2001) showed that the SREC is expressed at the beginning of stretching applied to the wrist flexors (up to 0.03 rad (1.7 °) in Figure 6 (Axelson & Hagbarth, 2001). It has also been shown that the SREC disappears after the first cycle of stretching (e.g. Figure 6, implying a dependence on loading history of the mechanical behavior of muscle. It results in a decrease in hysteresis which was understood as a decrease in viscosity. Several authors have concluded from these results that the muscle has a thixotropic behavior (Axelson & Hagbarth, 2001; Proske, Morgan, & Gregory, 1993; Proske & Morgan, 1999; Whitehead, Gregory, Morgan, & Proske, 2001). Some authors have interpreted this behavior of the muscle by the presence of actin-myosin bridges weakly attached that could contribute to the development of passive tension in the first phase of the stretch. These bridges are broken from a certain level of stretching explaining, at least in part, the dependence on loading history of the mechanical behavior of muscle (Proske & Morgan, 1999). However, the contribution of actin-myosin bridges during passive stretching is still under discussion since Mutungi & Ranatunga (1996a, 2000) showed that the SREC is dependent on the speed of stretch (Mutungi & Ranatunga, 1996b, 1996c) and temperature (Mutungi & Ranatunga, 1998). These authors then proposed mechanisms to explain purely viscoelastic of SREC expression, regardless of the presence of residual bridges in a passive stretch. Further studies are needed to quantify the potential contribution of actin-myosin bridges in the production of passive muscle strength, particularly during a stretching protocol performed *in vivo*.

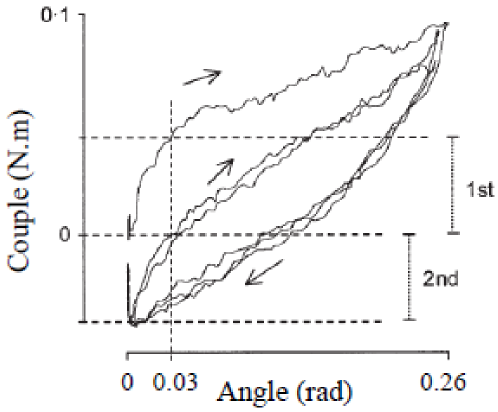

Figure 6. Relationship between the torque-angle developed in resistance to stretch and the angle joint during five passive stretching maneuvers with low amplitudes of the flexor muscles of the wrist (Hagbarth and Axelson, 2001).
During the first cycle (1st), the increase in torque is biphasic. The first phase (0-0.03 rad) indicates the presence of the Short Range Elastic Component (SREC). This component disappears in the following cycles.

II.2.3.1. Filamentary resting tension

The passive resistance that may result from stretching stable interactions or cross-links between the actin and myosin filaments was first proposed by Hill and expanded by others. These stable links can be explained by a very low level of actively generated resting tension that apparently is responsible for passive resistance (actin-myosin cross-bridges resist the stretch a short distance from the stable position before the contacts slip and reattach at other binding sites). “Cross-bridge Population Displacement Mechanism” also allowed an expansion of this proposal (Campbell & Lakie, 1998; Gajdosik, 2001). According to it, in a relaxed muscle, the actin and myosin filaments are linked by a relatively small number of cross-bridges, each of which behaves as a linear spring for both extension and compression; only cross-bridges with displacements corresponding to a defined interaction range can bind between the filaments; the attachment probability is increased by interfilamentary movement; unstrained cross-bridges are stable and relatively long-lived, whereas cross-bridges with high strains detach more rapidly; interfilamentary displacement skews the cross-bridge distribution; and the mean cross-bridge displacement of an undisturbed population is slightly positive. This bias produces a force which acts to increase filament overlap and contributes to the muscle’s resting tension (Campbell & Lakie, 1998). Indeed, if this very low level of activity exists in completely relaxed muscles, it seems to be clear that electromyographic surface signal might be not detected. Instead, the passive state in human muscles is operationally defined by the presence of minimal or negligible EMG activity.

X-ray diffraction studies have shown that actin and myosin filaments show extensibility properties that contribute to the stiffness of an active muscle. The evidence for low compliance in actin filaments originally came from stiffness measurements of sarcomeres during activation of intact muscles at various lengths. The spacing changes of reflections in X-ray diffraction patterns, which also depend on the mechanical load on the muscle, indicate that elongation is accompanied by slight changes of the actin helical structure possibly because of the axial force exerted by the actomyosin cross-bridges. Moreover, the effects of steady lengthening applied to contracting muscles on the actin and myosin filament-based reflections are described, showing extensibility of the myosin filaments too (Gajdosik, 2001; Purslow, 1989; Wakabayashi et al., 1994). Thus, actin-myosin cross bridges may also contribute to some of the passive resistance in relaxed muscles.

II.2.4. Parallel elastic components contribution

II.2.4.1. Connective tissues and sarcolemma

Connective tissue (epimysium, perimysium, endomysium) and sarcolemma (cell membrane) are different layers of muscle. We can consider that they are placed in parallel contractile structures. If they all contribute to the development of passive tension in the muscle, the greater part appears to be due to the perimysium (Gajdosik, 2001; Purslow, 1989). The diffraction imaging showed that networks constituents of collagen fibers, which are folded at rest, moved into direction of the stress applied in the course of stretching

(Purslow, 1989). This probably contributes to the exponential increase of the stress during stretching of a muscle (Gajdosik, 2001).

II.2.4.2. Sarcomere elastic component

The sarcomere (Figure 7) also exhibits elastic components in the development of resistance to stretching produced by muscles (Anderson et al., 2002). The works of Horowitz et al. (1986) have highlighted the involvement of titin filaments during passive stretching. On a fiber peeled, these authors showed that passive tension from stretching is greatly reduced when titin is degraded by radiation. These filaments connecting the myosin to Z-discs have elastic properties and bring back the sarcomere to its initial position when it is stretched. They also ensure the maintenance of the structural organization of the sarcomere (Horowits et al., 1986). Intermediate muscle filaments, mainly desmin and nebulin, are resistant proteins filaments. For example, the main function of desmin filaments is to ensure the cohesion of the myofibril sarcomeres transversely, connecting them (Figure 8).

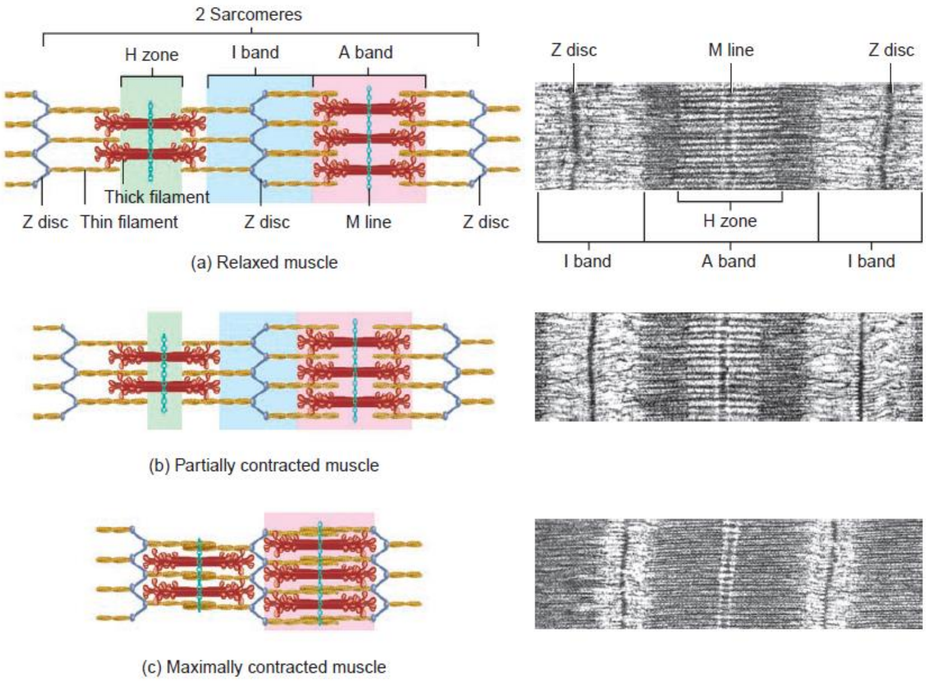

Figure 7. Structural organization of the sarcomere.
Titin, elastic filament, connects with myosin at Z line. Adapted from (Tortora & Derrickson (2009).

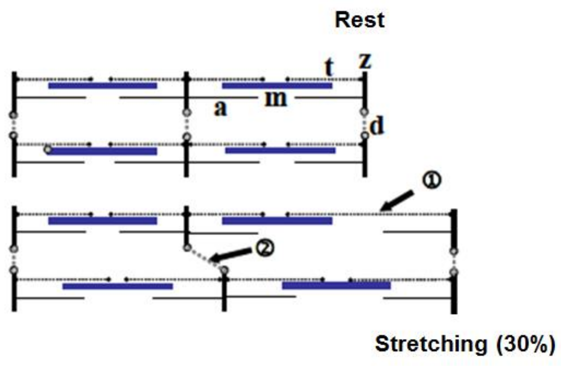

Figure 8. Diagram of the sarcomere at rest and during a stretch of 30% in eccentric contraction.
a – actin; m – myosin; t – titin; d – desmin; z – line z. ① and ② show the elongation of filaments of titin and desmin which will produce a greater resistance to stretching (adapted from Allen, 2001).

II.2.4.2.1. Sarcomere cytoskeletons

Much of the passive resistance of a stretched relaxed muscle has been given to non-contractile filamentous connections within two sarcomeric cytoskeletons, termed the endosarcomeric and exosarcomeric cytoskeletons. Filamentous connections between the thick myosin filaments and the Z-discs of the sarcomere have been shown to contribute to this passive resistance, particularly when the sarcomere is stretched beyond the actin and myosin overlap. The filamentous connections of the endosarcomeric cytoskeleton are comprised of large, thin filaments of a giant protein that has been named titin (Gajdosik, 2001; Herzog, Leonard, Jinha, & Herzog, 2012; Horowits et al., 1986).Titin filaments span a half-sarcomere from the Z-line to the M-line and is believed to be major sub-celular component of the endosarcomeric cytoskeleton that resists passive lengthening of a relaxed muscle. Slow twitch muscle fibers (type I) have greater passive stiffness than fast twitch muscle fibers (type II), and the differences may reflect different isoforms of titin within each fiber type (Gajdosik, 2001).

Intermediate sized protein filaments, with diameters of about 10nm, midway between actin (6nm) and myosin (16nm), contribute to the exosarcomeric cytoskeleton of the muscle fibers. One protein, called desmin, is the major subunit of the intermediate protein filaments forming the Z-discs. It serves to inter-connect Z-discs transversally, and to connect Z-discs with organelles, but not with the T-tubule system. Desmin also extends longitudinally from Z-discs to Z-discs outside of the sarcomere and because of this longitudinal arrangement between Z-discs outside of the sarcomere, the proteins contributes to exosarcomeric exoskeleton. Desmin lengthens as the sarcomere is stretched, so its elasticity is thought to contribute to the passive resistance of stretched muscle (J. Anderson et al., 2002; Gajdosik, 2001; Herzog et al., 2012; Mutungi & Ranatunga, 2000; Purslow, 2010).

II.2.5. Series elastic components of contractile tissues contribution

During muscle contraction, the force produced by the muscle is transmitted to the bone via structures arranged in series of the contractile component, mainly tendon and aponeurosis. They are often considered to be stiffer than muscle. When two series elastic components are stretched, if the stiffness of one of the two elements is greater than that of the other element, we can neglect the contribution of the steepest rise. Also, some authors have ignored the contribution of the production of passive forces from structures that are placed in series of contractile tissues (J. Anderson et al., 2002; Gajdosik, 2001).

However, it has recently been shown through ultrasonography that during passive *in vivo* stretching of plantar flexor muscles, only 27% of the extensibility of the muscle-tendon complex of *gastrocnemii* was effectively transmitted to the fascicles (Herbert, Moseley, Butler, & Gandevia, 2002). This suggests that about 70% of the change in length of the muscle-tendon lengthening is explained by the series elastic components. In this study, the low variation in length of *gastrocnemii* muscle fascicles is explained by tendon length that is much larger (i.e. about 10 times) than fascicles which underlines the importance of measuring the deformation instead of extensibility.

Kubo et al. (2005) showed that, in a passive stretching of the ankle plantar flexor muscles, deformation of the *gastrocnemius medialis* muscle fascicles is about five times larger than the deformation of the tendon and ten times greater than the deformation of the aponeurosis. A simple explanation with a model of two linear springs in series can show that if the deformation of one of the two elements is about five times lower than the other through a passive stretch, the error in neglecting the spring is then steeper of the order of 20%. The results of the study of Kubo et al. (2005) can therefore be understood that, during passive stretching, the contribution of structures placed in series with the muscle and tendon, are probably not completely negligible (Kubo, Kanehisa, & Fukunaga, 2005).

II.2.6. Joint contribution

Many studies used passive stretching of muscle groups to characterize passive mechanical properties. In this context, it is commonly considered that passive stretching helps to determine passive viscoelastic properties of muscle (Gajdosik, 2001), muscle-tendon unit (Magnusson, 1998) or tissues crossing the joint (McNair & Portero, 2005). These studies did not focus about the role of the contribution of the type of joint. However, the joints are not perfectly spherical, and thus they can produce additional resistance to stretching (Riemann et al., 2001). In addition, a study on *ex vivo* wrist joint of cat showed that during passive stretching, joint produced 47% of the passive torque (Johns & Wright, 1962). This animal model was chosen because the properties of the wrist cat seem to be similar to the human wrist. However, although the joint geometry and muscle volumes being different from one joint to another, the relative contribution of the joint is likely to be different depending on the joint in question (Riemann et al., 2001). However, the relative contribution of the knee, ankle and spine column during hamstring passive stretching has, to our knowledge, never been specifically determined. Therefore, in this study, we will not neglect the contribution of these joints.

Traditionally, it is considered that the torque developed in passive resistance to stretch is mainly produced by the muscular structures, internal or external to the sarcomere, placed in parallel with contractile tissues. However, we have seen that the tendon, contractile proteins, and joint can also have a significant contribution in this context. That is why we consider that stretching can characterize the overall mechanical properties of the entire MAC including all muscles, tendons and joints (Figure 9).

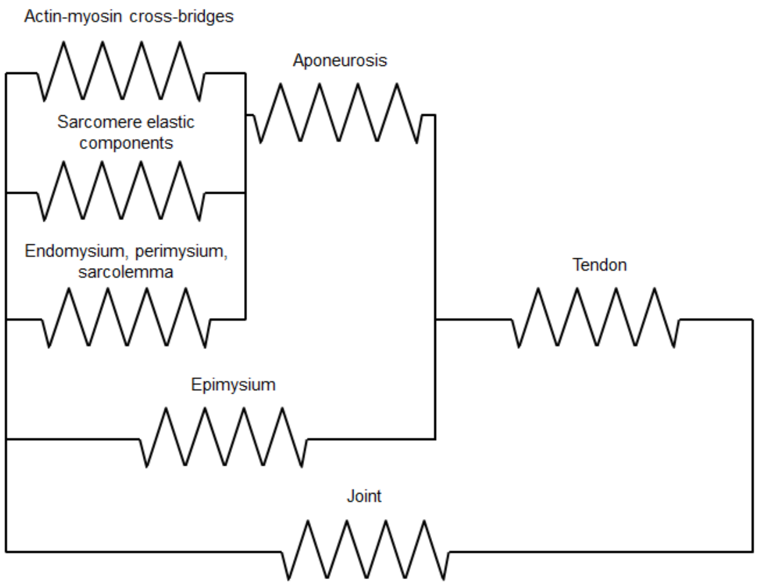

Figure 9. Rheological model proposed to describe the elastic behavior of the entire musculo-articular passive terms (adapted from Huijing, 1994).
The different structures of the musculo-articular complex are placed in series or in parallel contractile structures (actin-myosin cross-bridges).

II.2.7. Connective tissues

The extracellular matrix, and particularly the connective tissue with its collagen, links tissues of the body together and plays an important role in the force transmission and maintenance of tissues structure especially in tendons, ligaments, bone, and muscle.

Thereby, the amounts and composition of the various intramuscular extracellular matrix (or intramuscular connective tissue, or muscle fasciae) structures in living tissue represent a dynamic balance between deposition, growth, remodeling and degradation, which is affected by the interplay between functional demands on the tissue and the mechanical environment. Extensible connective tissues (e.g. muscle, skin, blood, vessels, and fascia) contain networks of fibrous collagen in an amorphous matrix. It is the reorientation of the collagen fibers within these networks that allows large extensions of the tissues and is responsible for their non-linear stress-strain curves (Purslow, Wess, & Hukins, 1998; Purslow, 2010).

As a muscle is stretched, the passive resistance is also influenced by a lengthening deformation of the connective tissues of the endomysium, perimysium, and epimysium of the muscle belly.

Intramuscular connective tissue has multiple functions (Kjaer, 2004). First it provides a basic mechanical support for vessels and nerves. Second, the connective tissue ensures the passive elastic response of muscle. Third, it is now clear that force transmission from the muscle fibers not only is transformed to tendon and subsequent bone via the myotendinous junctions but also via lateral transmission between neighboring fibers and fascicles within a muscle (Kjaer, 2004).

It has been shown that tension developed in one muscle part can be transmitted via shear links to other parts of the muscle, and that even the cutting of an aponeurosis in a pennate muscle still maintains much of the force transmission. The perimysium is especially capable of transmitting tensile force. Although studies have also demonstrated a potential of the endomysium for force transmission, the orientation and curvilinearity of the collagen fibers provide high amounts of elasticity and thus not sufficient stiffness to function optimally as a force transmitter. The thickness of the endomysium as a whole varies with muscle length, becoming thicker at short muscle lengths and thinner as the muscle is extended (Purslow, 2010).

II.3. Joint passive torque-angle measurement methods

Regarding literature, hamstrings and plantar flexors are the most *in vivo* studied muscle groups during passive stretching. Hamstrings passive stretching are the most often performed by researchers through varying the angles of the knee (Magnusson, 1998) or hip (Halbertsma, Mulder, Göeken, & Eisma, 1999), while the ankle is usually mobilized by stretching the plantar flexors (McNair, Dombroski, Hewson, & Stanley, 2001; McNair et al., 2002). Two methods are commonly described in the literature to perform stretching and then measure the response of muscle-articular system. The first one is to mobilize a joint manually. An electronic goniometer was used to measure joint angle and a cell force was used to measure passive resistance to stretch (Harvey, McQuade, Hawthorne, & Byak, 2003; Hoang, Gorman, Todd, Gandevia, & Herbert, 2005; M. McHugh, Magnusson, Gleim, & Nicholas, 1992; Riener & Edrich, 1999). It has been shown that this method allows reproducible measurements (Harvey et al., 2003; Hoang et al., 2005). It is nevertheless difficult to standardize the stretching velocity with this kind of experimental setup, and results may depend on the examiner who mobilizes the joint. It is possible to overcome these difficulties by using automatic dynamometers as isokinetic dynamometer (e.g. Biodex®, Kin-Com®, Cybex®...) or a device implemented specifically for this purpose (Tognella, Mainar, Vanhoutte, & Goubel, 1997). These apparatuses allow measurements of articular angle and passive torque from MAC in response to passive stretching with constant and programmed velocity (Figure 10**Error! Reference source not found.**).

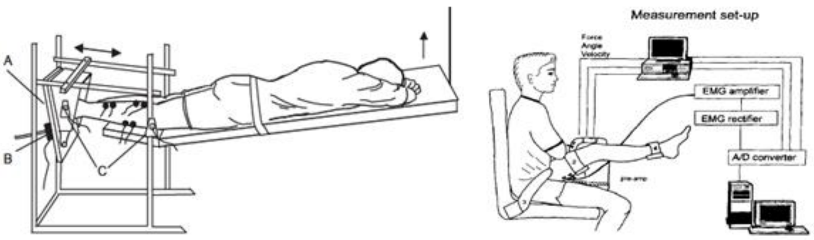

Figure 10. Examples of experimental setups used to characterize the passive mechanical properties (Hoang et al., 2005; Magnusson, 1998).

Low stretching velocities (<0.44 rad.s^{-1} (<25°.s^{-1})) are generally chosen to limit the acceleration and deceleration of the system and avoid stretch-induced reflexes. However, some studies have used higher stretch velocities – 3.14 rad.s^{-1} (180°.s^{-1}) (Lamontagne, Malouin, Richards, & Dumas, 1997; Rabita et al., 2005) – to determine the stretching velocity dependence of muscle-articular complex response. Frequently velocities lower than 0.09 rad.s^{-1} (5º.s^{-1}) should be done in order to avoid reflexes.

The stretching velocity is also an important factor to consider since the level of electromyographic activity (EMG)[3], voluntary or reflex, may contribute to modify the

[3] The surface electromyographic activity corresponds to the electrical potential difference that can be detected between two points near the surface of the superficial muscles using various electrode arrangements. A calculation of root mean square (RMS) of EMG signal then determines the level of muscle activity examined.

mechanical properties (e.g. increased stiffness) of the mobilized muscles. Thus, as part of the study of mechanical properties of MAC through passive stretching, it is necessary to ensure that the tested muscles do not produce a voluntary or reflex electrical activity through measuring their surface electromyographic activity (sEMG).

Some authors have recommended that the level of activity of measured muscles should be less than 1% of the activity of surface EMG obtained during maximal isometric contractions (Gajdosik, 2001; P. McNair et al., 2002; P. J. McNair & Portero, 2005). However, some authors have observed values between 3% and 5% (Magnusson, 1998; M. P. McHugh et al., 2012).

Finally, it was shown that isokinetic dynamometers allow do reproducible measurements of passive mechanical properties of MAC (Gajdosik, 2001; Magnusson, Simonsen, Aagaard, Sørensen, & Kjaer, 1996; Magnusson, 1998). However, a good reproducibility of passive mechanical properties can not be understood that the measurement error of the different sensors is negligible. The torque sensor of an isokinetic dynamometer is generally designed mainly to measure the capacity of force of a muscle group during contractions. However, the torque levels measured during passive stretching are much lower than those observed during contractions. It is therefore possible that measurement error disturbs passive torque assessment, for example due to energy dissipation at the rotation axis.

Additionally, many attempts have been made by different researchers to improve the accuracy of assessment, through development of new processing data procedures (D. Anderson, Nussbaum, & Madigan, 2010; Hoang et al., 2005; Antoine Nordez et al., 2010; Antoine Nordez, Cornu, & McNair, 2006). Recently, Freitas et al. (2012) suggest that direct measures should be done instead of indirect, and a strict control should be taken by researchers about tested a non-tested body segments positioning, since this affects considerable the torque-angle outputs.

II.4. Neurodynamic features of the peripheral nervous system

II.4.1. Neurodynamics

Neurodynamics is now a more accepted term and as a specific concept was first presented in a paper published in 1995 (Shacklock, 1995). It refers to the integrated biomechanical, physiological and mechanical functions of the nervous system (Butler, 2000; Shacklock, 1995, 2005; Walsh, 2005). The nervous system has a vital role as bidirectional transport system carrying information to and from different body systems in order to perceive, process and activate human movement. In doing so, nervous system must therefore be able to cope with the mechanical and physiological stresses that are imposed upon it from neighboring tissues in order to operate effectively. Specialized interdependent neuroanatomical and neurophysiological features allow the nervous system to maintain optimum function. Mechanical events at one site in the nervous system can produce a cascade of related events along the system. For example, passive neck flexion produces tension in the lumbosacral nerve roots, wrist extension can produce tension in the brachial plexus and dorsiflexion of the ankle moves the sciatic nerve. Regardless of the underlying construct, it is vital that the nervous system is able to adapt to mechanical loads, and it must undergo distinct mechanical events such as elongation, sliding, cross-sectional change, angulation, and compression. If these dynamic protective mechanisms fail, the nervous system is vulnerable to neural edema, ischemia, fibrosis, and hypoxia, which may cause altered neurodynamics (Butler, 2000; Shacklock, 1995).

II.4.1.1. Neural structures and biomechanical properties

The neural structures are simply those that constitute the nervous system. Included are the brain, cranial nerves and spinal cord, nerve rootlets, nerve roots and peripheral nerves (including the sympathetic trunks) and all their related connective tissues. Therefore, the structural organization of peripheral nerves allows axons to conduct impulses that facilitate an individual’s interactions with the world while directing and tolerating the myriad postures of the trunk, head, and limbs. Axons within a peripheral nerve are the lengthy extensions of cell bodies located in the dorsal root ganglia (sensory neurons), autonomic ganglia (autonomic neurons), or the ventral horn of the spinal cord or brain stem (motoneurons). Because their terminals are quite distant from the cell bodies, axons are insulated from each other, bundled together, and protected by 3 connective tissue layers – the endoneurium, the perineurium, and the epineurium (Figure 11). Axons, Schwann cells, and endoneurial components are bundled by a sheath of perineurium to form a nerve fascicle. Several fascicles are held together by epineurial tissue to form a nerve.

The nervous system possesses a natural ability to move and withstand mechanical forces that are generated by body movements. For the nervous system to move normally, it must successfully execute three primary mechanical functions; withstand tension, slide in its container, and be compressible. It is important to the development of the present work to discuss the relevance of these neurodynamic features in order to understand neural biomechanics behavior. Although it is easier to discuss each of the neurodynamic features in isolation, it is important to note that these features are interdependent.

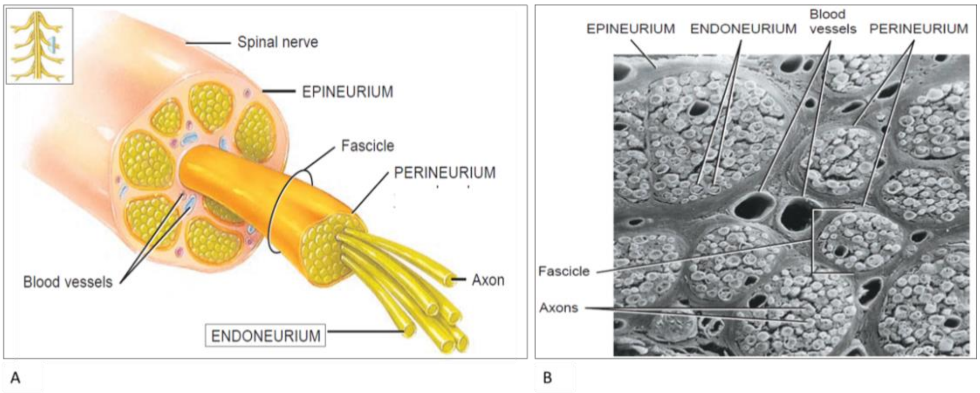

Figure 11. Nerve anatomy. Structural organization of 3 connective tissue layers – the endoneurium, the perineurium, and the epineurium. Adapted from Tortora & Derrickson (2009)

II.4.1.2. The ability of nerve to move or slide

From an extrinsic mechanical perspective, the PNS must be able to move and slide in relation to, and independently, its surrounding tissues (Butler, 2000; Dilley et al., 2003; Shacklock, 2005). Thus, body is the container of the nervous system in which the musculoskeletal system presents a 'mechanical interface' (also called the 'nerve bed' or 'neural container') to the nervous system. It consists of anything that resides next to the nervous system, such as tendon, muscle, bone, intervertebral discs, ligaments, fascia and blood vessels. Although the mechanical interface is not a direct layer or feature of the specific anatomy of the PNS, it must be taken into account when discussing nerve biomechanics and nerve pathology.

Excursion of a nerve refers to the movement of the neural structures relative to their adjacent tissues (McLellan & Swash, 1976; Wilgis & Murphy, 1986), and it occurs in the nerves longitudinally and transversely. Therefore, this sliding capacity allows the nerve to adapt to changes imposition and length of the nerve bed imposed by limb movements (Byl et al., 2002; Dilley et al., 2007; Erel et al., 2003; McLellan & Swash, 1976; Topp & Boyd, 2006). Excursion is an essential aspect of neural function because it serves, in all planes, to dissipate tension in an attempt to equalize pressure along the length of the nerve tract (Breig, 1978; Shacklock, 2005; Topp & Boyd, 2006; Walsh, 2005). Significant mechanical forces can be generated as peripheral nerves slide against the mechanical interface.

The routes of most peripheral nerve trunks fall beyond the movement plane of many joints (Phillips, Smit, De Zoysa, Afoke, & Brown, 2004). Dissipation of tensile forces via elongation and sliding must occur relatively evenly throughout the length of the nerve. The ability to slide may protective against increases in intraneural pressure caused by excesses of tension.

Sliding of peripheral nerves in their bed is provided by specialized connective tissues layers (Millesi et al., 1995) The mesoneurium is the external sheath surrounding the whole nerve. This allows sliding of the nerve relative to the mechanical interface (Butler, 2000; Shacklock, 2005). The external epineurium, which also consists of soft and loose connective tissues, allows nerve sliding in relation to the mesoneurium and nerve bed. The capacity of nerves to function in this way means that they can respond internally as well as externally to the forces to which they are subjected (Shacklock, 2005). This internal movement is facilitated by the internal epineurium and endoneurium (Butler, 2000; Walsh, 2005).

II.4.1.3. The ability of nerve withstand stretch

The ability of the PNS to dissipate tensile load is critical to maintain nerve function (Coppieters et al., 2006; Phillips et al., 2004). PNS also adapts to lengthening by the development of tension or increased pressure within the tissues, i.e., increased intraneural pressure, or increased intradural pressure. This pressure develops as a consequence of elongation and occurs in all tissues and fluids enclosed by an including the epineurium and the dura mater (Butler, 2000).

The neural connective tissue layers have an important role to play in nerve elongation. The collagen fibers of the epineurium and endoneurium have an undulating configuration which allows some slack to accommodate initial elongation forces (Topp & Boyd, 2006). The perineurium provides the primary resistance to elongation/stretching and it is effectively the cabling in the peripheral nerve. Perineurium possesses considerable longitudinal strength and elasticity and it can accept 18-22% strain before a peripheral nerve structurally fails (Rydevik et al., 1990). So, the perineurium has a strong protective ability. The spinal nerve roots do not have a perineurial layer and are therefore less resistant to elongation compared to peripheral nerves (Singh, Kallakuri, Chen, & Cavanaugh, 2009).

II.4.1.4. The ability of nerve to withstand compression

Compression of a nerve segment causes displacement of its internal contents in transverse and longitudinal directions. The epineurium is the padding of the nerve and is what protects the axons from excessive compression and it acts as a shock-absorber to dissipate compressive forces (Butler, 2000). It consists of finer and less densely packed connective tissue than the perineurium, a feature that gives the nerve spongy qualities and enables the nerve to spring back when pressure is removed. Both connective tissue density (Keir & Rempel, 2005; Rempel, Dahlin, & Lundborg, 1999) and fascicle numbers

(Sladjana, Ivan, & Bratislav, 2008) have been shown to be greater at regions of increased mechanical load, i.e. adjacent to a joint.

II.4.2. Peripheral nervous system responses to movement

II.4.2.1. Dynamics between neural tension and neural movement

The more complex mechanical events that occur during human movement are merely combinations of tension, sliding and compression. These events occur in both peripheral and central nervous systems. However, they are often achieved in different ways because of the existence of regional differences in anatomy and biomechanics. Always, each of the component mechanical events will interact with the others.

Multiple joint movements and body position significantly influence the relationship between nerve excursion and strain (Coppieters & Butler, 2008). Nerve excursion that is present with single joint movement will diminish with additional joint movements that increase tension (Coppieters et al., 2006; Wright et al., 1996). Significant increases in nerve strain will occur once sliding diminishes and elongation occurs. It is during this elongation that nerve tension increases exponentially and, potentially, a similar increase in intraneural pressures (Topp & Boyd, 2006; Wright et al., 1996). For example, cadaver studies have demonstrated that tibial, medial and lateral plantar (Alshami, Babri, Souvlis, & Coppieters, 2008) and median (Coppieters & Alshami, 2007; Coppieters & Butler, 2008) nerve strain significantly increased with additional joint movements that elongated the nerve bed.

Tibial nerve strain significantly increased, during ankle dorsiflexion, as the proximal aspect of the nerve tract was elongated (by adding hip flexion and knee extension). The same phenomenon was seen in the medial and lateral plantar nerves, whereby nerve strain was significantly higher in a pre-tensioned position (ankle dorsiflexion) compared to an unloaded position (ankle plantarflexion) (Alshami et al., 2008).

Clinically, the influence of additional neural strain and tension can be seen during neurodynamic testing where joint ROM decreases as tension is progressively added to the PNS. This is a key premise of neurodynamic testing. For example, studies which have examined knee ROM during a slump test have concluded that significantly less knee extension occurred with the addition of cervical flexion (Herrington et al., 2008; Johnson & Chiarello, 1997; Tucker et al., 2007). The explanation given for the reduction in knee extension was that cervical flexion imposed additional tension upon the neuromeningeal structures at the spinal cord and nerve roots which led to a reciprocal increase in tension further down to the sciatic nerve tract (Johnson & Chiarello, 1997; Yeung et al., 1997).

Knee extension increases the length of the sciatic nerve bed by up to 60 mm, and accounts for 49% of nerve bed elongation (Shacklock, 2005). The sciatic and tibial nerves converge toward the knee, sliding distally and proximally respectively (Coppieters et al., 2006). This will cause the plantar nerves to slide proximally. Knee extension is useful clinically because the joint offers a large range of motion through which changes in symptoms can be easily observed. Hence, this movement can be used to mobilize nerves that are located a large distance from the knee, such as the posterior tibial nerve (Shacklock, 1995, 2005) or the lumbosacral nerve roots, without producing excessive mechanical stresses in these structures.

Dorsiflexion of the ankle has been shown to increase tension in the tibial nerve (Alshami et al., 2008; Coppieters et al., 2006) and, at the height of the SLR, has at surgery been observed to produce movement in the lumbosacral nerve roots (Butler, 2000; Shacklock, 2005). Clinically, dorsiflexion is frequently a valuable differentiation and sensitizing maneuver for the SLR because of its ability to produce movement in the sciatic nerve tract as far proximally as the lumbosacral nerve roots.

Because the *in situ* strain is a direct reflection of cumulative nerve positioning across multiple joints, one must consider the effect of trunk, neck, and limb positioning during clinical assessment and intervention to minimize physical stress on an injured nerve.

Lew and Briggs (1997) recorded the hamstring EMG response and tension in the biceps tendon in the slump position with and without cervical flexion and concluded that this maneuver did not further elongate the hamstring muscle group and did not elicit a hamstring EMG response (Blackburn et al., 2004; Lew & Briggs, 1997; M. McHugh et al., 1992).

II.5. Conclusion

We conclude that regarding literature there is a lack of evidence about contribution of PNS on muscles extensibility, particularly under passive conditions. Therefore, the possibility that torque-angle, EMG and stretching tolerance during passive stretch of skeletal muscle in healthy subjects is limited by extensibility of the neural tissues has not been examined previously.

III. Methods

III.1. Design

An experimental design study was used with a convenience sample. Only healthy male subjects were chosen because it usually exhibits higher torque values and less maximal joint angles than female population (Kubo, Kanehisa, & Fukunaga, 2003). This study was approved by the local ethics committee and it was conducted according to the Helsinki Statement (1964).

III.2. Subjects

Ten male subjects (27.97±5.72 years, weight: 78.40±13.10kg, height 1.77±0.04m, 25.06±3.97 *body mass index*, leg length: 38.14± 2.57 cm, hip length: 39.31 ± 4.61), with maximum knee extension test deficit (i.e. lower than 70º), volunteered to participate in this study. Subjects practiced recreational sports, but did not participate in any strength or flexibility training at the time of the study. None of subjects reported any known current or ongoing musculoskeletal lower limbs (i.e. ankle, knee and hip) and spine injuries, neuromuscular diseases, or orthopaedics-related problems. Consequently, no subjects had sustained a recent injury that may have affected the findings and the purposes of this study. The subjects were informed of the nature and the aim of this study before they signed an informed consent form.

III.3. Protocol Procedures

The experimental setup used was adapted and it, i.e. procedures and instruments to data collection and its processing, has been previously well described by Freitas et al. (2012). Subjects came to the Faculty of Human Kinetics Laboratory in two distinct moments.

First, a familiarization session was performed at least two hours before the main testing session so as to prepare the subjects for the test protocols. Then, a short explanation about apparatus, articulate device to hold cervical and thoracic positions and security norms was told to the subjects and they were instructed to do not perform vigorous exercise before the testing sessions. Lastly, subjects did some trials in the passive knee extension setup until report confidence with the experimental condition (i.e. test angular velocity, maximal stretching tolerance, and specific cervical and thoracic spine positions).

In the second moment, even before the experimental protocol, anthropometric measures were determined, procedures for surface electromyography (sEMG) were done and reflective markers were placed in specific anatomical references. Taping was made to the right ankle with the aim of restrain it in a predefined static angle (according with test condition) and then avoid movement of the foot during knee extension test.

Afterwards, subjects were instructed to execute the experimental protocol. It consisted of a comparison of maximal tolerated PKE (i.e. passive dynamic stretching of hamstring) in six distinct test conditions each one with different combinations of ankle and upper body (i.e. cervical and thoracic spine column) positions (see details in Figure 12): **i.** neutral position (N); **ii.** maximal active ankle dorsiflexion (AD); **iii.** maximal active cervical and thoracic flexion (CTF); **iv.** maximal active cervical and thoracic flexion with ankle on maximal active dorsiflexion (CTFAD); **v.** maximal active cervical extension with thoracic flexion and ankle on maximal active dorsiflexion (CETFAD); **vi.** maximal cervical extension with thoracic flexion (CETF). The sequence of six test conditions was performed in a balanced random order, and during the tests it was carefully observed that the pelvis did not modify position when the position of the upper body and ankle was changed. The accurate alignment of lower limb and pelvis was assured with two tape-measures placed on both sides (i.e. left and right) over the articulate device. The shift between test positions did therefore not affect structures relating mechanically to the knee. So it was performed one repetition of each test condition in total of six, *per* subject. The stoppage between repetitions was the minimum necessary to shift subject placements for the following test condition according with randomized order. No test was performed immediately before first test condition.

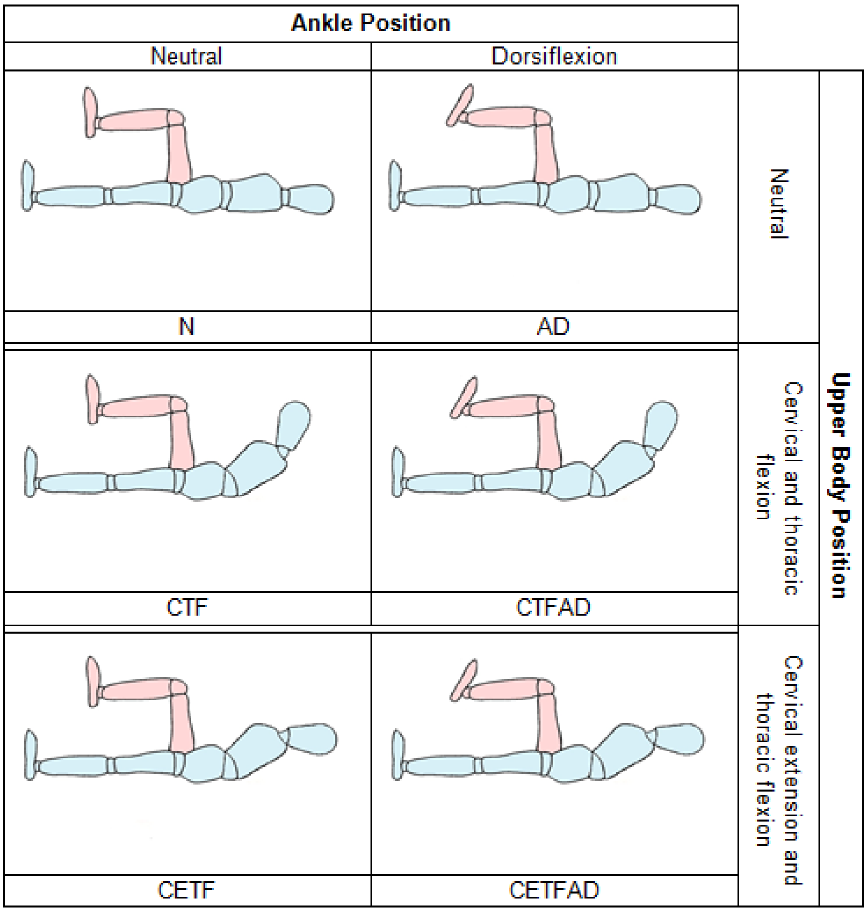

Figure 12. Experimental test conditions according with ankle and upper body position.
Legend: N - neutral position; AD - maximal active ankle dorsiflexion; CTF - maximal active cervical and thoracic flexion; CTFAD - maximal active cervical and thoracic flexion with ankle on maximal active dorsiflexion; CETF - maximal cervical extension with thoracic flexion; CETFAD - maximal active cervical extension with thoracic flexion and ankle on maximal active dorsiflexion.

In each situation, subjects were lying in a supine position on an articulate device specifically designed to this study to hold maximal active positions of cervical and thoracic spine according with test condition protocol, with the right thigh flexed at 90° and fixed to a solid plaque with a velcro with 11cm wide. This was attached to a force sensor fixed to the plaque were the thigh was supported (see below in outcomes section - i.e. thigh fixation). The hip flexion caused the bi-articular hamstring muscles to limit the knee extension, so the subjects could not tolerate full extension of the knee. Left lower limb was controlled in a neutral position with straps around the limb to avoid hip flexion and external rotation, and to attenuate pelvic girdle movement. The right leg of the subjects was firmly strapped by a velcro with 13cm long, to the arm of an apparatus design and described by Freitas et al. (2012). The apparatus (see further details in outcomes section - i.e. passive force) fitted in a dynamometer shaft so that could produce a passive knee extension. The right medial malleolus was aligned to a specific marker of the apparatus arm in all testing conditions. All repetitions started with the apparatus initially positioned parallel to the ground, so the leg could also be at 90° to the thigh as the maximum as possible (i.e.

assumed as slack length position). The passive extension of knee was imposed at an angular velocity of $2°.s^{-1}$ ($0.035\ rad.s^{-1}$) by isokinetic dynamometer. On test conditions where cervical and thoracic spine are not both in neutral positions, subjects performed maximal active movements firstly and then we positioned and stop the articulate device (with cervical and thoracic spine segments according with test condition) in order to hold it in that position (see more information forward - i.e. articulate device). Subjects were instructed to do not perform movements during the experimental protocol apart from the changes in upper body and ankle positions. We particularly instruct subjects to avoided any thoracic and cervical spine flexion and test contra lateral hip flexion and external rotation, and to report during the test the maximum knee range of motion without feeling pain or discomfort in the back of the tight or knee by saying "ok". In all repetitions, an examiner stopped the apparatus lever according subjects indication, and recorded the stretching intensity perception in a visual analog scale (VAS). In this way ROM was limited by the pain/stretch tolerance of the subject. At the end of experimental session, subjects performed three maximal voluntary isometric contraction (MVIC) for knee flexors and extensors, with a contraction duration of 5 seconds and 10 seconds between repetitions, so that could determine the maximal surface EMG activity of the muscles tested. No attempt was made to obscure the vision of the subjects.

III.3.1. Articulate device

This device was designed and built to this specific study. Briefly, it was an iron device (i.e. to ensure greater hardiness and stability) lined with slabs of wood (Figure 13). It allows free and independent movements of cervical (i.e. flexion and extension in sagittal plane from neutral position) and thoracic spine (i.e. flexion in sagittal plane from neutral position) according with test condition. This device was created to be adjustable to any subject. Therefore, we defined the following control anatomical points for the correct alignment of the body parts of subjects when lying on device: **a.** 7th spinous process of cervical vertebrae; **b.** 12th spinous process of thoracic vertebrae; **c.** 5th spinous process of lumbar vertebrae. Firstly, it were marked all landmarks described above and was measure the length between ***b.*** and ***c.*** points in standing position. Thereafter, we positioned subjects lying in a supine position on articulate device with **c.** on top of first wooden slab and **b.** point at the base of second wooden slab, according with length measure between both. The device had a bearing system and hand brakes to adjust positioning and to hold correct alignment of thoracic and cervical spine.

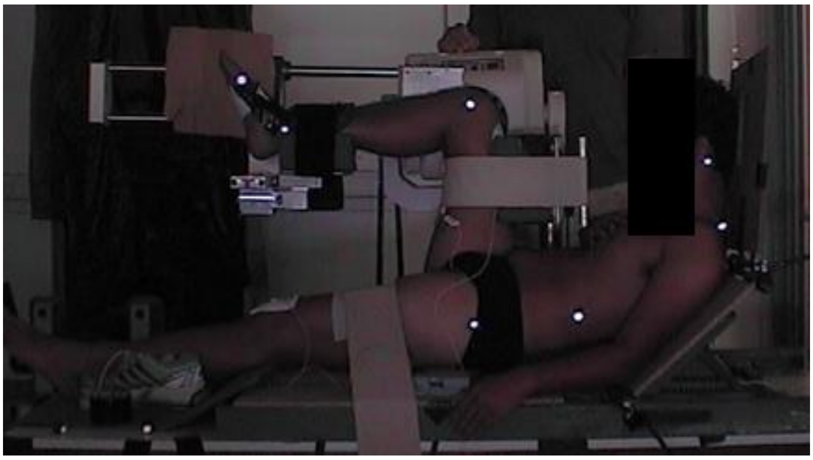

Figure 13. Articulate device allowing maximal cervical and thoracic flexion positions during CTF experimental condition.

III.4. Main outcomes

III.4.1. Passive force

The apparatus described in Figure 14 was specifically designed by Freitas et al. (2012) to measure resistance to passive knee extension (F_P). It was designed to fit the isokinetic dynamometer so it has a slot for the Biodex® axis, and it is composed by two veins with rollers sliding by a system of spheres connected a leg support platform. This allows an independent and free movement of the leg platform relatively to the lever device moved by the dynamometer with a minimum of friction. The apparatus was then projected to ensure that the distance from the F_P measurement site to the knee axis was not affected by the misalignment between Biodex shaft and knee axis (lateral condyle of femur), since this alteration affect torque assessment. A force sensor (platform load cell 1042, Sensor Techniques Ltd, UK) was incorporated in the leg support platform running with a perpendicular direction to subject’s leg. The load cell ranged from 0 to 100kg, measured force with two decimals places, and it was previously calibrated to the data collection with different known weights (0, 2.5, 5, and 10kg). Raw force data were sampled at a 50Hz rate and amplified by a DAS 72.1 (Flintec, Sweden). Force was then smoothed with a low pass filter of 5 Hz.

Figure 14. Apparatus system of spheres sliding freely in two veins in order to accommodate to lever arm length variations along knee extension.

III.4.2. EMG

To monitor if the passive knee extension was imposed without any activity in the muscles around knee joint, surface EMG for the hamstring and quadriceps (i.e. *semintendinousus* - ST_{EMG} - and *quadriceps vastus medialis* - VM_{EMG}) was used to record gross muscle activity. So average of surface EMG was measured during all assessments. For purposes of EMG signal normalization, three repetitions of 5-second maximal voluntary isometric muscle contractions (MVIC) were made for both knee extension and flexion with the knee at 90° in a Biodex, after each protocol session. Surface bipolar electrodes (Analog Devices mod. AD620, gain of 1000±2%) were placed horizontally 20 mm center to center over the mid-portion of each muscle, according to SENIAM guidelines, after abrading and cleaning the skin. The skin was therefore previously shaved, slightly roughened through sandpaper abrasion and cleaned with alcohol. The ground electrode was fixed over the left patella. The EMG signals were amplified (Input Impedance = 5MΩ; Bandpass Filters = 10-500Hz; CMRR >80dB) and A/D converted (MP100 – Biopac™ Systems, 16bits) with a sample rate of 800Hz. Once the EMG data were recorded and after visual inspection, the raw EMG signals were digitally band-pass filtered (20 to 400 Hz), fullwave rectified, low pass filtered with a Butterworth 2nd order and a frequency cut-off of 30 Hz. The average amplitude of the EMG signal was measured during a window of 20 miliseconds. EMG amplitude was then normalized using as reference the EMG of the maximum MVIC for both muscles. A value of EMG than 3% of MVIC was considered the cutoff value to ensure a passive condition.

III.4.3. Joint angle

Ankle (α_{Ankle}), knee (α_{Knee}), hip (α_{Hip}), thoracic ($\alpha_{Thoracic}$) and cervical ($\alpha_{cervical}$) angles were assessed by collecting kinematic data in a sagittal plan (i.e. *2D*) using a digital camera (JVC, GR-DVL9800U).

Skin reflective markers (see at Figure 13) were placed over the following anatomic points in order to determine aforementioned joint angles: head of 1st metatarsal, medial femoral condyle, and medial tibial malleolus of the right lower limb; great trochanter of the left femur; in the left side of the trunk at the intersection of the transverse line passing over the spinous process of the 1st lumbar vertebrae and the line linking the greater trochanter and the midaxilar point; acromion of the left upper limb as a projection of the 7th cervical vertebra; and mastoid apophysis of the left side of cranium. The digital camera was aligned perpendicular to Biodex axis at a distance of 3.02 meters, and thus perpendicular to subject position. The sites of these markers were chosen to allow video recording of overall body segments in a sagital plan, and assure operational conditions to execute the stretching protocol. Data were sampled at a 50Hz rate and processed using Ariel Performance Analysis System (APAS®) to get joint angles. Additionally, α_{Knee} was also assessed during the stretching protocols through the position output from Biodex with a 50Hz sample rate (as well Biodex torque), by aligning knee axis (lateral femoral epicondyle) as close as possible to dynamometer fulcrum. Ankle angle of the right lower limb was measured by a digital goniometer before and after each stretching repetition in the starting position, in order to assure equal ankle angles among test conditions with the same protocol (i.e. Neutral ankle position or maximal active ankle dorsiflexion). Goniometer fulcrum was placed over the medial tibial malleolus, and arms were aligned with the imaginary line between medial tibial malleolus and the femur medial condyle and head of 1st metatarsal.

III.4.4. Thigh stabilization

A force sensor (platform load cell 1042, Sensor techniques Ltd, UK) was attached to the platform that stabilized the thigh so that could record the force produced by the velcro to fasten the thigh. Force collecting data was done using similar equipment and procedures used to collect the F_p. The thigh clamping force was first determined when setting up the subject for the first repetition ensuring the least possible movement of the thigh and the most comfort of the subject. For the follow repetitions, the examiner reproduced as closely as possible the force applied in the first repetition by reading immediately after determining clamping force, because it was seen that this force tends to decrease over time after tightening due to the viscoelastic nature of the soft tissues. The thigh clamping force was continuously recorded during the stretching maneuvers in all repetitions.

III.4.5. Anthropometric measures

Height, weight and leg length was accessed according ISAK guidelines, and convectional instruments. Leg length (L_{leg}) was determined by the distance between medial tibial malleolus and femur medial condyle.

III.4.6. Data processing

All data were synchronized and recorded using BIOPAC MP100 Acquisition System Version 3.5.7 (Santa Barbara, USA), with the exception of α_{Knee} assessed from the digital camera that used a manual trigger linked to Acknowledge Software to synchronize. Data was then synchronized and processed by a specific designed automatic routine using the MATLAB® v7.0 software (The Mathworks Inc, Natick Massachusetts, USA). This routine processed data from both direct measures (i.e. APAS and passive force).

This routine completed the follow steps: **i.** Knee passive torque (PT) for direct measure were determined by multiplying passive resistance to knee extension (F_p) by the leg length (L_{leg}); **ii**. Torque data was gravity corrected by subtracting the leg-foot-device weight (W_{LFD}) to the torque of direct measures, using a simple cosine function (M. McHugh et al., 1992). The W_{LFD} was previously determined in the testing start position, by measuring the average force in one-second interval just previous to the beginning of dynamic phase (i.e. passive knee extension). This was done assuming that leg-foot-device center of mass was horizontally aligned to dynamometer shaft, and tissues mobilized in this stretching maneuver were in a slack length at the starting position. Summarily, processing data of these two steps were executed by the follow equation:

$$PT = (F_P \, . \, L_{Leg}) - (\cos \alpha_{Knee} \, . \, W_{LFD} \, . \, L_{Leg})$$

The routine processed the data with the knee angle (α_{Knee}) and direct torque (T_{Knee}) obtained from digital camera and force sensor separately for further treatment and comparison. Then, **iii.** a specific designed mathematical model were fitted to the torque-angle data for the dynamic phase so that could eliminate forces artifacts. This model consisted on a specific mathematical model proposed by Bruno, Freitas, & Vaz (2012) that was fitted to the torque-angle data for dynamic phase (Bruno et al., 2012). Briefly, dynamic phase was fitted with an exponential model given by the equation:

$$f_D(t) = -b_0 \times \left(1 - \exp\left(\ln\left(1 + \frac{A_1}{b_0}\right)\frac{t}{T_1}\right)\right), \quad 0 \le t \le T_1$$

Where b_0 is a parameter to be estimated, T_1 is the last time of dynamic phase, and A_1 is the true observed ordinate of T_1.

Besides force and ROM measures processing, the automatic routine also normalized the EMG activity of the muscles tested to the maximal EMG obtained in MVIC for knee extension.

III.5. Statistical analysis

Data were processed in IBM SPSS Statistics 20.0 (IBM Corporation, New York, USA) software. In general, descriptive statistics were reported as mean and standard deviation (mean±SD). Data were tested for normality with the Shapiro-Wilk and Kolmogorov tests (P>0.05) and no serious violations were noted. A two-way repeated measures ANOVA [ankle position (neutral vs dorsiflexion) x upper body position (neutral vs thoracic and cervical flexion vs thoracic flexion and cervical extension)] was conducted. Post-hoc analysis was carried out with Bonferroni test. The additional assumption of sphericity was assessed by the Mauchly's test and was in general confirmed (when it was violated, the degrees of freedom were corrected using Greenhouse-Geisser estimates). Intraclass correlation coefficients ($ICC_{3,1}$, was chosen according Chen & Barnhart 2008) and their 95% confidence limit (CI) were computed to determine the reproducibility of the torque-angle parameters between measures. The ICCs were classified as follows: 0.90-0.99, high reliability; 0.80-0.89, good reliability; 0.70-0.79, fair reliability; <0.70, poor reliability (Sekir et al., 2008). Statistical significance was set at p-value<0.05.

IV. Results

IV.1. Experimental condition

IV.1.1. Test condition

Articular angles, i.e. ankle, knee, hip, thoracic and cervical, from start positions were calculated for each test condition in order to evaluate and ensure reliability, between tests conditions, in setup devices and approaches used to hold maximum active required positions during experimental tests.

IV.1.1.1. Knee angle

The mean values of knee start angle were similar for all protocol conditions. Start angles of knee varies about 4.5º (5.2%) between maximum (i.e. N) and minimum (i.e. CETF) averaged values of test conditions (Table 1). No significant differences were found between interaction measures of ankle and trunk positions on all test conditions in knee start angle variable ($F_{(2,18)}=1.31$, $P>0.05$). When cervical flexion and extension, combined with thoracic flexion, components were added to neutral position (i.e. N), a decrease in start angle position was observed.

Table 1. Knee, hip and thoracic spine start angles according with test condition

Test Condition	N	AD	CTF	CTFAD	CEFT	CEFTAD
Knee start angle	86.0 ± 6.1º	84.9 ± 6.1º	82.1 ± 6.5º	82.5 ± 8.4º	81.5 ± 6.3º	82.3 ± 6.3º

Values are given as mean ± SD.

IV.1.1.2. Hip angle

Hip start angles differed between experimental setup conditions (Table 2). The average scores of hip start angles varied about 8.1º (9.2%) between experimental conditions. No significant differences were found between measures taken on all test conditions in hip start angle variable ($F_{(2,18)}=0.48$, $P>0.05$). However, correlations had poor reliability (ICC=0.593 ranged from 0.243 to 0.858; CI=95%). When statistics were made only for hip positions according with upper body positions (i.e. neutral position; cervical and thoracic flexion; cervical extension and thoracic flexion), we observed significant differences between all of them ($F_{(2,18)}=42.54$, $P<0.001$).

Table 2. Average scores of hip start angles for each test condition

Test Condition	N	AD	CTF	CTFAD	CETF	CETFAD
Hip start angle	88.1 ± 5.1º	87.5 ± 4.0º	84.3 ± 4.3º	84.0 ± 5.0º	80.0 ± 6.1º	80.2 ± 5.5º

Values are given as mean ± SD.

IV.1.1.3. Ankle angle

There were statistically significant differences ($F_{(1,18)}$=36.41, P<0.001) for ankle start angles between overall test conditions with dorsiflexion of the ankle and tests with ankle in neutral position (Table 3). There were no statistically significant differences ($F_{(2,18)}$=2.37, P>0.05) for ankle start angles for three different upper body test conditions (neutral position vs cervical and thoracic flexion vs cervical extension and thoracic flexion).

Table 3. Ankle start angle according with test condition and ankle position requirements

Test Condition	Ankle neutral			Ankle dorsiflexion		
	N	CTF	CETF	AD	CTFAD	CETFAD
Ankle start angle	127.3±5.1º	127.1±4.4º	128.9±4.7º	118.6±9.2º	114.4±7.1º	115.9±9.1º

Values are given as mean ± SD.

IV.1.1.4. Cervical angle

Cervical start angles of test conditions (Table 4) were statistically significant different ($F_{(2,18)}$=222.01, P<0.001) for upper body test conditions (neutral position vs cervical and thoracic flexion vs cervical extension and thoracic flexion). When comparisons were made for ankle position (ankle dorsiflexion vs ankle neutral) start angles significant differences were not found ($F_{(1,9)}$=0.88, P>0.05).

Table 4. Cervical start angle according with test condition and cervical required position

Test	Cervical neutral		Cervical flexion		Cervical extension	
	N	AD	CTF	CTFAD	CETF	CETFAD
Cervical	188.1±25.5º	188.1±24.7º	109.9±7.8º	117.1±8.6º	214.0±19.8º	215.1±18.4º

Values are given as mean ± SD.

IV.1.1.5. Thoracic angle

Significant statistical differences ($F_{(2,18)}$=236.31, P<0.001) among upper body measurements were seen in thoracic start angle between conditions (neutral position vs cervical an thoracic flexion vs cervical extension and thoracic flexion) (Table 5). No significant differences among thoracic start angle measurements were seen for ankle position factor ($F_{(1,9)}$=0.14, P>0.05) (dorsiflexion vs neutral) and for interaction of upper body and ankle positions ($F_{(2,18)}$=0.01, P>0.05).

Table 5. Thoracic spine start angles according with test condition

Test Condition	N	AD	CTF	CTFAD	CETF	CETFAD
Thoracic angle	181.3 ± 4.1º	181.1 ± 4.6º	153.0 ± 5.2º	152.9 ± 5.0º	163.5 ± 4.8º	163.1 ± 2.9º

Values are given as mean ± SD.

IV.1.2. Rest intervals between repetitions

The mean time between assessments was 4.3±4.0 minutes (mean±SD). The time for repositioning subjects decreased throughout the experimental protocol (i.e. 1^{st} to 2^{nd} repetition was 6.3 minutes; 2^{nd} to 3^{rd} repetition was 4.8 minutes; 3^{rd} to 4^{th} repetition was 4 minutes; 4^{th} to 5^{th} repetition was 3.5 minutes; and 5^{th} to 6^{th} was 2.8 minutes) (Table 6). No significant differences were found between rest intervals of all experimental conditions ($F_{(1,9)}$=2.00, P>0.05).

Table 6. Time mean between assessments

	Repetitions					
	1-2	**2-3**	**3-4**	**4-5**	**5-6**	**Total**
Time (minutes)	6.3 ± 7.4	4.8 ± 3.6	4.0 ± 3.0	3.5 ± 1.4	2.8 ± 0.9	4.3 ± 4.0

Values are given as mean ± SD.

IV.1.3. Thigh clamping force

No significant differences among measurements were seen in thigh clamping force values (mean±SD; 10.9±1Kg).

IV.2. Maximum values

IV.2.1. Knee angle

Changes in the mean scores of maximum ROM accepted by the subjects were observed between test conditions (Table 7). Maximum ROM was greater (between 7.5 and 10.4%) for neutral position (N) versus all other conditions: AD, CTF, CTFAD and CEFTAD. Between AD, CTF, CTFAD, CETF and CETFAD conditions, maximum ROM values ranged about 1.6º (3.1%) (Figure 15). No significant differences ($F_{(2,18)}$=42.93, P>0.05) among maximum ROM measurement were seen for ankle factor and upper body factor positions interaction. For ankle position factor (ankle dorsiflexion vs ankle neutral) we observed significant differences for maximum ROM accepted by subjects ($F_{(1,9)}$=84.73, P=0.047).

Table 7. Mean values of maximum ROM per each test condition

	Test conditions					
	N	**AD**	**CTF**	**CTFAD**	**CEFT**	**CEFTAD**
Maximum ROM*	55.9 ± 10.9º	50.3 ± 6.9º	50.8 ± 8.3º	50.9 ± 8.4º	51.7 ± 7.8º	50.1 ± 8.3º

Values are given as mean ± SD.
ROM, range of motion; *Higher value = more flexible/extended knee.

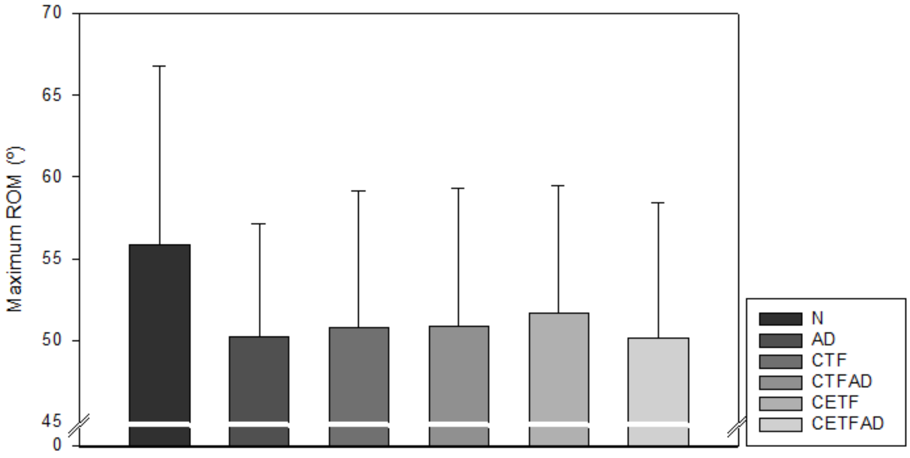

Figure 15. Mean values of maximum ROM per each test condition

IV.2.2. Peak torque

When ankle dorsiflexion was added (i.e. AD, CTFAD and CEFTAD), we observed a decrease in maximum torque compared to the same test with ankle in neutral position (*i.e.* N, CTF, CEFT) (Table 8). Maximum torque accepted by subjects were significant different for ankle position (ankle dorsiflexion vs ankle neutral) ($F_{(1,9)}$=5.25, P=0.048). Maximum torque values were not statistically significant for upper body and ankle position factors interaction ($F_{(2,18)}$=0.31, P>0.05).

Table 8. Mean values of maximum torque per each test condition

	Test conditions					
	N	**AD**	**CTF**	**CTFAD**	**CEFT**	**CEFTAD**
Maximum torque (N m)	44.7 ± 13.0	39.8 ± 9.7	45.8 ± 17.0	44.2 ± 15.6	45.6 ± 13.2	43.2 ± 14.1

Values are given as mean ± SD.

IV.2.3. Visual Analogic Scale

VAS did not differ for interaction of ankle position with upper body position ($F_{(2,18)}$=0.86, P>0.05). It varies about 8.5% between minimum (i.e. AD test condition) and maximum (i.e. CEFTAD test condition) average values accepted by subjects for each condition (

Table 9).

Table 9. Mean values of VAS at maximum ROM

Test Condition	N	AD	CTF	CTFAD	CEFT	CEFTAD
VAS at maximum ROM	70.0 ± 22.0	64.70 ± 24.6	68.9 ± 19.1	68.3 ± 22.2	65.4 ± 27.2	70.7 ± 23.7

Values are given as mean ± SD.
ROM, range of motion; VAS. Visual Analogic Scale.

IV.2.4. EMG

Table 10. Mean values of EMG response for ST_{EMG} and VM_{EMG} at maximum ROM per each test condition

	Test conditions					
	N	AD	CTF	CTFAD	CEFT	CEFTAD
VM_{EMG} (%)	0.05±0.07	0.04±0.06	0.07±0.12	0.06±0.10	0.04±0.06	0.06±0.10
ST_{EMG} (%)	0.05±0.04	0.05±0.04	0.05±0.05	0.05±0.06	0.04±0.03	0.05±0.04

Values are given as mean ± SD.
EMG, electromyography; ST_{EMG}, semintendinousus electromyography; $VM_{EMG,}$ quadriceps vastus medialis.

No significant differences were seen on ST_{EMG} and VM_{EMG} between ankle and upper body positions.

IV.2.5. Influence of ankle dorsiflexion on maximum torque-angle values

Table 11. Averaged results from 10 subjects; changes in knee-joint range of motion (ROM) and in passive resistance to knee extension (torque) between ankle neutral and dorsiflexion position for three upper body positions

Mean change	ROM		Peak Torque	
	Mean	*P*-value	Mean	*P*-value
N vs AD	5.6±7.7º	0.05	4.9±6.4 N m	0.04
CTF vs CTFAD	-0.1±3.4º	0.96	1.6±7.6 N m	0.54
CETF vs CETFAD	1.6±7.3º	0.52	2.4±11.9N m	0.54

Values are given as mean ± SD.

Changes in maximum ROM accepted by the subjects were observed between neutral and dorsiflexion position of ankle. Furthermore, the peak torque was observed to change in accordance with ROM. The decrease in mean ROM, when changing from neutral to dorsiflexion ankle position, was observed between N vs AD and CETF vs CETFAD condition (P>0.05). Peak torque decreased with the addition of ankle dorsiflexion to neutral position (Table 11).

IV.2.6. Influence of upper body position on maximum torque-angle values

Table 12. Averaged results from 10 subjects; changes in knee-joint range of motion (ROM) and in passive resistance to knee extension (torque) between upper body positions for data related to shift in ankle position

Mean change		ROM		Peak Torque	
		Mean	***P*-value**	**Mean**	***P*-value**
Ankle neutral	N vs. CTF	5.1±9.9º	0.14	-1.1±9.6 N m	0.73
	N vs. CETF	4.2±9.3º	0.19	-0.9±9.2 N m	0.76
	CTF vs. CETF	-0.8±6.4º	0.69	0.2±12.6 N m	0.97
Ankle dorsiflexion	AD vs. CTFAD	-0.6±4.1º	0.64	-4.4±7.4 N m	0.10
	AD vs. CETFAD	0.1±3.0º	0.88	-3.4±6.1 N m	0.11
	CTFAD vs. CETFAD	0.8±4.9º	0.62	1.0±7.2 N m	0.68

Values are given as mean ± SD.

The upper body position was also reflected in peak torque and ROM values. The accepted torque was generally found to be higher (*P*>0.05) when cervical and thoracic components were added to neutral position (N) (Table 12). No significant differences were found on changes in maximum ROM and peak torque between neutral positions and upper body modified positions. A seemingly clear statistic results were found when the ankle joint was fixed in dorsiflexion compared to neutral position.

IV.3. Submaximal torque-angle curves

The increase in resistance to stretch with the increasing stretch was progressively greater for the tests with cervical and thoracic flexion (i.e. CTF and CTFAD) *versus* tests with cervical and thoracic spine in neutral position (i.e. N and AD).

Table 13. Results of torque-angle parameters in assessments by different upper body and ankle positions

Ankle Position [a]	**Neutral** [a1]			**Dorsiflexion** [a2]		
Trunk Position [b]	**N** [b1]	**CTF** [b2]	**CETF** [b3]	**AD** [b1]	**CTFAD** [b2]	**CETFAD** [b3]
T100	0.75±0.26	0.91±0.42	0.82±0.26	0.76±0.33	0.83±0.28	0.80±0.28
T200	1.61±0.54	1.94±0.86	1.75±0.54	1.63±0.69	1.77±0.59	1.72±0.59
T300	2.58±0.58	3.11±1.34	2.80±0.85	2.61±1.09	2.84±0.93	2.76±0.93
T400	3.69±1.19	4.41±1.86	3.99±1.20	3.73±1.53	4.05±1.31	3.94±1.30
T500	4.95±1.56	5.89±2.41	5.35±1.59	5.01±2.01	5.43±1.72	5.28±1.70
T600	6.37±1.97	7.55±3.00	6.89±2.01	6.46±2.54	7.00±2.17	6.81±2.13
T700	8.00±2.41	9.43±3.63	8.64±2.48	8.12±3.13	8.78±2.67	8.55±2.61
T800	9.84±2.89	11.56±4.30	10.63±3.00	10.01±3.76	10.80±3.22	10.52±3.12
T900	11.94±3.42	13.96±5.01	12.89±3.58	12.15±4.46	13.09±3.82	12.78±3.69
T1000	14.33±4.00	16.67±5.75	15.45±4.22	14.60±5.22	15.70±4.47	15.34±4.29

Values are reported as mean±SD. Legend: T100 - Torque at 3°; T200 - Torque at 7°; T300 - Torque at 10°; T400 - Torque at 13°; T500 - Torque at 16°; T600 - Torque at 19°; T700 - Torque at 23°; T800 - Torque at 26°; T900 - Torque at 29°; T1000 - Torque at 32°.

[1] - Significant differences were found between trunk position (b) for T300, T400, T500, T600, T700, T800, T900 and T1000 for torque variable. Bonferroni multiple comparisons shows significant differences between b1 vs b2 trunk positions for ankle in neutral position (a1).

[2] - No significant differences were found between measures taken in T100, T200, T300, T400, T500, T600, T700, T800, T900 and T1000 in a*b interaction for torque variable.

[3] - No significant differences were found between measures taken in T100, T200, T300, T400, T500, T600, T700, T800, T900, andT1000 for ankle position [a = (a1 vs a2)] factor in torque variable.

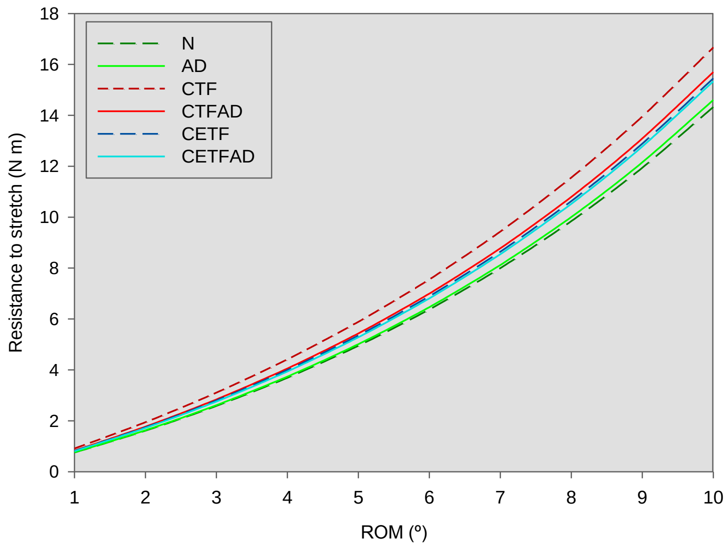

Figure 16. Resistance to stretch throughout the range of motion (ROM) for the six test conditions.
Legend: ROM1 - 3°; ROM2 - 7°; ROM3 - 10°; ROM4 - 13°; ROM5 - 16°; ROM6 - 19°; ROM7 - 23°; ROM8 - 26°; ROM9 - 29°; ROM10 - 32°.

V. Discussion

The main finding in this study was that adding neural tension components (namely thoracic and cervical flexion – CTF and CTFAD), during a hamstring stretch, significantly increased the resistance to stretch, observed on torque-angle curves (for submaximal ROM), compared with a hamstring stretch with the spine in a neutral position (N and AD). Observed effects were, for example, 14% significantly higher for resistance to stretch at the maximum common angle between CTF vs N test conditions. Therefore we shown that trunk position (i.e. namely cervical and thoracic segments) significantly affects torque-angle response during PKE test. Although without significant differences, increased resistance to stretch was also proved to be clearly higher on cervical extension and thoracic flexion (CETF and CETFAD) compared with spine in neutral position (N and AD) but lower when it was compared with cervical and thoracic flexion (CTF and CTFAD) (Figure 16). Maximal flexion of the all spine is used in the slump test as described by Maitland (1985). Similarities can be found between hamstring torque-angle assessment, e.g. experimental setup used by Magnusson (1998) and slump test. We can also find relationships between this assessment and positions of test conditions used in this study. Spinal flexion, including cervical flexion, places traction on the lumbosacral nerve roots and sciatic nerve. Breig & Marions (1963) and Ko et al. (2006) reported that the dura, root sleeves, cord, and nerve roots move cranially during maximum flexion of the trunk. Full spinal flexion, or flexion of the cervical, thoracic, and lumbar regions of the spine, produces lengthening of the vertebral canal. Thereafter, when the vertebral canal is elongated, the spinal dura is stretched, transmitting tension to the spinal cord, lumbosacral nerve root sleeves, and nerve root (Breig & Marions, 1963; Kleinrensink et al., 1995; Reid, 1960). During full spinal flexion, the cauda equina becomes taut and the lumbosacral nerve roots and root sleeves are pulled into contact with the pedicle of the superior vertebra. However, when extension of the cervical spine is introduced, the dura and the nerve roots slacken as the vertebral canal begins to shorten (Breig & Marions, 1963; Reid, 1960). In other hand, cervical flexion places additional tension on the lumbosacral nerve roots by placing traction on the neuromeningeal tract and therefore can influence nerve root/sciatic tension (Breig & Marions, 1963; Butler, 2000; Shacklock, 2005). We verified in this study that cervical position (flexion vs extension) have direct influence on resistance to stretch of hamstring muscle for the same thoracic flexion position during PKE when it was compared with neutral position, since in both (CTF and CETF) thoracic was in the same position. Thus it was shown that cervical flexion caused higher torque values than cervical extension for submaximal ROM. Accordingly, it can be assumed that the cervical and thoracic components play an important role in the torque-angle relationship during PKE caused by the possible tension that they can apply on the neural tract according with their positioning. Tensioning of the neural structures during passive stretch is the basis of the slump test. We observed that in the absence of significant contractile activity (low EMG signal) an increased resistance to hamstring stretch in CTF/CTFAD condition can be attributed to tensile force applied along neural tract.

The increased resistance to stretch was not due to a change in the contractile response to stretch. The contractile response did not differ between test conditions. The small EMG response (*semintendinousus* and *quadriceps vastus medialis*) to passive knee extension among test conditions is consistent with previous work (Laessøe & Voigt, 2004; McHugh et al., 2012) and emphasizes that resistance is primarily due to passive extensibility of the tissues under stretch.

Ankle position significantly affects resistance to stretch on maximum ROM. However, differences between tests conditions on resistance to stretch were not observed when interaction ankle × upper body positions was made. Nevertheless, a dorsiflexion in the ankle joint is believed to add to a stretch on the sciatic nerve (Breig, 1978). Therefore, another finding was that ankle dorsiflexion position during a hamstring stretch resulted in a significant decreased in the maximum ROM that the subject could achieve. Thus, a shift in ankle position from neutral to dorsiflexion caused a decrease in ROM during passively imposed knee extension. These observations could therefore support the view that elongation of the peripheral nerve tissue plays a role in stretch tolerance. Thus this finding supports the view that ROM can be influenced by stretch tolerance and that the stretch tolerance apparently is acutely diminished when an ankle dorsiflexion is assumed. It is known that dorsiflexion also limits maximum active knee extension ROM in slump test (Johnson & Chiarello, 1997). These results are agreement with those achieved by Laessøe & Voigt (2004) and McHugh et al. (2012) studies. Furthermore, *gastrocnemius* are biarticular muscles since they cross the knee and ankle joint. Therefore, passive stretch of those muscles (with ankle in dorsiflexion) would provide additional passive stretch to the hamstring because tension in the *gastrocnemius* muscles would tend to pull the knee into extension.

The experimental setup has still some limitations on the measures taken as well describe Freitas et al. (2012): **i.** the weight of device may have produced transverse forces during knee extension, and thus may have affected the measure of force perpendicular to the leg; **ii.** the marker of tested trochanter was assumed to be aligned with non-tested trochanter, and that may have affected the exact positioning of hip/thigh tested in testing sessions; **iii.** the clamping force of leg fixation was not controlled; **iv.** in some individuals it was observed some tibial external rotation during knee extension, that may have affected the force measurement; **v.** some subjects reported foot numbness, probably originated from leg and thigh fixation, and this may have affected subject perception and consequently maximum knee angle performed; **vi.** the system of spheres incorporated in the device might have affected the torque measurement by the dynamometer. Furthermore, the articulate device used to set upper body positions could lead a misalignment of pelvis between six experimental conditions since there was not any type of pelvic or lumbar fixation. Thus a difference in sagittal plane pelvic rotation between the six stretch techniques is an important confounding factor in the experimental intervention used in this study. Because marked differences in submaximal resistance to stretch were substantial between test conditions, the confounding factor was probably not a limiting factor. However, O'Sullivan et al. (2006) documented posterior pelvic tilt in slump test position which is also compatible with cervical and thoracic in flexion position. These positions may decrease the stretch on the hamstring and therefore torque-angle measurement.

Moreover, since cervical position can influence submaximal torque-angle results, also pressure imposed to cervical in order to hold it in flexion position can change results. Cervical flexion has been widely advocated as an additional maneuver, usually executed by an external forces (e.g. manually by physiotherapists), to the slump test in order to further tension the neural system leading to further neural sensitization and symptom reproduction (Breig & Troup, n.d.; Butler, 2000; Shacklock, 2005). In this study we try to avoid this bias instructing subjects to do maximal active cervical flexion and then we hold it through articulate device properties. Therefore, we supposed that pressure over occipital bone was constant during tests ensuring no variations between experimental conditions. In other studies accessing PKE, it is common to see Magnusson's experimental protocol with the subject seated in the dynamometer chair and the tested thigh flexed 30° to a horizontal line (Laessøe & Voigt, 2004; Magnusson, 1998; McHugh et al., 2012). Freitas et al. (2012) showed that altering the non-tested thigh position affects the torque-angle response and stretching tolerance. Therefore we speculate if reliability it is not affected in that testing experimental position since non-tested thigh was not stabilized. Another issue it is regarding studies that argue that flexibility gains are consequence of stretch tolerance mechanisms. As seen in the present study, either stretch intensity and torque-angle outcomes changed with the position of foot and upper body segments. We speculate if in that testing posture in which the position of the body segments easily changes without real perception by the tester that will not affect the reliability of the test.

Other elements such as back muscles and spinal ligaments are mechanically effected by the shift in upper-body position. For example, extension of the lumbodorsal fascia occurs when a slump position is assumed, and also the back muscles would be involved. Within the passive structures, it appears that there is an elongation in the tendon-aponeurosis complex and muscle fascia during passive stretch. Those factors could also contribute to stretch tolerance and resistance to passive torque during PKE with various upper body positions.

Prospective studies on hamstring injuries have reported that injured elite soccer players had significantly reduced hamstring flexibility in comparison with un-injured elite soccer players (Bradley & Portas, 2007; Witvrouw et al., 2003) and older retrospective studies reported that previously injured athletes had significantly lower hamstring flexibility in comparison to un-injured athletes (Jönhagen et al., 1994; Worrell, Perrin, Gansneder, & Gieck, 1991). Worrell et al. (1991) reported an asymmetry between legs (i.e. injured and non-injured leg) in hamstring flexibility during rehabilitation after injury, with the injured leg being significantly less flexible. This lack in injured leg flexibility can be attributed to adverse neural tension which was described as abnormal physiological and mechanical responses produced from the nervous system structures when their normal range of movement and stretch capabilities are tested (Butler, 2000; Shacklock, 2005; Turl & George, 1998). Our results showed increased tension during changes of upper body position compatible with PNS manipulation. Traditional static stretching techniques that involve the combination of cervical flexion, hip flexion and dorsiflexion have been shown to increase neural tension (Turl & George, 1998). Since tensioning of the neural structures during passive stretch is the basis of the neural tension tests (Butler, 2000), increased tension in injured athletes may be due to resistance to elongation of the neural tissues (intraneural) or adhesions between the neural tissues and the surrounding tissues preventing the neural tissues from gliding freely (extraneural) (Turl & George, 1998). In order to assess hamstring flexibility and avoid neural tension Hunter & Speed (2007) recommend the active knee extension test as opposed to the straight leg raise. However in this study we verified that positions of non-mechanically direct related segments, e.g. ankle and upper body positions, should be taken in account during these kind of exercises in order to avoid additional tension on hamstring muscle (for example, during rehabilitation protocol).

V.1. Perspectives

In this study the increase in passive torque to hamstring stretch for submaximal ROM on cervical and thoracic flexion, in absence important or EMG activity, indicates that manipulation of body segments positions, which are not mechanically related with knee joint, can contribute to musculoskeletal flexibility. Therefore we can hypothesize that elongation of neural tract can influence hamstring flexibility.

We observed that voluntary accepted knee joint ROM and torque could be influenced by stretch of structures that there were not mechanically related to the knee, namely ankle position. Therefore stretch tolerance may be affected by positioning of those segments and clear by local mechanical structures as described in review of literature.

These findings are relevant outcomes both to researchers, i.e. for torque-angle experimental assessments, and to clinicians, e.g. physiotherapists and sport specialists, in order to better assess hamstring injuries and define rehabilitations protocols.

VI. References

Alshami, A. M., Babri, A. S., Souvlis, T., & Coppieters, M. W. (2008). Strain in the tibial and plantar nerves with foot and ankle movements and the influence of adjacent joint positions. *Journal of applied biomechanics*, *24*(4), 368–76. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/19075306

Alter, M. (2004). *Science of Flexibility* (Third.). USA: Human Kinetics Publishers.

Anderson, D., Nussbaum, M., & Madigan, M. (2010). A new method for gravity correction of dynamometer data and determining passive elastic moments at the joint. *Journal of biomechanics*, *43*(6), 1220–3. doi:10.1016/j.jbiomech.2009.11.036

Anderson, J., Li, Z., & Goubel, F. (2002). Models of skeletal muscle to explain the increase in passive stiffness in desmin knockout muscle. *Journal of biomechanics*, *35*(10), 1315–24. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12231277

Axelson, H. W., & Hagbarth, K. E. (2001). Human motor control consequences of thixotropic changes in muscular short-range stiffness. *The Journal of physiology*, *535*(Pt 1), 279–88. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2278763&tool=pmcentrez&rendertype=abstract

Blackburn, J. T., Padua, D. a, Riemann, B. L., & Guskiewicz, K. M. (2004). The relationships between active extensibility, and passive and active stiffness of the knee flexors. *Journal of electromyography and kinesiology : official journal of the International Society of Electrophysiological Kinesiology*, *14*(6), 683–91. doi:10.1016/j.jelekin.2004.04.001

Bradley, P. S., & Portas, M. D. (2007). The relationship between preseason range of motion and muscle strain injury in elite soccer players. *Journal of strength and conditioning research / National Strength & Conditioning Association*, *21*(4), 1155–9. doi:10.1519/R-20416.1

Breig, A. (1978). *Adverse Mechanical Tension in the Central Nervous System*. Stokholm: Almquist & Wiksell.

Breig, A., & Marions, O. (1963). Biomechanics of the lumbosacral nerve roots. *Acta radiologica: diagnosis*, *1*, 1141–60. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/14086403

Breig, A., & Troup, J. D. (n.d.). Biomechanical considerations in the straight-leg-raising test. Cadaveric and clinical studies of the effects of medial hip rotation. *Spine*, *4*(3), 242–50. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/157532

Brodie, T. G. (1895). The Extensibility of Muscle. *Journal of anatomy and physiology*, *29*(Pt 3), 367–388.3. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1328411&tool=pmcentrez&rendertype=abstract

Bruno, P. M., Freitas, S. R., & Vaz, J. R. (2012). Adjustment of knee passive extension torque-angle raw data by different mathematical models. *Clinical Biomechanics*.

Butler, D. S. (2000). *The Sensitive Nervous System*. Adelaide, Australia: Noigroup Publications.

Byl, C., Puttlitz, C., Byl, N., Lotz, J., & Topp, K. (2002). Strain in the median and ulnar nerves during upper-extremity positioning. *The Journal of hand surgery*, *27*(6), 1032–40. doi:10.1053/jhsu.2002.35886

Caiozzo, V. J. (2002). Plasticity of skeletal muscle phenotype: mechanical consequences. *Muscle & nerve*, *26*(6), 740–68. doi:10.1002/mus.10271

Campbell, K. S., & Lakie, M. (1998). A cross-bridge mechanism can explain the thixotropic short-range elastic component of relaxed frog skeletal muscle. *The Journal of physiology*, *510 (Pt 3*, 941–62. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2231083&tool=pmcentrez&rendertype=abstract

Coppieters, M. W., & Alshami, A. M. (2007). Longitudinal excursion and strain in the median nerve during novel nerve gliding exercises for carpal tunnel syndrome. *Journal of orthopaedic research : official publication of the Orthopaedic Research Society*, *25*(7), 972–80. doi:10.1002/jor.20310

Coppieters, M. W., Alshami, A. M., Babri, A. S., Souvlis, T., Kippers, V., & Hodges, P. W. (2006). Strain and Excursion of the Sciatic , Tibial , and Plantar Nerves during a Modified Straight Leg Raising Test, (September), 1883–1889. doi:10.1002/jor

Coppieters, M. W., & Butler, D. S. (2008). Do “sliders” slide and “tensioners” tension? An analysis of neurodynamic techniques and considerations regarding their application. *Manual therapy*, *13*(3), 213–21. doi:10.1016/j.math.2006.12.008

Dilley, A., Lynn, B., Greening, J., & DeLeon, N. (2003). Quantitative in vivo studies of median nerve sliding in response to wrist, elbow, shoulder and neck movements. *Clinical Biomechanics*, *18*(10), 899–907. doi:10.1016/S0268-0033(03)00176-1

Dilley, A., Summerhayes, C., & Lynn, B. (2007). An in vivo investigation of ulnar nerve sliding during upper limb movements. *Clinical biomechanics (Bristol, Avon)*, *22*(7), 774–9. doi:10.1016/j.clinbiomech.2007.04.004

Ellis, R. F., Hing, W. a, & McNair, P. J. (2012). Comparison of Different Neural Mobilization Exercises Upon Longitudinal Sciatic Nerve Movement: An In-Vivo Study Utilizing Ultrasound Imaging. *The Journal of orthopaedic and sports physical therapy*, *42*(8), 667–675. doi:10.2519/jospt.2012.3854

Ellis, R., Hing, W., Dilley, A., & McNair, P. (2008). Reliability of measuring sciatic and tibial nerve movement with diagnostic ultrasound during a neural mobilisation technique. *Ultrasound in medicine & biology*, *34*(8), 1209–16. doi:10.1016/j.ultrasmedbio.2008.01.003

Enoka, R. (2002). *Neuromechanics of Human Movement* (3rd ed.). Champaign, IL: Human Kinetics Publishers.

Erel, E., Dilley, A., Greening, J., Morris, V., Cohen, B., & Lynn, B. (2003). Longitudinal sliding of the median nerve in patients with carpal tunnel syndrome. *Journal of hand surgery (Edinburgh, Scotland)*, *28*(5), 439–43. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12954253

Freitas, S., Vaz, J., Bruno, P., Valamatos, M. J., & Mil-Homens, P. (2012). Comparison of different knee extension passive torque-angle assessments. *Clinical Biomechanics*.

Fung, Y. C. (1983). *Biomechanics. Mechanical Properties of Living Tissues*. Springer-Verlag.

Gajdosik, R. L. (2001). Passive extensibility of skeletal muscle: review of the literature with clinical implications. *Clinical biomechanics (Bristol, Avon)*, *16*(2), 87–101. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11222927

Garrett, W. E., Nikolaou, P. K., Ribbeck, B. M., Glisson, R. R., & Seaber, A. V. (n.d.). The effect of muscle architecture on the biomechanical failure properties of skeletal muscle under passive extension. *The American journal of sports medicine*, *16*(1), 7–12. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/3344884

Garrett, W. E., Safran, M. R., Seaber, A. V, Glisson, R. R., & Ribbeck, B. M. (n.d.). Biomechanical comparison of stimulated and nonstimulated skeletal muscle pulled to failure. *The American journal of sports medicine*, *15*(5), 448–54. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/3674268

Gleim, G. W., & McHugh, M. P. (1997, November). Flexibility and its effects on sports injury and performance. *Sports medicine (Auckland, N.Z.)*. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/22596073

Goubel, F., & Lensel-Corbeil, G. (2003). *Biomécanique: éléments de mécanique musculaireTitle*. Paris: Masson.

Halbertsma, J. P., & Göeken, L. N. (1994). Stretching exercises: effect on passive extensibility and stiffness in short hamstrings of healthy subjects. *Archives of physical medicine and rehabilitation*, *75*(9), 976–81. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8085933

Halbertsma, J. P., Mulder, I., Göeken, L. N., & Eisma, W. H. (1999). Repeated passive stretching: acute effect on the passive muscle moment and extensibility of short hamstrings. *Archives of physical medicine and rehabilitation*, *80*(4), 407–14. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10206602

Halbertsma, J. P., Van Bolhuis, a I., & Göeken, L. N. (1996). Sport stretching: effect on passive muscle stiffness of short hamstrings. *Archives of physical medicine and rehabilitation*, *77*(7), 688–92. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8669996

Harvey, L. A., McQuade, L., Hawthorne, S., & Byak, A. (2003). Quantifying the magnitude of torque physiotherapists apply when stretching the hamstring muscles of people with spinal cord injury. *Archives of physical medicine and rehabilitation*, *84*(7), 1072–5. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12881837

Haycraft, J. B. (1904). The elasticity of animal tissues. *The Journal of physiology*, *31*(5), 392–409. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1465594&tool=pmcentrez&rendertype=abstract

Herbert, R. D., Moseley, A. M., Butler, J. E., & Gandevia, S. C. (2002). Change in length of relaxed muscle fascicles and tendons with knee and ankle movement in humans, 637–645. doi:10.1013/jphysiol.2001.012756

Herrington, L., Bendix, K., Cornwell, C., Fielden, N., & Hankey, K. (2008). What is the normal response to structural differentiation within the slump and straight leg raise tests? *Manual therapy*, *13*(4), 289–94. doi:10.1016/j.math.2007.01.013

Herzog, J. a, Leonard, T. R., Jinha, A., & Herzog, W. (2012). Are titin properties reflected in single myofibrils? *Journal of biomechanics*, *45*(11), 1893–9. doi:10.1016/j.jbiomech.2012.05.021

Heslinga, J. W., & Huijing, P. A. (1990). Effects of growth on architecture and functional characteristics of adult rat gastrocnemius muscle. *Journal of morphology*, *206*(1), 119–32. doi:10.1002/jmor.1052060112

Hill, D. K. (1968). Tension due to interaction between the sliding filaments in resting striated muscle. The effect of stimulation. *The Journal of physiology*, *199*(3), 637–84. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1365364&tool=pmcentrez&rendertype=abstract

Hill, A. V. (1938). The Heat of Shortening and the Dynamic Constants of Muscle. *Proceedings of the Royal Society B: Biological Sciences*, *126*(843), 136–195. doi:10.1098/rspb.1938.0050

Hill, A. V. (1951). The Effect of Series Compliance on the Tension Developed in a Muscle Twitch. doi:10.1098/rspb.1951.0025

Hoang, P. D., Gorman, R. B., Todd, G., Gandevia, S. C., & Herbert, R. D. (2005). A new method for measuring passive length-tension properties of human gastrocnemius muscle in vivo. *Journal of biomechanics*, *38*(6), 1333–41. doi:10.1016/j.jbiomech.2004.05.046

Horowits, R., Kempner, E. S., Bisher, M. E., & Podolsky, R. J. (1986). A physiological role for titin and nebulin in skeletal muscle. *Nature*, *323*(6084), 160–4. doi:10.1038/323160a0

Hough, A. D., Moore, A. P., & Jones, M. P. (2007). Reduced longitudinal excursion of the median nerve in carpal tunnel syndrome. *Archives of physical medicine and rehabilitation*, *88*(5), 569–76. doi:10.1016/j.apmr.2007.02.015

Hunter, D. G., & Speed, C. A. (2007). The assessment and management of chronic hamstring/posterior thigh pain. *Best practice & research. Clinical rheumatology*, *21*(2), 261–77. doi:10.1016/j.berh.2006.12.002

Jewell, B., & Wilkie, D. (1958). An analysis of the mechanical components in frog's striated muscle. *The Journal of physiology*, *143*(3), 515–40. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1356730&tool=pmcentrez&rendertype=abstract

Johns, R. J., & Wright, V. (1962). Relative importance of various tissues in joint stiffness. *Journal of Applied Physiology*, *17*(5), 824–828.

Johnson, E. K., & Chiarello, C. M. (1997). The slump test: the effects of head and lower extremity position on knee extension. *The Journal of orthopaedic and sports physical therapy*, *26*(6), 310–7. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9402567

Jönhagen, S., Németh, G., & Eriksson, E. (1994). Hamstring injuries in sprinters. The role of concentric and eccentric hamstring muscle strength and flexibility. *The American journal of sports medicine*, *22*(2), 262–6. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8198197

Keir, P. J., & Rempel, D. M. (2005). Pathomechanics of peripheral nerve loading. Evidence in carpal tunnel syndrome. *Journal of hand therapy : official journal of the American Society of Hand Therapists*, *18*(2), 259–69. doi:10.1197/j.jht.2005.02.001

Kjaer, M. (2004). Role of extracellular matrix in adaptation of tendon and skeletal muscle to mechanical loading. *Physiological reviews*, *84*(2), 649–98. doi:10.1152/physrev.00031.2003

Kleinrensink, G. J., Stoeckart, R., Vleeming, a, Snijders, C. J., & Mulder, P. G. H. (1995). Mechanical tension in the median nerve. The effects of joint positions. *Clinical biomechanics (Bristol, Avon)*, *10*(5), 240–244. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11415560

Knudson, D. (2007). *Fundamentals of Biomechanics* (2nd ed.). New York: Springer.

Ko, H.-Y., Park, B. K., Park, J. H., Shin, Y. B., Shon, H. J., & Lee, H. C. (2006). Intrathecal movement and tension of the lumbosacral roots induced by straight-leg raising. *American journal of physical medicine & rehabilitation / Association of Academic Physiatrists*, *85*(3), 222–7. doi:10.1097/01.phm.0000200386.28819.6a

Kubo, K., Kanehisa, H., & Fukunaga, T. (2001). Is passive stiffness in human muscles related to the elasticity of tendon structures? *European Journal of Applied Physiology*, *85*(3-4), 226–232. doi:10.1007/s004210100463

Kubo, K., Kanehisa, H., & Fukunaga, T. (2003). Gender differences in the viscoelastic properties of tendon structures. *European journal of applied physiology*, *88*(6), 520–6. doi:10.1007/s00421-002-0744-8

Kubo, K., Kanehisa, H., & Fukunaga, T. (2005). Comparison of elasticity of human tendon and aponeurosis in knee extensors and ankle plantar flexors in vivo. *Journal of applied biomechanics*, *21*(2), 129–42. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16082014

Kubo, K., Kanehisa, H., Kawakami, Y., Fukunaga, T., Fouré, A., Nordez, A., Cornu, C., et al. (2011). Influence of static stretching on viscoelastic properties of human tendon structures in vivo Influence of static stretching on viscoelastic properties of human tendon structures in vivo, 520–527.

Laessøe, U., & Voigt, M. (2004). Modification of stretch tolerance in a stooping position. *Scandinavian journal of medicine science in sports*, *14*(4), 239–244. Retrieved from http://www3.interscience.wiley.com/journal/118785899/abstract

Lamontagne, A., Malouin, F., Richards, C. L., & Dumas, F. (1997). Impaired viscoelastic behaviour of spastic plantarflexors during passive stretch at different velocities. *Clinical biomechanics (Bristol, Avon)*, *12*(7-8), 508–515. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11415761

Lew, P. C., & Briggs, C. A. (1997). Relationship between the cervical component of the slump test and change in hamstring muscle tension. *Manual therapy*, *2*(2), 98–105. doi:10.1054/math.1997.0291

Lieber, R. L., & Bodine-Fowler, S. C. (1993). Skeletal muscle mechanics: implications for rehabilitation. *Physical therapy*, *73*(12), 844–56. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8248293

Magnusson, S. P. (1998). Passive properties of human skeletal muscle during stretch maneuvers. A review. *Scandinavian journal of medicine & science in sports*, *8*(2), 65–77. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9564710

Magnusson, S. P., Simonsen, E. B., Aagaard, P., Sørensen, H., & Kjaer, M. (1996). A mechanism for altered flexibility in human skeletal muscle. *The Journal of physiology*, *497 (Pt 1*(1996), 291–8. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1160931&tool=pmcentrez&rendertype=abstract

Maïsetti, O., Hug, F., Bouillard, K., & Nordez, A. (2012). Characterization of passive elastic properties of the human medial gastrocnemius muscle belly using supersonic shear imaging. *Journal of biomechanics*, *45*(6), 978–84. doi:10.1016/j.jbiomech.2012.01.009

Maitland, G. D. (1985). The slump test: examination and treatment. *Aust J Physiother.*, *31*, 215–219.

McHugh, M., Magnusson, S., Gleim, G., & Nicholas, J. (1992). Viscoelastic stress relaxation in human skeletal muscle. *Med Sci Sports Exerc.*, *24*(12), 1375–82.

McHugh, M. P., Johnson, C. D., & Morrison, R. H. (2012). The role of neural tension in hamstring flexibility. *Scandinavian journal of medicine & science in sports*, *22*(2), 164–9. doi:10.1111/j.1600-0838.2010.01180.x

McLellan, D. L., & Swash, M. (1976). Longitudinal sliding of the median nerve during movements of the upper limb. *Journal of neurology, neurosurgery, and psychiatry*, *39*(6), 566–70. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=492349&tool=pmcentrez&rendertype=abstract

McNair, P., Dombroski, E., Hewson, D., & Stanley, S. (2001). Stretching at the ankle joint: viscoelastic responses to holds and continuous passive motion. *Medicine and science in sports and exercise*, *33*(3), 354–8. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11252058

McNair, P., Hewson, D., Dombroski, E., & Stanley, S. (2002). Stiffness and passive peak force changes at the ankle joint: the effect of different joint angular velocities. *Clinical biomechanics (Bristol, Avon)*, *17*(7), 536–40. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12206945

McNair, P. J., & Portero, P. (2005). Using isokinetic dynamometers for measurements associated with tissue extensibility. *Isokinetics and Exercise Science*, *13*, 53–56.

Millesi, H., Zöch, G., & Reihsner, R. (1995). Mechanical properties of peripheral nerves. *Clinical orthopaedics and related research*, (314), 76–83. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7634654

Mutungi, G., & Ranatunga, K. W. (1996a). The viscous, viscoelastic and elastic characteristics of resting fast and slow mammalian (rat) muscle fibres. *The Journal of physiology*, *496 (Pt 3*, 827–36. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1160867&tool=pmcentrez&rendertype=abstract

Mutungi, G., & Ranatunga, K. W. (1996b). The visco-elasticity of resting intact mammalian (rat) fast muscle fibres. *Journal of muscle research and cell motility*, *17*(3), 357–64. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8814555

Mutungi, G., & Ranatunga, K. W. (1996c). Tension relaxation after stretch in resting mammalian muscle fibers: stretch activation at physiological temperatures. *Biophysical journal*, *70*(3), 1432–8. doi:10.1016/S0006-3495(96)79702-3

Mutungi, G., & Ranatunga, K. W. (1998). Temperature-dependent changes in the viscoelasticity of intact resting mammalian (rat) fast- and slow-twitch muscle fibres. *The Journal of physiology*, *508 (Pt 1*, 253–65. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2230871&tool=pmcentrez&rendertype=abstract

Mutungi, G., & Ranatunga, K. W. (2000). Do cross-bridges contribute to the tension during stretch of passive muscle? A response. *Journal of muscle research and cell motility*, *21*(3), 301–2. doi:10.1023/A:1005633931146

Nordez, A, Casari, P., & Cornu, C. (2008). Effects of stretching velocity on passive resistance developed by the knee musculo-articular complex: contributions of frictional and viscoelastic behaviours. *European journal of applied physiology*, *103*(2), 243–50. doi:10.1007/s00421-008-0695-9

Nordez, A, Gennisson, J. L., Casari, P., Catheline, S., & Cornu, C. (2008). Characterization of muscle belly elastic properties during passive stretching using transient elastography. *Journal of biomechanics*, *41*(10), 2305–11. doi:10.1016/j.jbiomech.2008.03.033

Nordez, Antoine, Casari, P., Mariot, J., & Cornu, C. (2009). Modeling of the passive mechanical properties of the musculo-articular complex: acute effects of cyclic and static stretching. *Journal of biomechanics*, *42*(6), 767–73. doi:10.1016/j.jbiomech.2008.12.019

Nordez, Antoine, Cornu, C., & McNair, P. (2006). Acute effects of static stretching on passive stiffness of the hamstring muscles calculated using different mathematical models. *Clinical biomechanics (Bristol, Avon)*, *21*(7), 755–60. doi:10.1016/j.clinbiomech.2006.03.005

Nordez, Antoine, Fouré, A., Dombroski, E., Mariot, J.-P., Cornu, C., & McNair, P. (2010). Improvements to Hoang et al.'s method for measuring passive length-tension properties of human gastrocnemius muscle in vivo. *Journal of biomechanics*, *43*(2), 379–82. doi:10.1016/j.jbiomech.2009.07.034

Özkaya, N., & Nordin, M. (1999). *Fundamentals of Biomechanics: Equilibrium Motion, and Deformation* (2nd ed.). New York: Springer.

Phillips, J. B., Smit, X., De Zoysa, N., Afoke, A., & Brown, R. A. (2004). Peripheral nerves in the rat exhibit localized heterogeneity of tensile properties during limb movement. *The Journal of physiology*, *557*(Pt 3), 879–87. doi:10.1113/jphysiol.2004.061804

Proske, U., & Morgan, D. L. (1999). Do cross-bridges contribute to the tension during stretch of passive muscle? *Journal of muscle research and cell motility*, *20*(5-6), 433–42. doi:10.1023/A:1005573625675

Proske, U., Morgan, D. L., & Gregory, J. E. (1993). Thixotropy in skeletal muscle and in muscle spindles: a review. *Progress in neurobiology*, *41*(6), 705–21. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8140258

Purslow, P. (1989). Strain-induced reorientation of an intramuscular connective tissue network: implications for passive muscle elasticity. *Journal of biomechanics*, *22*(1), 21–31. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/2914969

Purslow, P. (2010). Muscle fascia and force transmission. *Journal of bodywork and movement therapies*, *14*(4), 411–7. doi:10.1016/j.jbmt.2010.01.005

Purslow, P., Wess, J., & Hukins, W. (1998). Collagen orientation and molecular spacing during creep and stress-relaxation in soft connective tissues. *The Journal of experimental biology*, *201*(Pt 1), 135–42. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9390944

Rabita, G., Dupont, L., Thevenon, A., Lensel-Corbeil, G., Pérot, C., & Vanvelcenaher, J. (2005). Quantitative assessment of the velocity-dependent increase in resistance to passive stretch in spastic plantarflexors. *Clinical biomechanics (Bristol, Avon)*, *20*(7), 745–53. doi:10.1016/j.clinbiomech.2005.04.002

Reid, J. D. (1960). Effects of flexion-extension movements of the head and spine upon the spinal cord and nerve roots. *Journal of neurology, neurosurgery, and psychiatry*, *23*, 214–21. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=497411&tool=pmcentrez&rendertype=abstract

Rempel, D., Dahlin, L., & Lundborg, G. (1999). Pathophysiology of nerve compression syndromes: response of peripheral nerves to loading. *The Journal of bone and joint surgery. American volume*, *81*(11), 1600–10. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10565653

Ridehalgh, C., Moore, A., & Hough, A. (2012). Repeatability of measuring sciatic nerve excursion during a modified passive straight leg raise test with ultrasound imaging. *Manual therapy*, 1–5. doi:10.1016/j.math.2012.06.002

Riemann, B. L., Demont, R. G., Ryu, K., & Lephart, S. M. (2001). The Effects of Sex, Joint Angle, and the Gastrocnemius Muscle on Passive Ankle Joint Complex Stiffness, *36*(4), 369–377.

Riener, R., & Edrich, T. (1999). Identification of passive elastic joint moments in the lower extremities. *Journal of biomechanics*, *32*(5), 539–44. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10327008

Ryan, E. D., Beck, T. W., Herda, T. J., Hull, H. R., Hartman, M. J., Costa, P. B., Defreitas, J. M., et al. (2008). The time course of musculotendinous stiffness responses following different durations of passive stretching. *The Journal of orthopaedic and sports physical therapy*, *38*(10), 632–9. doi:10.2519/jospt.2008.2843

Rydevik, B. L., Kwan, M. K., Myers, R. R., Brown, R. A., Triggs, K. J., Woo, S. L., & Garfin, S. R. (1990). An in vitro mechanical and histological study of acute stretching on rabbit tibial nerve. *Journal of orthopaedic research : official* *publication of the Orthopaedic Research Society*, *8*(5), 694–701. doi:10.1002/jor.1100080511

Sekir, U., Yildiz, Y., Hazneci, B., Ors, F., Saka, T., & Aydin, T. (2008). Reliability of a functional test battery evaluating functionality, proprioception, and strength in recreational athletes with functional ankle instability. *European journal of physical and rehabilitation medicine*, *44*(4), 407–15.

Shacklock, M. O. (1995). Neurodynamics. *Physiotherapy*, *81*(3), 9–16.

Shacklock, M. O. (2005). *Clinical Neurodynamics: A New System of Neuro-musculoskeletal Treatment*. Oxford, UK: Butterworth-Heinemann.

Singh, A., Kallakuri, S., Chen, C., & Cavanaugh, J. M. (2009). Structural and functional changes in nerve roots due to tension at various strains and strain rates: an in-vivo study. *Journal of neurotrauma*, *26*(4), 627–40. doi:10.1089/neu.2008.0621

Sladjana, U. Z., Ivan, J. D., & Bratislav, S. D. (2008). Microanatomical structure of the human sciatic nerve. *Surgical and radiologic anatomy : SRA*, *30*(8), 619–26. doi:10.1007/s00276-008-0386-6

Speich, J. E., Quintero, K., Dosier, C., Borgsmiller, L., Koo, H. P., & Ratz, P. H. (2006). A mechanical model for adjustable passive stiffness in rabbit detrusor. *Journal of applied physiology (Bethesda, Md. : 1985)*, *101*(4), 1189–98. doi:10.1152/japplphysiol.00396.2006

Stolov, W. C., & Weilepp, T. G. (1966). Passive length-tension relationship of intact muscle, epimysium, and tendon in normal and denervated gastrocnemius of the rat. *Archives of physical medicine and rehabilitation*, *47*(9), 612–20. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/5919253

Tabary, J. C., Tabary, C., Tardieu, C., Tardieu, G., & Goldspink, G. (1972). Physiological and structural changes in the cat's soleus muscle due to immobilization at different lengths by plaster casts. *The Journal of physiology*, *224*(1), 231–44. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1331536&tool=pmcentrez&rendertype=abstract

Tardieu, C., Tabary, J. C., Tabary, C., & Tardieu, G. (1982). Adaptation of connective tissue length to immobilization in the lengthened and shortened positions in cat soleus muscle. *Journal de physiologie*, *78*(2), 214–20. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7131334

Taylor, D. C., Dalton, J. D., Seaber, A. V, & Garrett, W. E. (1990). Viscoelastic properties of muscle-tendon units. The biomechanical effects of stretching. *The American journal of sports medicine*, *18*(3), 300–9. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/2372082

Tognella, F., Mainar, A., Vanhoutte, C., & Goubel, F. (1997). A mechanical device for studying mechanical properties of human muscles in vivo. *Journal of biomechanics*, *30*(10), 1077–80. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9391877

Topp, K. S., & Boyd, B. S. (2006). Structure and biomechanics of peripheral nerves: nerve responses to physical stresses and implications for physical therapist practice. *Physical therapy*, *86*(1), 92–109. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16386065

Tortora, G., & Derrickson, B. (2009). *Principles of anatomy and physiology*. USA: John Wiley & Sons.

Tucker, N., Reid, D., & McNair, P. (2007). Reliability and measurement error of active knee extension range of motion in a modified slump test position: a pilot study. *The Journal of manual & manipulative therapy*, *15*(4), E85–91. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2565640&tool=pmcentrez&rendertype=abstract

Turl, S. E., & George, K. P. (1998). Adverse neural tension: a factor in repetitive hamstring strain? *The Journal of orthopaedic and sports physical therapy*, *27*(1), 16–21. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9440036

Wakabayashi, K., Sugimoto, Y., Tanaka, H., Ueno, Y., Takezawa, Y., & Amemiya, Y. (1994). X-ray diffraction evidence for the extensibility of actin and myosin filaments during muscle contraction. *Biophysical journal*, *67*(6), 2422–35. doi:10.1016/S0006-3495(94)80729-5

Walsh, M. T. (2005). Upper limb neural tension testing and mobilization. Fact, fiction, and a practical approach. *Journal of hand therapy : official journal of the American Society of Hand Therapists*, *18*(2), 241–58. doi:10.1197/j.jht.2005.02.010

Weppler, C. H., & Magnusson, S. P. (2010). Increasing muscle extensibility: a matter of increasing length or modifying sensation? *Physical therapy*, *90*(3), 438–49. doi:10.2522/ptj.20090012

Whitehead, N. P., Gregory, J. E., Morgan, D. L., & Proske, U. (2001). Passive mechanical properties of the medial gastrocnemius muscle of the cat. *The Journal of physiology*, *536*(Pt 3), 893–903. Retrieved from http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2278914&tool=pmcentrez&rendertype=abstract

Wilgis, E. F., & Murphy, R. (1986). The significance of longitudinal excursion in peripheral nerves. *Hand clinics*, *2*(4), 761–6. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/3025228

Witvrouw, E., Danneels, L., Asselman, P., D'Have, T., & Cambier, D. (2003). Muscle flexibility as a risk factor for developing muscle injuries in male professional soccer players. A prospective study. *The American journal of sports medicine*, *31*(1), 41–6. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12531755

Wolff, A. V, Niday, A. K., Voelker, K. A., Call, J. A., Evans, N. P., Granata, K. P., & Grange, R. W. (2006). Passive mechanical properties of maturing extensor digitorum longus are not affected by lack of dystrophin. *Muscle & nerve*, *34*(3), 304–12. doi:10.1002/mus.20588

Worrell, T. W., Perrin, D. H., Gansneder, B. M., & Gieck, J. H. (1991). Comparison of isokinetic strength and flexibility measures between hamstring injured and noninjured athletes. *The Journal of orthopaedic and sports physical therapy*, *13*(3), 118–25. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/18796850

Wright, T. W., Glowczewskie, F., Cowin, D., & Wheeler, D. L. (2001). Ulnar nerve excursion and strain at the elbow and wrist associated with upper extremity motion. *The Journal of hand surgery*, *26*(4), 655–62. doi:10.1053/jhsu.2001.26140

Wright, T. W., Glowczewskie, F., Wheeler, D., Miller, G., & Cowin, D. (1996). Excursion and strain of the median nerve. *The Journal of bone and joint surgery. American volume*, *78*(12), 1897–903. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17415752

Yeung, E., Jones, M., & Hall, B. (1997). The response to the slump test in a group of female whiplash patients. *The Australian journal of physiotherapy*, *43*(4), 245–252. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11676694

Appendixes

Appendix I

This work was accepted for presentation in a poster session at the 60th Annual Meeting and 4th World Congress on Exercise is Medicine of the American College of Sports Medicine being held at the Indiana Convention Center in Indianapolis, Indiana, May 28 - June 1, 2013.

Andrade R, Freitas S, Vaz J, Pezarat-Correia P. “Effect Of Head, Trunk And Foot Position On Knee Passive Extension Torque-angle Response”, Medicine and Science in Sports and Exercise, Volume 45:5 Supplement (2013).

Abstract

Effect of head, trunk and foot position on knee passive extension torque-angle response

Ricardo Andrade, Sandro R. Freitas, João R. Vaz, Pedro Pezarat-Correia
Univ Tecn Lisboa, Fac Motricidade Humana, P-1100 Lisbon, Portugal

Appendix II

This work was presented in a moderated poster session at PRACTICE 2012 congress held at the Lusófona University, Lisbon, October 28 and 29, 2012.

--

EFEITOS MECÂNICOS DA POSIÇÃO DO PÉ, TRONCO E CABEÇA NA RELAÇÃO `MOMENTO-ÂNGULO´ DA EXTENSÃO PASSIVA DO JOELHO

Ricardo Andrade[1], Sandro R. Freitas[1], João R. Vaz[1], Pedro Pezarat-Correia[1]

[1] Univ Tecn Lisboa, Fac Motricidade Humana, P-1100 Lisboa, Portugal

Appendix III

Articulate device design specifically for this study with the aim to hold maximal active positions of cervical (flexion, neutral and extension) and thoracic (flexion and neutral) spine.