EFFECT OF TISSUE COMPRESSION ON THE HOFFMANN REFLEX:

COMPARISON BETWEEN THE ISCHIAL TUBEROSITY

AND POSTERIOR THIGH

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EFFECT OF TISSUE COMPRESSION ON THE HOFFMANN REFLEX: COMPARISON BETWEEN THE ISCHIAL TUBEROSITY AND POSTERIOR THIGH

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DISSERTATION ABSTRACT

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COMPARISON BETWEEN THE ISCHIAL TUBEROSITY

AND POSTERIOR THIGH

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This projected monitored nerve function during 9-minutes of compression at 210 mmHg and in the ensuing 10-minute recovery period. Compression was produced by a purpose-built plunger. A within-subjects design was used to compare the effects of tissue compression at two different locations: the middle posterior thigh, to replicate previous research, and the ischial tuberocities, to serve as control. Ten (5 men and 5 woman) apparently healthy adults (M = 22.44, SD = 3.13) were the participants in this study. Throughout testing, a 50% H_{max} stimulus was administered during the onset of each minute. Thermography and oxygenation measures showed that total limb blood flow and oxygenation, respectively, did not change from baseline values throughout testing. Four 2 (Location) x 5 (Time) repeated measures ANOVAs, with alpha set at the .05 level, were conducted to investigate the influence of location and time on the H-wave amplitude, H-

wave latency, two-point discrimination test, and subjective comfort. The times of interest were pre-compression, the ninth (i.e., final) minute of compression, and the first, fifth, and tenth minutes post-compression. A significant main effect for the time emerged for the H-wave amplitude indicating that the H-wave amplitude was significantly higher during the last minute of compression than at baseline and the first minute post-compression. Subjective comfort was significantly lower during the last minute of compression than during pre- and post-compression. This study demonstrated that tissue compression impacted the Hoffmann Reflex H-wave amplitude generated at the tibial nerve and recorded at the soleus. The findings suggest that compression in the absence of full limb ischemia may provide different effects on nerve function than a pneumatic cuff inflated around the circumference of the thigh. Future research should replicate these findings and identify the mechanisms responsible for producing the increase of H-wave amplitude responses, regardless of location, observed during compression.

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INTRODUCTION

The impetus for this study is the search for an objective measure to evaluate seating comfort. Comfort is an elusive quality. Subjectively, it is possible to identify sitting surfaces with relative levels of comfort and discomfort; however, subjective evaluations are prone to biases and require a large sample of various sized users sitting on candidate seat designs for extended periods of time. Recent advances in electronics have made possible thin flexible mats of capacitors that map the pressure distribution of the seated individual. Pressure distribution is a function of the material properties of both the seat and the occupant. Thus, important questions include what happens to the visco-elastic material of human soft tissue when compressed by sitting loads, and could an element of this be used as an objective measure of seat comfort?

Sitting involves a tissue load that compresses the areas of the buttocks and posterior thigh. The magnitude of compression is dependent upon many factors including an individual's anthropometry, the posture adopted during sitting, and the material properties of the sit surface. Skin, adipose tissue, muscle, fascia, and nerve tissues are all compressed during sitting. Compression reduces fluid flow through living tissue. The fluid flow through living tissue is influenced by blood pressure and to a lesser extent concentration gradients. Compression serves to limit the supply of nutrient laden fluid to the tissue being compressed, producing ischemia in the compressed tissue. During ischemic conditions, oxygenation decreases, serving to increase metabolite byproducts

that are harmful to living tissue. The impact compression has upon any single tissue is related to force magnitude and the duration of continuous application. Tissue compression intensities greater than blood pressure will produce ischemic conditions and over time can produce tissue necroses, with intensities greater than systolic pressure requiring less time than intensities greater than diastolic pressure. The impact of tissue compression upon skin and muscle has been well studied in relation to the development of pressure ulcers in immobile patients (Bouten, Oomens, Baaijens, & Bader, 2003; Gefen, Gefen, Linder-Ganz, & Margulies, 2005; Herrman, Knapp, Donofrio, & Salcido, 1999). However, to date the literature does not present significant information regarding the impact of tissue compression upon function of the mixed peripheral nerve (Kiernan, & Bostock, 2000; Lin, Kuwabara, Cappelen-Smith, & Burke, 2002; Zakutansky, Kitano, Wallace, & Koceja, 2005; Zhu, Starr, Haldeman, Chu, & Sugerman, 1998).

One possible method for assessing comfort objectively may be to utilize nerve function. Pressure on a peripheral nerve, of sufficient intensity, applied for enough time, leads to the temporary paresthesia (a limb falling asleep). Compression of peripheral nerves reduces the flow of nutrients near and along the axons, and is accompanied by reductions in oxygenation which diminish sodium potassium pump function, serving to alter membrane potential. Altered membrane potential is responsible for errant action potential propagation associated with the characteristic uncomfortable sensations of pins and needles.

The peripheral nerve that experiences compression during sitting is the sciatic nerve. The sciatic nerve arises from the sacral plexus, passes through the sciatic notch between the ischium and the trochanter, courses down the posterior thigh, and then

divides into the tibial and common peroneal nerves. To assess function, two testing methods are available: the first is the Hoffmann Reflex and the second is the two-point discrimination test.

The Hoffmann Reflex technique is a valid method for studying nervous system function outside the brain (Palmieri, Ingersoll, & Hoffman, 2004). The Hoffmann Reflex uses a short duration electric stimulus to depolarize axons of the mixed peripheral nerve near the skin to generate an action potential that causes an obligatory muscle twitch measurable by electromyography (EMG). Two distinct twitches are observable in the EMG. In terms of latency from time of stimulus presentation, the first twitch in the EMG signal is the muscle response or M-wave, associated with the smaller diameter efferent axons, while the second is the Hoffmann Reflex or H-wave, generated by the larger diameter afferent axons. The absolute latency of each twitch is determined by path length. In the case of the M-wave, latency is dependent upon the distance between the stimulation electrode and the EMG electrode because the M-wave travels directly along efferent motor axon to the monitored muscle. The H-wave appears later on the EMG output because it traverses a longer path. The H-wave travels from the stimulation site along the Ia afferent into the spinal cord where it directly synapses with the efferent alpha motor nerve (α–mn). The message then travels down the α–mn to the muscle and produces an obligatory muscle twitch, known as the later H-wave in the EMG trace.

The amplitude of each of these twitches is dependent upon the level of current used and the task performed by the participant during stimulus administration. Due to fiber diameter differences of the axons in the mixed nerve, the intensity of the current used for the electric stimulation produces different amplitudes of each muscle twitch.

Low levels of current depolarize only the larger afferent axons and produce a small amplitude H-wave. Increasing the stimulus intensity increases the amplitude of the H-wave and at some point results in direct stimulation of the motor axons leading to the presence of the M-wave in the EMG output. By using a low enough intensity stimulus the afferent sensory neurons are stimulated to generate an H-wave before the motor axons are activated. As stimulus intensity is increased, the H-wave increases to maximum amplitude (H_{max}), then decreases, and ultimately disappears from the EMG output. The reduction and disappearance of the H-wave is caused by the propagation of an antidromic action potential up the α–mn. The refractory period following the antidromic action potential cancels the orthodromic action potential, known as antidromic collision. While increasing stimulus intensity past H_{max} reduces and ultimately eliminates the H-wave altogether, the M-wave amplitude continues to increase until all α–mn are recruited. Once all α–mn are recruited, further increases of stimulus intensity do not impact M-wave amplitude.

The H-wave of the tibial nerve Hoffmann Reflex propagates through the area that experiences tissue compression during sitting. Alterations to the latency and amplitude of the H-wave would be indicative of the influence tissue compression has upon nerve function. The tibial nerve is accessible for electric stimulation in the popliteal fossa with EMG measurable in the soleus, while common peroneal nerve function may be observed by cutaneous sensitivity using the two-point discrimination test administered to the lateral shank. The two-point discrimination test is a clinical test used to identify the just noticeable distance between two points touched to the skin simultaneously (Lundborg & Rosen, 2004).

To date, studies that have evaluated the Hoffmann Reflex in the presence of tissue compression have used a full limb occlusion method (Zakutansky, et al., 2005; Zhu, et al., 1998). These studies used a pneumatic cuff inflated to a pressure greater than systolic blood pressure. Zhu et al. (1998) used the constant stimulus value of H-wave amplitude maximum (H_{max}) to stimulate the tibial nerve every 5 minutes during 20 minutes of occlusion. It was reported that within 15 minutes of full limb ischemia the latency of both the M- and H-wave was lengthened by approximately 2 ms, accompanied with a noticeable reduction of amplitudes. At the 20 minute data point the M-wave was still present, at reduced amplitude, while the H-wave was absent. The behavior of the M- and H-waves after the release of compression/ischemia was not reported.

Zakutansky et al. (2005) used a method that performed full recruitment curves during compression induced ischemia of the lower limb and at 5 minutes after release. Latency values were not reported in this study. At the end of the 5 minute compression induced full limb ischemia, the maximum amplitude of the M-wave was unchanged while the H_{max} was attenuated 18%. Even though H_{max} amplitude was reduced during ischemia, less stimulus intensity was required to generate both the M- and H-waves compared to baseline, indicating that action potential threshold decreased, making the membrane more susceptible to depolarization. After 5 minutes post occlusion the M-wave remained unchanged while the H-wave was still altered, but to a lesser extent than during ischemia, suggesting recovery back towards normal function.

In both of these studies, occlusion pressure was applied by a pneumatic cuff positioned around the circumference of the middle thigh. Pressure at this location would have also provided tissue compression to the sciatic nerve. It may be possible that the

observed findings were brought about by the compression of the pneumatic cuff instead of by the effects of ischemia. The location of the cuff would have compressed the portion of the sciatic nerve responsible for the transmission of the H-wave, while the M-wave section was only susceptible to ischemic conditions. In each study the H-wave showed greater change than did the M-wave, suggesting the influence was greater for compression than for ischemia.

Statement of the Purpose

The influence of externally applied tissue compression upon nerve function, in the absence of ischemia, is currently unknown. Therefore, the purpose of this study was to investigate the impact of tissue compression over the sciatic nerve in the posterior thigh. To help support the hypothesis that compression over the sciatic nerve produced nerve function changes, a control location was used. The control location was directly over the ischial tuberosity of the same leg used for the middle posterior thigh condition. Between the skin and the pelvis prominence of the ischial tuberosity there is minimal tissue so there was a limited chance of compressing the nerves that influence the soleus H-wave of the Hoffmann Reflex. The intensity of application was 210 mmHg, which was chosen because it was greater than systolic blood pressure for healthy people. The duration used was nine minutes. Pilot testing showed this intensity and duration to be safe.

Hypotheses

I. Significant interaction effects will emerge among the independent variables, location and time, for the three dependent variables, H-wave amplitude, H-wave latency, and the two-point discrimination task. Specifically,

   a. The H-wave amplitude associated with the middle posterior thigh location will be significantly attenuated during compression and gradually return to baseline amplitude over the 10 minute time period post-compression. In contrast the H-wave amplitude associated with the ischial tuberosity location will remain unchanged over time.

   b. The H-wave latency associated with the middle posterior thigh location will be significantly lengthened during compression and gradually return to baseline over the 10 minute time period post-compression. In contrast the ischial tuberosity location will remain unchanged over time.

   c. The two-point discrimination test associated with the middle posterior thigh location will be significantly reduced from baseline during compression and gradually return to baseline over the 10 minute time period post-compression. In contrast the ischial tuberosity location will remain unchanged over time.

II. Subjective comfort will be significantly lower than baseline during compression for both locations during the last minute of compression. Subjective comfort would then return to baseline over the10 minute post-compression period.

III. A significant relationship will be found during the last minute of compression between the distance, in centimeters, the plunger surface will deflected into the tissue at each location and H-wave amplitude value.

Limitations

1. The location of the sciatic nerve was not objectively determined. To locate the sciatic nerve within the posterior thigh, an imaging technique is required. The expense associated with imaging techniques with the fidelity necessary to identify the sciatic nerve was prohibitive.

2. It was assumed that tissue oxygenation under the plunger would change over time, but the rate of change and absolute saturation values were not measured.

3. Attentional focus can alter the amplitude of the Hoffmann Reflex response. Test sessions were conducted following a consistent protocol. However, attentional focus was not measured.

4. Stiffness, k, was not determined by application of multiple forces with deflection measured at each force. Rather, stiffness was inferred by deflection, total displacement of the plunger surface into the tissue. The force used was 210 mmHg. Displacement was determined as the difference between initial contact with the skin surface and final displacement into the tissue. A lower value of displacement was indicative of a stiffer material while a higher value of displacement was interpreted as less stiff.

Definition of Terms

Compression surface - the round shaped, flat surfaced, aluminum plunger used to produce tissue compression. The surface area of the compression area is 10.16 cm^2 (4.0 in^2).

Deflection - the crude approximation of stiffness. Deflection was determined as the difference between initial contact with the skin surface and final displacement into the tissue. A lower value of deflection was indicative of a stiffer material while a higher value of deflection was interpreted as less stiff.

$\%H_{max}$ amplitude - the unit used for the normalized H-wave amplitude.

H-wave - the electrically generated muscle twitch analogous to the spinal stretch reflex. The action potential travels up the afferent axon from the site of stimulation, synapses directly onto the alpha motor nerve to produce an obligatory muscle twitch. The H-wave is the second or late wave observable in the EMG trace and is associated with the low to moderate electric stimulus intensity. Latency from stimulus onset is determined by axon conduction velocity, synapses transmissibility, and length of travel. Amplitude is determined by stimulus intensity.

Ischial tuberosity - bony projection at the junction of the lower end of the body of the ischium and its ramus; this is a weight-bearing point in the sitting position; provides attachment for the sacrotuberous ligament and is the site of origin of the hamstring muscles.

Middle posterior thigh - the mid point between the medial and lateral edges of the posterior thigh at the location equidistance from the greater trochanter and the lateral epicondyle.

Motor neuron pool - the group of motor neurons originating in the spinal cord that innervate a muscle.

M-wave - the measurable muscle twitch produced by direct stimulation of the alpha motor nerve axons in the mixed peripheral nerve.

Normalize - divide an individual participant's data point(s) acquired during treatment by that individual's baseline value(s). This removes bias produced by individual differences and converts data to unitless values relative to baseline performance. Normalized values can be pooled for group analyses.

Stiffness - k = slope of the line of best fit over the elastic region of a load-deformation graph.

Tissue compression - is the result of external pressure applied to the surface of the skin.

50% H_{max} - one half the amplitude of the H-wave maximum amplitude.

LITERATURE REVIEW

The purpose of the following project was to use an established nerve conduction technique to evaluate the impact of externally applied tissue compression on nerve function in healthy participants. The following literature review discusses normal peripheral nerve function, the spinal stretch reflex, and the Hoffmann Reflex, including the variables of amplitude and latency. Following normal function, the impact of ischemia and compression upon the Hoffmann Reflex and other nerve conduction techniques are reviewed. Finally, mechanisms impacting nerve function during compression and ischemia are discussed. The chapter concludes with a brief summary of the literature.

Peripheral Nerve Function

First, although the specific details are beyond the scope of this review, it is important to briefly describe nerve function under normal conditions. Much of our understanding of normal nerve function comes from a series of studies using squids (Hodgkin & Huxley, 1952a, 1952b, 1952c, 1952d; Hodgkin, Huxley, & Katz, 1952). The axon diameter of these invertebrates exceeds 0.5 mm, allowing for the introduction of electrodes and easy measurement. By adjusting the external membrane environment by known quantities and observing internal membrane potential changes, numerous

functions were delineated; however, only the membrane potential and action potential will be detailed in the next section. The following two sections were summarized from Guyton and Hall (1996).

The Membrane Potential

The axon membrane is comprised of a lipid bi-layer, with membrane proteins throughout that transport materials across the membrane. The lipid bi-layer acts like an insulator separating two conducting media, the external medium of the axon and the internal medium, axoplasm. There are two types of ion channels in the membrane, gated and nongated. Gated channels only open and close in response to specific electrical, mechanical, or chemical signals. Nongated channels are always open and are not influenced significantly by extrinsic factors. Nongated channels are primarily important in maintaining the resting membrane potential. Since ion channels recognize and select among specific ions, the actual distribution of ionic species across the membrane depends on the particular distribution of ion channels in the cell membrane.

Selective permeability of the membrane develop ion gradients with intracellular concentration high in K+ compared to the extra cellular space which is high in Na+ and Cl-. The potential across the membrane when the cell is at rest is termed resting potential. By convention, the potential outside the cell is arbitrarily defined as zero, and given the relative excess of negative charges inside the membrane; the potential difference across the membrane is expressed as a negative value typically between -60 to -70 mV. The charge separation across the membrane, and therefore the resting membrane potential, is disturbed whenever there is a net flux of ions in or out of the cell. A reduction of the

charge separation is called depolarization; an increase in charge separation is called hyperpolarization. For the membrane to depolarize, during the action potential, either cations (positive) enter the cell or anions (negative) leave the cell. For hyperpolarization, cations leave the cell and anions enter. Typical ion concentrations (millimols / liter) maintained in human motor neurons for the rest potential are intracellularly: Na+ 15; K+ 150; Cl- 10; nonpermeable anions 65; and extracellularly: Na+ 150; K+ 5; Cl- 110; nonpermeable anions 0.2. Two equations describe membrane ion concentration relationships; they are the Nernst Equation and the Goldman-Hodgkin-Katz Constant Field equation.

Two forces act on ions, the concentration gradient and the electrostatic force. The driving force of the chemical concentration gradient tends to move ions down the gradient; while the electrostatic force tends to move ions in a direction determined by its particular charge associated with the charge separation across the membrane. Thus, for instance, K+ ions concentrated inside the cell tend to move extracellularly down its concentration gradient through nongated K+ channels. However the relative excess of positive charge outside the membrane pushes it back into the cell. Eventually, equilibrium can be reached so that the actual ratio of intracellular and extracellular concentration is reduced to 1 or concentration establishes full equilibrium, eliminating the concentration gradient. Therefore, to prevent this from occurring active transport of ions is necessitated.

Dissipation of ion concentration gradients is ultimately prevented by Na-K pumps. These pumps remove Na+ from the cell while moving in K+, at the ratio of 3 Na+ pumped out for every 2 K into the cell. Because the pump moves Na+ and K+ against

their net electrochemical gradients, energy is required. The energy necessary for this process is obtained from the hydrolysis of ATP, produced in the presence of oxygen; thus ischemia/edema must have some form of negatively impact nerve function. Upon depletion of stored ATP nerve function is negatively impacted due to sustained energy requirements leading to anaerobic processes. This produces an increase in damaging catabolites. Limited or no oxygen ultimately causes the membrane potential to dissipation below functional levels. Oxygen depletion can impact the entire axon or occur at a specific location along the axon. Regardless of the cause of ischemia, ischemia inhibits proper propagation of the action potential.

The Action Potential

The propagation of a nerve impulse along an axon begins when the synapses receive neurotransmitters from nearby nerve endings. An increase in internal potential sets off a chain of events which is repeated for each Node of Ranvier as the nerve impulse travels along the axon. This process is referred to as "saltatory" conduction, (or leaping or dancing conduction). Voltage gated Na+ channels open when the membrane potential increases about 20mV above the rest potential at which point threshold is reached. Na+ ions rush in for about 1 ms and positive feedback keeps the channels open until the cell interior becomes positively charged. A membrane potential of + 30mV closes Na+ channels, and until the membrane potential returns to below threshold, the neuron cannot react to further stimulus, known as the refractory period.

Refractory allows for the unidirectional propagation of action potentials. Sodium ions exiting the axon cause the local interstitial fluid to become positive. The increased

positive ion concentration inside the node increases the membrane potential at both neighboring nodes only. Therefore, the downstream node reaches threshold and the process continues there while the upstream node has an elevated threshold and can not fire again. In this way, the nerve impulse propagates down the axon, maintaining its intensity until it causes the release of neurotransmitters at the nerve endings. To this accord, nerve messages are transmitted by the frequency of action potentials.

The strength of the action potential is an intrinsic property of the nerve cell axon and is always of the same intensity regardless of stimulus. Conduction velocity depends on the fiber diameter and degree of myelination. Larger axons transmit faster than small diameter axons mostly due to the membrane to cytosol ratio. The closer to a 1:1 ratio, as in larger axons, the faster conductance. Myelination speeds conduction, myelinated axons are faster than unmyelinated axons. Table 1 provides a list of fiber type, fiber diameter, myelination level, conduction velocity, and function for each axon of the peripheral nervous system.

Table 1. Characteristics of the nerves of the peripheral nervous system.

Fiber Type	Diameter (micrometers)	Degree of Myelination	Conduction Velocity (m/sec)	Function
IA	12-22	Greatest	70-120	Somatic motor,
αMN				Muscle-spindle afferent
IB	5-13		30-70	Touch, Kinesthesia, Pressure,
				Tendon tension
II	5-15		30-80	Sensory, Encapsulated end
				organs, Muscle Spindles
Gama	3-8		15-40	Motor to Muscle Spindle,
				Fusimotor Drive
III	1-5		12-30	Pain, Temperature
B	1-3		3.0-5.0	Autonomic
C	0.2-1.2		0.2-2.0	Pain, Reflexes
C	0.3-1.3	Unmyelinated	0.7-2.3	Postganglionic sympathetic

Nerves of the Peripheral Nervous System

Stuart (2000) and Schmidt (2003) described peripheral nerves a mix of myelinated and unmyelinated axons. The mixed nerve is comprised of fascicles of individual nerves embedded in the epineurium, which consists of collagenous connective tissue, scattered fat cells, and blood vessels. Each fascicle is deliminted from the epineurium by the perinerium. The perineurium consists of several laminae of flattened perineurial cells with numbers of laminae decreasing proximally in parallel with

diminishing fascicular size. Perineurial cells, the innermost layers of which are joined together by tight junctions, restrict entry of foreign materials into the endoneurium. Several septae of perineurial cells may subdivide individual fascicles. Other cellular elements within the endoneurium are small capillaries and venules, scattered fibroblasts, occasional macrophages and monocytes, and scattered mast cells.

The Sciatic Nerve

Bundled as part of the Sciatic Nerve, along with the common peroneal, the tibial nerve originates at L4, L5, and Sacral 1, 2, and 3. As detailed by Gray (1918), the Sciatic supplies nearly the whole of the skin of the leg, the muscles of the back of the thigh, and those of the leg and foot. It is the largest nerve in the body, measuring 2 cm. in breadth, and is the continuation of the flattened band of the sacral plexus. It passes out of the pelvis through the greater sciatic foramen, below the piriformis muscle. It descends between the greater trochanter of the femur and the tuberosity of the ischium, and along the back of the thigh to about its lower third, where it divides into two large branches, the tibial and common peroneal nerves. This division may take place at any point between the sacral plexus and the lower third of the thigh.

The Spinal Stretch Reflex of the Soleus

The spinal stretch reflex is the only monosynaptic reflex in the human body. As summarized by Blackburn (2004) this mechanism has been observed to occur consistently between 30 and 95 ms in the soleus muscles. The spinal stretch reflex is comprised from an interaction between skeletal muscle and the central nervous system.

Within the muscle, the muscle spindle is the sensory receptor responsible for initiating the reflex loop. The muscle spindle is sensitive to length, and change in length, and rate of change of length, of the muscle. When muscle length is increased rapidly, a stretch is registered by the muscle spindle. Activation of the spindle, in turn, initiates an action potential up the Ia afferent neuron. Within the spinal cord the Ia afferent forms a monosynaptic connection with an α-motorneuron (α-mn) innervating the same muscle. After synapsing with the α-mn, an action potential then travels to the homonymous muscle, causing the associated fibers to contract in opposition of the increased change in muscle length.

The time course of the soleus spinal stretch reflex has been outlined by Eldred (1967). Approximately 1 ms is necessary for the mechanical signal (change in length) to be transmitted to the muscle spindle, and for the muscle spindle to generate propagation of an action potential up the Ia afferent. The following example is the approximate conduction velocity and time for an individual with a distance of 1 meter between the soleus and the spinal cord. At a maximal nerve conduction velocity of 70-120 m/s (See Table 1), approximately 10 ms is required for the afferent impulse to travel the 1 meter distance from soleus to the spinal cord. A small amount of time is required for transmission of central delay as the signal crosses the single synapse of the Ia afferent and the homonymous α-mn. The efferent signal travels down the motor unit which is comprised of the α-mn and associated muscle fibers. Transmission time includes the 10 ms for the α-mn length, transmission of the message to the muscle fibers across the Musculoskeletal junction, and the time necessary for the signal to propagate along the muscle fibers. The peripheral nervous system of the spinal stretch reflex is rather straight

forward. Activation of the spindle generates an obligatory reaction, action potentials travel up the Ia afferent, form a synapse directly with the motor unit comprised of the α-mn and associated muscle fibers, causing a twitch. However, with in the spinal cord of the central nervous system a complex set of activities is present.

The spinal stretch reflex has been found to interact with a number of central mechanisms of the spinal cord. The effects include reciprocal inhibition, synergistic innervation, recurrent inhibition, and descending command all of which have implications for the spinal stretch reflex. In the case of reciprocal inhibition, when the Ia afferent transmits to the α-mn it also stimulates Ia inhibitory interneurons associated with the antagonistic musculature. The Ia inhibitory interneuron subsequently inhibits activation of the antagonist α-mn pool within the spinal cord, limiting antagonist activity. Stimulation of the Ia afferent also results in activation of agonist synergistic muscles. This connection, labeled by Ciullo and Zarins (1983) as synergistic innervation, allows muscles which perform similar actions as the agonist to provide additional force output. Interneurons of the spinal cord provide a complex mechanism known as recurrent inhibition by which excessive agonist activation is prevented, which acts to protect the muscle and tendon unit from overload. In recurrent inhibition the Renshaw cell provides a negative feedback loop with the agonist α-mn pool by controling its firing rate, and regulating the strength of reciprocal inhibition. Activation of the agonist α-mn pool stimulates Renshaw cells; Renshaw cells synapse back onto the α-mn pool which inhibits further activation of α-mn pool. Therefore, agonist activity itself is responsible for reducing agonist activity by increased stimulus to the Renshaw cell. In addition, the Renshaw cell inhibits the Ia inhibitory interneuron of the antagonist, thus serving to turn

off inhibition which has been termed disinhibition. Disinhibition is the inhibition of an inhibitory interneuron which permits the activity of the neuron that was inhibited to be active. Activity of the spinal stretch reflex is also controlled by descending command of the CNS. The complexity of the interaction between descending commands from CNS and the lower level activity of the spinal cord, such as the spinal stretch reflex, is beyond the scope of this investigation.

Hoffmann-reflex

Originally described by Paul Hoffmann in 1910 (as cited in Palmieri, Ingersoll, & Hoffman, 2004), and later given his name, the Hoffmann Reflex (H-reflex) is an electrically induced reflex analogous to the mechanically induced spinal stretch reflex described above. The primary difference between the H-reflex and the spinal stretch reflex is that the H-reflex is induced by electrical stimulation, bypassing the muscle spindle. For the soleus muscle, electrical stimulation is delivered to the tibial nerve transcutaneously in the popliteal space. Electromyography (EMG) is used to measure the compound action potential generated by the electrical stimulus in the muscle of interest. There are three main variables associated with the H-reflex, namely 1) Intensity of the electric stimulus used—most often normalized for each individual, 2) Amplitude of the compound action potential as recorded by EMG expressed in millivolts (mV), and 3) Latency of the compound action potential as recorded by EMG expressed in milliseconds (ms).

Normal Function

The H-reflex can be elicited in any muscle, as long as the peripheral nerve can be conveniently stimulated (Buschbacher, 1999; Palmieri et al., 2004). Although reported for many muscles, difficulty exists in eliciting H-reflexes in many of the muscles due to accessibility. The most widely reported muscles used for H-reflex studies include soleus, quadriceps, and flexor carpi radiali.

The H-reflex measures the efficacy of transmission of an electronic pulse stimulus traveling up afferent fibers, through dorsal root ganglia into the spinal α-mn pool of the corresponding muscle, out the ventral root, and down the efferent fibers to the muscle, where this volley is measured by EMG. The afferent portion of the H-reflex begins at the point of electric stimulation and results in action potentials traveling along afferent fibers until they monosynaptically meet the α-mn in the spinal pool. The efferent portion of the H-reflex pathway includes action potential propagation down the α-mn to the Musculoskeletal junction, which produces an obligatory contraction of the associated muscle fibers and can be recorded by EMG. On the EMG output the H-reflex is known as the H-wave. The H-reflex is a compound action potential, or a group of almost simultaneous action potentials, from many muscle fibers in the region near the EMG electrode.

In addition to the pathway of the H-wave, electric stimulation of the peripheral nerve causes direct activation of the efferent fibers sending an action potential directly to the muscle from the point of stimulation. This volley of activity also causes a muscle contraction, but because it does not pass through the spinal cord, it is not referred to as a reflex. The efferent arc produces a measurable response in EMG known as the muscle

response or M-wave. On the EMG output the M-wave always precedes the H-wave. The amplitudes and latency of the H- and M- waves are interdependent upon stimulus intensity and axon fiber diameter. The appearance of the M- and H-waves in the EMG trace is determined by the distribution of Ia and α-mns axons and the order of neural fiber recruitment (Palmieri et al., 2004). Recruitment order is determined by fiber diameter, with larger diameter fibers having less resistance to stimulation than smaller fibers; thus larger fibers are stimulated at lower intensities. As detailed in Table 1, Ia afferent fibers have larger diameters than the efferent α-mns, and are, therefore, stimulated at lower stimulus intensities.

Amplitude

Increasing the stimulus intensity increases the amplitude of the H-wave and at some point results in direct stimulation of the motor axons leading to the presence of the M-wave in the EMG output. It is highly possible to stimulate the Ia sensory neurons before the motor axons are activated by using a low intensity stimulus. This produces a low amplitude response of the H-reflex. To obtain maximum H-reflex (H_{max}) some motor axon activation is always present. The presences of the M-wave during H_{max} is due to Ia afferents and α-mns of similar fiber diameter. The maximum of the M-wave is generated by a stimulus that recruits all of motor axons in the nerve. The M-wave appears before the H-reflex on the EMG because it has a shorter path. The threshold of the motor axons is higher than that for the Ia afferent neurons due to the generally smaller axon diameter of the α-mns. It is not possible to illicit a maximum H-reflex amplitude void of all M-wave response. In contrast, when the maximum M-wave response (M_{max}) is generated the

H-reflex is completely suppressed due to antidromic collision (see below). Due to the large diameter of some of the motor axons as compared to all of the Ia afferent axons, M_{max} always has a larger amplitude than H_{max}.

Latency

The H-wave latency depends on afferent conduction velocity, central delay, efferent conduction velocity, transmission of signal across the Musculoskeletal junction, propagation through the muscle fibers, and absolute length of this pathway. Prolongation of latency may result from dysfunction in any portion of this loop, by either peripheral and/or central mechanisms. M-wave latency is dependent upon efferent conduction velocity, transmission of signal across the Musculoskeletal junction, propagation through the muscle fibers, and absolute length of this pathway. Latency measurements can be used to assess the response of the nervous system to various neurologic conditions, musculoskeletal injuries, application of therapeutic modalities, pain, exercise training, and performance of motor tasks.

The time of onset for the H-wave always follows onset time of the M-wave in the EMG signal. This relative latency is caused by the distance the action potentials must travel. For the H-wave the action potential travels up (away from the muscle) the afferent axons, synapses in the spinal cord and then propagates back down the efferent axon to the muscle whereas for the M-wave the action potential travels from the site of stimulation to the muscle. The distance each must travel accounts for the observed relative latency.

Limb length is the primary factor influencing overall latency of both the H-wave and the M-wave, with longer limbs associated with longer latencies. Similarly, the closer the muscle under investigation is to the spinal cord, the shorter the latency.

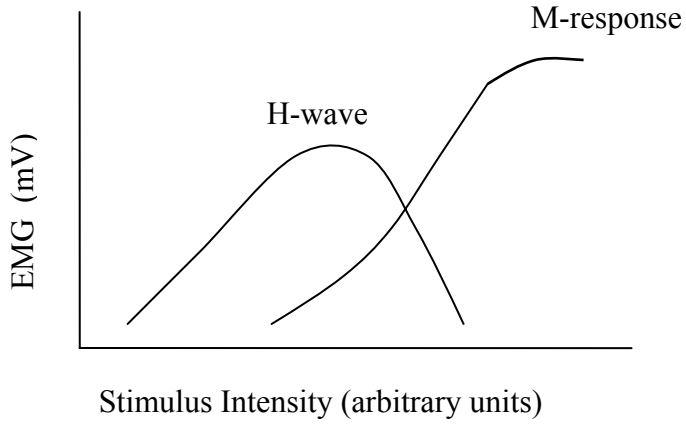

Figure 1. The H-wave and M-wave curves.

Recruitment Curve

Figure 1 illustrates typical recruitment curves where the amplitude of the H-wave and M-wave appears on the y-axis, and the stimulus intensity required to elicit these responses depicted on the x-axis. The H-wave tracing begins to appear on the EMG at low levels of stimulation. When the stimulus is low enough, neither the H-wave nor the M-wave is generated, because the firing threshold for both types of fibers is not reached. Increasing stimulus intensity recruits the Ia afferent fibers first, producing only a H-wave in the EMG trace. As the stimulus intensity increases, the depolarization threshold for the

motor axons is reached, causing an M-wave to appear in the EMG simultaneously with the H-reflex. Continuing to increase the stimulus intensity eventually results in the H-wave reaching its maximum, then decreasing and finally disappearing from the EMG. In contrast the M-wave achieves a maximum plateau; with further increases in stimulus intensity producing no further effect. The H-reflex peak determines the maximum H-wave amplitude (H_{max}). Just prior to the stimulus required to ellicit H_{max} the M-wave tracing began to appear with a low amplitude. Further increasing the stimulus intensity beyond that needed to elicit the H_{max} resulted in the H-wave amplitude decreasing and the M-wave amplitude increasing. This pattern (H-wave decrease and M-wave increase) continues until the H-wave disappears altogether and terminates with the M-wave reaching a plateau, termed M-wave maximum (M_{max}).

The M-wave typically displays an S shape due to the distribution of efferent axon diameters. At low stimulus intensities sufficient to stimulate efferent fibers, the largest of the α-mns are recruited, resulting in the initial appearance of the M-wave. However, the numbers of large fibers are relatively small, thus the amplitude of the M-response is small, and the rate of response increase in relation to increases in stimulus intensity is small. With increasing stimulus intensity, progressively smaller fibers with higher activation thresholds are stimulated, resulting in a large, rapid increase in M-response amplitude, due to the fact that these fibers are relatively more numerous. As stimulus intensity approaches M_{max} levels, the smallest fibers with the greatest stimulus threshold are stimulated. These levels of stimulation result in a gradual M-wave amplitude increase to maximum, due to the fact that smaller fibers are less numerous.

Standardizing Values

The ratio between the H_{max} and M_{max} amplitudes, or $H_{max}\backslash M_{max}$ ratio, can be used to standardize values among individual participants. To compare treatment changes differentially affecting the H-wave or M-wave the ratio of amplitudes is used as a single score. To ensure that a normalized stimulus intensity is administered across individuals, a percent of stimulus necessary to elicit either H_{max} or M_{max} is used. For example half the stimulus required to generate M_{max} is depicted as 50% M_{max}. Use of a normalized stimulus permits individuals to be grouped and comparison to be made between groups. Additionally, findings are often presented as a ratio between the H- and M- waves induced by a standardized stimulus intensity. The H/M ratio shows the relative impact treatment effect by central processing and by peripheral changes. Specifically, when the H-wave shows change and the M-wave stays constant, central processing changes at the spinal cord level is often present.

Orthodromic and Antidromic Propagation

Electronic stimulation causes action potential propagation in both directions along the axon from the application point. This is true for both afferent and efferent axons. When the propagation travels normally, it is termed orthodromic. In the case of the propagation in the direction opposite to which it normally travels, afferent to the muscle spindle and efferent to the spinal cord, it is denoted as antidromic. Orthodromic propagation is responsible for producing the H- and M-waves. Antidromic propagation has minimal impact on the muscle spindle of the afferents. However, antidromic

propagation is responsible for reducing and ultimately eliminating the H-wave during increases in stimulus intensity due to antidromic collision.

Antidromic Collision

The reason for the disappearance of the H-wave is an effect known as antidromic collision. As the antidromic volley travels the wrong direction, up the motor axon toward the spinal cord, it collides with the reflexive orthodromic volley, which has proceeded up the efferent and has passed through the spinal cord along the typical monosynaptic pathway. When the two action potentials, orthodromic and anitdromic meet along the same axon they effectively cancel out one another. As an action potential propagates along the axon an increase in membrane depolarization results which causes a transient refractory period along the membrane. This increased depolarization of the membrane ensures one way transmission of the action potential along the length of the axon. This is achieved by rendering the area just behind the leading edge of the action potential unresponsive to further stimulation. Therefore, when two action potentials meet, the non-responsive region following each, eliminates further propagation in either direction, serving to terminate both.

Antidromic collision cancels the action potential along individual axons. The different diameters of Ia afferents and α-mn is responsible for the H-wave reduction observed in recruitment curves (see Figure 1). Antidromic collision occurs when the Ia afferent and its monosynaptically linked α-mn efferent are both stimulated by the same stimulus. The two action potentials meet and cancel each other out, while all the Ia volleys associated with motor axons that have not reached threshold, are not effected.

Antidromic collision is influenced by the stimulus intensity. When intensity is low, as when only the H-wave is present, little to no antidromic collision is present. As stimulus intensity increases and the number of efferent axons stimulated beyond threshold also increases, leading to increased antidromic collisions and a reduction in observable H-wave. At a high enough stimulus intensity, antridromic volley is present in all α-mn associated with all Ia afferents and the H-wave is completely suppressed. Finally, antidromic collision does not affect the M-wave, leading to the plateau M_{max} in the presence of sufficient stimulus intensity.

Indices of System Function

Measurable differences in the H-reflex are caused by the parameters that influence afferent and efferent recruitment (Hilgevoord, Bour, Koelman, & Ongerboer de Visser, 1996). Alertness, anxiety, medication, turning of the head, eye closure, duration and intensity of the stimulus also have dramatic impact on results (Oh, 1993). Even stimuli such as loud and unexpected sound may affect the response (Enoka, 1994). Other key factors that impact the H-reflex during the present study include reciprocal, presynaptic, and recurrent inhibitions, as well as vestibular influences (Hoffman & Koceja, 1995). In the case of the soleus, impact of knee angle, posture, and baseline EMG activity are important factors that affect amplitude of the H-and M-waves (Alrowayeh, Sabbahi, & Etnyre, 2005). Therefore, consistency during data collection must be maintained for all study participants to ensure data quality. Additionally, muscle level activity must remain constant as well for all conditions.

Diagnostic Applications

Numerous researchers have tried to use the H-reflex protocol to identify pathological conditions; unfortunately, limited success has been documented in the literature. Similarly, there exists no clinical application of this method for diagnosing problems or disease conditions. However, the influence compression has on the H-reflex has been investigated.

Compression Influence on Limb

Force-deflection

The property of a visco-elastic material, such as human tissue, can be characterized by a force-deflection curve. A force-deflection curve plots the deflection of a material against the force used to produce that deflection. Forces of differing magnitude are used, with plunger size, application rate, hold time, and maximum deflection varying based on test procedure. The line of best fit over the elastic region of the curve for the force-deflection graph is termed, the stiffness constant (k).

The visco-elastic property of human tissue produces a *k* value that is non-linear. When placing an external force upon the body, the skin and underlying tissue deflect. Anatomical location and an individual's tissue distribution influences the amount and rate of deflection. Additionally, deflection is a function of force application time. Linder-Ganz and Gefen (2004) used finite element modeling to explore the distribution of force, stress, and strain of sitting related externally applied tissue compression. The finite element formulas used included coefficients for skin, fat, muscle, and bone density and elastic modulus. The densities used were 1.056 g/cm^2 for skin, 1.200 for fat, 1.056 for

muscle, and 1.900 for bone. The elastic modulus used were 695 kPa for skin, 80 kPa for fat, 75 kPa for muscle, and 18 GPa for all bone structures. These values indicate that fat was considered to be denser than muscle and skin and that bone, fat and muscle were more deformable than skin.

Anatomical Location and Individual's Tissue Distribution

The relative composition of skin, adipose, muscle, and other tissues determines an individual's force-deflection value. The absolute deflection for each individual depends upon the depth of tissue and the primary composition of that material. The ischial tuberosity is composed primarily of skin, adipose, and connective tissues. The middle thigh location is composed primarily of adipose and muscle tissue with the femur located deep in the tissue.

Application Time of Force

Human tissue is primarily comprised of fluid held in place by a semi-rigid semi-closed cell lattice structure. Initially, force placed upon this type of material produces deformation of the lattice structure and generates increased pressure within each semi-closed cell. The pressure gradient created, while maintaining a constant force, causes the fluid to diffuse from areas of higher to lower pressure. The rate of movement is dependent upon the application force, with higher forces generating more rapid movement rates than lower force. Indicating that measurement of force and displacement must be time matched until the equilibrium is re-established. Also this indicates that the

displacement of the force applicator must increase at the rate of diffusion to maintain a constant force upon the tissue.

Vascular Tissue

In the case of tissue pressures, interstitial fluid pressure is in the range of -2.6 to -7.1 mmHg which is balanced by a positive solid tissue pressure, resulting in a total tissue pressure of 0 mmHg (Schell & Wolcott, 1966). The term "tissue load" refers to the distribution of pressure, friction, and shear on the tissue (Bergstrom, Bennett, Carlson, et al., 1994). It is widely documented that high concentrated pressures applied to human tissue, particularly over bony prominences, can cause loss of blood and nutrient flow and over time lead to ischemia.

Landis (as cited in Williams, Wasserman, Rawlinson, Kitney, Smaje, & Tooke, 1988) used a microinjection method to cannulate the arteriolar limb of capillaries in human fingernail beds to study capillary blood pressure. He reported an average pressure of 32 mm Hg in the arteriolar limb, 22 mm Hg in the midcapillary bed, and 12 mm Hg on the venous side. Williams, Wasserman, Rawlinson, Kitney, Smaje, and Tooke (1988) reported slightly different values of 37+/- 3 mmHg in the arteriolar limb when testing capillary blood pressure. Fronek and Zweifach (1975) recorded pressures in all types of vessels including capillaries of the deep hamstring muscle in cats. These authors report that systemic blood pressure was maintained in arterial vessels down to a diameter of approximately 100 micrometers. In the beginning of the capillaries, it was 40 mmHg and at the end of the capillaries about 20 mmHg. In a vein of 100 micrometers, pressure was slightly above 11 mmHg. These values indicate that pressures as low as 11 mmHg impact

the proper draining of tissue by the veins, and that when pressures increase to over 20 mmHg, blood flow to the tissue is restricted. Continued increases in pressure stops the flow in capillaries and precapillary vessels resulting in a standstill in the entire vascular network. With time this condition leads to ischemia in the associated tissue.

Tissue Compression Over Time

Both surface and deep vascular tissues are impacted by externally applied tissue loads with the degree of impact related to length of application periods. In other words, an inverse relationship exists between acceptable pressure and application time. In daily life the static posture of sitting is when people experience a tissue load sufficient to occlude blood flow from vascularized tissue. During sitting the skin, adipose, muscle, and nerves are subjected to internal and external tissue load. Skin, particularly that over the bony structures of sacrum and ischial tuberocities, is subjected to the greatest tissue load. Muscles, including the gluteus, piriformis, and hamstrings, experience a load. The nerves that innervate the entire lower extremity traverse through the posterior thigh and are subjected to the effects of pressure as well.

Skin Compression

Pressure and pressure changes are detected by four types of mechanoreceptors namely, Meissner, Pacinian, Merkle, and Ruffini. Two quickly adapt to pressure, Meissner and Pacinian, making them sensitive to changes in intensity (and vibration), while Merkle and Ruffini adapt slowly, producing sensitivity to sustained pressure. When electrically stimulating the afferent axons of these mechanoreceptor, only the Merkle

produces the perception of pressure (Macefield, 2005). Additionally, unmyelinated polymodal nociceptors responsive to mechanical, chemical, and thermal stimuli are present (Besson, 1999). Therefore pain is detected when nociceptors are activated from excessive pressure, sufficient tissue deformation, tissue damage, or extremes in temperature. More importantly with repeated exposure to a stimulus, nociceptors exhibit a reduced threshold for activation or an increase in the magnitude of the response. Spontaneous activity has also been recorded during repeated exposure to a noxious stimulus such as pressure. Besson (1999) reports that increased activity and the activation of sleeping category of nociceptors is caused by a messenger system activated by the release of several inflammatory mediators (bradykinin, prostaglandins, serotonin, and histamine). Another fascinating aspect of nociceptor activity is that over time the central nervous system becomes hypersensitive to pain, such that the same level of activity becomes more painful with time.

Pressure on skin, only if high enough and applied for long enough duration, can lead to cutaneous ulceration. Extensive research has been conducted on the formation of cutaneous pressure ulcers because of their prevalence in clinical patients (Bergstrom, et al., 1994). Early studies used pressure levels significantly higher than those experienced during sitting, in excess of 550 mmHg, while more recent research has investigated the intensity and duration needed to cause ulcer formation. Kosiak (1959) conducted a series of experiments exposing fully intact dog tissue to pressure ranges of 60 to 550 mmHg for periods of 1 to 12 hours. For the lowest pressure and shortest duration Kosiak found, histological lesions, were present and included inflammatory cell infiltration, extravasation, and hyaline degeneration. Tissues subjected to higher pressures for longer

periods of time also showed significant lesions, including muscular degeneration and venous thrombosis. More rigorously controlled trials of pressure application on animals have shown that 2 rather than 1 hour of application is needed for ischemia to cause damage at pressures between 55 and 61 mmHg. Histology studies show significant cell damage only begins to occur when applied for over 2 hours (i.e., Herrman, Knapp, Donofrio, & Salcido, 1999).

Muscle Compression

Tension in muscle is registered by both the muscle spindle and the golgi tendon organelle. Externally applied pressure, if sufficient, increases muscle tension. The spindle is sensitive to changes in tension and absolute tension and the golgi tendon organelle registers excessive absolute tension. Tension and pressure can both reduce the flow of nutrients to muscle and increases the accumulation of metabolites leading to damage that activate nociceptors.

Recent research of cutaneous pressure sores etiology has identified the existence of deep pressure sore, which develop in skeletal muscles from damage caused by external tissue load and by internal loads (Bouten, Oomens, Baaijens, & Bader, 2003; Gefen, Gefen, Linder-Ganz, & Margulies, 2005; Linder-Ganz & Gefen, 2004). Deep pressure sores are most often recognized as occurring in tissue compressed under bony prominences resulting in local occlusion of blood supply for a critical time period. For the body to remain in static posture when external pressure is applied, an internal force must be generated (see Figure 2). At the interaction point of these two opposing forces, compression of vascularized tissue occurs. In other words, two tissue loads are present,

one externally and the second internally concentrated at the rigid structures of the body, with the tissue (skin, adipose, and muscle) between these two solid surfaces experiencing compression.

Animal models and clinical evidence in which external compression of soft tissue layers composed of skin, subcutaneous fat, and muscle have shown that cell death occurs in muscle tissue first; additionally, increases in muscle stiffness have been found (Gefen, et al., 2005; Linder-Ganz & Gefen, 2004). The external pressures used in animal models showed that a minimal value of 262 mmHg applied for more than 2 hours is necessary to produce muscle cell damage and in vivo stiffening, while an 86 mmHg application for up to 8 hours has had no effect.

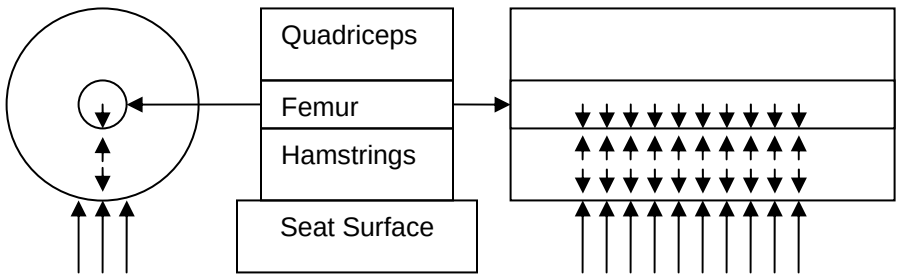

Figure 2. Stylized distribution of internal tissue load experienced during sitting. The seat surface exerts a force up into the hamstrings and the femur produces a force down into the hamstrings. The tissue in-between the two solid surfaces of the femur and seat surface experience deformation due to compression.

Internal load may be produced by both rigid structure (bone) pressing into muscle and by inflammation or swelling of tissue. For internal tissue load on muscle, produced by the rigid structures of the human body during seated posture, it is currently not possible to ascertain specific values. However, given the surface area of the ischial tuberocities, sacrum, and femur relative to the surface area of the buttocks and thigh, it is reasonable to assume that muscles near these structures are subjected to a higher load than the load on the skin (see Figure 2). The extent of the damage to muscle produced during seat posture is a current topic of research. Verver, van Hoof, Oomens, Wismans, & Baaijens (2004), Gefen, Gefen, Linder-Ganz, and Margulies, (2005), and Linder-Ganz and Gefen (2004) have developed computer models which can demonstrate and estimate impact of internal load on compressible tissues. Validations of these models with animals have shown cell death and muscle stiffness to occur. This internal load mechanism for damage may also compress nerves and account for numbness and burning expressed by individuals who remain in a static posture for an extended time.

Nerve Compression

The sciatic and other nerves of the lower extremity are located in the sit area and are subjected to the effects of tissue compression during sitting. This compression may cause errant function or damage anywhere along the nerve. These nerves originate from lumbosacral plexus, pass through the buttock—with most positioned through the sciatic notch between the ischial tuberosity and the greater trochanter, and continue distally deep in the posterior thigh. Tissue load may impact these nerves. The deep location of these nerves may protect them from external loads but may subject them to loads from internal

sources. Other factors may also contribute to nerve compression during static seated posture, such as, but not exclusively, anthropometry, workload, and posture.

Tissue compression can impact nerve conduction in two ways. Compression can directly compress the nerve and produce localized ischemia around the nerve, both of which are undesirable. To date the method typically used to assess compression impact on nerve function is to use a pneumatic cuff applied at a constant pressure for different lengths of time. Both animal and human studies have been conducted.

Animal studies have shown that the degree of damage a nerve suffers is directly proportional to its ability to recover, with myelinated and unmyelinated axons impacted differently. Stewart (2000) points out that axon function is interpreted by acute and severe crush, when a nerve is compressed for a longer period of time, or compressed over a great part of its length. Distributing pressure over a large area of the nerve axon impacts its function and suggests that long duration sitting may contribute to nerve problems of the lower extremity.

Human nerve compression studies of the lower extremity have applied blood pressure cuffs to the circumference of middle thigh inflated to a level sufficient to produce full limb ischemia (Zakutansky et al., 2005; Zhu et al., 1998). Although these studies show that compression and ischemia alter nerve conduction, they do not directly apply to sitting because it does not cause full limb ischemia. A method that only compresses the posterior thigh is needed to better understand the impact compression has on nerve conduction.

Prolonged pressure against the buttocks or posterior thigh can damage the sciatic nerve by direct compression and pressure-induced swelling and necrosis of muscles

within the posterior of the thigh. For example nerve damage produced by compression may be caused by prolonged sitting on hard surfaces or in one position (Vogel, Albin, & Alberts, 1991) or habitually sitting on an object located in a rear pocket of one's pants (Gould, 1974). In one case, an individual who spent over 4 hours in a static seated posture with pressure applied to the mid posterior thigh, suffered significant sciatic nerve damage (Tyrrell, Feher, & Rossor, 1989).

This is an important factor to consider because nerve damage to the sciatic nerve is not uncommon in the adult population (Heliovaara, Makela, Knekt, Impivaara, & Aromaa, 1991). New diagnostic technology has demonstrated that many cases of sciatica are symptoms secondary to herniation of a lumbar disc (Moore, Tsuruda, & Dailey, 2001; Patrick, Deyo, Atlas, Singer, Chapin, & Keller, 1995; Stuart, Morgan, & Persing, 1989). Paresthesias, discomfort, pain, and other symptoms of sciatica have been more specifically diagnosed as piriformis syndrome, ischial tunnel syndrome, pudendal nerve entrapment with referred pain, distal sciatic entrapment, sciatic tumor, lumbosacral plexus entrapment, nerve root injury, and inadequate spinal nerve root decompression (Filler, Haynes, Jordan, Prager, Villablanca, Farahani, McBride, Tsuruda, Morisoli, Batzdorf, & Johnson, 2005). Other damage may also be induced by compression including acute localized crush, traumatic neuroma, and injury to the connective tissue components of the nerves (Stewart, 2000).

As with muscle damage, nerve injury may be caused by external and internal compressive forces. Regardless of the source of the compression, pressure on axons can have many effects. These include: 1) interrupted transmission of action potentials; 2) errant action potential propagation originating from the point of compression; 3)

interrupted axoplasm transport (both anterograde—from the nerve cell body and retrograde—from the axon to the cell body); 4) reduced or prevented the normal gliding of the nerve, both the bundle relative to surrounding tissue and of individual axon fascicles relative to each other within the bundle; 5) prevented proper blood flow to, from, and within the nerve which can produce ischemic conditions and prevent proper draining (blood pressure in the small vessels surrounding nerves can be as low as 11 mmHg); and 6) crushing of all or a limited number of axons within the bundle.

Compression of the Sciatic Nerve: Impact on the H-reflex

To date limited research has investigated the impact of compression on the H-reflex. Most studies in this area have used compression to induce full limb ischemia and have monitored the impact before, during, and post ischemia. Other research areas that also lend evidence to the present research study include animal, nerve conduction, and compression pathologies in humans. This section details information about the impact of compression on nerve function with primary focus on the H-reflex. Information from other pertinent methodologies is also reviewed.

Zhu et al. (1998) produced unilateral ischemia of the lower limb by a blood pressure cuff placed around the thigh 15 cm above the knee and inflated to a pressure of 300 mmHg for 20 minutes. Stimulation sites included the traditional popliteal fossa and the novel location of the S1 foramen. At the popliteal fossa location, latencies for both the H- M-waves were found to lengthen by approximately 2 ms at the end of compression/ischemia. Although quantified amplitude values were not provided, at 15 minutes only a small H-wave amplitude was observable and at 20 minutes the H-wave

was completely suppressed, while a small amplitude M-wave was still present. S1 root stimulation were tested at the onset of the ischemia and again at 20 minute. The amplitude of H_{max} elicited from the S1 stimulation was reduced approximately 50% at 20 minutes.

The high pressure and the length of time used in this study presents significant health risk to the participants making it difficult to justify a replication study implementing a similar protocol. Blood pressure cuff levels over 250 mmHg will induce full limb ischemia and provide enough force to cause tissue damage. Occlusion of the femoral artery was guaranteed at this pressure level, as was occlusion of the femoral veins, associated with the risk of blood clot formation.

In addition, this study did not follow the recovery process of post-compression. The amplitude and latency post-compression are important because they indicate the impact compression has on nerve function for some time after the adverse stimulus has been removed. The time following compression is of interest because H-wave and M-wave behavior may provide insight about the recovery process, lingering after effects, and the longer-term effects of compression on function. A more recent project conducted by Zakutansky, Kitano, Wallace, and Koceja (2005) evaluated the responses post-compression.

Zakutansky et al. (2005) examined the H-reflex in 12 healthy participants prior to 5 minutes of ischemia, during ischemia created by femoral artery occlusion, and again after a 5-minute post-ischemia recovery period. Ischemia was produced in the right leg with a pneumatic blood pressure thigh cuff and inflated to 50 to 60 mmHg above resting systolic pressure. Ischemia was confirmed when tissue oxygenation fell to 0%, as

measured by near infrared spectroscopy. All participants reached ischemia within 3 to 4 minutes at which point the 5 minute ischemia trial began. After the 5 minutes, but while occlusion was maintained, a recruitment curve was obtained and when completed the pressure was released. Within the first minute of recovery, the tissue oxygenation returned to baseline for all participants. At 5 minutes post ischemia, another recruitment curve was obtained.

After 5 minutes of ischemia, the maximum amplitude of the H-wave, H_{max}, was significantly lower than baseline values. The difference between baseline (M = 6.08, SD = 2.20 mV), and during ischemia (M = 5.18, SD = 2.18 mV) was a reduction of H_{max} by 15%. At the 5 minute post-ischemia recovery time H_{max} returned to baseline (M = 6.35, SD = 0.55 mV).

The authors concluded that acute ischemia decreases afferent and efferent axon thresholds in healthy individuals with a longer lasting effect for the H-wave. This H-wave effect indicates that compression or ischemia, since both were present, has a longer post-compression impact on sensory nerves and/or spinal level synapse transmission than on motor nerves. Ischemia may have produced this effect or this effect may have been caused by the location of the compression. This study compressed the mid-thigh therefore the reason the H-wave recovery did not return to pre-ischemia levels while the M-threshold did, may be due to the lingering effects of direct compression. The mid-thigh location would seemingly only impact the H-wave because the orthodromic electric pulse along the Ia afferents traveled through the region of compression, while the M-wave response pulse traveling down the α-mn did not pass through the compression site. Compression impact on the motor axons could not be evaluated through this method

because the H-wave utilizes both sensory and motor axons during transmission, and it is currently not possible to differentiate the effects of each. Another limitation of this study was that the recovery time used was not sufficient for the H-wave to return to baseline. This finding indicates that ischemia or direct compression have an impact on nerve function that lingers after recovery. Therefore, monitoring recovery for a longer duration is warranted. The amount of time until H-wave returns to baseline is important to know because it indicates how long it takes for recovery.

The impact of pressure on the nerve itself may induce different changes than do those of the reduced oxygen of the entire limb. The following section on threshold tracking and electrophysiological techniques have shown that axon function is altered by direct pressure.

Proposed Mechanisms

The intensity and duration of the compression applied interact to produce the severity of the tissue damage or nerve function impairment. The reduced oxygenation, increased accumulation of catabolites, and externally applied pressure in the tissue impacts the nerve in two ways by (1) altering the function of the myelin sheath and (2) changing the normal membrane potential of axon membrane.

Myelin Sheath Interference

Stuart (2000) and Schmidt (2003) details peripheral nervous system damage. Three forms of peripheral nerve lesions caused by localized pressure are reported in the literature, namely neurapraxia, axonotmesis, and neurotmesis. These three lesions

increase in severity. Neurapraxia is the mildest type of focal nerve lesion that produces clinical deficits, most often caused by localized focal pressure. It is characterized by localized loss of conduction along a nerve without axon degeneration. This is caused by a focal lesion and with time is often followed by a complete recovery. Axonotmesis is the interruption of the axons of a nerve followed by complete degeneration of the peripheral segment, without severance of the supporting structures of the nerve. Such a lesion may result from pinching, crushing, or prolonged pressure to the nerve. Neurotmesis, a type of axon loss lesion resulting from focal peripheral nerve injury in which, at the lesion site, the nerve stroma is damaged to varying degrees, as well as the axon and myelin, which degenerate from that point distally. With the most severe neurotmesis lesions, the gross continuity of the nerve is disrupted.

The earliest impact of injury is that axoplasm diffuses from the axon into the surrounded area, including the endoneurial sheath and cytoplasm of the myelin. Immediately following lesion conduction of the nerve ceases. At this time differences emerge between the axon distal and proximal to the lesion. Distally, there is a rapid decrease in cholinesterase, succinic dehydrogenase, choline acetylase, and the ability to synthesize these enzymes. Other distal changes include disintegration of neurofilaments, swelling of mitochondria, and fragmentation of endoplasmic reticulum rows of small vesicles. While proximally, there is an accumulation of materials typically transported from the cell body down the axon (McMinn & Pritchard, 1969).

Degeneration characteristics of axons are determined by fiber diameter and degree of myelination. Typically small unmyelinated fibers show signs of degenerated first at a slow rate, while the large myelinated fibers begin degeneration later but proceed at a

faster rate. In the case of the unmyelinated axons the degeneration is rather straight forward, the membrane of the axon disintegrate from the lesion sight. The loss of unmyelinated axons may result in the formation of numerous collagen pockets in which longitudinal bundles of collagen are held by Schwann cells in place of axons (Schmidt, 2003). The degeneration of myelinated axons is much more complex.

Myelinated Axon Degeneration

At two days post injury, degeneration of the myelin is noticeable by light microscopy. This particular form of degeneration has been labeled Wallerian degeneration after the English physiologist A.V. Waller (1819-1870) for his initial description of this process (Waller, 1850). Although other axonal degeneration processes may account for axon loss, Wallerian degeneration is the stereotypical process of myelinated axon degeneration. After injury, initial observable histological change is the retraction of myelin from the nodes of Ranvier (Causey & Palmer, 1952, 1953). The nodes first affected are those nearest the lesion with the phenomenon spreading peripherally. This destruction and catabolism of degenerating myelin and axons occur within a few days in the Schwann cell. Within 3-4 days additional macrophages, to those resident in the endoneurium, are present and speed-up degeneration. Simultaneously, early regenerative events begin with the proliferation of new Schwann cells, which accumulate within the original basal lamina of the axon-Schwann cell unit as bands of Bungner. Bungner bands are formed when the myelin begins to break up or segment into small units which are tube shaped and have a strong tendency to unite with one another.

At the same time axonal regeneration begins, with the sprouting of the proximal viable portion of the axon and the proximodistal growth of regenerative sprouts within the bands of Bungner. Schwann cells composing regenerative bands form by the presence of nerve growth factor and other neurotropic substances. Subsequently, several axons begin to mature within a single Schwann cell tube, giving rise to small groups of thinly myelinated axons. Over time one dominant axon emerges and the others regress. Functional recovery eventually may occur, although reconstitution of the original complement of axons is uncommon.

Traumatic Neuropathy

Acute damage of nerve is characterized by focal loss of myelin and is repaired by the remyelination process detailed above. Chronic nerve compression may result in demyelination and axonal degeneration (Schmidt, 2003). Traumatic damage to peripheral nerve with loss of continuity may cause the development of traumatic neuroma which is a combination of degenerative and regenerative processes. Axons proximal to the injury form numerous regenerative sprouts that may become disoriented and disorganized at the injury site, resulting in the formation of a mass composed of large numbers of minifascicles in a colleagenous matrix and surrounded by a few layers of perineurium. Treatment for this painful condition includes resection of the neuroma and apposition of separated proximal and distal nerve ends or placement of a nerve graft across the site of injury. It is important to note that degenerative and regenerative changes that have been outlined above take place in injured nerves with intact endoneurial sheaths as well as when complete severance has occurred (McNinn & Pritchard, 1969).

Traditional electrophysiological tests of nerve function focus on the number of conducting fibers and their conduction velocity. These techniques include the H-reflex method described above and simple nerve conduction tests in which a single nerve is stimulated and measured directly either with surface or indwelling electrodes (Oh, 1993). Any condition such as disease or injury affecting the myelin sheath impacts the amplitude and velocity and generally as the condition severity increases conduction decreases.

Membrane Potential Changes

The mechanisms that may impact conduction amplitude and latency is the impact of compression and ischemia on the axon membrane itself. Bostock, colleagues, and others (Bostock, Baker, Grafe, & Reid, 1991; Bostock, Baker, & Reid, 1991; Bostock, Cikurel, & Burke, 1998; Kiernan, & Bostock, 2000; Kiernan, Guglielmi, Kaji, Murry, & Bostock, 2002; Lin, Kuwabara, Cappelen-Smith, & Burke, 2002; Mogyoros, Kiernan, Burke, & Bostock, 1998) have used a threshold tracking technique to investigate interstitial environmental changes and their impact on the axon membrane under the myelin sheath. This method uses conventional EMG for recording nerve and muscle action potentials, a constant current stimulator pulse, and a purpose built computer for threshold tracking. The typical procedure is as follows: a 1 ms stimulus is applied to the ulnar nerve at the wrist at regular intervals, the compound muscle action potential is recorded from the hypothenar muscle (either surface or indwelling EMG), and the stimulus intensity is increased systematically until a maximum response is recorded. A target value determined as a percent of maximum response is set by the researcher. The software is designed to automatically adjust to elicit this target value based on the

preceding response. If the response was above the target value, the electric stimulus is reduced for the next stimulation, and if below the target value the stimulus is increased for the next stimulation. The output measure is the stimulus level required to generate the target value and the latency of the electric pulse as recorded by EMG.

In these studies a pressure of 200 mmHg for durations ranging from 3 minutes (Mogyoros et al., 1998) to 13 minutes (Lin, et al., 2002) has been used. The threshold tracking technique has shown compression induced ischemia to decrease threshold. While post compression, threshold increased above pre-compression intensity. Latency differences also emerge, with compression induced ischemia increasing latency and reducing latency post-compression.

These authors have reported that the membrane potential changes during ischemia and compression, and that this effect is most likely caused by interference of electrogenic ion pumps and increased activation of ion channels. Specifically, this indicates that the Na+K+ pump activity is reduced in the limited oxygen of ischemia leading to the accumulation of extracellular K+. Thus the membrane is hyper polarized which inadvertently causes voltage regulated channels to become permeable and paradoxically causing a net reduction in resting potential. This reduced membrane potential is indicated by the reduced level of stimulus necessary to generate the target response.

Summary of the Literature

The preceding sections explored the research literature on nerve function and the Hoffmann Reflex technique. Specifically, the review of literature explored an innovative application of the Hoffmann Reflex as a potential objective measure of extended duration

seat comfort. Seated posture compresses the tissue of the buttocks and posterior thigh, through which courses the sciatic nerve. From the review of literature it was concluded that a 50% H_{max} Hoffmann Reflex stimulus administered to the tibial nerve at the popliteal fossa and recorded at the soleus, would permit testing of nerve function throughout extended sitting. A 50% H_{max} stimulus administered to the tibial nerve sends a reflex volley up afferent and down the efferent axons coursing through the compressed tissue.

There currently is a paucity of research literature available evaluating the effect tissue compression has on nerve function. The literature available (Bostock, Baker, Grafe et al., 1991; Bostock, Baker, & Reid, 1991; Bostock et al., 1998; Kiernan, & Bostock, 2000; Kiernan et al., 2002; Lin et al., 2002; Mogyoros et al., 1998; Zakutansky et al., 2005; Zhu et al., 1998) has used a total limb ischemia research paradigm. These studies have used a pneumatic cuff inflated around the circumference of the limb to produce ischemia. This method confounds the effects of ischemia with those of tissue compression produced by the cuff. The preceding sections reviewed the research literature pertinent to normal nerve function, the impact externally applied tissue compression has on nerve function, and the possible mechanism responsible for these research findings. Additionally, the impact of ischemia and compression upon the Hoffmann Reflex and other nerve conduction techniques were reviewed and possible mechanisms were discussed.

METHODS

Participants

Based on Zakutansky et al. (2005), healthy individuals aged 19 to 40 were recruited to participate in this project. A total of 10 participants (5 men and 5 women) average age 22.4 +/- 3.1 years of age were evaluated in this study. Table 2 details the descriptive statistics for the sample studied. Participants were recruited from healthy men and women available for two separate visits to the Musculoskeletal Research Laboratory at Auburn University (see Appendixes B and C). All procedures were first approved by the Auburn University Institutional Review Board for the Protection of Human Subjects in Research.

Table 2. Descriptive statistics of study participants.

Category	Mean	Standard Deviation	Maximum	Minimum
Age in Years	22.44	3.13	31	19
Height (cm)	172.47	9.67	185.42	157.48
Weight (kg)	70.67	14.61	90.72	50.80
Body Mass Index	24.26	3.21	28.82	19.12
Hip Circumference (cm)	97.23	8.04	107.50	83.50

Category	Mean	Standard Deviation	Maximum	Minimum
Leg Length (cm)	37.37	3.09	41.50	33.00
Thigh Length (cm)	44.13	2.40	49.50	40.00
Lower Limb length (cm)	81.00	5.15	90.00	74.00
Middle of Thigh Circumference (cm)	53.00	4.00	61.00	47.50
Thigh Skin Fold	18.10	7.36	31.00	7.33
Deflection Ischial Tuberosity location (cm)	5.63	0.73	6.90	4.20
Deflection Medial Thigh location (cm)	4.83	0.61	6.20	3.80
Systolic Blood Pressure (mmHg)	117.30	11.4	135.00	100.00
Diastolic Blood Pressure (mmHg)	79.20	6.55	85.00	60.00

Participants were selected based on the following inclusion criteria:

1) in general good health;
2) free of injury in both the right and left lower limb for 6 months prior to participating;
3) no history of chronic injury to either right or left lower limb;
4) no history of surgery in either the right or left lower limb;

5) no prior history of neurological and/or vascular disorder;
6) no history of vascular disorder including chronic high (>220/110) or low (<90/60) blood pressure;
7) had no bleeding disorders;
8) demonstrated a measurable Hoffmann Reflex;
9) a BMI less than 30;
10) if a woman was taking birth control hormones, she was a non-smoker;
11) physically active. Physical activity was defined as a minimum of 3 times per week for at least 20 minutes per exercise bout.

Design and General Procedure

This study used a repeated measures design to apply tissue compression to two locations on two separate visits to the Musculoskeletal Research Laboratory. Data were collected prior to baseline, during compression, and post compression. Application order was counter balanced to control for possible order effects. The soleus H-wave was produced using 50% H_{max} stimulus intensity administered to the tibial nerve. The common peroneal nerve was monitored using the two-point discrimination task, administered to the lateral side of the shank. During testing, subjective comfort/discomfort was monitored. A pulse oximeter was attached to a phalange of the tested limb and monitored continuously during compression to assure the absence of full limb ischemia. Additionally, a temperature sensitive camera system was used to monitor skin surface temperature.

For this study there were three variables of interest: location, intensity, and duration. Pilot testing indicated that for both the ischial tuberosity and middle thigh location, 9 minutes of 210 mmHg application was safe. More specifically, it was found that all safety measures indicated no immediate or prolonged adverse health effects were experienced by any participant.

Location. One site of compression was the ischial tuberosity, while the second was the middle of the posterior thigh. The middle of the posterior thigh was defined as the mid point between the greater trochanter and the lateral epicondylus and the medial and lateral edges of the thigh.

Intensity. For this study, 210 mmHg was used for application intensity. The pressure intensity was based on Zakutansky et al. (2005) and was equivalent to peak contact pressure values measured using an Xsensory H2 pressure mapping system at the United States Army Aeromedical Research Laboratory, Fort Rucker, AL. The average interface pressure for an adult seated on a solid surface, such as a bleacher seat, was approximately 50 mmHg, while the peak pressure experience at this interface was found to exceed 210 mmHg. The average and peak pressure interface for an adult seated on a soft surface, such as a Herman Miller Aeron® chair, was close to 30 mmHg average and 90 mmHg peak. These values also match those published in other seat human interface studies (Stubbs et al., 2005).

Duration. Nine minutes of application was used in this study. This duration was deemed acceptable by the Auburn University Institutional Review Board for the Protection of Human Subjects in Research because Zakutansky et al. (2005) found

changes in the H-wave amplitude with this duration. The review board indicated that longer duration was excessive risk.

Procedures

Data collection was conducted during two separate visits to the Musculoskeletal Research Laboratory at Auburn University. During the first scheduled appointment at the laboratory, the participant completed the Informed Consent (see Appendix A) and Health Assessment (see Appendix D) documents. After obtaining Informed Consent and completing the Health Assessment documents—and being deemed within the health inclusion criteria for this study, the participant put on spandex type athletic shorts for the testing session. Anthropomorphic measures were taken (see Appendix E) and excessive hair was removed and the skin cleaned and prepared for electrodes.

Following attachment of electrodes, the participant was shown the procedures to be used during the lying prone portion of the study. The participant was then provided with verbal instructions as to his or her responsibility during testing. Specifically, the participant was told to lie still during all testing and stay as relaxed as possible. Additionally, each measure was explained. All equipment was attached and tested to ensure proper function. Next, the participant assumed the prone position upon the padded surface (massage table) and a bolster was inserted under the ankles for comfort.

Electrodes were attached to the dominant leg, as determined by the preferred leg used to kick a ball or manipulate an object with the toes. Stimulating electrodes for the Hoffmann Reflex were attached to the posterior and anterior of the knee and EMG electrodes attached to the soleus with a reference electrode placed over the tibia

tuberosity. The availability assessment of the Hoffmann Reflex was completed and all participants demonstrated a measurable Hoffmann Reflex. A set of 20 pulses was delivered with gradually increasing intensity at a frequency of 1 every 10 to 20 seconds to allow the individual to become comfortable with the protocol. Following this familiarization with the protocol, baseline maximum motor response (M_{max}) and Hoffmann Reflex (H_{max}) amplitude were determined. To establish baseline nerve function, up to 4 sets of 20 pulses were conducted with increasing intensity, administered with a frequency of 1 every 10 to 30 seconds. From the H_{max} amplitude the 50% H_{max} amplitude stimulus was determined and used for all subsequent stimulations.

After establishing baseline Hoffmann Reflex values, the 50% H_{max} amplitude stimulus was administered 3 times every minute approximately 10 seconds apart for approximately 5 minutes to allow the participant to become familiar with the experience. Throughout all following testing the Hoffmann Reflex was elicited 3 times every minute approximately 10 seconds apart and the ensuing muscle twitch recorded by custom software for later analyses offline. During testing the Hoffmann Reflex was recorded by EMG and visually monitored via an oscilloscope. Following the familiarization period, baseline values were collected for the dependent measures, a process that took approximately 5 minutes.

After the baseline period, the compression surface was manually lowered to the skin surface over the proper location. This location served as the zero point for the deflection variable. Then the surface was lowered into the tissue until the displacement produced a value 210 mmHg, as indicated by the load cell through the computer software. Time of application began when the compression surface made contact with the

participant. Due to the visco-elastic properties of the tissue being compressed, the pressure intensity was constantly monitored and intensity was adjusted manually as necessary. At the end of the ninth minute of compression, the displacement was recorded and the compression released.

During compression, limb oxygenation and blood flow were monitored continuously. Each minute throughout the compression period, the 50% H_{max} stimulus was presented 3 times a minute and ensuing EMG activity, subjective comfort, pulse oximeter value, and thermo imagery data was recorded. At the last minute of compression, and at the first, fifth, and tenth minute post-compression, the two-point discrimination test was conducted. Compression was released after 9 minutes, or if '3 Extreme Discomfort' was surpassed on the subjective report, or if the participant wished the trial to be terminated for any other reason. After compression release, monitoring of the Hoffmann Reflex, limb oxygenation and blood flow, subjective comfort, and the two-point discrimination test continued for 10 minutes. After 10 minutes of recovery time electrodes were removed and the participant scheduled a second appointment to repeat the above procedure. Each participant responded to the follow-up questionnaire 24- and 48- hours after each visit to the laboratory (see Appendix H).

Apparatus, Measures, and Instrumentation

Compression apparatus load cell, elapsed time, and deflection. Tissue compression was produced by a purpose built plunger (see Figure 3). The compression surface was constructed of aluminum with a flat surface that was circular in shape with a surface area of 10.16 cm^2. The apparatus was manually controlled, and the application

surface could be angled to match the angle of surface to ensure uniform pressure distribution. A load cell was interfaced with a computer to provide real-time compression intensity and to display elapsed time.

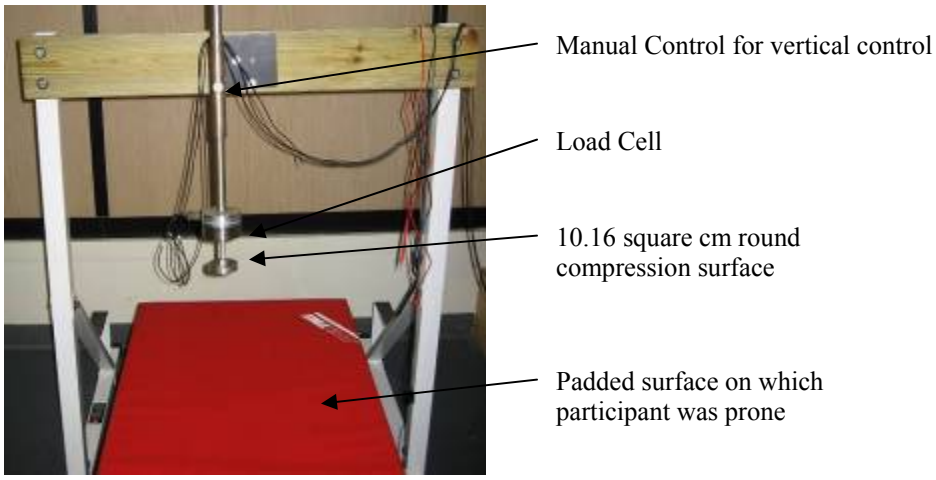

Figure 3. Front view of the purpose built compression device.

The load cell measured compression intensity with the real-time values displayed on a computer screen. Time recording began when the compression surface contacted the individual. Custom Visual Basic Version 6.0 user interface using Measurement Studio data acquisition software was used to display and record time, load cell, and EMG data. Load cell information was recorded with EMG at 2000 samples per second using a National Instruments 6020E analog to digital converter.

Deflection was measured using a tape measure permanently fixed to the stationary base and to the application surface. The initial displacement value was when the plunger initially contacted the surface of the skin and the load cell registered a force between 1 and 5 mmHg. The final displacement value was recorded at the end of the ninth minute of compression. The distance between first contact and final displacement was used in

analyses. This was considered a crude estimate of stiffness, with lower values equating to a stiffer tissue and higher values indicating a less stiff tissue.

Electromyography. A recording electrode was placed over the soleus. Proper electrode placement and minimal crosstalk was verified via manual muscle tests (Hislop & Montgomery, 1995). A DelSys, Inc., (Boston, MA) electromyography system was used to collect Hoffmann Reflex latency and amplitude. This system uses silver bar electrodes with an interelectrode distance of 10 mm, a CMMR @ 60 Hz > 80 dB, and input impedance > 1015//0.2 ohm//pF. Custom Visual Basic Version 6.0 user interface using Measurement Studio data acquisition software was used to record the EMG. EMG was recorded at 2000 samples per second using a National Instruments 6020E analog to digital converter. Data was stored and analyzed off-line after the testing conditions concluded.

Hoffmann Reflex. The soleus Hoffmann Reflex was elicited via transcutaneous stimulation of the tibial nerve, in the posterior part of the knee, using a Grass Telefactor S44 system (Astro-Med, Inc., West Warwick, RI), with electrodes placed over the popliteal fossa and superior to the patella. Specifically, the stimulus consisted of a 1 millisecond pulse using a 0.80 cm diameter stimulating electrode. A 4 cm diameter dispersive electrode was placed on the anterior thigh just superior to the patella over the quadriceps tendon (just above the knee cap). Initially, the stimulus intensity was adjusted so that a small amplitude Hoffmann Reflex was elicited as measurable on EMG output.

Two-point discrimination test. The two-point discrimination test is a well established clinical test that measures skin sensitivity to touch. This test used a two pointed compass lightly touched to the skin surface. The two points were moved closer or

further apart to determine the distance at which the participant just began to recognize the two points as only one point. The distance between the points was measured and recorded onto the data collection sheets (see Appendix F).

Subjective comfort scale. Participants were asked to rate their level of comfort related to the compression (see Appendix G). Participants were asked to verbally indicate their level of comfort with 10—extremely comfortable, 7—comfortable, 5—neutral perception of neither comfortable nor uncomfortable, 3—associated with extremely uncomfortable, and 1 equating to pain and distress. This was administered each minute during pressure application. In addition, continuous verbal communication was maintained with the participant throughout testing.

Pulse oximeter. A pulse oximeter (Nonin Onyx II, Model 9550, Plymouth MN) was used to measure full limb oxygenation. This clinical instrument provides real time information about blood oxygenation level. A small, light emitting, unit was attached by friction to the toe of the leg being tested and was interfaced directly with the measuring unit. The purpose for using a pulse oximeter was to verify that blood oxygenation to the entire lower limb was normal

Thermo imagery camera. This system uses a temperature sensitive camera interfaced with a computer for data collection and storage (Thermal Image Processor, Release 29.1.2, Computerized Thermal Imaging Inc, Ogden UT). The camera was focused on the lower limb. This system detects changes in temperature which is indicative of changes in blood flow (Pascoe, Mercer, & de Werd, 2006). However, for each individual and each specific testing session it was not be possible to infer precise blood flow changes. Rather relative changes were observable.

Blood pressure. A researcher, trained according to the ACSM guidelines, measured blood pressure using a blood pressure cuff and stethoscope. Blood pressure was taken for screening purposes and for sample descriptive statistics.

Weight, height, and body mass index. A standard balanced scale was used for weight and the attached ruler was used for height. BMI was calculated by the formula Weight [in kilograms] / (Height [in meters])^2.

Follow-up questions. Follow-up information was collected by email 24 and 48 hours after participation (see Appendix H). Participants were asked to describe any lingering effects associated with participating in this study.

Data Reduction and Statistical Analysis

Data Reduction

Hoffmann Reflex. For the Hoffmann Reflex variable, custom software recorded the EMG data onto computer at a rate of 1000 samples per second. For each minute, 25 seconds of EMG data was collected during which time the three 50% H_{max} amplitude stimuli were administered. Custom software was used to process the raw data into H-wave latency and amplitude. Latency was calculated as the elapsed time between the maximum peak of the stimulus onset and the first peak of the H-wave on the EMG trace. Amplitude was calculated as the voltage difference of the maximum and minimum peak-to-peak deviation of the H-wave. Latency was reported in milliseconds (ms) and amplitudes were reported in millivolts (mV). H-wave amplitude was normalized for each participant by dividing each H-wave by the H_{max} amplitude established for each

participant at each of the two separate visits to the laboratory. After establishing normalized data, minute by minute data was pooled and used for subsequent analysis.

Two-point discrimination test. For the two-point discrimination test, data was normalized to each individual's baseline value. Baseline was determined during both visits to the laboratory. Normalization was established by dividing each subsequent data point by baseline and multiplying that value by 100. Normalized data were used for inferential statistics.

Subjective comfort. Subjective comfort was reported as the number indicated by each participant.

Statistical Analysis

Separate repeated measures 2 (Location) x 5 (Time) ANOVAs were performed to for each of the dependent variables: H-wave amplitude, H-wave latency, 2-point discrimination test, and subjective comfort. The two locations were the ischial tuberosity and the middle posterior thigh and the five times used were baseline, the last minute of compression, the first, fifth, and tenth minutes post-compression. Significance was denoted by an alpha of .05. Follow-up tests were to include paired sample t-tests for location for each of 5 times and simple planned comparisons for each measure across time. The last minute of compression was used for the planned comparison tests for each measure over time.

Deflection. The influence of tissue stiffness, as measured by displacement of the plunger surface into the tissue, on the Hoffmann Reflex H-wave was investigated by a Pearson's correlation coefficient.

RESULTS

Safety Measures

As with the pilot testing of this protocol, all safety measures indicated no immediate or prolonged adverse health effects experienced by any participant. During testing, no trial was terminated prematurely. As can be reviewed in Appendix I, no participant reported a subjective comfort score lower than uncomfortable and pulse oximetery values did not change from baseline. Throughout testing, all participants demonstrated pulse oximeter values greater than 92%, the criterion value considered acceptable for normal systemic blood oxygenation. The lowest individual value observed was 97% oxygenation, and this score was recorded during baseline for that individual. Additionally, the thermo camera system showed no changes in blood flow. Both the thermo camera data and the pulse oximeter data indicated that no participant experienced any reduction in full limb oxygenation during any trial.

Data Manipulation

SPSS for Windows Release 11.0.0 was used for all data analyses. The raw data used in the following statistical analyses is available in Appendix I. A total of 600 data points were compiled to generate the scores for the H-wave amplitude and latency variables. Specifically, 10 participants were exposed to two locations; data was generated

at five test times of interest throughout treatment at each location, and at each test time, three samples were produced for the two variables, H-wave amplitude and the H-wave latency. Out of the possible 600 data points, two were not available for processing. In each case one out of three samples per minute was missing. Therefore, a sample mean of the two data points was used in place of the mean calculated with three. No minute was missed completely in any condition and thus group mean substitutions were unnecessary.

A total of three research assistants participated in the data reduction process for the H-wave amplitude and latency variables. To assure consistency between research assistants, inter- and intra- rater reliability tests were performed. Each research assistant evaluated the same ten H-wave data sets twice. The two sets of 30 data points were compared within and between each research assistant. Results indicated that inter- and intra- rater reliability was acceptable with all r values reaching statistical significance values of 1.00, $p < .05$.

Treatment Order Effect

One limitation inherent in the repeated measures investigation is the possibility of an order effect. The treatment locations in this study were counterbalanced to reduce the likelihood of an order effect. To statistically test for a treatment order effect, a one-way ANOVA was performed. Treatment order was entered as the independent variable and the H-wave amplitude during the last minute of compression as the dependent variable. No significant effect emerged, $F(1, 18) = 0.49$, $p = .50$, indicating that an order effect was not present.

H-wave Amplitude Hypothesis

A 2 (Location) x 5 (Time) repeated measures ANOVA was conducted to test the effects of location and time on the H-wave amplitude. The two locations of interest were the ischial tuberosity and middle posterior thigh. The five times used were the last minute of baseline, the ninth (last) minute of compression, and the first, fifth, and tenth minutes following compression (see Table 3). The data used in this analysis were established by averaging the three H-waves amplitudes collected each minute, normalized for each participant to his or her H-wave maximum. The values presented in Table 3 represent the percent of the H-wave maximum.

Table 3. Means and standard deviations of the Hoffmann Reflex amplitude presented as percent H-wave maximum amplitude. The two locations ischial tuberosity (IT) and middle posterior thigh (MP) are presented for each of the five times used in the repeated measures ANOVA.

	Pre-		During-		Post- Compression minute					
	Baseline		Last Minute		First		Fifth		Tenth	
Location	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
IT	52.53	19.02	72.84	14.97	63.83	17.25	64.94	14.26	67.53	19.85
MP	50.59	15.45	65.04	20.81	51.45	22.99	55.91	14.88	52.51	21.92

A significant main effect for location emerged, $F(1, 9) = 5.55$, $\eta^2 = .38$, $p = .04$. The ischial tuberosity ($M = 67.28$, $SD = 4.59$) location produced a larger increase in the H-wave amplitude than did the middle posterior thigh ($M = 56.86$, $SD = 4.04$) location. A significant main effect for time was also found, $F(4, 36) = 2.80$, $\eta^2 = 0.24$, $p = .04$. Simple follow-up planned comparisons revealed that when collapsing the two locations together, the last minute of compression ($M = 68.95$, $SD = 4.99$) produced a significantly larger H-wave amplitude than baseline ($M = 51.56$, $SD = 4.96$), $F(1, 9) = 8.91$, $\eta^2 = 0.48$, $p = .02$. Similarly, the last minute of compression also had a significantly larger H-wave amplitude than did the first minute post-compression ($M = 57.64$, $SD = 5.00$), $F(1, 9) = 5.58$, $\eta^2 = 0.38$, $p = .04$. The last minute of compression was not found to be significantly different from the fifth ($M = 60.43$, $SD = 3.71$), $F(1, 9) = 3.08$, $\eta^2 = 0.26$, $p = .11$, and tenth ($M = 60.03$, $SD = 5.76$), $F(1, 9) = 2.95$, $\eta^2 = 0.25$, $p = .12$, minutes post-compression.

An interaction between location and time was hypothesized for the H-wave amplitude. The ischial tuberosity location was predicted to remain unchanged over time while the middle posterior thigh location was expected to be attenuated during compression and gradually return to baseline amplitude over the 10 minute time period post-compression. However, no interaction effect between location and time was found for the H-wave amplitude, $F(4, 36) = 0.86$, $\eta^2 = .09$, $p = .49$. To explore possible extraneous variables responsible for the non-significant interaction effect of the H-wave amplitude, 6 ANCOVAs were performed. The same 2 (Location) x 5 (Time) ANOVAs used above were repeated with BMI, deflection of the plunger, hip circumference, thigh circumference, skin fold of the thigh, and gender entered as separate covariates. Results

revealed no single variable emerged as a significant covariate for the H-wave amplitude, namely, BMI, $F(4, 32) = 0.87$, $\eta^2 = 0.10$, $p = .49$; deflection of plunger the plunger, $F(4, 32) = 0.53$, $\eta^2 = 0.06$, $p = .71$; hip circumference, $F(4, 32) = 1.55$, $\eta^2 = 0.16$, $p = .21$; thigh circumference, $F(4, 32) = 0.63$, $\eta^2 = 0.07$, $p = .52$; skin fold of the thigh, $F(4, 32) = 0.55$, $\eta^2 = 0.06$, $p = .70$; and gender $F(4, 32) = 1.10$, $\eta^2 = 0.12$, $p = .37$.

H-wave Latency Hypothesis

To test the effects of location and time on the Hoffmann Reflex latency a 2 (Location) x 5 (Time) repeated measures ANOVA was conducted. The two locations assessed were the ischial tuberosity and middle posterior thigh. The five times used were the last minute of baseline, the ninth (last) minute of compression, and the first, fifth, and tenth minutes following compression (see Table 4). The data used in this analysis were established by averaging the three H-waves latencies collected each minute. Values presented in Table 4 are reported in milliseconds (ms).

Table 4. Means and standard deviations of H-wave latency in milliseconds. The two locations ischial tuberosity (IT) and middle posterior thigh (MP) are presented for each of the five times used in the repeated measures ANOVA.

	Pre-		During-		Post- Compression minute					
	Baseline		Last Minute		First		Fifth		Tenth	
Location	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
IT	31.50	1.98	31.50	2.10	31.44	2.09	31.46	2.16	31.57	2.06
MP	31.50	2.07	31.50	1.81	31.50	1.93	31.50	1.90	31.43	2.04

Significant main effects for location and time did not emerge, $F(1, 9) = 0.43$, $\eta^2 = 0.04$, $p = .53$, and $F(4, 36) = 0.07$, $\eta^2 = 0.01$, $p = .99$, respectively. An interaction between location and time was hypothesized for the H-wave latency. The ischial tuberosity location was predicted to remain unchanged over time while the middle posterior thigh location was anticipated to be lengthened during compression and gradually return to baseline over the 10 minute time period post-compression. No interaction effect between location and time was established for the H-wave latency $F(4, 36) = 0.49$, $\eta^2 = 0.05$, $p = .74$.

Two-point Discrimination Test Hypothesis

For one participant, there were incomplete data for the two-point discrimination test for the middle posterior thigh. This participant requested the two-point discrimination

test to be stopped due to strong dislike for the sensation produced by repeated exposure of the points to the skin. This participant was excluded from the two-point discrimination data analysis.

To test the effects of location and time on the two-point discrimination test a 2 (Location) x 5 (Time) repeated measures ANOVA was conducted. The two locations assessed were the ischial tuberosity and middle posterior thigh. The five times used were the last minute of baseline, the ninth (last) minute of compression, and the first, fifth, and tenth minutes following compression (see Table 5). The data used in this analysis were established by normalizing to baseline; individual participants' minute values were divided by baseline value and multiplied by 100. Values presented in Table 5 are reported in percent baseline value for each minute of interest.

Table 5. Means and standard deviations for the two-point discrimination test. The two locations ischial tuberosity (IT) and middle posterior thigh (MP) are presented for each of the five times used in the repeated measures ANOVA.

	Pre-		During-		Post- Compression minute					
	Baseline		Last Minute		First		Fifth		Tenth	
Location	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
IT	100	0.00	98.6	37.0	93.3	41.0	93.1	40.9	92.7	38.6
MP	100	0.00	85.3	24.3	100.7	27.2	94.7	24.8	104.0	36.1

A significant main effect for location, $F(1, 8) = 0.04$, $\eta^2 = 0.00$, $p = .85$, and time, $F(4, 32) = 0.53$, $\eta^2 = 0.06$, $p = .71$ were not found. An interaction between location and time was expected for the two-point discrimination task. The ischial tuberosity location was predicted to remain unchanged over time while the middle posterior thigh location was predicted to be reduced during compression and gradually return to baseline over the 10 minute time period post-compression. No interaction effect between location and time emerged, $F(4, 32) = 0.49$, $\eta^2 = 0.09$, $p = .88$.

Subjective Comfort Hypothesis

To test the effects of location and time on subjective comfort a 2 (Location) x 5 (Time) repeated measures ANOVA was conducted. The two locations assessed were the ischial tuberosity and middle posterior thigh. The five times used were the last minute of baseline, the ninth (last) minute of compression, and the first, fifth, and tenth minutes following compression (see Table 6). Values presented in Table 6 represent means and standard deviations of self-reported comfort with anchor perceptions equivalent to 10 (Extremely comfortable), 7 (Comfortable) 5 (Neutral: Neither comfortable nor uncomfortable), 3 (Extremely uncomfortable), 1 (Pain and distress).

Table 6. Means and standard deviations for subjective comfort. The two locations ischial tuberosity (IT) and middle posterior thigh (MP) are presented for each of the five times used in the repeated measures ANOVA.

Location	Pre- Baseline		During- Last Minute		Post- Compression minute First		Fifth		Tenth	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
IT	10.00	0.00	7.80	1.68	8.60	1.07	9.80	0.30	9.80	0.42
MP	10.00	0.00	7.00	1.41	7.60	2.01	9.50	0.60	9.70	0.48

It was hypothesized that for both locations subjective comfort would be lower at the last minute of compression than at baseline. Subjective comfort would return back to baseline over the10 minute post-compression period. A main effect for location approached significance, $F(1, 9) = 3.65$, $\eta^2 = 0.28$, $p = .09$, while a significant main effect for time was found, $F(4, 36) = 29.79$, $\eta^2 = 0.77$, $p < .05$. Simple follow-up planned comparison revealed that the last minute of compression differed significantly from baseline, $F(1, 9) = 37.07$, $p < .05$; the first, $F(1,9) = 12.25$, $p < .05$; the fifth, $F(1,9) = 32.25$, $p < .05$; and the tenth, $F(1,9) = 34.22$, $p < .05$, minutes post-compression. No interaction effect between location and time emerged, $F(4, 36) = 1.22$, $\eta^2 = 0.12$, $p = .32$.

Deflection Hypothesis

A Pearson's product moment correlation coefficient was calculated to test the strength of the relationship between the distance, in centimeters, the plunger surface deflected into the tissue at each location, and the H-wave amplitude value during the last minute of compression. No significant relationship was found between H-wave amplitude and deflection for the ischial tuberosity and middle posterior thigh, r = -.26, p = .46, and r = .29, p = .49, respectively. Although not part of hypotheses testing, further exploration of stiffness was investigated to provide further descriptive information for this sample. By location, the middle thigh (M = 4.83 cm, SD = 0.61) was not stiffer than the ischial tuberosity (M = 5.63 cm, SD = 0.73), $t(9) = 2.12$, p = .06. Values by sex showed no significant difference for either location, namely, the middle thigh location men (M = 4.88 cm, SD = 0.90), and women, (M = 4.78 cm, SD = 0.33), $t(4) = 0.26$ p = .80, and the ischial tuberosity location men (M = 5.82 cm, SD = 0.75), and women (M = 5.44 cm, SD = 0.82), $t(4) = 1.25$ p = .28.

DISCUSSION

This investigation was conducted to determine the effects of tissue compression on nerve function in the absence of full limb ischemia. Previous research in this area has used a pneumatic cuff inflated around the circumference of the middle thigh to produce ischemia distally in order to determine the influence of total limb ischemia on nerve function as measured by the Hoffmann Reflex H-wave and M-wave, amplitudes and latencies. This method confounds ischemia with the physical compression produced by the pneumatic cuff. The present study incorporated a novel laboratory device to test the effects of tissue compression on nerve function without producing full limb ischemia. Compression was administered over the sciatic nerve at the same location, middle thigh, as previous research and to a control location, the ischial tuberosity. The present findings showed this new research equipment to be safe for the intensity and duration applied during the testing protocol.

Throughout this study full limb blood flow and total limb oxygenation did not change. Blood flow and total limb oxygenation remained at baseline values for all 10 participants during and post- compression. This finding indicates that observed changes in the dependent variables are not attributable to full limb circulation reductions. Previous research has used full limb occlusion to study nerve function in the ischemic limb, whereas this study evaluated nerve function in a more normal limb.

It was hypothesized that the dependent measures for H-wave amplitude, H-wave latency, and two-point discrimination would be altered, from baseline values, for the middle thigh location and be unaffected at the ischial tuberosity location during compression. Specifically, it was hypothesized that the dependent measures for the middle posterior thigh location would gradually return to baseline values post-compression, that is, the H-wave amplitude would be significantly attenuated during compression and gradually return to baseline amplitude over the 10 minute time period post-compression. Unfortunately, a significant interaction effect did not emerge for any of these three measures. However, an interesting main effect for the H-wave amplitude was found. This main effect finding revealed that compression, regardless of location, amplified the H-wave amplitude.

H-wave Amplitude

Although the proposed interaction effect for the H-wave amplitude hypothesis was not significant, main effects for time and location were found. The main effect for location revealed that compression at the ischial tuberosity location produced a 9% larger H_{max} amplitude H-wave than the middle thigh location. In addition, the main effect for time showed that the last minute of compression produced a 17% larger H_{max} amplitude than baseline. The first minute post-compression was significantly different than the last minute of compression while the fifth and the tenth minutes post compression had similar amplitudes as the last minute of compression. These findings are difficult to explain. Although a main effect for time was not hypothesized, it would make sense that, similar to the first minute post-compression, the fifth and tenth minutes post-compression would

be comparable to baseline and not similar to the last minute of compression. Instead, the main effect for time indicated that extended recovery was similar to compression for both locations. This suggests that the compression amplitude and extended recovery time produced a similar effect on the H-wave amplitude.

The non-significant interaction for the H-wave amplitude suggests that compression over the sciatic nerve, as used in this study, did not have the expected impact on nerve function. It was expected that the compression would produce a change in nerve function, measurable as a reduction in H-wave amplitude. The mechanism suggested to produce this effect was membrane potential changes caused by direct pressure and reduced nutrient flow to the axon. The potential changes of the membrane were believed to inhibit the normal saltatory conduction of action potentials along some axons which would manifest as reduced H-wave amplitude in the EMG trace during compression. However, attenuation was not found, rather amplification of the H-wave amplitude in response to compression was revealed. Additionally, the control location also experienced the amplification, suggesting a completely different mechanism than the one proposed.

Two possible explanations may account for the non-significant interaction and the main effect findings: (1) cutaneous stimulation increasing the excitability of the alpha motor pool, and (2) descending excitation produced by attention focused on the compression experience. The first explanation suggests that cutaneous sensory receptors provided an increase in motor pool excitation in the spinal cord. Animal research has found that in the presence of strong cutaneous stimulus, the number of motor units recruited was larger than when no stimulus was administered to the skin (Clark, Dacko,

& Cope, 1993; Kanda, Burke, & Walmsley, 1977). It is possible that the intensity used for compression was adequate to activate cutaneous sensors which served to increase the number of motor units recruited during compression. An increase in the number of motor units recruited would have manifested as a larger amplitude H-wave, as was found. The finding that both locations experienced amplitudes larger than baseline during compression suggests that this mechanism could be a plausible explanation for the results. Future research should attempt to verify the presence of this cutaneous effect by using a similar method to the one used in the present study, but instead of applying compression, the application of a strong cutaneous stimulus (i.e., a pinch), could be the independent variable. This first mechanism suggests that peripheral afferent excitation increases activity in the spinal motor neuron pool because this phenomenon was revealed in the decerebrate cat (Clark, et al., 1993; Kanda, et al., 1977). The next two plausible explanations for the findings of the present study indicate that descending control from the brain produced increased activity in the motor neuron pool.

The second explanation raises the possibility that the compression surface provided a location upon which to focus attention leading to descending control from the brain to increase activity of the motor neuron pool. Attentional focus has been established as a confounding variable associated with the Hoffmann Reflex (Blackburn, 2004; Palmieri, et al., 2004). It is possible that the current main effects could be explained by descending excitation messages generated by conscious thought. At baseline there was no single location on which to focus conscious attention, but during compression the plunger surface provided a location upon which attention could be focused. During post-compression there may have been a lingering sensation upon which to focus. However,

because focus was not assessed in this study, future research could monitor attentional focus.

H-wave Latency

Zhu, et al. (1998) found that the H-wave latency was prolonged by 2 ms after 15 minutes of full limb ischemia. Based on the findings of Zhu, et al. (1998), the H-wave latency was expected to increase during the ninth (final) minute of compression in the present study. However, no significant finding emerged for latency. Moreover, the latency means did not change over time; all mean values in each cell used for statistical analyses were 31 ms. One possible explanation for this finding is that the compression was not of sufficient duration to produce a measurable impact on the latency of the H-wave. The Zhu, et al. (1998) study used a higher pressure intensity, 300 mmHg, as compared to 220 mmHg used in the present study. Compression alone may have an impact on H-wave latency; however, the application time of nine minutes may be insufficient to produce a measurable difference. The choice to use nine minutes of compression at an intensity of 210 mmHg was deemed by the scientific ethics committee as the maximum values with the lowest associated risk. Future research using extended duration and increased intensity would be likely to find changes in H-wave latency.

Two-point Discrimination Test

The two-point discrimination test is typically used in clinical settings as an inexpensive early assessment of nervous system dysfunction. Asymmetries between limbs or the absence of sensation is a sign of nervous system dysfunction and warrants

further testing. The two-point discrimination test was included as a secondary measure of nerve function, in addition to the Hoffmann Reflex. It was hypothesized that compression would alter the function of the sciatic nerve, thus reducing cutaneous sensitivity measured as increased distance for the just noticeable difference.

Unfortunately, significant information was not generated by the two-point discrimination test. Moreover, repeated exposure to this measure was reported by many participants to be unpleasant. In follow-up questions most participants indicated the test to be uncomfortable and required a high level of attentional focus to perform. One participant disliked the experience so intensely that a request was made to stop performing the test, which was obliged by the researchers. This test is an invaluable clinical assessment of nerve function; however, in this application it proved unnecessary. Significant knowledge would not be lost by excluding this variable from future research. Participants did not like the sensation and the data derived from the assessment was ambiguous. Finally, it is possible that this assessment contributed to the increase of the H-wave amplitude in that it provided an additional cutaneous stimulus.

Subjective Comfort

Subjective comfort was hypothesized to be lowest during compression followed by a return to baseline after release. This prediction was supported by a main effect for time. During compression, comfort was reported to be lower than during baseline, although still within the comfortable range of the scale. Post-compression comfort returned to baseline. Overall the subjective comfort findings indicate that the experience

was not unpleasant and that minimal discomfort was produced during and after release of compression.

Deflection

The hypothesis relating deflection with the H-wave amplitude was made to account for differences in tissue density between individuals. Visceral fat and muscle density were believed to alter the distribution of force transmitted from the skin's surface through to the sciatic nerve. It was expected that tissue density would be correlated with the H-wave amplitude; however significant correlations did not emerge. This study used a crude surrogate in lieu of establishing a force-deflection curve; the displacement of the plunger surface into the tissue was used. This method provided a single value, in centimeters, to suggest the stiffness of a location. Lower values were indicative of a stiffer material and higher values a less stiff material.

The displacement or stiffness of the tissue being compressed may contribute significantly to the effect upon the sciatic nerve. Although no significant finding emerged in this study further investigation is warranted. Future research should incorporate an animal model to systematically explore the influence cutaneous level compression has at the nerve level of the tissue as a function of the tissue density between the skin surface and precise locations along various nerves.

This research project was the first step in evaluating the use of nerve function as a potential metric of seat performance. Sitting provides tissue compression over a large area and generates a range of intensities throughout. The method employed purposefully limited the extraneous variables of area and range of intensities associated with sitting. A

purpose built plunger was used to administer known tissue compression intensity (210 mmHg) to exact locations (ischial tuberosity and middle posterior thigh) for a prescribed duration (9 minutes).

This study also contributes to the literature in two important ways. First, this study assessed nerve function during normal blood flow and full limb oxygenation unlike previous research which used a full limb ischemia method. The middle thigh location of the tissue compression replicated the location and intensity produced by ischemia method in previous research (Zakutansky, et al., 2005; Zhu, et al., 1998). Secondly, a control location was used in this study. The ischial tuberosity location, proved invaluable because it revealed that a mechanism additional to tissue deformation and ischemia may contribute to these findings and those of previous research. Previous research has not employed a control location because it may not be possible to use a control location and still produce full limb ischemia.

The primary dependent variables of interest were the H-wave amplitude and latency. The influence of tissue deformation and local ischemia, under and around the plunger, were the mechanisms driving the interaction hypotheses for the H-wave variables. Deformation was measured by displacement into the tissue and was shown to be unrelated to the H-wave amplitude. Ischemia may have been present during compression however; it was not possible to measure the oxygenation saturation of the compressed tissue, due to a lack of instrumentation. However, this may be unimportant, because ischemia may not have contributed to the H-wave amplitude findings, the data did not support the interaction hypotheses. The findings indicated that both locations

produced an increase in the H-wave amplitude during and post compression while neither location impacted latency.

The finding that both locations increased H-wave amplitude indicates that deformation and/or ischemia may not account for amplitude changes. There is not a plausible mechanism to account for deformation or ischemia of the tissue at the ischial tuberosity responsible for producing amplitude increase. In normal human anatomy the sciatic nerve does not course over the ischial tuberosity, thus compression at the ischial tuberosity did not directly impact the sciatic nerve. Deformation and ischemia were anticipated to alter nerve function at the middle posterior thigh location because compression was directly over the nerve. To account for these findings another mechanism must be responsible for the amplitude increases observed. Two candidate mechanisms were identified, namely, cutaneous stimulation (Clark, et al., 1993; Kanda, et al., 1977) and attention focus (Blackburn, 2004; Palmieri, et al., 2004) these may have acted independently or synergistically. Further research is required to investigate the Hoffmann reflex amplitude increases observed in this study.

Conclusions

Based on the findings of this study the following conclusions were made:

1. Regardless of location, nerve function is not altered as hypothesized. Specifically, a tissue compression area of 10.16 cm^2 administered at 210 mmHg for 9 minutes, does not produce the hypothesized influences on H-wave amplitude, H-wave latency, and the two-point discrimination task.

2. Subjective comfort is significantly reduced from baseline by a tissue compression area of 10.16 cm^2 administered at 210 mmHg for 9 minutes and does return to baseline value within 10 minutes of recovery time.

3. Tissue deflection is not significantly related to the H-wave amplitude.

Future Research

Future researchers should consider extending the duration of the compression application used in this study. The nine minutes of compression at 220 mmHg, was found to be safe for all participants. Bruising, soreness, or other undesirable side effects were not indicated immediately following each trial, or at the 24- and 48- hour follow-up. Additionally, during the trials the participants reported the experience to be no worse than comfortable, mean comfort scores for the final minute of compression were 7.0 and 7.8 for the middle posterior thigh and ischial tuberosity, respectively.

Another suggestion for future research is to use comfort as the primary independent variable for determining compression release. The research question guiding this project was to evaluate the effectiveness of using the Hoffmann Reflex H-wave amplitude and latency as an objective metric for comfort. However, the duration of compression application used was not sufficient to generate uncomfortable sensations. Changing the criterion for compression release from elapsed time, to perceptions of comfort/discomfort, would better address the research question. Sitting for extended periods of time is an activity most people have experienced. Requesting participants to experience compression until the sensation was similar to an uncomfortable sitting

experience, may elicit the desired conditions. Increasing the range of comfort scores reported could reveal a meaningful and significant relationship.

In addition to altering the independent variable used for compression release, future researchers should develop a more sophisticated plunger device to produce compression. Two improvements suggested for the plunger include the addition of an automatic control mechanism, and inclusion of a tissue oxygenation monitoring system. The plunger used in this study was manually operated. Manual operation provided a low cost device that required human operators to maintain proper compression intensity. This was not a difficult task to perform, but continuous human operation of a system is prone to possible error. To eliminate the error associated with manual control, a motor to drive displacement and a software control mechanism for pressure could be employed.

Pilot testing of the plunger incorporated a 16 channel Near Infrared Spectroscopy (NIRS) system to monitor the oxygenation of the tissue near the plunger surface. This system was used to show that the plunger did not produce a significant change in tissue oxygenation near the contact location. One limitation of both the pilot and this present study was the assumption that tissue oxygenation under the plunger would change over time, while the rate of change and absolute saturation values were not measured. Future research should incorporate tissue oxygen saturation monitoring into both the plunger surface and the area near the contact location. Incorporation of a NIRS into the plunger compression surface would provide information about tissue oxygenation directly under the compression surface. This improvement would permit investigation of the relationship between changes in the Hoffmann Reflex during known changes in oxygen saturation. This information is currently unavailable in the literature.

A final suggestion for future research would be to ensure that the protocol used eliminates any external distraction. To limit external distractions, sensory deprivation is recommended. Visual and auditory stimuli were held constant in this study but were still present. Use of sensory deprivation would control the visual and auditory stimuli experienced during the test session. This would not serve to control for the physical contact of the plunger, which would still provide a source for focusing attention; it would, however, ensure a higher degree of consistency between participants and conditions.

The use of a control location, namely the ischial tuberosity, is recommended for future research investigations of tissue compression and ischemia influences on nerve function. The findings from the control location provided useful information in this study. If only the treatment location, the middle posterior thigh, had been used, the findings of this study would suggest that compression, without full limb blood flow occlusion, produced amplification of the H-wave amplitude by more than 10% H_{max}. However, the finding that the control location, the ischial tuberosity, also produced amplification suggests that the compression, placed over the sciatic nerve coursing through the posterior thigh, was not responsible for the observed changes from baseline. Rather, another mechanism altogether contributed to these findings. Additionally, there might be other interesting phenomena associated with systematically investigating the influence various locations along the same limb or locations along different limbs might have on nerve response. Inter and intra limb control locations should be tested and evaluated.

A major portion of work associated with this project was gaining approval to use the plunger device with human participants. Prior to conducting the study reported above, pilot testing was required to determine a safe set of intensities and durations for the two

locations. The intensities and locations chosen were based on previous research using the full limb blood occlusion. The absolute values used were those with the lowest associated risk of producing tissue injury. This study indicates that future work could systematically extend the window of intensity and duration used and still be within acceptable risk for human participants.

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APPENDICES

Appendix A—Informed Consent Letter

INFORMED CONSENT
for a Research Study Entitled
EFFECT OF TISSUE COMPRESSION ON THE HOFFMANN REFLEX:
COMPARISON BETWEEN THE ISCHIAL TUBEROSITY AND POSTERIOR THIGH

You are invited to participate in a research study to assess how the pressure of sitting impacts the nerve function of the lower limb. This study is being conducted by Paul St. Onge a graduate student under the supervision of Dr. Mary Rudisill, department chair of the Auburn University Health and Human Performance Department. We hope to learn how different pressure intensities and application locations impact nerve function. You were selected as a possible participant because you are not currently afflicted by peripheral neurological or vascular disorders, are 19 to 40 years of age, and are in good overall health.

If you decide to participate you will be required to visit the Musculoskeletal Research Laboratory twice. The second visit must be at least 48-hours after the first visit. During testing the nerve that controls the muscles of your calf, in your dominant leg, will be stimulated and monitored during two different conditions conducted on two separate days. Electromyography and nerve stimulation electrodes will be attached to your skin. The electromyography electrodes will be attached to your calf and the stimulation electrodes will be attached to the front and back of your knee (see Image 1 and 2). The nerve stimulation test uses a very brief (1 millisecond) weak electric pulse that has been described as a “carpet shock”. The result of the stimulation is a brief contraction of the calf muscles. While the stimulus may seem uncomfortable at first, most people quickly get used to the sensation. This is a safe procedure that is used frequently for medical screening and rehabilitation purposes. You will be given 20 small stimuli to allow you to get used to the stimulus. If you do not get used to the stimulus, or if you feel uncomfortable with the test at any time, the experience can be halted.

Once the 20 familiarization trials have been performed, you will lie on a padded surface facedown for approximately 30 minutes. At this point measurement will begin. An initial nerve test will be conducted 4 times. These 4 initial tests will use various stimulus levels to determine the strongest your calf muscle can contract. Due to the short duration of the stimulus, you will most likely not be able to determine a difference between each stimulus level but may notice a more vigorous calf contraction. When this test is complete, pressure will be applied to one of two locations. The maximum pressure applied is similar to that experienced while sitting on a firm surface such as a folding chair or a bleacher seat.

One pressure intensity will be applied to one location one day and upon returning to the laboratory a second time, the same pressure will be applied to the second location. The two different locations are the ischial tuberosity and the middle of the posterior thigh (see Image 3 and 4). The ischial tuberosity, is also known as the sit bone and is located in the buttocks. The middle thigh will be determined by measuring the length and width of your thigh.

To determine the location of anatomical landmarks for this study a trained researcher will be required to touch areas of your lower limb, buttocks, and hip. Specifically, a researcher will touch the outside of your hip, the ischial tuberocities in the buttocks, the front and back of your knee, and your lower calf. Both male and female researchers will be available to perform these tasks. Please inform the researchers if this is uncomfortable for you.

_____
Participant’s Initials

While the pressure is applied, a series of nerve tests will be conducted using a stimulus determined from the initial test. The stimulus will be given 3 times at the beginning of each minute, at approximately 10 second intervals. Additionally, blood oxygenation and flow to your leg will be monitored continuously and you will be asked to report your level of comfort each minute. Blood oxygenation will be measured at the toe by a pulse oximeter. Blood flow will be monitored by a thermo imagery camera that will detect changes in skin temperature. There will also be a test of skin sensation every few minutes in which you will be asked to report if you feel one or two items touching your leg. If at any point the pressure applied to your leg becomes too uncomfortable or painful or if your blood flow is interrupted, the pressure will be released. Also, if you do not want the pressure to remain applied, or if you feel uncomfortable with the test at any time, the experience can be stopped. If a test is stopped for any reason you will still receive compensation.

After the pressure is released, the nerve test will continue for a few more minutes. Additionally, your level of comfort, blood oxygenation and flow, and skin sensation will continue to be monitored. This same procedure will be repeated during the second visit to the laboratory.

At the end of all testing, electrodes will be removed and you will be asked to respond to follow-up questions 24 and 48 hours later. These questions will ask you to name and describe all bruising, discomfort, pain, or other sensation you are experiencing related to this study.

You will spend approximately two hours in the lab for all procedures to be conducted. Responding to the follow-up questions will take you approximately five minutes for the two days following each visit to the laboratory.

The risk associated with participating in this study is minimal. Mild skin irritation may occur due to the process used to prepare the surface for attaching the electrodes for electromyography collection. Mild discomfort may occur during the nerve stimulation. You will be asked every minute how you feel, and if you are uncomfortable, all effort will be made to reduce the unpleasantness. Please inform the investigators if you experience any uncomfortable sensations or wish to end the testing procedures at any point. You have the right to withdraw from the study at any time.

To ensure proper coupling of the measurement equipment to you, it may be necessary to remove excess hair from the lower limb to be tested. Specifically, the area right above your knee, on your lower calf, on the top of your shin, and posterior thigh may need to be shaved. If shaving is necessary, a new razor will be used and extreme care will be taken to reduce the discomfort the hair removal process may produce. If this procedure is too uncomfortable you have the right to withdraw from this study at any time.

The tissue compression used in this study will provide a tissue load that may cause tissue damage. Tissue damage may appear as a bruise, muscle stiffness, burning, tingling, or numbness. If you experience any of this these symptoms of tissue damage please inform the researchers immediately. If any of these develop during testing, the test will be terminated immediately. Tissue compression will also reduce the nutrient flow into and out of the tissue compressed which can produce ischemia. With sufficient time, ischemia can cause tissue death. The risk of tissue death occurring in this study will be minimal because the pressure intensities and the application times used are not sufficient to produce tissue death due to ischemia.

_____
Participant's Initials

There is no risk of injury due to electric shock during this experiment. The equipment being used is designed to isolate the electricity drawn from the wall socket, and contains a series of fuses that are automatically set off in the event of a power surge. When these fuses are set off, current flow to the device is immediately stopped. In this manner, electrical surges in the building's electrical supply cannot reach you.

In addition, there may be uncommon or previously unrecognized risks that might occur. You will be given any new information gained during the course of the study that might pose a risk and affect your willingness to continue your participation.

You should not participate in this study if you are not in general good health, have sustained an injury to either your right or left legs in the past 6 months, if you have any chronic peripheral nervous system or vascular or bleeding problems, or if you are not physically active (exercise at least 3 times per week for 20 minutes or more), or if you are a female smoker currently taking birth control hormones.

Any information obtained in connection with this study that can be identified with you will remain confidential. You will be assigned an ID number by which you will be referred to at all times. Your name will not be used for any purpose throughout this study. Only the principal and co-investigators will have access to files that contain information about you. Details that can identify you will not be printed in any report or publication about this study. Although every effort will be made to keep research records private, there may be times when federal or state law requires the disclosure of such records, including personal information. This is very unlikely, but if disclosure is ever required, Auburn University will take all steps allowable by law to protect the privacy of personal information.

In the event of personal injury resulting directly from these research procedures, financial compensation cannot be provided by Auburn University. In spite of all precautions, you might develop medical complications from participating in this study. You will be responsible for the cost of medical services. The Auburn University medical clinic is open from 8:00AM to 6:00PM, Monday thru Friday and 8:00AM to 12:00PM Saturday; otherwise you may see your private physician or arrange for other medical services.

You can withdraw from this study at any time, without penalty. If you withdraw you will still receive your compensation. The investigators also have the right to stop your participation at any time. If testing is stopped for any reason you will still receive your compensation. This could be because you have had an unexpected reaction, or have failed to follow instructions, or because the entire study has been stopped. If you are a student, your decision to terminate participation in the study will have no effect on your academic evaluation. Your decision whether or not to participate will not jeopardize your future relations with Auburn University or the Health and Human Performance Department.

You will receive $15.00 compensation for participation in this study. If for any reason you stop participating in this study or the study is stopped you will still receive your financial compensation. Additionally, you will be provided with participation verification to be used in a course in which the instructor provides credit for participating in research studies. Participation will not guarantee course credit because that is up to the each individual instructor's discretion. If this is pertinent to you, verify with your professor(s) before participating, if you withdraw from this study or if the study is stopped you will still receive verification of participation.

_____
Participant's Initials

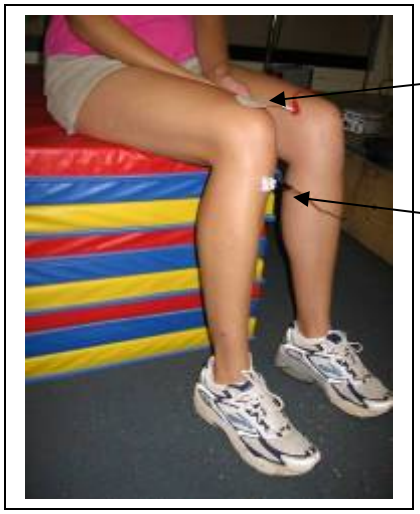

Image 1. Electrode placements on the front of the lower limb. Superior to the patella is one electrode for the Hoffman Reflex. Inferior to the knee, over the boney prominence of the tibia is the ground electrode for the EMG.

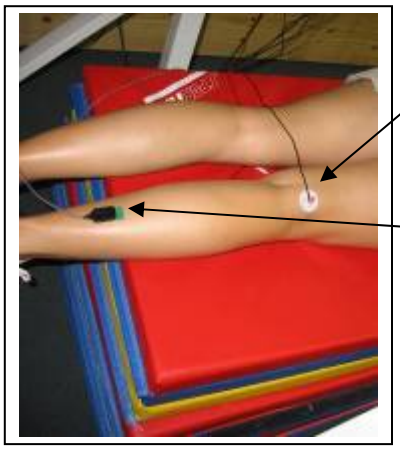

Image 2. Electrodes on the back of the lower limb. The Hoffman Reflex stimulus electrode is located on the back of knee. The soleus collection EMG electrode is located over the lower calf.

_____
Participant's Initials

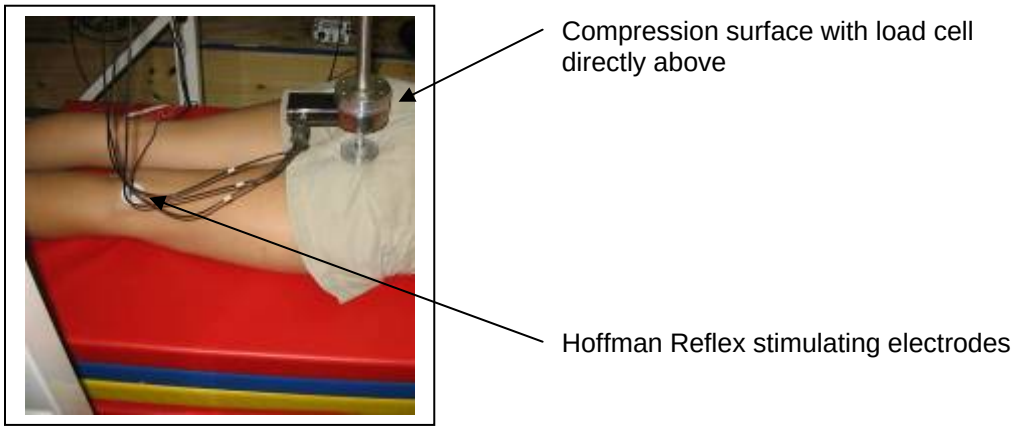

Image 3. Compression at the ischial tuberosity location. The compression surface is centered over the ischial tuberosity.

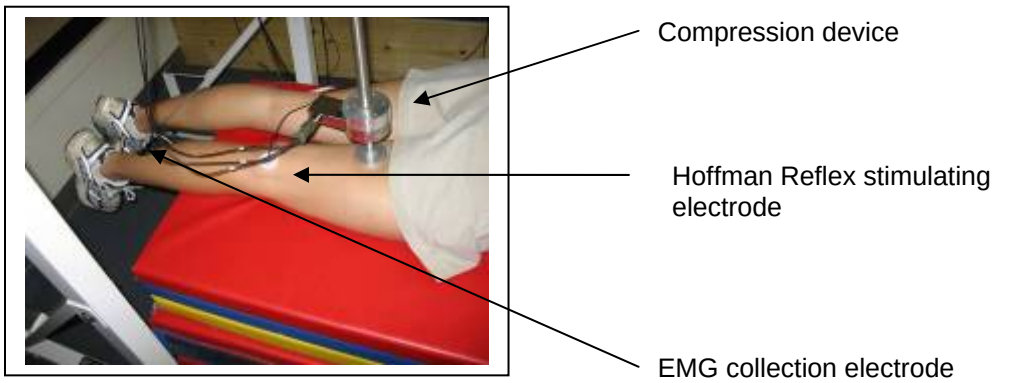

Image 4. Compression at the middle posterior thigh location. The contact surface is applied to the middle posterior thigh location in this figure. The middle posterior thigh is determined as the mid point between the greater trochanter and the lateral epicondylus and the medial and lateral edges of the thigh.

_____
Participant's Initials

You have the right to ask, and have answered, any questions you may have about this research. If you have further questions, or if a research-related injury occurs, please contact Paul St. Onge at (334) 844-1480 or by e-mail at stongep@auburn.edu, or Dr. Rudisill at (334) 844-1458 or by e-mail at rudisme@auburn.edu. You will be provided a copy of this form to keep for your records.

For more information regarding your rights as a research participant you may contact the Auburn University Office of Human Subjects Research or the Institutional Review Board by phone (334)-844-5966 or e-mail at hsubjec@auburn.edu or IRBChair@auburn.edu.

HAVING READ THE INFORMATION PROVIDED, YOU MUST DECIDE WHETHER OR NOT YOU WISH TO PARTICIPATE IN THIS RESEARCH STUDY. YOUR SIGNATURE INDICATES YOUR WILLINGNESS TO PARTICIPATE.

_____	_____
Participant's signature Date	Investigator obtaining consent Date
_____	_____
Print Name	Print Name

Appendix B—Recruitment Flyer

Research Study

With the Health and Human Performance Department: Musculoskeletal Research Laboratory located in Haley Center on Auburn University Campus, Auburn, AL

Study Title: EFFECT OF TISSUE COMPRESSION ON THE HOFFMANN REFLEX: COMPARISON BETWEEN THE ISCHIAL TUBEROSITY AND POSTERIOR THIGH

Have you ever sat in an extremely uncomfortable chair which caused your leg to fall asleep? Did you know that when your arm or leg falls asleep it is because of nerves and not blood flow? Experiencing pins and needles is not caused by cutting off blood flow rather they are a product of nerve function being altered.

Purpose

- The purpose of this research study is to develop a test method for assessing human/seat interface pressure on nerve function without causing the limb to fall asleep.
- Health screening will be used to determine eligibility to participate.

What you have to do during this study

- You will have to lie still on your stomach for 30 minutes
- Pressure similar to sitting will be applied to the buttocks and back of your thigh
- Your lower limb reflex will be tested during the application of different levels of pressure

What you get

- $15 for participating in this study
- Participating may provide extra credit with some instructors

Contact: Paul St.Onge at stongep@auburn.edu or call 334-844-1480

Appendix C—Recruitment Script

Study Title: EFFECT OF TISSUE COMPRESSION ON THE HOFFMANN REFLEX: COMPARISON BETWEEN THE ISCHIAL TUBEROSITY AND POSTERIOR THIGH

You are invited to participate in a research study to assess how the pressure of sitting impacts the nerve function of the lower limb. This study is being conducted by Paul St. Onge a graduate student under the supervision of Dr. Mary Rudisill, department chair of the Auburn University Health and Human Performance Department. We hope to learn how different pressure intensities and application locations impact nerve function.

If you decide to participate you will be required to visit the Musculoskeletal Research Laboratory twice. The laboratory is located in Haley Center on the first floor. During testing the nerve that controls the muscles of your calf, in your dominant leg, will be stimulated and monitored during two different conditions conducted on two separate days. Electromyography—EMG, and nerve stimulation electrodes will be attached to your skin. The EMG electrodes will be attached to your calf and the stimulation electrodes will be attached to the front and back of your knee. The nerve stimulation test uses a very brief (1 millisecond) weak electric pulse that has been described as a “carpet shock”. The result of the stimulation is a brief contraction of the calf muscles. While the stimulus may seem uncomfortable at first, most people quickly get used to the sensation. This is a safe procedure that is used frequently for medical screening and rehabilitation purposes. You will be given 20 small stimuli to allow you to get used to the stimulus. If you do not get used to the stimulus, or if you feel uncomfortable with the test at any time, the experience can be halted.

Once the 20 practice trials have been performed, you will lie on a padded surface facedown for approximately 30 minutes. At this point measurement will begin. An initial nerve test will be conducted 4 times. These 4 initial tests will use various stimulus levels to determine the strongest your calf muscle can contract. Due to the short duration of the stimulus, you will most likely not be able to determine a difference between each stimulus level but may notice a more vigorous calf contraction. When this test is complete, pressure will be applied to one of two locations. The maximum pressure applied is similar to that experienced while sitting on a firm surface such as a folding chair or a bleacher seat.

One pressure intensity will be applied to one location one day and upon returning to the laboratory a second time, the same pressure will be applied to the second location. The two different locations are the ischial tuberosity and the middle of the posterior thigh. The ischial tuberosity, is also known as the sit bone and is located in the buttocks. The middle thigh will be determined by measuring the length and width of your thigh.

While the pressure is applied a series of nerve tests will be conducted using a stimulus determined from the initial test. The stimulus will be given 3 times at the beginning of each minute, at approximately 10 second intervals. Additionally, blood oxygenation and flow to your leg will be monitored continuously and you will be asked to report your level of comfort each minute. Blood oxygenation will be measured at the toe by a pulse oximeter. Blood flow will be monitored by a thermo imagery camera that will detect changes in skin temperature. There will also be a test of skin sensation every few minutes in which you will be asked to report if you feel one or two items touching your leg. If at any point the pressure applied to your leg becomes too uncomfortable or painful or if your blood flow is interrupted, the pressure will be released. Also, if you do not want the pressure to remain applied, or if you feel uncomfortable with the test at any time, the experience can be stopped. If a test is stopped for any reason you will still receive compensation.

After the pressure is released, the nerve test will continue for a few more minutes. Additionally, your level of comfort, blood oxygenation and flow, and skin sensation will continue to be monitored. This same procedure will be repeated during the second visit to the laboratory.

At the end of all testing, electrodes will be removed and you will be asked to respond to follow-up questions 24 and 48 hours later. These questions will ask you to name and describe all bruising, discomfort, pain, or other sensation you are experiencing related to this study.

You will spend approximately two hours in the lab for all procedures to be conducted. Responding to the follow-up questions will take you approximately five minutes for the two days following each visit to the laboratory.

You should not participate in this study if you are not in general good health, have sustained an injury to either your right or left legs in the past 6 months, if you have any chronic peripheral nervous system or vascular or bleeding problems, or if you are not physically active (exercise at least 3 times per week for 20 minutes or more), or if you a female smoker currently taking birth control hormones.

You will receive $15.00 compensation for participation in this study. If for any reason you stop participating in this study or the study is stopped you will still receive your financial compensation. Additionally, you will be provided with participation verification to be used in a course in which the instructor provides credit for participating in research studies. Participation will not guarantee course credit because that is up to the each individual instructor's discretion. If this is pertinent to you, verify with your professor(s) before participating, if you withdraw from this study or if the study is stopped you will still receive verification of participation.

The inclusion criteria for this are the following:

1) be in general good health;
2) be free of injury in both the right and left lower limb for 6 months prior to participating;
3) have no history of chronic injury to either right or left lower limb;
4) have no history of surgery in either the right or left lower limb;
5) have no prior history of neurological and/or vascular disorder;
6) have no history of vascular disorder including chronic high (>220/110) or low blood (<90/60) pressure;
7) have no bleeding disorders;
8) demonstrate a measurable Hoffmann Reflex;
9) have a BMI less than 30;
10) if woman, not be a smoker taking birth control hormones—for this provides elevated risk for blood clots; and
11) be physically active. Physical activity will be defined as a minimum of 3 times per week for at least 20 minutes per exercise bout.

If you are interested in participating in this study please email Paul St. Onge at stongep@auburn.edu.

He will respond to your email with scheduling information and will answer any additional questions you may have.

FOLLOW-UP EMAIL FOR RECRUITMENT

Date DD,MM,2006

Hello,

Thank you for considering participating in the research study titled: Influence of tissue load location on nerve function: Implications for seat design. This study is being conducted by Paul St.Onge, a graduate student in the Health and Human Performance department, under the advisement of Dr. Mary Rudisill, acting department head for Health and Human Performance.

This study will require two separate visits the Musculoskeletal Research Laboratory located on ground floor of Haley center. These two visits must be at least two day apart and each will last about 1 hour.

If you decide to participate, when you come to the laboratory please bring a properly fitting sized pair spandex type athletic shorts to wear during testing. If you do not bring a set of shorts, a clean, appropriate sized pair will be available for you to use.

Additionally, after both testing session you will be asked to respond to a set of follow-up questions by email one and two days after each test session..

Currently available dates and times are:

Date Times

Dates and times will be placed here when established.

Please respond to this email with a date and time you would like to visit the laboratory for your first visit.

If you have any further questions pertaining to this study please respond to this email.

Sincerely,

Paul St.Onge
Graduate Student HHP
stongep@auburn.edu

Appendix D—Health Screening

HEALTH SCREENING

Auburn University Musculoskeletal Research Laboratory

Study:
EFFECT OF TISSUE COMPRESSION ON THE HOFFMANN REFLEX: COMPARISON BETWEEN THE ISCHIAL TUBEROSITY AND POSTERIOR THIGH

Principal Investigator
Paul St. Onge
stongep@auburn.edu

Name _____ Age: _____
Email _____
Phone number __(____)_____

Emergency contact
Name _____
Phone number __(____)_____

Health history related to the present study

		YES	NO
1.	Have you been diagnosed with a chronic peripheral nervous system illness? Such as neuropathy or multiple sclerosis or restless leg syndrome?	_____	_____
2.	Have you recently been diagnosed with a peripheral nervous system illness? Such as Saturday Night Palsy or Sciatica?	_____	_____
3.	In the past 6 months have you been diagnosed with a peripheral nervous system illness or disorder?	_____	_____
4.	Are you current taking any medication for a peripheral nervous system disorder? Not including antidepressants, anti-anxiety, or hyperactivity medications.	_____	_____
5.	Have you been diagnosed with a chronic vascular system condition? Such as high or low blood pressure, blood clots, or hemophilia.	_____	_____

		YES	NO
6.	Are you currently living with a chronic illness or medical condition? Such as diabetes, cancer, arthritis, or heart disease.	_____	_____
7.	In the past 6 months have you been diagnosed with a vascular system illness, condition, or disorder?	_____	_____
8.	Are you currently taking medications for any blood disorders?	_____	_____
9.	Are you currently experiencing pain in the either leg or hip?	_____	_____
10.	Have you had injury to or surgery on either leg in the past 6 months?	_____	_____
11.	Do you bruise easily?	_____	_____
12.	Women: Are you a smoker currently taking birth control hormones?	_____	_____
13.	Is there any reason you should not be included in this study?	_____	_____

Height _____ Weight _____ Calculated BMI* _____
* (BMI =Weight [in kilograms] / (Height [in meters])^2

Physical Activity

How many days a week do you engage in purposeful physical activity?

On average how long do you spend engaged in purposeful physical activity per session?

What type of purposeful physical activities do you engage?

According the Surgeon General of the United States, all Americans should engage in at least 30 minutes of moderate physical activity daily. These activities can include, but are not limited to, walking, cleaning the house, yard work, washing the car, or traditional exercises. Do you engage in at least 30 minutes of moderate physical activity daily?

Yes_____ No _____

Appendix E—Anthropomorphic Information

ANTHROPOMORPHIC INFORMATION

Auburn University Musculoskeletal Research Laboratory

Study
EFFECT OF TISSUE COMPRESSION ON THE HOFFMANN REFLEX: COMPARISON BETWEEN THE ISCHIAL TUBEROSITY AND POSTERIOR THIGH

Principal Investigator
Paul St. Onge

Leg used to kick a ball or manipulate object _____

	1	2	3	AVG
Height	_____			
Weight	_____			
Hip Circumference	_____	_____	_____	_____
Right Leg length	_____	_____	_____	_____
Right Thigh Length	_____	_____	_____	_____
Right Lower Limb Length	_____	_____	_____	_____
Right Mid point of Thigh Circumference	_____	_____	_____	_____
Right Length from lateral to medial side of thigh	_____			
Left Leg length	_____	_____	_____	_____
Left Thigh Length	_____	_____	_____	_____
Left Lower Limb Length	_____	_____	_____	_____
Left Mid point of Thigh Circumference	_____	_____	_____	_____
Left Length from lateral to medial side of thigh	_____			

Appendix F—Procedural and Data Collection Materials

PROCEDURAL AND DATA COLLECTION MATERIALS

Auburn University Musculoskeletal Research Laboratory

Study
EFFECT OF TISSUE COMPRESSION ON THE HOFFMANN REFLEX: COMPARISON BETWEEN THE ISCHIAL TUBEROSITY AND POSTERIOR THIGH

Principal Investigator
Paul St. Onge

Date _____
Start Time _____

Researchers present
_____

_____

Testing Procedures
Blood Pressure
____/____

EMG
____ Attach ground electrode
____ Attach Electrodes to soleus of right leg
____ Check for placement
____ Check for crosstalk without muscles

Hoffmann Reflex
____ Attach 4 cm diameter dispersive electrode will be placed on the anterior thigh just superior to the patella over the quadriceps tendon
____ Attach 0.80 cm diameter stimulating electrode in the popliteal fossa
____ Test electrode placement
____ Position participant in prone position

Sweep 1

Number	Stimulus Intensity	H-Wave		M-Wave	
		Latency	Amplitude	Latency	Amplitude
1					
2					
3					
4					
5					
6					
7					
8					
9					
10					
11					
12					
13					
14					
15					
16					
17					
18					
19					
20					

H-max _____

M-max _____

Sweep 2

Number	Stimulus Intensity	H-Wave		M-Wave	
		Latency	Amplitude	Latency	Amplitude
1					
2					
3					
4					
5					
6					
7					
8					
9					
10					
11					
12					
13					
14					
15					
16					
17					
18					
19					
20					

H-max _____

M-max _____

Sweep 3

Number	Stimulus Intensity	H-Wave		M-Wave	
		Latency	Amplitude	Latency	Amplitude
1					
2					
3					
4					
5					
6					
7					
8					
9					
10					
11					
12					
13					
14					
15					
16					
17					
18					
19					
20					

H-max _____

M-max _____

Sweep 4

Number	Stimulus Intensity	H-Wave		M-Wave	
		Latency	Amplitude	Latency	Amplitude
1					
2					
3					
4					
5					
6					
7					
8					
9					
10					
11					
12					
13					
14					
15					
16					
17					
18					
19					
20					

H-max _____

M-max _____

1 H-max _____
2 H-max _____
3 H-max _____
4 H-max _____

Average H-max _____
50% H-max Average _____

Compression condition _____ **Intensity** _____
Location _____

_____ Set stimulator unit to stimulus necessary to produce 50% H-max
_____ Apply pulse oximeter **Start Time**
_____ Activate Thermography system _____
_____ Apply tissue load to appropriate location **End Time**
_____ Value applied in mmHg _____
_____ Verify proper pressure on computer monitor from load cell

Minute		H-Wave		M-wave		Subjective Comfort	Therm blood flow	Pulse O2	2-point
		Latency	Amplitude	Latency	Amplitude				
1	a								
	b								
	c								
2	a								
	b								
	c								
3	a								
	b								
	c								
4	a								
	b								
	c								
5	a								
	b								
	c								
6	a								
	b								
	c								
7	a								
	b								
	c								
8	a								
	b								
	c								
9	a								
	b								
	c								

Minute		H-Wave		M-wave		Subjective Comfort	Therm blood flow	Pulse O2	2-point
		Latency	Amplitude	Latency	Amplitude				
10	a								
	b								
	c								
11	a								
	b								
	c								
12	a								
	b								
	c								
13	a								
	b								
	c								
14	a								
	b								
	c								
15	a								
	b								
	c								
16	a								
	b								
	c								
17	a								
	b								
	c								
18	a								
	b								
	c								

Minute		H-Wave		M-wave		Subjective Comfort	Therm blood flow	Pulse O2	2-point
		Latency	Amplitude	Latency	Amplitude				
19	a								
	b								
	c								
20	a								
	b								
	c								
21	a								
	b								
	c								
22	a								
	b								
	c								
23	a								
	b								
	c								
24	a								
	b								
	c								
25	a								
	b								
	c								
26	a								
	b								
	c								
27	a								
	b								
	c								

Minute		H-Wave		M-wave		Subjective Comfort	Therm blood flow	Pulse O2	2-point
		Latency	Amplitude	Latency	Amplitude				
	a								
	b								
	c								
	a								
	b								
	c								
	a								
	b								
	c								
	a								
	b								
	c								
	a								
	b								
	c								
	a								
	b								
	c								
	a								
	b								
	c								

End of testing when 50% H-max latency and amplitude return to baseline
_____ Time of day baseline returned

_____ **End of Test**

_____**Minute Compression was Applied**
_____**Minute Compression was Released**

Notes:

Appendix G—Subjective Comfort Scale

SUBJECTIVE COMFORT SCALE

Auburn University Musculoskeletal Research Laboratory

Study
EFFECT OF TISSUE COMPRESSION ON THE HOFFMANN REFLEX: COMPARISON BETWEEN THE ISCHIAL TUBEROSITY AND POSTERIOR THIGH

Subjective Comfort Scale

10 Extremely Comfortable

9

8

7 Comfortable

6

5 Neutral: Neither comfortable nor uncomfortable

4

3 Extremely Uncomfortable

2

1 Pain and Distress

Appendix H—Follow-up Questions

FOLLOW-UP QUESTIONS

Thank you for your participation in study: Influence of tissue load location on nerve function: Implications for seat design. To finish this study please respond to this email by answering the following questions. If you have further question, comments, suggestions please do not hesitate to contact me immediately.

Please answer the following questions with as much detail necessary.

1. Are you experiencing bruising at any of the four application locations?
If yes, please describe the extent of discoloration, the size of the bruising, and how tender the area is to touch.

2. Are you experiencing muscle stiffness at any of the four application locations?
If yes, please describe the magnitude of the stiffness.

3. Are you experiencing pain at any of the four application locations?
If yes, please describe the sensation.

4. Are you experiencing burning, tingling, or numbness in either of your lower limbs?
If yes, please describe the location of the sensation and the sensation itself.

5. Have you noticed any discomfort or other undesirable sensations from your participation in this study?

6. If you have experienced any of the above, what have you done to relieve the discomfort?

Thank you for your participation in this study,
Paul St.Onge
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334-844-1480

Appendix I—Raw Data Used in Analyses

	PART # 01 Male	VISIT 1	Hmax 6.5	Location Ischial	Tuberosity		Total Displ 5.9	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**	
B	1	2.515	52.410	33	99	10	42	
	2	4.150		33				
	3	3.555		33				
A	**APP**		**APP**			**APP**		
9	1	3.960	65.128	33	100	10	45	
	2	4.326		33				
	3	4.414		33				
R	**RELE**		**RELE**			**RELE**		
10	1	3.828	61.077	33	100	10	44	
	2	3.936		33				
	3	4.146		33				
15	1	4.419	65.231	33	100	10	37	
	2	4.346		33				
	3	3.955		33				
20	1	4.302	70.210	33	100	10	35	
	2	5.151		33				
	3	4.238		33				

	PART # 01 Male	VISIT 2	H_{max} 3.16	Location Middle	Thigh		Total Displ 5.1	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**	
B	1	1.416	52.437	33	100	10	20	
	2	2.046		33				
	3	1.509		33				
R	**APP**		**APP**			**APP**		
9	1	1.943	58.713	33	100	6	15	
	2	1.694		33				
	3	1.929		33				
R	**RELE**		**RELE**			**RELE**		
10	1	2.627	67.173	33	100	6	23	
	2	1.934		33				
	3	1.807		33				
15	1	1.641	57.078	33	100	10	27	
	2	2.017		33				
	3	1.753		33				
20	1	0.186	51.867	33	100	10	31	
	2	1.958		33				
	3	2.773		31				

	PART # 02 Female	VISIT 2	Hmax 2.84	Location Ischial	Tuberosity		Total Displ 5.3	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**	
B	1	0.122	24.754	29	99	10	20	
	2	0.698		28				
	3	1.289		29				
A	**APP**		**APP**			**APP**		
9	1	1.802	70.728	29	99	8	35	
	2	1.411		29				
	3	2.813		28				
R	**RELE**		**RELE**			**RELE**		
10	1	1.260	63.838	29	99	9	28	
	2	1.733		29				
	3	2.446		28				
15	1	1.475	69.871	29	99	9.5	22	
	2	2.251		29				
	3			29				
20	1	0.269	27.406	29	99	10	22	
	2	1.060		29				
	3	1.006		29				

	PART # 02 Female	VISIT 1	Hmax 2.18	Location Middle	Thigh			Total Displ 5.0	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	1.538	47.154	29	99	7	25		
	2	1.377		29					
	3	0.169		29					
A	**APP**		**APP**			**APP**			
9	1	2.012	87.645	29	99	6	21		
	2	1.694		30					
	3	2.026		29					
R	**RELE**		**RELE**			**RELE**			
10	1	1.655	62.997	29	98	6	24		
	2	1.108		29					
	3	1.357		29					
15	1	0.688	27.691	30	99	8.5	25		
	2	0.718		30					
	3	0.405		29					
20	1	1.230	48.593	29	99	10	20		
	2	0.952		29					
	3	0.996		29					

	PART # 03 Male	VISIT 2	Hmax 2.32	Location Ischial	Tuberosity		Total Displ 5.0	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**	
B	1	1.821	77.586	34	99	10	25	
	2	1.733		35				
	3	1.846		34				
A	**APP**		**APP**			**APP**		
9	1	2.402	91.552	35	98	10	30	
	2	1.885		35				
	3	2.085		31				
R	**RELE**		**RELE**			**RELE**		
10	1	1.812	81.868	35	100	10	32	
	2	1.855		34				
	3	2.031		34				
15	1	2.090	90.647	34	99	10	47	
	2	2.305		34				
	3	1.914		34				
20	1	2.202	85.934	34	99	10	44	
	2	1.909		34				
	3	1.870		34				

	PART # 03 Male	VISIT 1	Hmax 4.84	Location Middle	Thigh			Total Displ 4.8	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	2.236	44.920	34	100	10	41		
	2	2.134		35					
	3	2.153		35					
A	**APP**		**APP**			**APP**			
9	1	3.408	70.985	34	100	8	57		
	2	3.408		34					
	3	3.491		31					
R	**RELE**		**RELE**			**RELE**			
10	1	2.622	50.544	34	98	10	56		
	2	2.368		34					
	3	2.349		34					
15	1	1.963	36.963	34	99	10	57		
	2	1.685		33					
	3	1.719		33					
20	1	1.597	33.636	34	99	10	61		
	2	1.851		35					
	3	1.436		34					

	PART # 04 Male	VISIT 1	Hmax 4.08	Location Ischial	Tuberosity			Total Displ 5.9	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	2.378	54.730	30	99	10	60		
	2	2.119		31					
	3	2.202		31					
A	**APP**		**APP**			**APP**			
9	1	2.407	56.291	31	99	9	33		
	2	2.344		31					
	3	2.139		31					
R	**RELE**		**RELE**			**RELE**			
10	1	1.841	53.815	31	99	9	35		
	2	2.305		31					
	3	2.441		31					
15	1	2.539	64.028	31	99	10	41		
	2	2.583		31					
	3	2.715		31					
20	1	2.646	52.688	31	99	10	31		
	2	1.279		31					
	3	2.524		31					

	PART # 04 Male	VISIT 2	Hmax 3.28	Location Middle	Thigh			Total Displ 4.3	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	0.894	40.447	31	97	10	33		
	2	0.913		31					
	3	2.173		32					
A	**APP**		**APP**			**APP**			
9	1	1.279	37.754	30	99	8	na		
	2	1.064		31					
	3	1.372		31					
R	**RELE**		**RELE**			**RELE**			
10	1	1.572	57.104	31	98	8	na		
	2	2.192		32					
	3	1.855		31					
15	1	2.275	60.783	31	98	10	na		
	2	1.909		31					
	3	1.797		31					
20	1	0.410	19.004	31	98	10	na		
	2	0.898		31					
	3	0.562		32					

	PART # 05 Female	VISIT 1	Hmax 3.76	Location Ischial	Tuberosity		Total Displ 5.5	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**	
B	1	2.773	63.236	30	100	10	51	
	2	2.075		30				
	3	2.285		30				
A	**APP**		**APP**			**APP**		
9	1	3.027	79.867	30	98	9	35	
	2	2.925		30				
	3	3.057		30				
R	**RELE**		**RELE**			**RELE**		
10	1	1.602	62.287	30	98	9	35	
	2	2.319		30				
	3	3.105		31				
15	1	1.240	48.005	30	100	10	35	
	2	1.875		30				
	3	2.300		30				
20	1	2.803	79.566	30	98	10	35	
	2	3.330		30				
	3	2.842		31				

	PART # 05 Female	VISIT 2	Hmax 4.56	Location Middle	Thigh			Total Displ 4.4	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	3.354	77.595	32	99	10	21		
	2	3.628		32					
	3	3.633		32					
A	**APP**		**APP**			**APP**			
9	1	2.598	69.744	32	100	9	20		
	2	3.276		32					
	3	3.667		32					
R	**RELE**		**RELE**			**RELE**			
10	1	1.396	31.908	32	100	10	21		
	2	1.499		33					
	3	1.470		33					
15	1	0.820	40.439	33	100	10	23		
	2	2.813		32					
	3	1.899		32					
20	1	0.679	49.898	32	100	10	14		
	2	3.789		32					
	3	2.358		32					

	PART # 06 Female	VISIT 2	Hmax 2.88	Location Ischial	Tuberosity		Total Displ 4.2	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**	
B	1	1.226	44.734	33	99	10	130	
	2	1.309		32				
	3	1.330		33				
A	**APP**		**APP**			**APP**		
9	1	2.095	61.944	33	100	7	87	
	2	1.509		33				
	3	1.748		33				
R	**RELE**		**RELE**			**RELE**		
10	1	1.289	53.912	33	100	7	86	
	2	1.709		32				
	3	1.660		32				
15	1	1.123	45.833	33	98	9	85	
	2	1.162		33				
	3	1.675		33				
20	1	2.070	59.167	33	99	9	87	
	2	1.421		32				
	3	1.621		32				

	PART # 06 Female	VISIT 1	Hmax 3.01	Location Middle	Thigh			Total Displ 4.8	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	1.768	47.367	30	100	10	42		
	2	1.260		30					
	3	1.235		30					
A	**APP**		**APP**			**APP**			
9	1	0.371	20.556	31	99	8	34		
	2	0.776		31					
	3	0.703		30					
R	**RELE**		**RELE**			**RELE**			
10	1	0.996	35.156	30	99	9	44		
	2	1.045		30					
	3	1.123		30					
15	1	1.772	55.389	29	100	9	45		
	2	1.250		30					
	3	1.963		30					
20		1.948	59.840	30	100	9	43		
		1.641		30					
		1.797		30					

	PART # 07 Male	VISIT 2	Hmax 4.8	Location Ischial	Tuberosity			Total Displ 5.6	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	2.920	62.542	32	97	10	30		
	2	3.288		33					
	3	2.798		33					
A	**APP**		**APP**			**APP**			
9	1	3.843	86.771	33	98	7	35		
	2	4.126		33					
	3	4.526		33					
R	**RELE**		**RELE**			**RELE**			
10	1	3.818	81.007	32	97	7	30		
	2	3.965		33					
	3	3.882		32					
15	1	3.501	79.347	32	98	10	28		
	2	3.931		32					
	3	3.994		32					
20	1	4.277	89.854	33	98	10	30		
	2	4.453		33					
	3	4.209		32					

	PART # 07 Male	VISIT 1	Hmax 4.64	Location Middle	Thigh			Total Displ 6.2	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	2.847	58.405	31	100	10	30		
	2	3.203		31					
	3	2.080		31					
A	**APP**		**APP**			**APP**			
9	1	3.970	81.588	31	100	8	27		
	2	3.495		31					
	3	3.892		31					
R	**RELE**		**RELE**			**RELE**			
10	1	4.229	89.454	31	100	8	32		
	2	4.092		31					
	3	4.131		31					
15	1	3.545	81.164	30	99	9	31		
	2	3.857		31					
	3	3.896		31					
20	1	4.019	86.121	30	100	9	40		
	2	4.048		31					
	3	3.921		30					

	PART # 08 Male	VISIT 1	Hmax 5	Location Ischial	Tuberosity		Total Displ 6.5	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**	
B	1	1.230	23.467	32	99	10	27	
	2	1.919		32				
	3	0.371		31				
A	**APP**		**APP**			**APP**		
9	1	4.370	84.440	31	100	5	26	
	2	4.297		32				
	3	3.999		31				
R	**RELE**		**RELE**			**RELE**		
10	1	3.799	85.513	31		9	20	
	2	4.375		31				
	3	4.653		31				
15	1	3.081	61.780	31		10	18	
	2	2.407		32				
	3	3.779		31				
20	1	4.331	80.600	31		10	15	
	2	3.828		32				
	3	3.931		32				

	PART # 08 Male	VISIT 2	Hmax 4.8	Location Middle	Thigh			Total Displ 4.5	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	2.192	43.056	30	100	10	30		
	2	1.318		30					
	3	2.690		31					
A	**APP**		**APP**			**APP**			
9	1	4.351	82.604	31	100	5	28		
	2	3.604		31					
	3	3.940		31					
R	**RELE**		**RELE**			**RELE**			
10	1	0.728	23.396	31	99	4	30		
	2	0.981		30					
	3	1.660		31					
15	1	2.075	44.319	31	99	10	32		
	2	1.704		31					
	3	2.603		31					
20	1	2.974	53.174	31	98	10	35		
	2	1.553		31					
	3	3.130		31					

	PART # 09 Female	VISIT 2	Hmax 3.24	Location Ischial	Tuberosity		Total Displ 5.7	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**	
B	1	2.427	80.031	29	100	10	40	
	2	2.554		29				
	3	2.798		29				
A	**APP**		**APP**			**APP**		
9	1	2.964	89.825	29	100	6	30	
	2	2.847		29				
	3	2.920		29				
R	**RELE**		**RELE**			**RELE**		
10	1	1.284	52.593	29	100	8	30	
	2	1.128		29				
	3	2.700		29				
15	1	1.533	71.327	29	99	9	28	
	2	2.480		30				
	3	2.920		29				
20	1	2.612	84.691	29	100	9	30	
	2	2.881		29				
	3	2.739		29				

	PART # 09 Female	VISIT 1	Hmax 3.6	Location Middle	Thigh			Total Displ 5.2	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	2.559	69.352	29	99	9	35		
	2	2.490		29					
	3	2.441		29					
A	**APP**		**APP**			**APP**			
9	1	2.988	78.759	30	98	5	20		
	2	3.076		30					
	3	2.998		29					
R	**RELE**		**RELE**			**RELE**			
10	1	2.500	83.907	29	99	6	21		
	2	2.061		29					
	3	2.695		29					
15	1	2.485	73.097	29	99	9	22		
	2	2.778		29					
	3	na		na					
20	1	2.979	78.407	29	99		29		
	2	2.847		29					
	3	2.642		29					

	PART # 10 Male	VISIT 1	Hmax 5.84	Location Ischial	Tuberosity			Total Displ 6.9	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	2.627	45.987	35	98	10	31		
	2	2.539		35					
	3	2.891		35					
A	**APP**		**APP**			**APP**			
9	1	3.627	47.123	35	99	7	22		
	2	1.890		35					
	3	2.739		35					
R	**RELE**		**RELE**			**RELE**			
10	1	2.529	33.973	35	98	8	22		
	2	2.344		35					
	3	1.079		35					
15	1	2.783	51.558	35	98	10	22		
	2	2.886		35					
	3	3.364		36					
20	1	2.783	48.967	35	98	10	30		
	2	3.042		35					
	3	2.754		36					

	PART # 10 Male	VISIT 2	Hmax 5.64	Location Middle	Thigh			Total Displ 3.8	cm
Min	**Pulse#**	**H-wave Amp**	**AVG %Hmax**	**H-wave Latency**	**Pulse O2 Sat**	**Sub Comf**	**Two-Point**		
B	1	3.110	43.428	34	97	10	22		
	2	1.816		35					
	3	2.422		34					
A	**APP**		**APP**			**APP**			
9	1	4.453	73.818	34	98	7	11		
	2	3.760		34					
	3	4.277		34					
R	**RELE**		**RELE**			**RELE**			
10	1	1.001	20.431	34	98	9	11		
	2	0.835		34					
	3	1.621		35					
15	1	2.495	41.903	35	97	10	12		
	2	2.251		35					
	3	2.344		35					
20	1	3.384	62.074	35	97	10	11		
	2	3.882		35					
	3	3.237		35					