THE PENNSYLVANIA STATE UNIVERSITY
SCHREYER HONORS COLLEGE

DEPARTMENT OF MECHANICAL AND NUCLEAR ENGINEERING

BLOOD PRESSURE AND ITS EFFECTS ON MUSCLE STIFFNESS MEASUREMENTS THROUGH ULTRASOUND ELASTOGRAPHY

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SPRING 2018

A thesis
submitted in partial fulfillment
of the requirements
for a baccalaureate degree
in Biomedical Engineering
with honors in Biomedical Engineering

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ABSTRACT

Acute compartment syndrome (ACS) is the increase of intramuscular pressure within a compartment. This condition limits blood flow to the compartment, which can permanently damage the limb if not treated. ACS is often diagnosed through a combination of clinical signs. If multiple symptoms related to the condition exist, the diagnosis is confirmed by measuring intramuscular pressure (IMP). The current methods for measuring IMP are invasive and can suffer from inaccuracies, making it not ideal for diagnosing compartment syndrome. The objective of this study is to determine if measuring muscle stiffness with shear wave elastography can confirm the change in intramuscular pressure. To do this, the experiments test the viability of our procedure for inducing compartment syndrome for a short period of time. The experiments also test our method on its viability for diagnosing the syndrome. The first experiment measures muscle stiffness using shear wave elastography at different leg elevations. The first experiment also measures muscle stiffness when applying different amounts of pressure to the patient's thigh. Changing leg elevation affects the blood pressure of the patient's leg, which we will test to see if muscle stiffness also changes as a result. Furthermore, we will test if changing the pressure applied to the patient's thigh will change the muscle stiffness in the leg. The second experiment tests if our methods for applying pressure to the patient's thigh will cause an increase in intramuscular pressure. The final experiment tests if increasing intramuscular pressure through saline injection can be differentiated by measuring muscle elasticity. We predict that both methods for simulating compartment syndrome will result in an increase in intramuscular pressure, which can be diagnosed by measuring muscular stiffness. If confirmed, muscle stiffness could potentially be used as a non-invasive marker for predicting intramuscular pressure.

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ACKNOWLEDGEMENTS

I would like to thank Dr. Daniel Cortes for allowing me to study and complete research in his lab. I could not have developed this thesis without the help of Dr. Cortes, Ali Sadeghi, Che-Yu Lin, and every other student in the Biomechanics and Imaging Lab. Their guidance throughout every step of the process helped me develop into the student I am today. I am very thankful to be a part of such an incredible lab. Additional thanks goes to my honors advisor, Dr. Nanyin Zhang, for helping me develop my thesis. Furthermore, thank you to Dr. Meghan Vidt for her assistance as a reader on my thesis committee.

Thank you to all of the incredible people who have touched my life and helped me grow into the person I am today. Thank you to my family for supporting me throughout the process and always encouraging me to be the best that I can be. Thank you to Marielle Ravally, Jamie Letender, Anuja Jonnalagadda, and the rest of the 2017 Homecoming Executive Committee for their constant support throughout the year and for being some of my best friends. Thank you to the 2017 Homecoming Merchandise Committee for keeping me sane during the busiest days of my life and for trusting in me as a leader. Thank you to the National Student Leadership Conference for teaching me how to be a great role model and for teaching me that I can still function with less than four hours of sleep. Thank you to Stacia Hollingsworth, Brienna Phillips, Dana Kyle, Alex Erdman, Jillian Susi, Phil Crompton, and Michael Kaplan for trusting in me, teaching me, and giving me the opportunity to make a difference in the lives of those around me. You all have shaped me into who I am today and this thesis would not have been possible without your help.

Chapter 1

Introduction

The human leg consists of numerous compartments to separate tissues from one another. Compartments consist of muscles, nerves, and blood vessels all surrounded by a membrane called fascia [1]. Fascia surrounds and goes through every structure in the body [2]. The purpose of fascia is to keep the tissues in place. Therefore, fascia is strong and does not stretch very much.

Compartment syndrome is a medical condition characterized by the increase in pressure within one of the body's compartments. Since the fascia does not stretch much, the increase in pressure within the compartment applies pressure to the capillaries carrying blood, thus limiting blood flow to the muscles. The increase in pressure occurs either through a reduction of volume within the compartment, or through an increase of debris and other matter within the compartment to make it expand [3]. When the pressure inside the compartment is elevated, blood flow through the capillaries slows down. If the blood flow to the compartment is limited enough, then the tissue will begin to swell. Edema, or the swelling of the tissue in a limb due to the accumulation of fluids, is painful and dangerous. If it continues for too long, blood flow will reach a level not sufficient to sustain cellular life, otherwise known as ischemia. The rate of ischemia increases with both time and pressure [4]. Too much ischemia will result in irreversible damage to the limb from cell death or necrosis.

Compartment syndrome can be caused by a one-time incident or by an ongoing overuse of the limb [5]. Acute cases of compartment syndrome can occur from a variety of reasons. Tight bandages, lying on the limb for an extended period, and other unnatural stresses on the limb can cause a decrease in compartment volume [6]. Any restriction to tissue expansion during swelling can also cause compartment syndrome to occur. Even with expansion of the compartment, a major increase in compartment pressure can also cause acute compartment syndrome. Bone fractures are the most common causes of acute

compartment syndrome. Arterial injuries are often seen in patients with acute compartment syndrome caused by severe trauma [7]. Intracompartmental pressure is elevated due to tissue swelling and haematoma secondary to fractures. While fracture may occasionally tear the fascia surrounding the compartment, the tears do not cause significant decompression of the compartment. Therefore, open and closed fractures should be closely monitored for their potential to develop into acute compartment syndrome.

Compartment syndrome can also be a chronic condition. Usually associated with athletics, chronic exertional compartment syndrome is muscular expansion that occurs during exercise [8]. Pain is the main symptom associated with this condition, but usually settles after exercise. For those who want to exercise without pain, the condition is best healed through the surgical release of the compartments, which is known as fasciotomy. This operation requires a long recovery period of little to no exercise, making the operation not ideal for most athletes with the condition.

Diagnosing and Treating Compartment Syndrome

Acute compartment syndrome is diagnosed by measuring the intramuscular pressure of the compartment and comparing its value with presence of symptoms [9]. However, measuring intramuscular pressure is an invasive process, which creates more risks and causes discomfort to the patient. Intramuscular pressure is measured continuously by inserting a catheter into the muscle [10]. There are many methods for performing this technique. However, many of the techniques do not produce accurate results, or are limited to certain types of studies (only patients at rest, only patients in a certain position, etc.) For example, a handheld needle manometer can be used for the measurement, but it requires saline injection to function continuously [10]. This could generate an increased pressure measurement in compartment and augment the compartment syndrome as a result. Therefore, measuring intramuscular pressure alone is not a sufficient way to diagnose acute compartment syndrome.

Another method used to determine if a patient has acute compartment syndrome is through clinical evaluation. Patients with acute compartment syndrome usually experience the five “P’s”, which are pain, paresthesia, pallor, paralysis, and high intramuscular pressure [11]. Paresthesia and pain occur early in most cases, but pain can be absent in later stages. However, the presence of all five symptoms at once usually does not occur until later stages. This can be bad because action should be taken early to ensure the patient does not undergo unnecessary ischemia due to action being taken too late. Furthermore, confirmation of these symptoms often requires the patient to communicate the presence of the symptoms, which is not possible in cases where the patient is unconscious. Therefore, waiting for confirmation of all symptoms may not be possible in many scenarios, and may lead to further damage to the patient than necessary.

When intramuscular pressure becomes too great and starts causing ischemia, a procedure known as fasciotomy is done. Fasciotomies release the intramuscular pressure of the compartments by surgically opening the fascia surrounding the compartment. If the condition is diagnosed and treated within 6 hours of occurring, it isn’t likely to cause overall functional impairment [12]. This process is currently the only effective way to treat acute compartment syndrome [13].

Muscle Stiffness and Acute Compartment Syndrome

Current methods for diagnosing acute compartment syndrome are often inaccurate and dangerous. In fact, a lack of symptoms is more useful for ruling out acute compartment syndrome than having symptoms is for diagnosing it [14]. Therefore, many methods have been developed to determine if there is a safer, noninvasive way to measure acute compartment syndrome. One of these methods involves testing muscle stiffness as a way to estimating intramuscular pressure. It has been demonstrated that muscle stiffness is correlated to intramuscular pressure in the myocardial muscles [15]. Therefore, muscle stiffness may represent a way to estimate intramuscular pressure in other muscles as well.

Shear wave elastography is an ultrasound elastography technique that is used to measure muscle stiffness [16]. This method transmits shear waves perpendicular to the direction of particle motion. Shear wave elastography calculates the shear modulus to measure the stiffness of the tissue. This occurs due to the properties of tissue that describe that an increase in shear-wave speed indicates a stiffer tissue. Shear-wave equations are solved quantitatively to estimate the properties of tissue mechanics. This method requires advanced mathematical modeling.

The transducer implements a SWE technique known as supersonic shear imaging. This technique was proposed by Bercoff and his colleagues to visualize soft tissue viscoelastic properties [17]. Supersonic shear imaging generates low-frequency shear waves in tissues through focused ultrasonic beams. The technique creates a source that moves at a supersonic speed. This source creates shear waves that interfere constructively to make two plane shear waves. Differences within tissues disrupt the propagating waves, and these distortions are imaged by the same scanner that generated the supersonic source. Shear elasticity is imaged quantitatively using inversion algorithms, which can occur in less than 20 ms. Shear compounding is also possible through this mechanism. This is done by tilting shear waves, which improves estimations for elasticity measurements.

When using the technique on muscles, the researcher must be sure that the transducer is oriented longitudinal to the muscle fibers. This is due to the anisotropic nature of the muscles. If the orientation is not correct, the technique could yield inaccurate results. Furthermore, when measuring with shear wave elastography, the position of the transducer must be the same for each trial. This is confirmed by marking on the patient's leg when the correct position is first located. The markings on the patient's leg make the study uniform and help to achieve results that are more consistent.

The purpose of this study is to demonstrate the relationship between blood pressure, intramuscular pressure, and muscle stiffness. For the first experiment, we hypothesize that increasing blood pressure by lowering the patient's leg will increase muscle stiffness. We also hypothesize that inflating a blood pressure cuff around the patient's thigh will cause an increase in intramuscular pressure in the second experiment.

This confirmation will be used in the first experiment to identify whether or not muscle stiffness increases when intramuscular pressure is increased. In the swine study, we aim to confirm the same results, but with the use of saline to increase intramuscular pressure instead of a blood pressure cuff. To our knowledge, no study has been conducted that measures the changes in muscle stiffness as a result of both blood pressure and intramuscular pressure changes. Should our hypotheses be proven true, measuring muscle stiffness using shear wave elastography could be developed as a method for estimating intramuscular pressure. These estimations could then potentially be used in the diagnosis of acute compartment syndrome.

Chapter 2

Methods

The Institutional Review Board at the Pennsylvania State University reviewed and approved the protocol for this study. The study was performed by the Mechanical and Nuclear Engineering Department of the Pennsylvania State University in University Park, PA.

Shear Wave Elastography

A Verasonics ultrasound system (Verasonics Inc., Redmond, WA) is used to perform shear wave elastography. The linear array transducer L7-4 implemented a SWE technique known as supersonic shear imaging. This technique was proposed by Bercoff and his colleagues to visualize soft tissue viscoelastic properties [17]. The transducer generates quasiplane shear waves at seven evenly spaced focal depths. The ultrasound push beams were generated by 64 elements at a frequency of 7.813 MHz. Each push beam performs 500 push cycles that last 100 μs. Wave propagation of the shear waves is calculated using plane wave imaging at 10,000 frames/s. Within the region of interest, the shear modulus map can be generated using the following equation:

$$\mu = \rho c^2$$

In this equation, μ is the shear modulus, ρ is the tissue density, and c is the shear wave speed. Tissue density is assumed to be 1000 kg/m^3. The accuracy of this technique was confirmed using calibrated homogeneous phantoms with varying shear moduli (Model 040GSE, CIRS, Norfolk, VA).

Intramuscular Pressure and Muscle Stiffness

The first experiment tests how changes in intramuscular pressure affect muscle stiffness. This study tests two different muscles in the patient's leg, the tibialis anterior muscle and the peroneus longus

muscle. These muscles are tested at three different elevations from the patient's heart. Furthermore, the study measures both muscle's stiffness values when a blood pressure cuff applies force to the patient's thigh. Ten volunteers were measured for this portion of the experiment.

The study starts with the patient lying in the supine position. One of the patient's legs lies flat on the bed, while the other leg is elevated on top of a 14 inch-tall box. On top of this box is a foam support that has a triangular indent in it. Any movement of the patient's foot during the experiment will cause a change in the stiffness of the muscle [18]. The foam support puts most of the pressure on the back of the ankle and keeps the leg in a comfortable position to limit changes to muscle stiffness throughout the experiment. The leg at heart level should stay straight throughout the duration of the exercise. This is achieved by maneuvering the patient's calf so that it points in the same direction as the patient's torso. Furthermore, the patient's knee and hips are adjusted so that the joints are at a 90° angle. A one-inch thick lid can be added to the box to ensure taller patients achieve these angles. A blood pressure cuff is placed around the patient's ankle, and the patient's ankle is placed on top of the foam support. The patient rests in this position for 15 minutes to normalize blood pressure. This position is imaged in Figure 1.

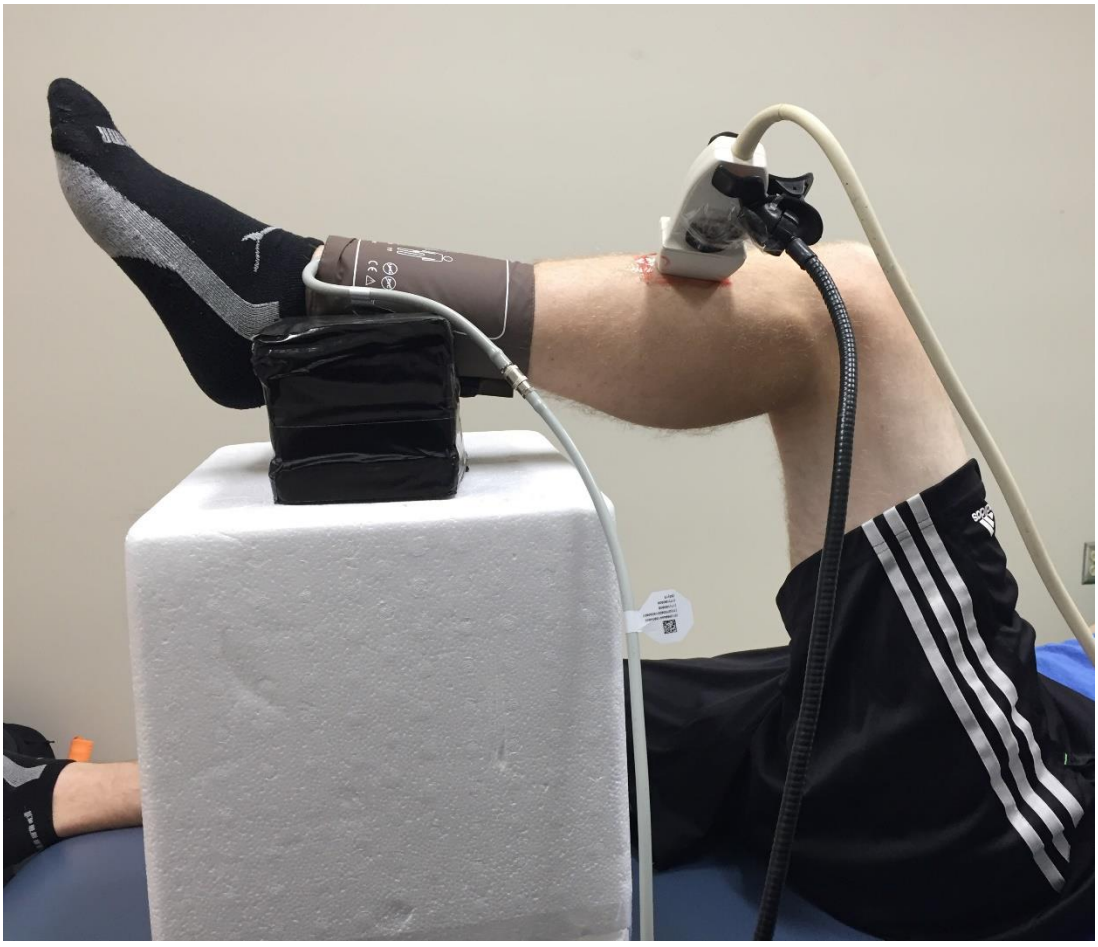

Figure 1: Patient position with leg above heart

During the 15-minute waiting period, the patient's tibialis anterior muscle is located and marked. To locate the tibialis anterior muscle, the transducer is placed in between the head of the fibula and the tip of the lateral malleolus. The transducer is placed so that the distance between the transducer and the tip of the lateral malleolus is twice as large as the distance between the transducer and the head of the fibula. The transducer is oriented longitudinal to the muscle fibers, while the correct transducer orientation and location is confirmed using B-mode imaging. The outline of the transducer is then marked on the patient's skin.

Once the 15 minutes are over, the blood pressure is recorded from the patient's ankle. The muscle stiffness of the patient is measured using shear wave elastography at the previously outlined position. The stiffness at this elevation is measured once.

The box is then removed from the patient bed, and the transducer is temporarily taken from the patient's leg. The patient is then placed in a supine position, with both legs fully extended on the bed. As any pressure on the calf may cause an inaccurate value for muscle stiffness, the leg is propped up to prevent the calf from touching anything. The same foam support is placed on the bed, and the patient's ankle is placed on top of the support. A pillow about 2 inches tall is then placed underneath the patient's knee to raise the patient's calf off the bed. A blood pressure cuff, connected to a palm style aneroid sphygmomanometer, is placed around the patient's thigh, but is kept deflated. The patient then waits for five minutes to normalize their blood pressure.

After 5 minutes, a blood pressure measurement is taken from the patient's ankle. The transducer is returned to the tibialis anterior muscle, and another muscle stiffness measurement is taken. Remaining in this position, the blood pressure cuff is then inflated to 40 mmHg, where another muscle stiffness measurement is taken. The cuff is then inflated to 80 mmHg, then 120 mmHg. A measurement is taken for each of these applied forces. The blood pressure cuff is then deflated and removed from the patient's thigh.

The patient then moves to a sitting up position on the bed. The patient's feet are then placed on a small box that rises 4 inches off the ground. The patient is instructed to remain as still as possible in this position and rest their feet on the box instead of putting a lot of pressure on the box. The patient then waits another five minutes to normalize their blood pressure. After the five minutes have passed, a blood pressure measurement is then recorded in the ankle. The transducer is placed on the tibialis anterior muscle, and a muscle stiffness measurement is taken.

The process is then repeated for the patient's other leg. The only difference now is that the peroneus longus muscle is located and measured instead of the tibialis anterior. The muscle stiffness is measured in all three positions, as well as for each applied pressure in the supine position.

Intramuscular pressure changes on muscle stiffness: A human model

An invasive blood pressure system is used to measure intramuscular pressure. This system consists of a 1 L bag of saline, a Cables and Sensors Reusable Pressure Infusion Bag, a 5 ft tall IV Pole, a JELCO® IV Catheter (22G x 1"), a BD connector compatible IBP disposable transducer, and an Edan IM50 Patient Monitor. The system is setup by spiking the saline bag with the spike of the transducer kit. The air left in the saline bag is forced out by squeezing the bag and allowing air to flow out the closest transducer stopcock. The saline bag is then placed inside the pressure infusion bag. The pressure infusion bag is inflated to around 300 mmHg, and is hung from the top of the IV pole. Air is then evacuated from the transducer by squeezing the white tab to open the line. This is done up to the first stopcock, where some of the saline is ejected from the open stopcock port to ensure as much air as possible is evacuated from the line. At this point, the stopcock is closed to the port, and the process is repeated to the next stopcock. The process ends when all the stopcocks are closed to their ports, and saline flows out of the catheter when the line is opened. Once the last of the air is ejected through the catheter, the airproof caps are then placed on the stopcock ports.

The pressure transducer then needs to be setup with the patient monitor. The transducer itself is taped to the IV pole. The transducer needs to be at the same height as the patient's heart; therefore, the transducer is taped about 2 inches above the height of the top of the patient bed. The transducer also needs to be oriented so that the end going to the patient is facing up. The BD connecting port of the transducer kit is then plugged into the patient monitor. With the stopcock closest to the transducer turned to the patient and the airproof cap removed, the transducer is zeroed on the monitor. The stopcock is turned off

to the open port, the airproof cap is returned, and the set-up of the invasive blood pressure system is complete.

The second experiment tests how intramuscular pressure changes due to applied pressure by a blood pressure cuff. The patient begins by lying supine on the bed, where they must remain for 5 minutes to normalize their blood pressure. A blood pressure cuff, connected to a palm style aneroid sphygmomanometer, is placed around the patient's thigh. Like in the first experiment, the patient's legs should be straight on the bed. The patient's ankle is placed on top of the foam support that is resting on the bed. A pillow is placed underneath the patient's knee to avoid applying pressure to the posterior compartments of the leg from the bed. The set-up of the invasive blood pressure system and the patient's legs is imaged in figure 2 below.

Figure 2: Invasive blood pressure system with catheter in patient leg

During the 5 minutes, the tibialis anterior of the patient is located using the same 2:1 length ratio as the previous experiment. The leg is sterilized, and a spray local anesthetic is applied to the patient's leg. An experienced clinician then inserts the catheter from the invasive blood pressure system into the patient's tibialis anterior at the site of anesthesia.

After stabilizing the catheter, a stopwatch is started. When the stopwatch reaches 2:00, the intramuscular pressure is recorded from the patient monitor. The blood pressure cuff is inflated to 40 mmHg, and the time is recorded when this point is reached. Once two minutes pass from the recorded time, the intramuscular pressure is recorded again. After this, the cuff is inflated to 80 mmHg. The measurement is then repeated at this applied pressure after two more minutes. The cuff is then inflated to 120 mmHg, and the measurements are repeated one last time. The cuff is then deflated back down to 0 mmHg. Once the intramuscular pressure returns to near baseline values, the catheter and cuff are removed to end the experiment.

Intramuscular pressure changes on muscle stiffness: A swine model

Our third experiment involves testing the muscle stiffness of swine by altering a previously successful procedure with shear wave elastography [19]. This experiment tests how changing the intramuscular pressure via saline injection affects muscle stiffness. Three swine legs were obtained, with the leg being cut right below the knee. The swine legs, which came frozen, were vacuum-sealed in a plastic bag. The legs were then placed in warm water for a few hours to thaw the legs.

After the few hours past, the leg was removed from the water and the plastic bag. The leg was placed on a sanitized table covered with surgical drapes to collect any spilled liquids. All of the researchers were required to wash their hands and wear gloves when dealing with the leg. The L7-4 transducer is then covered with an elastic material to protect it from touching the leg. The tibialis anterior

muscle of the swine is located on the swine using the B-mode imaging method of ultrasound. Once found, the location is marked for consistency by outlining the transducer on the skin of the swine.

The invasive blood pressure system is setup again in the same way as the second experiment. Before inserting the catheter into the compartment, some of the saline is ejected into a container. The catheter from this kit is then inserted into the tibialis anterior muscle approximately 5 cm proximal to the ultrasound transducer location.

At this point, intramuscular pressure and muscle stiffness can be continuously measured throughout the experiment. The initial intramuscular pressure is found using the invasive blood pressure system connected to the patient monitor. Five muscle stiffness measurements are found using shear wave elastography.

A syringe is then used to inject the saline into the compartment. Saline is injected approximately 5 cm posterior to the invasive blood pressure catheter. Saline is continually injected into the compartment until a constant value for intramuscular pressure is reached that is more than 5 mmHg above the initial value. The value for intramuscular pressure should be consistent for approximately 20 seconds to be considered constant. The intramuscular pressure is recorded in the compartment at this point, and five muscle stiffness measurements are taken. The process is repeated again to achieve a value for intramuscular pressure that is 5 mmHg larger than the previous value. If there is enough saline remaining, the process is repeated again a third time.

The invasive blood pressure kit is then disposed of, and the needle of the syringe is cleaned thoroughly. The elastic wrap around the transducer is removed and disposed of. The swine leg is then disposed of using IRB protocol.

Chapter 3

Results

From the initial measurements in the first experiment, we have determined that the supine and elevated leg positions provide consistent results throughout the muscle stiffness measurements taken. There is not much variation between the measurements in these positions, and the standard deviation is relatively low for these trials. However, we determined that the sitting up position was not very consistent, and we were unable to get quality data from this experiment. Therefore, we omitted this portion when performing the tests on the actual volunteers.

The data for the tibialis anterior muscle is seen in Table 1 and Figure 3 below. For the tibialis anterior muscle, the average stiffness increases significantly when lowering the leg. Furthermore, the muscle stiffness increases when increasing the pressure applied to the thigh via the blood pressure cuff. However, the applied pressure needs to be 80 mmHg or greater to get a significant increase in muscle stiffness from the zero pressure state. This significance was determined using the T-test between each of the situations with the supine position without pressure applied to the thigh.

				TA muscle								
	subject 1	subject 2	subject 3	subject 4	subject 5	subject 6	subject 7	subject 8	subject 9	subject 10	Average	STD
raising leg	3.710	3.865	3.327	3.817	3.575	3.120	3.249	2.935	3.382	3.766	3.4745	0.3194
0 mmHg	4.153	4.440	3.432	3.945	3.979	3.962	4.243	3.342	3.695	4.060	3.925	0.3461
40mmHg	4.042	4.573	3.605	4.628	4.487	4.015	4.219	3.562	3.763	4.749	4.1642	0.4353
80mmHg	4.367	4.567	3.695	4.727	5.118	4.450	4.379	3.632	3.937	5.012	4.3883	0.5085
120mmHg	4.779	4.636	3.661	4.823	5.837	4.474	4.678	3.722	3.928	5.044	4.5581	0.6599

Table 1: Muscle stiffness values from TA muscle of 10 individuals

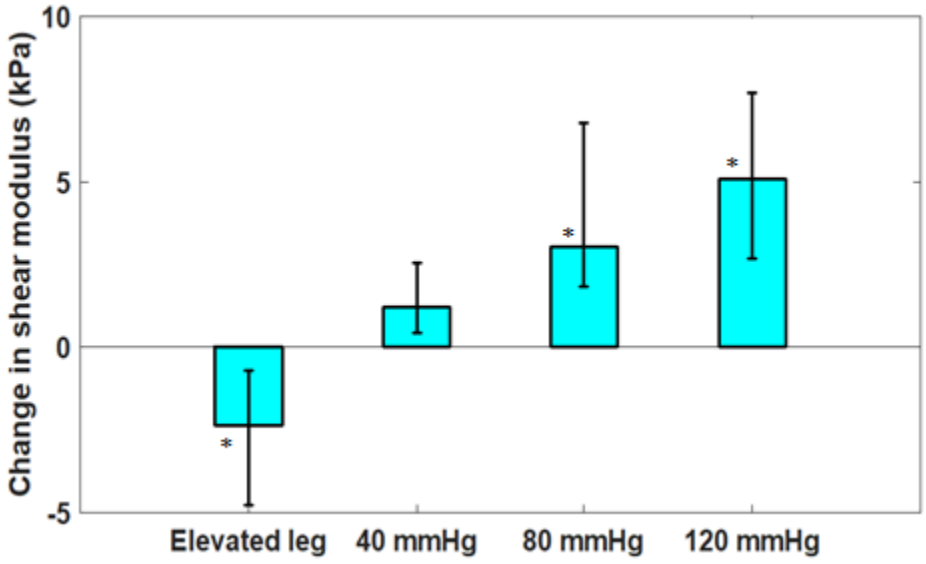

Figure 3: Shear modulus (kPa) of TA muscle (median, interquartile range) at different blood pressure levels (values at each pressure level graphed with respect to shear modulus at zero pressure level). Asterisk demonstrates statistical significance from the zero pressure level.

For the PL muscle, there was a nearly linear increase from the risen leg position to 40 mmHg. However, there was a larger jump to the 80 mmHg pressure level. Furthermore, the average for the 80 mmHg level was only slightly lower than the average for the 120 mmHg level. While the trend still exists, it is not as linear as the results from the TA muscle. Despite the non-linear results, there was still a positive trend where increasing the blood pressure in the leg made the muscle stiffer. The elevated leg position resulted in a significantly lower muscle stiffness than the supine position. Furthermore, applying 40, 80, and 120 mmHg of pressure to the patient's thigh caused an increase in muscle stiffness. Despite the large standard deviation in muscle stiffness measurements, all applied pressures to the thigh resulted in a significant increase in muscle stiffness from the zero pressure state. All of the data from the PL muscle is shown in Table 2 and Figure 4 below.

	subject 1	subject 2	subject 3	subject 4	subject 5	subject 6	subject 7	subject 8	subject 9	subject 10	Average	STD
raising leg	2.672	2.697	2.757	3.034	3.011	2.865	2.712	2.790	2.694	2.910	2.8141	0.134
0 mmHg	3.082	3.210	2.945	3.138	3.209	3.105	2.872	2.968	2.856	3.140	3.0524	0.1323
40mmHg	3.146	3.343	3.236	4.049	3.224	3.213	3.160	2.941	3.200	3.258	3.2768	0.2902
80mmHg	3.236	3.577	3.507	5.548	3.838	4.208	3.356	3.344	4.606	3.444	3.8664	0.7313
120mmHg	3.343	3.677	3.589	4.108	3.673	4.537	3.373	3.367	4.628	4.480	3.8775	0.5141

Table 2: Muscle stiffness values from PL muscle of 10 individuals

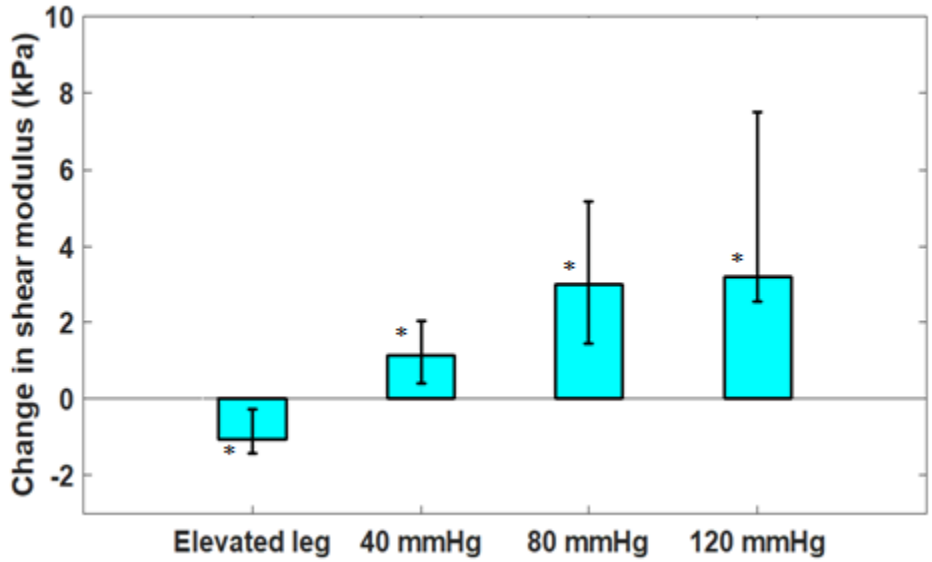

Figure 4: Shear modulus (kPa) of PL muscle (median, interquartile range) at different blood pressure levels (values at each pressure level graphed with respect to shear modulus at zero pressure level). Asterisk demonstrates statistical significance from the zero pressure level.

The second experiment tested five volunteers to record actual values for intramuscular pressure. Our results demonstrated an increase in intramuscular pressure for all volunteers whenever the blood pressure cuff pressure increased. The standard deviation is large, but that is mainly due to the variability of intramuscular pressure increase by each individual. The large standard deviation caused there to be no significant increase in any of the sequential mean pressure measurements. However, each individual demonstrated a trend of increasing intramuscular pressure with each step increase in blood pressure cuff pressure. Therefore, despite the lack of significance, it is still clear that increasing the applied pressure to the patient's thigh can cause an increase in intramuscular pressure. The data for this experiment is shown in figures 6-7 below.

	0 mmHg	40 mmHg	80 mmHg	120 mmHg
subject 1	13	17	22	27
subject 2	14	18	26	34
subject 3	14	19	23	25
subject 4	11	14	18	20
subject 5	10	12	14	16
mean	12.4	16	20.6	24.4
STD	1.816590212	2.915475947	4.669047	6.87749955

Table 3: Intramuscular pressure measurements for five subjects at applied blood pressure cuff values

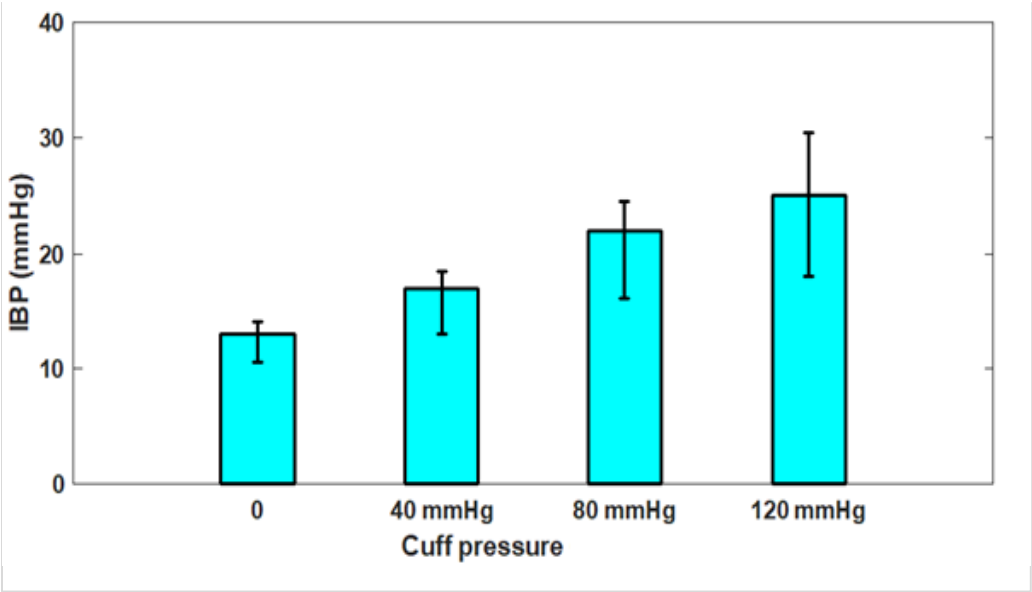

Figure 5: Intramuscular pressure results (median, interquartile range) for different levels of applied pressure

The third experiment did not achieve the results we were expecting. According to the procedure we were following, we were supposed to see an increase in the intramuscular pressure that stayed constant for minutes at a time [19]. Our experiments demonstrated an increase in the intramuscular pressure initially. However, the increased pressure dropped back to its original value a few seconds after the saline injection stopped. Because the intramuscular pressure did not stay at a constant value after injecting the saline, our measurements for muscle stiffness remained relatively the same as well. Our inability to change the intramuscular pressure to a consistent value makes this experiment inconclusive.

Therefore, the procedure needs to be adjusted to maintain a consistent value for intramuscular pressure after saline injection.

Chapter 4

Discussion

This section will mention the results of the experiments. When the results acted as expected, the reasoning behind these results will be argued. If the results were not what was expected, reasoning for the inaccuracies will be argued, as well as future experiments that could potentially provide better results.

First Experiment

The first experiment displayed the results we were expecting, with only minor issues. When leg elevation was changed from the elevated to level position, there was a significant increase in muscle stiffness for each muscle. This is due to the blood pressure inside of the muscles increasing. Lowering the leg elevation made it easier to pump blood to the patient's legs because gravity no longer worked against the flow of blood. Therefore, blood began to accumulate in the patient's legs when the patient was in the supine position. This accumulation caused an increase in blood pressure within the patient's leg. This increase in blood pressure caused there to be more pressure in to the muscles nearby. Therefore, the accumulation of blood in the lowered patient's leg caused the muscles to increase in stiffness.

This trend should have continued in the sitting position. However, preliminary tests demonstrated a large amount of variability in this position, so it was omitted from our actual studies. The reasoning for the variability is most likely due to the feet resting on the box. The feet must remain flat on the box at all times, but even while ensuring this, the patient can apply different amounts of pressure on parts of the box unknowingly. For example, the patient may press harder on their toes at the beginning of the study, but put more pressure on their heels at the end of the study. This change in pressure causes the muscles in the leg to be more or less tense, which affects muscle stiffness. Therefore, we predict that the patient is unknowingly changing their muscle stiffness throughout the experiment.

To counteract this unknowing change in muscle stiffness, another position should be tried. The bottom of the patient's feet should not be resting on a box like before. However, the patient's feel should be in a still and comfortable position to avoid unnecessary movement. One idea is to put the patient's knees at 135° angles when sitting on the bed, much like the position of dental patients. The foam pad could be secured to the bed and placed in a way that it keeps the calf from touching the bed. The patient will not have to put the bottom of their feet on anything, thus minimizing the amount of unnecessary pressure applied by the leg muscles. The patient could sit up, or lay at an angle to be comfortable. Preliminary tests will have to be done to determine which method achieves the most consistent results. If this position change were made, then this could provide additional information to the hypothesis that leg elevation affects muscle stiffness.

Applying pressure to the patient's thigh through blood pressure cuffs demonstrated an increase in muscle stiffness as well. There was a more linear trend when testing the TA muscle, but both muscles demonstrated an increase in muscle stiffness whenever the applied pressure increased. Every applied pressure caused a significant increase in muscle stiffness from the zero pressure state except for the 40 mmHg, TA muscle stiffness. This helped to argue our hypothesis that applying pressure to the patient's thigh will decrease the blood flow out of the limb. As a result, blood will accumulate in the limb, and the posterior muscles will become stiffer because they are filled with more blood. This muscle stiffness increase is differentiable using ultrasound.

From this experiment, we were able to prove two things. We proved that increasing the blood pressure in the patient's leg causes an increase in muscle stiffness. Furthermore, applying pressure to the patient's thigh cut of some of the blood flow to the patient's heart. This caused an accumulation of blood and increased muscle stiffness. Because blood pressure was a factor in both situations, we argue that the increase in blood pressure caused the muscles to experience more pressure due to the accumulation of blood in the legs. This caused the muscles to be stiffer as a result.

Second Experiment

The second experiment displayed a strong linear trend of increasing intramuscular pressure with an increase in applied pressure to the patient's thigh. In each subject, the baseline values of the patient's intramuscular pressure increased when pressure was applied to the patient's thigh. Furthermore, when the applied pressure was increased, there was an even further increase in intramuscular pressure for every patient. While there was not a significant increase in any of the steps, there was always an increase when applying more pressure to the patient's thigh. Therefore, it can be determined that applying pressure to the patient's thigh causes an increase in intramuscular pressure. All of the data averaged together also displayed this trend in a linear fashion.

Measuring an abnormal increase in intramuscular pressure is the clinically accepted way to diagnose compartment syndrome. Therefore, our results demonstrate that applying pressure to the patient's thigh is an accurate way to simulate compartment syndrome.

Third Experiment

Our third experiment had inconclusive results. We were unable to keep the pressure increase steady for any given period. We believe that the saline that was used to increase the pressure had too small particles to in the compartment. It likely diffused through the fascia or out of the cut portion of the muscle from the amputation. To get pressure increases that stayed for a longer portion of time, another solution should be used. The solution should have a larger molecule size to avoid diffusing through the fascia. If a blood analogous solution were used instead, the results should be a lot better. Another possible fix to the procedure would be to use a pig leg that has not been amputated. This would limit the amount of diffusion out of the amputation site. Better results would have helped argue that muscle stiffness can be used to diagnose potential compartment syndrome.

Chapter 5

Conclusion

Our results demonstrated that applying pressure to the patient's thigh using a blood pressure cuff causes an increase in intramuscular pressure. Because intramuscular pressure increase is the determining factor for compartment syndrome, we argued that the blood pressure cuff inflation around the patient's thigh simulates compartment syndrome. The first experiment used this method to simulate the syndrome on the patient. Because the blood pressure cuff limits blood flow back to the heart, applying the pressure cuff caused blood to accumulate in the posterior portions of the limb. This increase in blood caused an increase in blood pressure in the limb, much like changing the elevation of the limb did. From the results of the first experiment, it is clear that increasing the blood pressure in the limb causes an increase in the muscle stiffness in the TA and PL muscles. Therefore, by combining the results, it can be shown that compartment syndrome induction by inflating a blood pressure cuff around the thigh can be differentiated through muscle stiffness measurements. While the third experiment would have helped by providing another method for simulating compartment syndrome, our results still provide solid evidence to argue for the use of SWE ultrasound when diagnosing compartment syndrome. Further tests should be done with the method to determine its viability with actual cases of compartment syndrome. Regardless, the relation between compartment syndrome and muscle stiffness should be investigated further to determine if SWE ultrasound could become a viable method for diagnosing compartment syndrome.

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