INCREASED TIBIALIS POSTERIOR COMPARTMENTAL PRESSURE: A POSSIBLE CAUSE OF MEDIAL TIBIAL SYNDROME

BY

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ABSTRACT

Pain in the lower limb has been a controversial issue in past and present research. The term "shinsplints" is commonly used to define many of the lower leg pains. The purpose of this study was to try to associate a location of lower leg pain to a particular mechanism that produces pain. More specifically, the purpose of this study was to determine if a tibialis posterior compartment syndrome correlated with medial tibial syndrome.

Fifteen subjects were tested for compartment syndromes with a catheter placement into the tibailis posterior muscle compartment via an anterior approach. Eight subjects were patients who presented with medial tibial syndrome. Seven subjects represented the normal population. From the data collected, only seven subjects' results were analyzed. Seven subjects were eliminated from the study because of incomplete data. Catheter plugging was a problem throughout the study. One subject was eliminated from the study because of a lack of confidence in the instrumentation. Statistical analysis was performed with an experimental group of five and a control group of two.

Compartment pressures were analyzed in both the absolute and relative form. Absolute pressures were recorded directly off the monitor and analyzed. Relative pressures were recorded as mean arteriole blood pressure minus compartment pressure. Comparison of absolute and relative pressures between groups were performed using the Mann-Whitney U test statistic. Comparison to various literature cited criteria was also performed. There was a noticeable, though not

statistically significant, difference between the two groups, with those presenting with medial tibial syndrome having higher pressures. The criteria suggested in the literature to diagnose compartment syndromes may be too conservative. Results of this study indicate that a pressure of 30 mmHg. at any time would be strongly indicative of a compartment syndrome. Refinement to the instrumentation and/or procedure are needed so that complete results can be obtained from all subjects.

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TABLE OF CONTENTS

INTRODUCTION 1

Statement of the Problem 2

Hypothesis 2

Rationale for the Study 2

Delimitations 3

Definitions 3

REVIEW OF RELATED LITERATURE 4

Anatomy of the Lower Limb 4

Osseous structures 4

Musculature 5

Fascia 7

Supporting neurovascular vessels 8

Defining Lower Leg Pain 9

Defining Compartment Syndromes 10

Techniques for Measuring Compartment Syndromes 12

Pressure measurement 12

Long term pressure measurement 19

Reference Values for Compartment Syndromes 19

Absolute Pressure 19

Pressure Induced Ischemia 21

Etiology/Pathology of Compartment Syndromes 22

Tibialis Posterior: A Recognized Compartment 25

Identifying the compartment 25

Decompression 28

Medial Tibial Stress and Compartment Syndromes 29
METHODS AND PROCEDURES 32
Subjects 32
Subject Profile 32
Experimental Design 34
Data Collection 34
Data Analysis 36
RESULTS AND DISCUSSION 37
Compartmental Pressure Data 37
Comparison Between Groups 39
Comparison to Literature Criteria 44
SUMMARY, CONCLUSIONS AND RECOMMENDATIONS 47
Summary 47
Conclusions 48
Recommendations 49
BIBLIOGRAPHY 50
APPENDICES 55

LIST OF TABLES

Table 1 Origin insertion action innervation table of deep posterior muscles of lower leg 7

Table 2 Summaries of etiologies of compartment syndromes 23

Table 3 Intracompartmental pressure measurements in tibialis posterior muscle and the deep posterior compartment of the leg following exercise on a treadmill 27

Table 4 Compartment pressure (mmHg) 27

Table 5 Experimental subjects profile 33

Table 6 Control subjects profile 33

Table 7 P-Values (M-W U test) for comparison between absolute and relative pressures of experimental and control groups 40

Table 8 Contingency tables comparing literature criteria values to the seven subject's pressures 44

LIST OF FIGURES

Figure 1	Cross section through the lower leg, male	6
Figure 2	The solidstate transducer intra compartmental (STIC) catheter	15
Figure 3	The Stryker STIC pressure monitoring system	16
Figure 4	Pathologies of chronic compartment syndrome	24
Figure 5	Cross section of lower leg demonstrating placement of the needle for pressure measurements in the tibialis posterior muscle and the deep posterior compartment	26
Figure 6	Release of all five compartments via a single lateral approach	29
Figure 7	Release of the deep compartment and tibialis posterior via a medial approach	29
Figure 8	Area of tenderness resulting from medial tibial syndrome	30
Figure 9	Absolute compartmental pressures between control mean, experimental mean and control subject #3	38
Figure 10	Overall plot of absolute compartment pressures of control (N=2) and experimental (N=5)	41
Figure 11	Comparison of mean absolute compartmental pressures - compartment pressure between control (N=2) and experimental (N=5)	41
Figure 12	Overall plot of mean arteriole blood pressure of control (N=2) and experimental (N=5)	42
Figure 13	Comparison of mean arteriole blood pressure - compartmental pressure between control (N=2) and experimental (N=5)	42

CHAPTER 1

INTRODUCTION

Pain in the lower limb has been a topic of much controversy in past and present research. The lower limb is an area where many injuries occur with little known about the mechanisms which produce them. "Shinsplints" is a term which has been commonly used to define many lower leg pains. Medial tibial syndrome is one of many terms used to describe pain commonly referred to as "shinsplints". In the past, "shinsplints" has been ambiguously defined. More recently, the American Medical Association, as cited by Sherkin (1984), defined "shinsplints" as an "inflammation of muscles that lead from the shin to the foot resulting from unaccustomed repetitive activity of forcible use of foot flexors." This definition now classifies stress fractures and compartment syndromes as separate injuries (Barnish, 1986; Andrish, 1984).

Compartment syndromes can be diagnosed as acute or chronic in mechanism. Quarfordt, Christenson, Eklof, Ohlin and Saltin (1983) stated that the diagnosis and history of chronic compartment syndromes are remarkably similar to that of shinsplints. Chronic compartment syndrome, as defined by Rorabeck (1986), is a compartment pressure which is normally elevated during exercise and typically fails to return to normal with rest. The most helpful test in the diagnosis of compartment syndromes is tissue pressure measurement. The procedure for this measurement is invasion with a catheter into the suspected compartment.

The lower limb consists of four intramuscular compartments: anterior, lateral, superficial posterior and deep posterior (Moore, 1985). A

hidden fifth compartment in the deep compartment which encases the tibialis posterior muscle has only recently been explored by Davey, Rorabeck and Fowler (1984) and Rorabeck, Bourne and Fowler (1986). Aforementioned researchers have postulated that exertional compartment syndrome can occur involving the tibialis posterior muscle. It is this compartment that may be the cause of many of the pains found in the lower leg.

Statement of the Problem

The purpose of this study was to determine if increased pressure within the tibialis posterior compartment is associated with medial tibial syndrome.

Hypothesis

A compartment syndrome occurring in the tibialis posterior compartment is associated with medial tibial syndrome.

Rationale for the Study

The practical implication of this study is to link a particular compartment syndrome (tibialis posterior compartment) to a location of pain (medial tibial pain). Pressure monitoring, via invasive techniques, is a complicated procedure. It would be beneficial to relate compartment syndromes with clinical non-invasive techniques, such as pain location, before the actual catheter invasion process is used to confirm compartment syndromes. If the relationship can be verified, invasive techniques may not always be necessary.

Limitations

-A relatively new monitoring system was used to measure compartment pressures and little literature was available on the reliability and validity of the system.

-The difficult approach necessary to invade the deep tibialis posterior compartment allowed only an assumption that the catheter was placed within the proper compartment.

-A total sample size of 7 (5-experimental, 2-control) limited the statistical power of the comparison between the two groups.

Definitions

Medial tibial syndrome

As used in this paper, medial tibial syndrome is defined as pain along the posteromedial aspect of the tibia, ten to fifteen centimeters above the medial malleolus.

CHAPTER 2

REVIEW OF RELATED LITERATURE

Anatomy of the Lower Limb

Osseous Structures

The lower leg consists of two osseous structures, the tibia located on the anteromedial side and fibula lying posterolaterally (Moore, 1985). They are united at both ends by a bony articulation. The proximal tibiofibular joint consists of the mediosuperior head of the fibula and the posterolateral aspect of the lateral condyle of the tibia (Nicholas & Hershman, 1986). The distal tibiofibular joint consists of the lower end of the fibula, just superior to the lateral malleolus and the fibular facet on the posterolateral side of the distal end of the shaft of the tibia (Basmajian, 1982).

A binding between the shafts of the two bones, by the interosseous membrane, increases the stability of this unified structure. The membrane is attached to the interosseous borders of the tibia and fibula (Grant, 1983). The fibres of the membrane run laterally in a proximal to distal direction. Moore (1985) describes the interosseous membrane as having its fibres running in an oblique direction of approximately forty-five degrees from the horizontal. The interosseous membrane separates the anterior (extensor) surface from the posterior (flexor) surface.

The tibia is the second largest bone in the body, hence its primary function of weight bearing (Moore, 1985). Nicholas et al. (1986) describe the shaft of the tibia as being triangular, having three borders (anterior, medial and interosseous) and three surfaces (medial, posterior and lateral). The anterior border and medial surface are subcutaneous.

The fibula is a much thinner bone when compared to the tibia. It functions as an origin site for muscles, a pulley for tendons passing laterally into the foot, and as a lateral splint for support (Basmajian, 1982). Nicholas et al. (1986) described the body of the fibula as having three borders (interosseous, anterior and posterior) and three surfaces (medial, lateral and posterior).

Musculature

Peterson and Renstrom (1986) stated that the musculature of the lower leg is enclosed in four tight, inflexible compartments of connective tissue which are anchored on the tibia and fibula. The anterior compartment lies between the tibia and fibula and anterior to the interosseous membrane. The muscles enclosed in the anterior compartment are the tibialis anterior, extensor hallucis longus, extensor digitorum longus and peroneus tertius (Nicholas et al., 1986).

The lateral compartment lies laterally to the shaft of the fibula. It consists of two muscles, the peroneus longus and peroneus brevis (Nicholas et al., 1986).

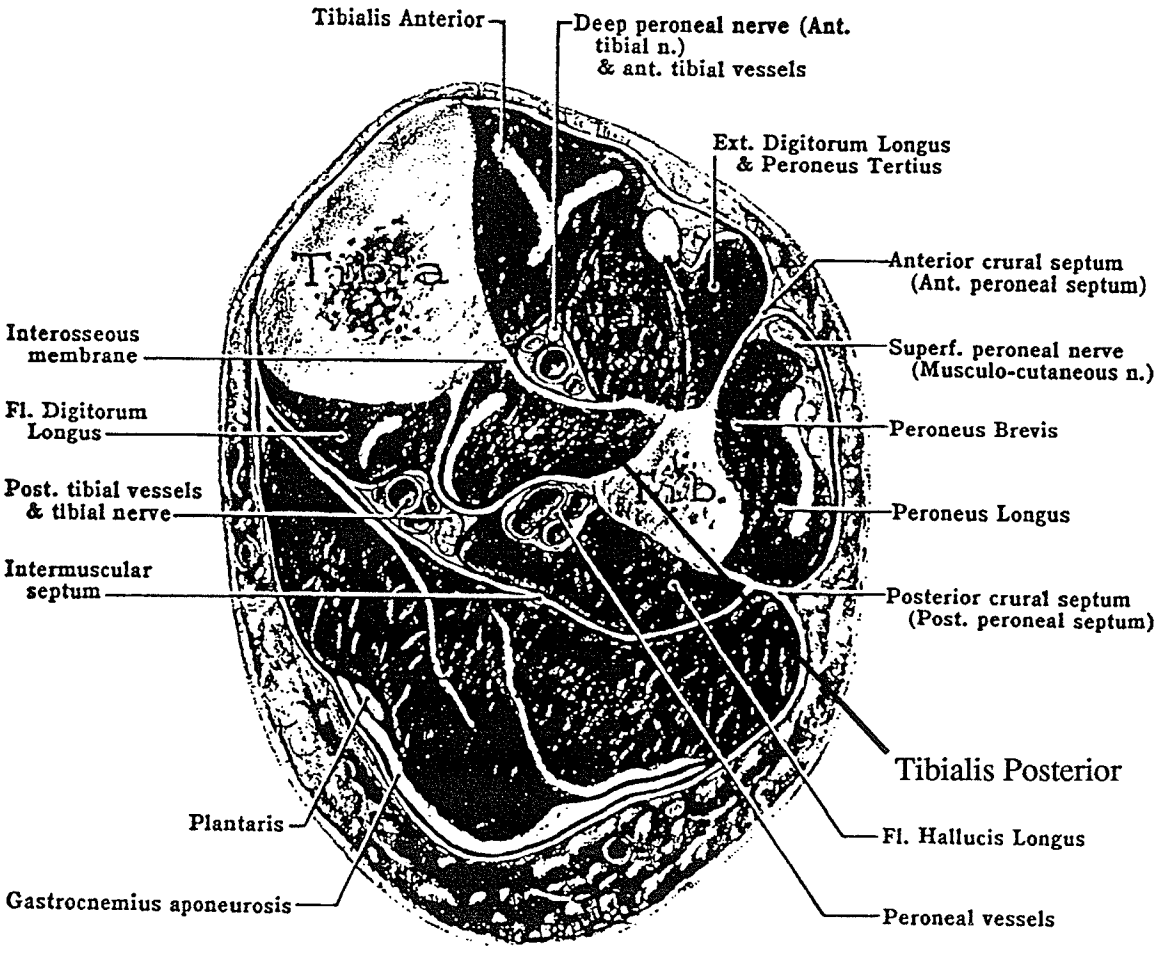
The last two compartments contain a superficial and deep group of muscles and both lie posterior to the tibia-interosseous membrane-fibula structure. The superficial group consists of the gastrocnemius, soleus and plantaris (Nicholas et al., 1986).

The deep posterior group lies between the tibia and fibula and posterior to the interosseous membrane. The musculature structures that are encased in the deep posterior compartment are the popliteus muscle (which acts on the knee), flexor hallucis longus (which acts on the ankle and greater toe), flexor digitorum longus (which acts on the ankle and lesser digits) and

the tibialis posterior (which acts on the ankle and supports the plantar arch) (Moore, 1985).

The tibialis posterior is the deepest muscle of the flexor group in the lower leg. It lies deep to both the flexor digitorum longus and flexor hallucis longus; especially deep to the latter (Gray, 1980) (see Figure 1 and Table 1).

Figure 1 Cross section through the lower leg, male



from Grant (1983).

Table 1 Origin insertion action innervation table of deep posterior muscles of the lower leg

Deep Muscles of Posterior Crural Compartment

Muscle	Origin	Insertion	Nerve Supply	Actions
Popliteus	Lateral surface of lateral condyle of femur and lateral meniscus	Posterior surface of tibia, superior to soleal line	Tibial nerve (L4, L5, and S1)	Flexes knee and unlocks the locked knee by rotating tibia medially on the femur
Flexor hallucis longus[1]	Inferior two-thirds of posterior surface of fibula and inferior part of interosseous membrane	Base of distal phalanx of great toe	Tibial nerve (S2 and S3)	Flexes great toe and plantarflexes foot
Flexor digitorum longus[1]	Medial part of posterior surface of tibia, inferior to soleal line	Bases of distal phalanges of lateral four digits		Flexes lateral four toes and plantarflexes foot
Tibialis posterior[1]	Interosseous membrane, lateral part of posterior surface of tibia, and superior two-thirds of medial surface of fibula	Tuberosity of navicular, cuneiforms, cuboid, and bases of second, third, and fourth metatarsals	Tibial Nerve (L4 and L5)	Plantarflexes and inverts foot

from Moore (1985).

Fascia

Basmajian (1982) described fascia as "dense fibrous tissue arranged in a sheet and most commonly found in association with muscles." (p.6). Deep fascia wraps individual muscles and groups of muscles together. It is the intermuscular septum that runs from the deep fascia to bone forming partitions between muscle groups. (Basmajian, 1982).

The fascia of the lower leg is a continuation of the fascia lata from the thigh. It attaches at the knee to the patella and patellar tendon, the tubercle and condyles of the tibia, and the head of the fibula (Gray, 1980). The fascia runs downward attaching to the periosteum of both the tibia and

fibula. On the lateral side of the fibula the fascia continues to become the anterior and posterior intermuscular septum (Gray, 1980). (see Fig. 1). Extensions from the septum enclose individual muscles. An important extension is the deep transverse intermuscular septum of the lower leg which functions as a divider between the superficial and deep posterior compartments of the leg.

A second deep transverse intermuscular septum is found in the deep posterior compartment. It runs from the lateral tibia to the medial fibula, separating the tibialis posterior muscle from the flexor digitorum longus and flexor hallucis longus (Moore, 1985). The deep posterior compartment is now seen as having two compartments within itself, the superficial-deep posterior and the deep-deep posterior compartment.

Supporting Neurovascular Vessels

The lower leg has three main neurovascular tracts running through it. The anterior tibial tract runs through the anterior compartment. It consists of the anterior tibial artery, its supporting veins and the deep peroneal nerve (Moore, 1985). The anterior tibial tract lies just anterior to the interosseous membrane and between the tibialis anterior and extensor hallucis longus (Grant, 1983).

The superficial peroneal nerve is the only supporting vessel in the lateral compartment. It lies anteriorly in the lateral compartment and superficial to the peroneous brevis (Grant, 1983).

The peroneal vessels, consisting of the peroneal artery and its supporting veins, lie in the superficial-deep posterior compartment. They lie along the posteromedial border of the fibula, between the tibialis posterior and flexor hallucis longus muscles (Moore,1985).

The posterior tibial tract also lies within the superficial-deep posterior compartment. The tract consists of the posterior tibial artery, its supporting veins, and the tibial nerve. The tibial nerve innervates all the muscles in the posterior compartments. This tract lies between both transverse intermuscular septum, surrounded by tibialis posterior anteriorly, flexor digitorum longus medially and flexor hallucis longus laterally. (Moore, 1985).

It can be seen in Figure 1 that all of the neurovascular supporting vessels are located along the border of the tibialis posterior muscle, with the exception of the superficial peroneal nerve. This makes it very difficult to test the physical characteristics of the tibialis posterior muscle in its surrounding compartment via invasive techniques.

Defining Lower Leg Pain

The source of many controversial injuries and pains has been the lower leg. The mechanism of injury most prevalent is low repetitive stresses causing an overuse-type of injury (Eggold, 1981). "Shinsplints" has been the much-used term to identify these injuries in past research. Only recently, within the past decade, have lower leg injuries been more accurately and concisely defined. The American Medical Association (AMA), as cited by Nicholas et al. (1986), defines "shinsplints" as any musculotendinous injury occurring in the lower leg.

Benas and Jokl (1978) categorized "shinsplint" pain into three possible mechanisms; 1) stress fractures (bony origin), 2) ischemia of the deep posterior compartment (vascular origin) and 3) myositis, fasciitis and periostitis (soft tissue origin).

Researchers do not agree when defining shinsplints, which is evident in the two previous examples. The deletion of stress fractures and compartment syndromes from the AMA's definition allows for a more precise although still quite ambiguous definition.

The location of "shinsplint" pain is also an area of uncertainty. It is generally accepted that the pain is centralized around the tibia (Jackson, 1978). There is little agreement as to the exact location of pain around the tibia.

DeLacerda (1980), Ryan (1977) and O'Donoghue (1970) cited "shinsplint" pain as along both the anterior lateral aspect and the posterior medial aspect of the tibia, particularly the proximal two-thirds of the bone and less frequently along the distal two thirds. Barnish (1986) defined the pain as radiating from the anterior tibia. Andrish (1984) located the pain over the posterior medial border of the tibia. Sherkin (1984) divided the pain into two different locations, posterior and anterior tibia: The former extending along the side or medial back of the lower leg and ankle and the latter centered in the lower front of the shin and down the outside or lateral border. Clark (1984) cited the pain at the lower one-third of tibia along the interosseous border. It can be seen that there are many locations along the tibia where pain can be found. Barnish (1986) hypothesized that each location of pain had its own injury and mechanism of production.

Defining Compartment Syndromes

Chronic compartment syndrome (Davey et al., 1984), exercise induced compartment syndrome (Mubarak, Gould, Lee, Schmidt & Hargens,

1982) and exertional compartment syndrome (Jones & James, 1987) are all interchangeable terms. Detmer (1980) stated that

> "acute and chronic compartment problems differ only in their degree of severity since both have the same underlying etiology. In susceptible individuals the fascial compartment is too small to accommodate for the 20% increase in size of the muscle mass which typically occurs during heavy exercise. The increase in pressure within the small unyielding compartments limits venous outflow by collapsing the veins. This increases capillary resistance, induces arterial vasospasm, decreases capillary perfusion and triggers an outpouring of edema fluid into the compartment which further raises compartment pressures and creates ischemic pain or tissue destruction or both." (p.142).

Another compartment syndrome definition, by Matsen (1975) stated a "condition in which the circulation and function of tissues within a closed space are comprised by increased pressure within that space."(p.8). Nicholas et al. (1986) defined compartment syndrome as a "pathologic condition of skeletal muscle characterized by increased interstitial pressure within an anatomically confined muscle compartment that interferes with the circulation and function of the muscle and neurovascular components of the compartment."(p.213).

There are four compartments in the lower leg where compartment syndromes can occur; anterior, lateral, superficial posterior and deep posterior. Barnish (1986) and Detmer (1980) cited the first as most common.

Next in frequency of occurrence was the deep posterior because it is encased by intermuscular septum and also enclosed between the osseous-membrane-osseous structure and the superficial posterior compartment. This causes an additional resistance to any possible expansion of the deep posterior compartment.

Davey et al. (1984) studied the tibialis posterior compartment, an overlooked compartment found in the deep posterior compartment. The study proved that an isolated compartment syndrome can occur in the tibialis posterior compartment.

Techniques for Measuring Compartment Pressures

Compartment syndromes have been and are presently under close observation as the causative factors for the previously cited controversial tibial pain. It is only recently that accurate techniques for measuring the intramuscular pressure within the compartments have been developed. It is important to measure compartmental pressure accurately in order to readily identify compartments that need decompression (Barnes, Gibson, Scott, Bently & Allen, 1985). An early diagnosis and effective decompression leads to less complications as continuing pressure causes further damage to tissue as time passes (Matsen, 1976).

Pressure Measurement

Invasion of the compartmental tissue is the most accurate way to measure compartmental pressure. There are presently three types of catheters used for intracompartmental pressure monitoring.

Mubarak, Hargens, Owen, Garetto and Akeson (1976) studied the use of a wick catheter for the measurement of compartmental pressure. The original technique consisted of braided cotton wicks with a fiber diameter of eight to sixteen micrometers, protruding out of an open bore needle (Scholander, Hargens & Miller, 1968). Mubarak et al. (1976) replaced the cotton wicks with soluble braided Dexon sutures. This modification kept the orifice of the catheter open.

The catheter was then attached to a pressure transducer (P23 Db Statham) (Hargens, Mubarak, Owen, Garetto and Akeson's, 1977). A PE-50 flexible tubing with an outside diameter of .9 mm and an inside diameter of .6 mm was used to connect the catheter to the pressure transducer (Hargens et al., 1977). Before insertion of the catheter into the compartment, the PE-50 connecting tube and the wick catheter were filled with a saline solution (Mubarak et al., 1976). The saline solution was used to transmit the pressure recordings from the catheter to the pressure transducer.

With the small inside diameter of the PE-50 connective tubing, minute pressure changes can be detected with minimal transmission time. Mubarak's et al. (1976) canine study positively tested the wick catheter method for accuracy and reliability. A graduated infusion column was inserted into the anterolateral compartment of the canines along with a wick catheter, needle nanometer device and a solid state probe. Canine plasma was infused into the compartment. Mubarak's et al. (1976) results proved the wick catheter to be very accurate and reliable. Results also found the solid state probe tended to produce higher pressure readings and the needle technique produced lower readings.

Shakespeare, Henderson and Clough (1981) found the wick catheter very accurate although tendencies to become plugged were noted. There was

also a problem of the Dexon wicks remaining in the tissue upon removal.

Rorabeck, Castle, Logan and Hardy (1980) studied a new technique for compartment pressure monitoring. This technique replaced Mubarak's et al. (1976) wick catheter with a slit catheter. The slit catheter consisted of PE-60 polyethylene tubing with five symmetrical slits extending about 2 mm along the length of the tubing. This catheter was then inserted into a cannula (approx. 16 gauge needle) and was ready to invade the compartment (Rorabeck et.al., 1980).

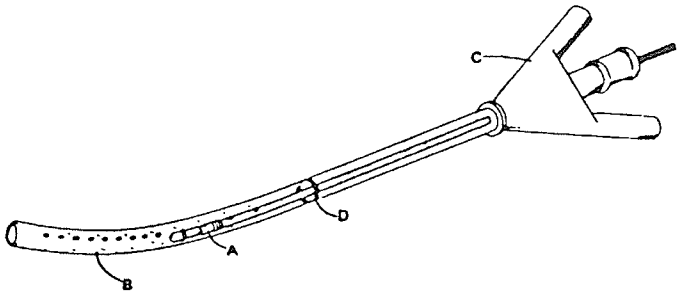
The catheter was attached to a pressure transducer and also an infusion pump (Barnes, Gibson, Scott, Bently & Allen, 1985). The transducer was then attached to a monitor which converted the pressure into mmHg. Logan, Rorabeck and Castle (1983) suggested a 90 MHz telemetry system to monitor pressure changes.

The catheter and tube were filled with a saline-heparin solution to prevent clotting at the catheter opening and thus eliminating any artifacts or inaccurate recordings (Logan et al. 1983).

Shakespeare et al. (1981) compared both the wick and slit techniques and found both to be accurate. The slit had an advantage over the wick in that its preparation was much simpler and it tended to clog less easily.

McDermott, Marble, Yabsley and Phillips (1982) studied the use of a Solid State Transducer Intra Compartmental (STIC) catheter in the measurement of compartmental pressure (see Figure 2). The catheter had a micro-transducer within its lumen which allowed for an extremely short response time to pressure changes. McDermott et al. (1982) stated that the STIC catheter had the ability to monitor pressure changes caused by systolic and diastolic blood pressure as compared to the slower wick technique.

Figure 2 Solidstate transducer intra compartmental (STIC) catheter



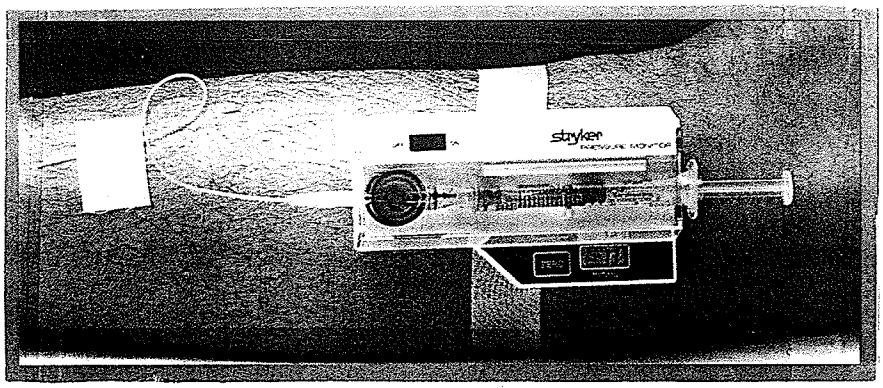
Solid-state transducer intracompartmental (STIC) catheter: (A) Millar transducer in lumen of catheter, (B) multiple side perforations and curved tip of catheter, (C) three-channel connector, and (D) catheter markings showing end-point for insertion.

from McDermott et al. (1982)

The positive attributes of this technique are dampened by the size of the catheter. The catheter size is similar to a 10 gauge needle and also has a blunt end. Difficulty may be found in exercising with the catheter placed in the compartment. Additional problems may be encountered with invasion of the deep compartments as an incision is made through the skin to introduce the STIC catheter to superficial compartments (McDermott et al. 1982). No research was found that uses the STIC to invade deep compartments.

Whiteside, in collaboration with Burgess, designed a portable Solid-State Transducer Intra Compartmental Pressure Monitoring System called the Stryker STIC (see Figure 3). It can be used as a hand held monitor or attached to a limb to monitor long term pressure recordings. The entire system weighs 150 grams with dimensions of 12.7cm x 7.6cm x 5.1cm. The Stryker STIC was specifically designed for measuring tissue fluid pressures. Whiteside's system uses a side ported needle for immediate pressure recordings and also has available an indwelling slit catheter for long-term continuous readings.

Figure 3 Stryker STIC pressure monitoring system



from Stryker STIC operating manual

This design allows for a pre-fill/flushing syringe to be attached to one side of the transducer chamber while a side ported needle or indwelling slit catheter is attached to the other side. The transducer chamber is set into the transducer well which is part of the monitoring system. An L.E.D. readout displays the recordings that the pressure transducer has measured. This system has an electronic "zeroing" device which allows for simple and accurate calibration functions.

The simplicity of this system allows for reduced assembly time. Awbrey, Sienkiewicz and Mankin, in an unpublished paper, studied assembly and complete measurement time of the digital monitor and needle manometer technique by using two physicians, each trained in the assembly of both systems. Each system was assembled ten times. The digital monitor was set up in a mean time of 2.3 minutes with a .7 minute standard deviation. The needle manometer technique mean set up time was 8.4 minutes with a 2.3 minute standard deviation.

Awbrey and associates, at the Massachusetts General Hospital,

studied the Stryker STIC digital monitor in four different situations; manometer study, pressure chamber study, animal study and clinical study. The purpose of the study was to determine the accuracy, reliability and efficiency of the Stryker STIC digital monitor.

Awbrey's et al. manometer study was designed to evaluate the accuracy of the digital monitor by comparing it directly against a standard water column. The digital monitor readings remained linear over the test range of 0 to 80 mmHg with a standard deviation of .8 mmHg. Of the 74 paired duplicate measurements, no single measurement was more than 1 mmHg from the true column pressure.

Awbrey's et al. second study, the pressure chamber study, was designed to evaluate the in vivo performance of the system under a controlled circumstance. A comparison between the digital monitor and Whiteside's needle manometer technique was observed. Both techniques were used to measure tissue pressure within the fascial compartments of bovine muscle. The muscle was sealed in a plexiglass air chamber. Pressure within the chamber could be consistently controlled between 0 and 150 mmHg. The results of 44 paired measurements at 12 different pressures ranging between 0 and 110 mmHg revealed that the digital monitor recorded pressures corresponded to the true pressure with a standard deviation of .9 mmHg. The needle manometer measurements corresponded to the true pressure with a standard deviation of 3.3 mmHg. Both methods produced measurements that behaved in a linear fashion. It was also noted that there was an increase of 2.5% in the standard deviation at pressures over 80 mmHg.

Awbrey's et al. third study consisted of the measurement of tissue pressure in the anterior, lateral and superficial posterior compartments of

both hind limbs of eight rabbits. Paired compartment pressures were taken by both the digital monitor and the standard needle manometer method. The results from the digital monitor and the standard needle manometer revealed standard deviations of 1.06 mmHg and 3.36 mmHg respectively.

The fourth investigation by Awbrey et al. was a clinical study using 51 patients presenting with various suspected compartment syndromes. Records by the digital monitor and needle manometer were taken. Replicate measurements of both systems were performed. The replicate digital monitor recordings were accurate up to 1.4 mmHg while the replicate needle manometer recordings were accurate to 2.8 mmHg.

The digital monitor was determined by the laboratory investigations to provide accurate, rapid and reproducible measurements of interstitial fluid pressure. The system was proven accurate in the standard water column test and in vitro muscle compartments contained within a pressure chamber. The animal investigation revealed that the digital monitoring technique was three times as precise as the old needle manometer technique. The clinical study confirmed the animal investigation results by revealing the digital monitoring measurements to be more than twice as precise in reproducing pressure results as the old needle manometer technique.

The Stryker STIC digital monitor, compared to other pressure monitors, is a less bulky, more compact system that has a much shorter set up time and complete measurement time. It is also a more accurate and reliable system, as zeroing and recordings are done electronically. The pressure transducer is also reusable, therefore reducing cost and eliminating any calibration differences.

Long Term Pressure Measurements

Pressure in a typical chronic compartment syndrome may be normal at rest, rise throughout exercise and gradually return to normal after exercise (Matsen, Mayo, Geoffrey, Sheridan & Krugmire, 1976). Therefore it is often necessary to monitor compartment pressures over a period of time. Previously mentioned techniques had difficulty in preventing clotting from occurring near the catheter opening. In long-term monitoring, this problem would be magnified.

Barnes et al. (1985) discussed a technique to prevent clotting in long-term monitoring. An infusion pump was attached to the connecting tube between the catheter and transducer via a three-way stop-cock fitting. A heparin-saline solution was infused into the connecting tube and down to the catheter at a rate of .1 ml per hour (Barnes et al., 1985). This rate was so minute that pressure readings were not significantly changed. The solution acts as a pressure transmitter from the catheter to the transducer and also prevent catheter blockage. Heparin, as defined by Dorland (1982), occurs naturally in the body and acts as an anticoagulant. Barnes et al. (1985) found that this technique maintained patency of the catheter for up to 24 hours in a compartment. An advantage of continuous intracompartmental pressure monitoring is that pre-exercise, exercise and post-exercise pressures can be monitored (Allen, Stirling, Crawshaw & Barnes, 1985).

Reference Values for Compartment Syndromes

Absolute Pressure

Research dealing with appropriate reference values for compartment syndromes is inconclusive. Allen and Barnes (1986) used a pressure greater

than 40 mmHg to define a compartment syndrome in the deep posterior compartment and a pressure greater than 50 mmHg for a compartment syndrome in the anterior compartment. The reason for different values is that the deep posterior compartment is more compliant than the anterior (Allen & Barnes, 1986). These researchers postulated that any pressure greater than 40 mmHg., six hours after exercise completion, should be classified as a compartment syndrome and a fasciotomy should be performed. Andrish (1984) stated that any pressure over 40 mmHg, in any compartment, should be classified as a compartment syndrome. Styf, Korner and Suurkula (1987) and Detmer (1980) indicated that intramuscular pressure that exceeded 30 mmHg at rest may elicit muscle ischemia. Wiederhielm and Weston (1973) and Burton (1951) found that a normal tissue pressure at rest was approximately zero mmHg.

Rorabeck's (1986) study suggested that a pressure greater than 10 mmHg., 20 minutes after exercise completion, would constitute a compartment syndrome. Styf (1987) also postulated that the 20-minute post-exercise interval was important and found that pressure was to return to normal by this time in order for a compartment syndrome to be ruled out. An additional criterion was also established where any pressure greater than 35 mmHg during muscle relaxation would be a suspected compartment syndrome.

Mubarak's (1987) criteria for a compartment syndrome were tissue pressure greater than 10 mmHg. at rest, or tissue pressure greater than 25 mmHg. 5 minutes after exercise completion.

Melberg (1989) discussed acknowledgement of a compartment syndrome when four criteria were met. The first criterion was a relaxation pressure of greater than 30 mmHg. This was followed by a post-exercise

pressure of greater than 30 mmHg. The third criterion was a time interval greater than six minutes for the compartment pressure to normalize. These three conditions must be met along with the fourth criterion, presence of pain and impaired muscle function.

Pressure Induced Ischemia

Dahn, Lassen & Weston (1967) found that bloodflow ceases in the microcirculation by the time tissue pressure, within a closed compartment, rose to a level equal to the diastolic blood pressure.

Ashton (1963) found that blood flow ceased or was ineffective even when tissue pressures were less than the diastolic blood pressure. This was shown by a study using the human forearm. The results revealed that there was a significant drop in blood flow when tissue compression increased to within 30 mmHg of the intravascular pressure.

Jenning (1964) reported cessation of blood flow at the arteriolar level when the tissue pressure outside the vessels increased to within the range of 10-20 mmHg of the normal intravascular pressures.

Heppenstall, Scott, Shewton & Chance (1985) determined that the change in pressure (mean arterial blood pressure minus compartment pressure) must be more than 40 to 50 mmHg for a safe compartment pressure reading. Therefore, a change in pressure less than 40 mmHg constituted a compartment syndrome and elicited ischemic responses.

Mean arterial blood pressure as defined by Fox & Mathews (1981), is the diastolic blood pressure plus one-third times the difference in systolic and diastolic pressure. [diastolic pressure + .33(systolic - diastolic)]. Awbrey et al. recommended a combination of the Stryker STIC monitoring system and the criteria established by Heppenstall's et al. (1985) study to

describe the "critical" differential between blood pressure and compartment pressure which is necessary for adequate perfusion of blood into a muscle compartment.

Etiology/Pathology of Compartment Syndromes

Increased tissue pressure is the central pathogenic factor that causes compartment syndromes. Tissue pressure may increase as a result of either a decrease in compartment size or an increase in the volume of its contents (Mubarak et al. 1976) (see Table 2). Tissue pressure increase as a result of decreased compartmental size is rare. It is the increase in compartmental contents that is a more common mechanism of compartment syndromes.

There are three proposed theories to explain the mechanisms of tissue perfusion compromise by increased pressure (Matsen, 1976).

1) Benjamin (1957) and Eaton and Green (1972), as cited by Matsen (1976), have clinically proved that arterial spasm can occur from increased compartmental pressure. Arterial spasms cause an irregular flow of blood to the capillaries, and limit blood supply to the muscles, causing ischemic pain.

2) Burton (1951), as cited by Matsen (1976) proposed a theory of critical closing pressure. In order for arterioles to maintain their patency, there must be a transmural pressure difference. If tissue pressure rises higher than arteriole pressure, the arteriole walls will collapse.

3) Burton's (1951) theory also applies to veins. With the flaccid walls of veins, tissue pressure can quite possibly rise until it passes venous pressure, causing a collapse of the venous walls. Both of Burton's (1951) theories explain a mechanism for ischemic pain.

TABLE 2 Summary of etiologies of compartmental syndromes

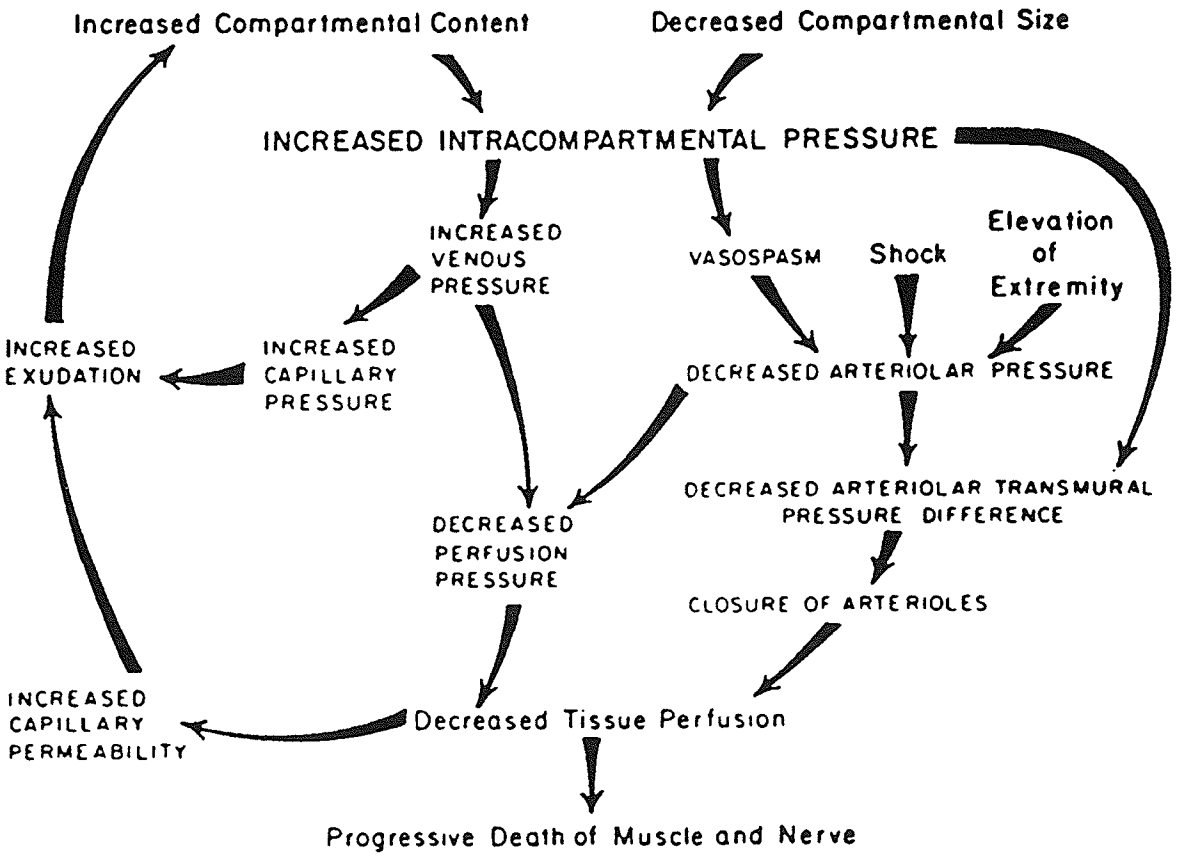
I. Decreased Compartmental Size
   A. Closure of the fascial defects
   B. Tight dressing
   C. Localized external pressure

II. Increased Compartmental Content
   A. Bleeding
      1. Major vascular injury
      2. Bleeding disorder
   B. Increased capillary permeability
      1. Post-ischemic swelling
      2. Exercise (seizures and eclampsia)
      3. Trauma (other than major vascular)
      4. Burns
      5. Intra-arterial drugs
      6. Orthopedic surgery
   C. Increased capillary pressure
      1. Exercise (see above II.B.2.)
      2. Venous obstruction (long leg brace)
   D. Muscle hypertrophy
   E. Infiltrated infusion
   F. Nephrotic syndrome

from Mubarak et al. (1976)

The pathogenesis of chronic compartment syndrome can be seen in Figure 4. Wiley, Clement, Dolye and Taunton (1987) discuss the difficulties in finding the concise pathology of chronic compartment syndrome. Much research is still needed in this area.

Figure 4 The pathogenesis of chronic compartment syndrome



from Matsen (1975)

Tibialis Posterior: A Recognized Compartment.

Identifying the Compartment

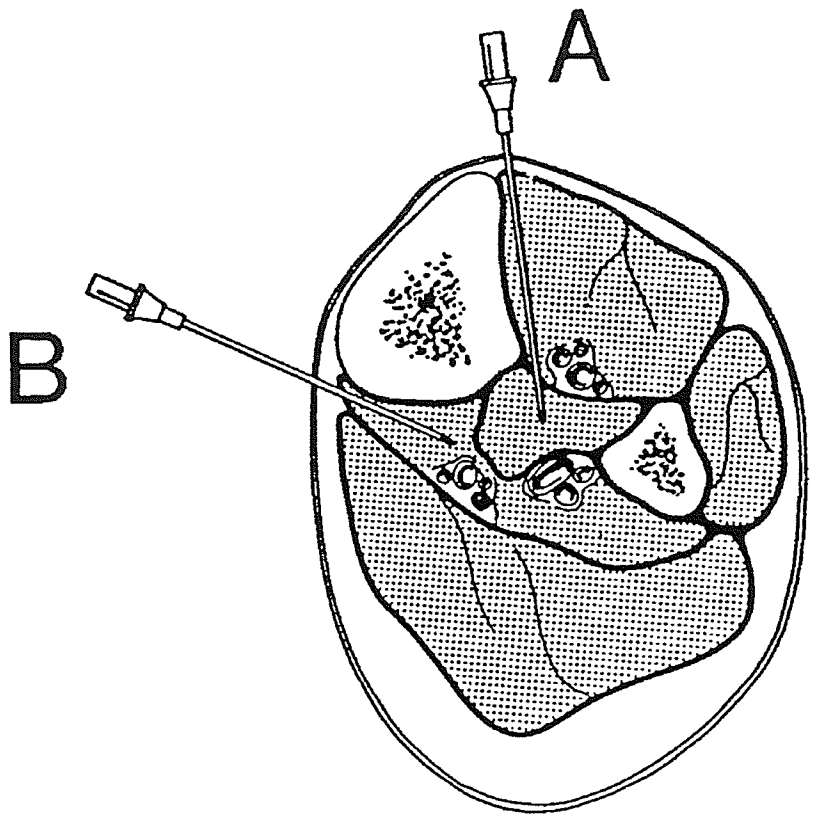
The tibialis posterior has recently been recognized as a separate compartment (Davey et al., 1984, and Rorabeck, 1986). As this compartment is surrounded by supporting vessels and also lies deep to all other compartments, measuring its physical characteristics via invasive techniques is very difficult.

Most studies measuring the deep posterior compartment invade the compartment medially just posterior to the tibia (Mubarak et al., 1982; Matsen, 1976; Puraneu, 1974). This places the catheter tip within the flexor digitorum longus muscle. With the deep transverse intramuscular septum separating the tibialis posterior from the flexor digitorum longus and flexor hallucis longus, pressure within the tibialis posterior compartment is overlooked.

Davey et al. (1984) discussed a technique to invade the tibialis posterior compartment. A slit catheter was introduced through the anterior compartment and interosseous membrane into the tibialis posterior muscle. Proper placement was acknowledged when a distinct pop was felt as the catheter penetrated the interosseous membrane and was positioned into the tibialis posterior compartment, posterior to the membrane (Davey et al., 1984).

With the use of cadavers, Davey et al. (1984) supported the hypothesis that the tibialis posterior was in a separate compartment. Invasion of the tibialis posterior via the previously mentioned technique and the flexor digitorum longus via Mubarak's et al. (1982) technique, allowed for a comparison of both compartment pressures (see Figure 5).

Figure 5 Cross section of leg demonstrating placement of catheter



Cross-section of leg demonstrating placement of the needle for pressure measurements in (A) the tibialis posterior muscle and (B) the deep posterior compartment.

from Davey et al. (1984).

Ten milliliters of lubricating jelly was injected into the tibialis posterior of three fresh cadaver specimens. An average increase in the pressures of the tibialis posterior compartment was 53 mmHg. A fasciotomy of the deep posterior compartment through a posteromedial incision (Mubarak & Owen, 1977) had no effect on the pressure measurements in the tibialis posterior muscle. A lateral incision along the flexor hallucis longus also had no effect. A sub-periosteal reflection of the flexor hallucis longus off the fibula had no effect. A release of the fascia enclosing the tibialis posterior muscle was the

only procedure that resulted in a fall of pressure, down to an average of 6 mmHg above starting pressures.

Davey et al. (1984) also proved differences in pressures from the two compartments in human studies, following treadmill exercise (see Table 3). Rorabeck (1986) also found differences between the two compartment pressures in human studies using five patient (see Table 4).

Table 3

Intracompartmental pressure measurements in the tibialis posterior muscle and the deep posterior compartment of the leg following exercise on treadmill

Patient	Compartment	Postexercise Pressure (mm Hg)		
		Insertion	15 minutes	1 hour
Control	Tibialis posterior muscle	10.6	9.5	
Case 1	Tibialis posterior muscle	22.5		11.5
	Deep posterior compartment	9.5		6.5
Case 2	Tibialis posterior muscle	15.5	9.5	
	Deep posterior compartment	1.3	1.2	

from Davey et al. (1984)

Table 4

Compartment Pressure (mmHg)

	Preexercise		*Postexercise*	
Patient	*Tib. Post.*	*D.P.C.*	*Tib. Post.*	*D.P.C.*
1	11	8	23	10
2	9	12	16	10
3	4	6	20	4
4	7	8	17	6
5	10	7	23	6

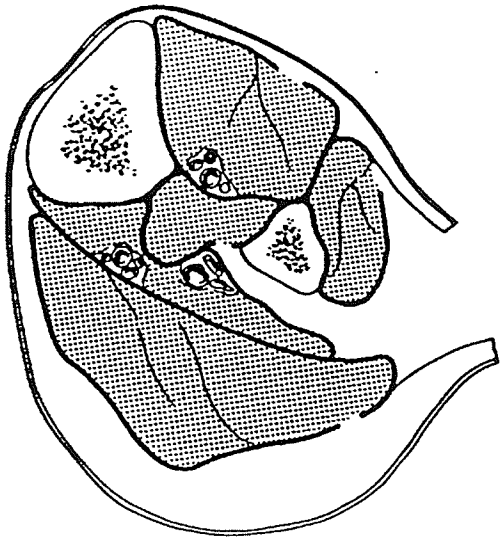
from Rorabeck (1986).

Decompression

As the tibialis posterior is now cited as being a separate and distinct compartment, it is necessary to produce safe and efficient techniques which will decompress this newly identified compartment. Davey et al. (1984) proposed compartmental release of all compartments could be performed through a single lateral incision (see Figure 6). This approach was quite efficient but carried some risk, as the incision was near many important neurovascular vessels. Mubarak's et al. (1977) double incision technique varies from Davey's et al. (1984) technique as incisions were made along both the medial and lateral border.

Rorabeck (1986) proposed a safer decompression of the tibialis posterior compartment. This was through a medial approach which was a continuation of the medial aspect of Mubarak's et al. (1977) technique. It consisted of an incision approximately 1 centimeter behind the posterior border of the tibia. The saphenous vein and nerve were protected while the fascia overlying the superficial posterior compartment was incised. The soleal bridge was released exposing the flexor digitorum longus muscle. The posterior tibial neurovascular bundle was identified distally and followed proximally and retracted with the flexor digitorum longus to expose the tibialis posterior. The fascia over the tibialis posterior was then incised. (see Figure 7). This approach seems much safer as there are no supporting vessels near the incision lines as in Davey's et al. (1984) technique.

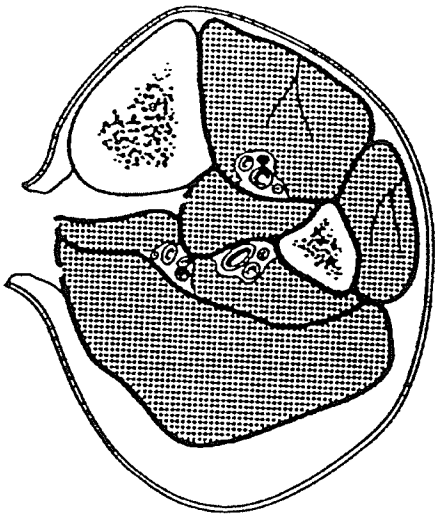
Figure 6



The interval between the superficial posterior and lateral compartments is developed. The flexor hallucis longus muscle is dissected subperiosteally off the fibula and retracted posteromedially. The fascial attachment of the tibialis posterior muscle to the fibula is identified and then incised decompressing the muscle.

from Mubarak et al. (1982)

Figure 7



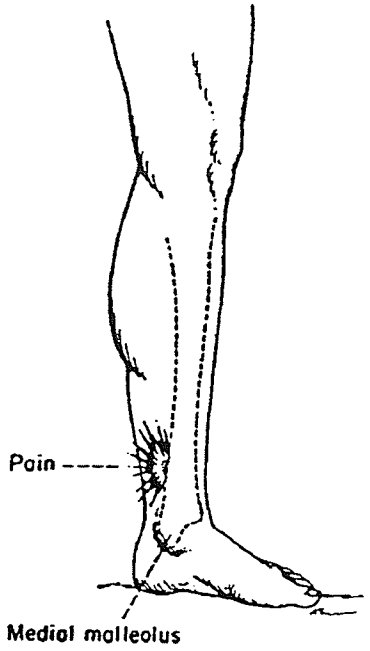
This illustrates the technique of fasciotomy of the deep posterior compartment. After retracting the flexor digitorum longus and identifying and protecting the posterior neurovascular bundle, the fascia overlying tibialis posterior is incised from its musculotendinous junction proximally.

from Rorabeck (1986)

Medial Tibial Stress and Compartment Syndromes

Medial tibial stress is defined as exercise-induced pain along the distal posterior-medial aspect of the tibia (Mubarak et al., 1982). (see Figure 9). Many researchers, Mubarak et al., 1982, Puranue, 1974, Matsen, 1975 and Rorabeck 1986, have hypothesized that the medial tibial stress is caused by a compartment syndrome in the deep posterior compartment. Most have failed in producing results to support this hypothesis, with the exception of Puranue (1974).

Figure 8 Area of tenderness resulting from medial tibial syndrome



Clinical findings of the medial tibial stress syndrome. There is a localized area of tenderness over the posteromedial edge of the distal one-third of the tibia (with permission from Mubarak SJ, and Hargens AR: *Compartment Syndromes and Volkmann's Contracture*. WB Saunders Co., Philadelphia, 1981).

from Mubarak et al. (1982)

Puranue (1974) studied the medial tibial syndrome and the mechanisms that produce it. Eleven subjects presented with clinical features of medial tibial syndrome. Each subject underwent an operation which consisted of a skin incision at the site of pain (along the medial border of the tibia). The deep and crural fascia was exposed. The fascial compartments of the flexor digitorum longus, flexor hallucis longus and tibialis posterior were split near the insertion on the medial border of the tibia. The skin was then closed.

All but one subject returned to pain-free activity after the operation was complete. Puranue (1974) concluded that the release of the deep crural

fascia and the fascia enclosing the flexor digitorum longus, flexor hallucis longus and tibialis posterior eliminated the medial tibial syndrome.

Puranue (1974) released all fascial compartments, including the tibialis posterior compartment. Thus, the study supported the hypothesis that medial tibial syndrome could be caused by a compartment syndrome in the deep posterior compartment.

The studies that did not support this hypothesis used pressure recordings from catheters that were placed in the flexor digitorum longus muscle via a medial approach. This placement does not consider the pressure of the tibialis posterior compartment as being unique to the pressure of the deep compartment. Increased pressure within the tibialis posterior compartment can be completely overlooked with a catheter inserted via a medial approach into the flexor digitorum longus muscle.

When considering the studies concerning medial tibial syndrome and compartment syndrome of the deep posterior compartment, a possible explanation to the controversial results could be that medial tibial syndrome is caused by a compartment syndrome occurring within the tibialis posterior muscle.

CHAPTER 3

METHODS AND PROCEDURES

Subjects

Each subject was required to read and sign an Informed Consent Form (see Appendix A) before becoming involved in the study. This form reviewed the tests to be carried out and also informed the subjects as to the possible risks present during the testing.

The experimental group consisted of patients who were recruited from the Pan Am Sports Medicine Centre and the University of Manitoba Athletic Therapy Clinic. Each experimental subject presented with medial tibial syndrome (pain along the posteromedial tibia). A pain questionnaire developed by the author (see Appendix B), along with a clinical diagnosis, were used to define and confirm the medial tibial syndrome. Bone scans were performed on subjects who were positively diagnosed as having medial tibial syndrome. Subjects with stress fractures were eliminated from the study. Clinical tests also eliminated any subjects presenting with soleus syndrome.

Literature did not reveal an accurate baseline for the comparison of the experimental group. Therefore, a control group was established to provide a baseline for comparison.

Subject Profile

Subjects from both groups were active running over 15 kilometers per week. The control group was matched as closely as possible to the

parameters of the experimental group. Comparison can be made from the subject profile tables below (Table 5 and Table 6).

TABLE 5
EXPERIMENTAL SUBJECTS PROFILE

SEX	AGE	HEIGHT	WEIGHT	TIME
M	25	175.0	70.9	15:10
F	24	172.5	70.5	17:00
F	19	165.0	60.9	16:23
F	21	170.0	61.4	14:00
F	23	167.5	61.4	14:16
Average	22.4	170.0	65.0	15:22
Range	19-25	165.0-175.0	60.9-70.9	14:00-17:00

Note: Time=time to onset of pain

TABLE 6
CONTROL SUBJECT PROFILE

SEX	AGE	HEIGHT	WEIGHT	TIME
F	21	155.0	51.8	16:00
F	22	155.0	50.0	17:10
F	23	162.5	55.5	19:00
Average	22	157.5	52.4	17:23
Range	21-23	155-162.5	50.0-55.5	16:00-19:00

Note: Time=time to exhaustion

The total sample group had an age range of 19 yrs to 25 yrs, a height range of 155 cm to 175 cm and a weight range of 50 kg to 70.9 kg. Exercise time parameters varied between groups as the experimental group mean was 15:22(range 14:00-17:00) and control group mean was 17:23 (range 16:00-19:00). A longer exercise regimen for the control group overall reveals that the control subjects had exercised as much or more than the

experimental group.

The procedure was performed on 15 subjects (8-experimental, 7-control). Data was recorded for 11 subjects (7-experimental, 4-control). Of these 11 subjects, 8 had complete data records (5-experimental, 3-control). Seven of the 15 subjects had missing data points. The causative factor for the missing data was the development of complications in the testing procedure, usually catheter plugging.

Experimental Design

The design of this study was a quasi experimental design, as the subjects were not randomly assigned but instead were patients who fit the definition of medial tibial syndrome. Control subjects were matched as closely as possible to the experimental group's paramters.

The research proposal was approved by the Committee on Research Involving Human Subjects on April 15, 1989, prior to the actual data collection (Appendix C).

Data Collection

The tested limbs underwent a slit catheter invasion into the deep tibialis posterior compartment via an anterior approach. A licensed physician performed the procedure.

Upon applying a local anesthetic to the superficial skin, the anterior approach introduced the needle and catheter anteriorly at an acute angle to the long axis of the limb, staying in contact with the lateral border of the tibia. The needle was pushed through the interosseous membrane after which

it was assumed to be lying in the belly of the tibialis posterior muscle. Literature cited a palpable "pop" could be felt by the physician while the catheter passed through the interosseous membrane. This distinct pop could be used to confirm correct placement. The palpable pop was not as distinct as literature cited with the subjects tested in this study. However, four cadaver trials were performed by the testing physician prior to this experiment to insure proper procedures were followed to ensure correct placement of the catheter. The catheter and the Stryker STIC portable transducer monitor were then fixed to the limb. The system was then flushed to exclude all air locks. The operating procedures for the catheter placement and hook-up to the Stryker STIC monitoring system can be found in Appendix D.

The system was allowed a 5 minute warm-up to eliminate any zeroing or calibration errors. It was then tested by squeezing the leg. A rise in pressure by the monitor confirmed that the system was functioning and the catheter was patent and correctly located.

The tested limb was also subjected to a number of passive and active movements before and after the catheter was placed. If both movements (before and after catheterization) elicited the same response, any pain caused by the catheter was eliminated.

If the system became blocked at any time during the test, (noticeable by a non-responsive change in pressure immediately after flushing) it was flushed with the heparenized solution placed within the system.

Once the catheter was placed and the equipment tested, a pre-exercise compartment pressure measurement and mean arteriole blood pressure were taken and recorded. The subject then began the exercise regimen which consisted of treadmill running until pain caused him/her to

stop. The treadmill protocol was the Bruce protocol (Fox & Mathews, 1981). Immediately after the completion of the exercise regimen, a compartment pressure measurement and mean arteriole blood pressure were taken.

The subjects then rested in a supine position with a cushion placed under the knee of the catheterized leg. This kept the posterior compartment of the lower leg off the table, eliminating any artifacts created by external pressure from the table. Post-exercise pressures were monitored at five minute intervals for a 30-minute post-exercise period. Pressures were taken every minute from exercise completion to the first five minute interval. Subject data sheets can be found in Appendix E. During the post-exercise period the experimental subjects determined the actual location of their specific pain on a body map (Appendix B).

Pressure measurements from the tibialis posterior compartment were compared to the various criteria found in the literature. The pressures were also subtracted from the mean arteriole blood pressure. If the difference was less than 40 mmHg a compartment syndrome was confirmed. Pressure differences greater than 40 mmHg ruled against a compartment syndrome.

Data Analysis

The complete data from seven of the eight subjects (5-experimental, 2-control) were compared as one subject's data was erroneously collected due to a low battery in the instrumentation. To determine significant differences between groups the Mann-Whitney U test statistic was used. This test was performed by comparing the two groups in both absolute and relative values. Contingency tables were also developed to evaluate the data according to the various criteria found in recent literature.

CHAPTER 4

RESULTS AND DISCUSSION

The purpose of this study was to determine if a compartment syndrome in the tibialis posterior was associated with medial tibial syndrome. The data was subjected to the various criteria found in recent literature. Comparisons between control and experimental groups in respect to both relative and absolute pressures were also performed.

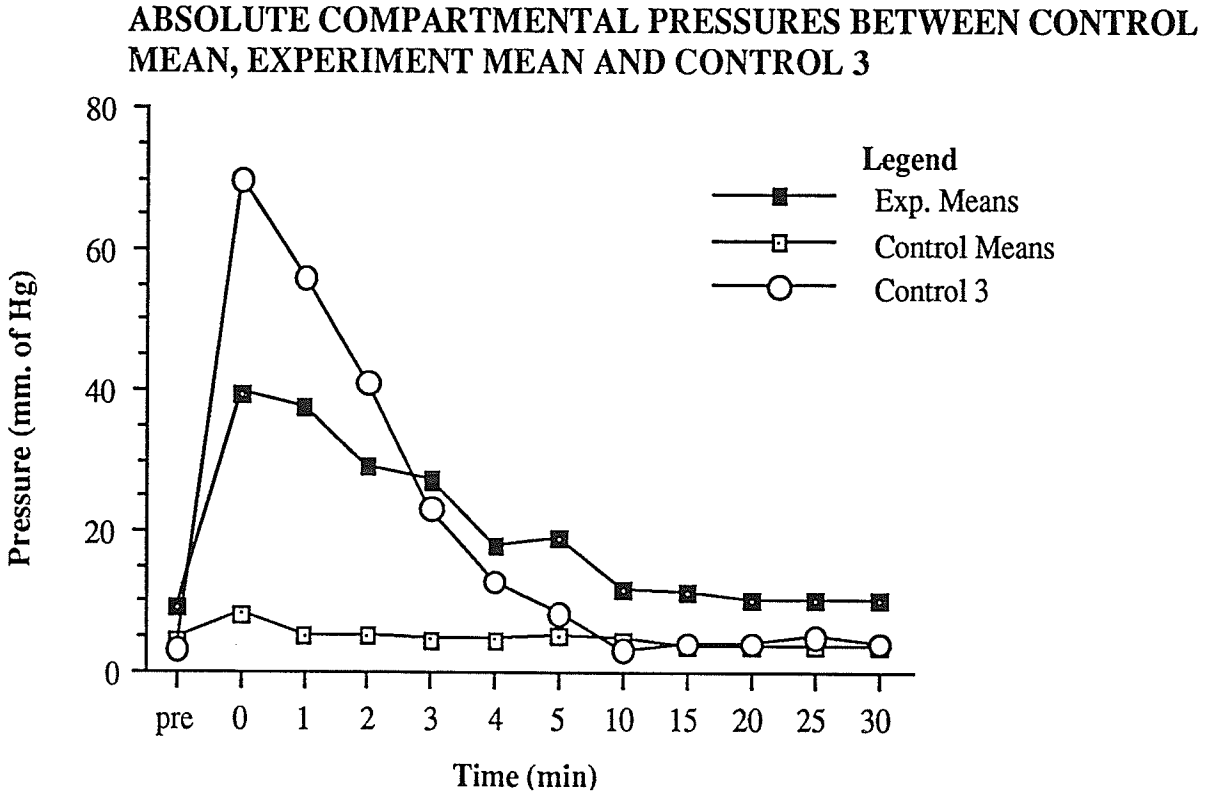
Compartmental Pressure Data

Fifteen subjects were initially tested (8-experimental, 7- control). Only eight of these subjects produced complete data for all data points. Four subjects did not produce any data as the catheter became plugged during the exercise regimen. Three more tested subjects completed the exercise regimen and data was obtained for parts of the recovery period. However, some data points were missing due to complications during recovery as again the catheter became plugged. Most plugging occurred during the exercise regimen and was rarely corrected through flushing. If the plugging occurred during recovery, the success of flushing the system to regain patency was quite good.

Eight subjects were tested with all data points recorded. One of these (control subject #3) was eliminated from the study due to instrumentation error. A comparison between experimental means, control means and Control #3 pressures can be found in Figure 9. A possible cause

for the deviation above both population means is that the pressure monitor was not functioning properly. The "low battery" warning signal came on during Control #3's testing. The only time that the signal presented was during Control #3's testing. All data from the other subjects were recorded in the absence of the "low battery" warning signal. The pressure monitor supposedly had the ability to record pressures for a period after the "low battery" signal had presented. The equipment operating procedures were unclear as to the length of time the monitor was able to record for after the signal had presented and the degree to which accuracy was maintained. Control #3 was excluded from the study because of a lack of confidence in the instrumentation.

Figure 9



The elimination of this subject left seven subjects with complete data points to analyze. Subject data tables for absolute and relative pressures of both experimental and control groups can be found in Appendix F.

Comparison Between Groups

Analysis of data was then performed on the remaining seven subjects. Compartmental pressures were analyzed as both absolute pressures and relative pressures. Absolute pressures were the actual pressures recorded from the monitor. Relative pressures were calculated by subtracting the absolute compartmental pressure from mean arteriole blood pressure [MABP = diastolic pressure + .33(systolic - diastolic)]. A comparison between experimental and control groups at both the absolute and relative pressures was performed by using the Mann-Whitney U test. Results revealed an overall p-value of .0528 when comparing the absolute scores and an overall p-value of .2453 when comparing relative scores. Tests at each time interval were also calculated to compare the two groups in both absolute and relative pressures. P-values can be found in Table 7, for all the time intervals of both the absolute and relative pressure scores.

The P-value of .0528 which represents the comparison of absolute pressure scores between experimental and control groups combined with a small sample size implies that there is an immense pressure difference between the experimental and control groups of this study. Statistically, a larger sample size would not need as great a difference between the two groups to produce significant p-values. Therefore, considering the data presented, a prediction of results from larger sample sizes would produce significant differences between groups, as such a small sample has produced near significant difference.

TABLE 7 P-Values (M-W U Test) for Comparison Between Absolute and Relative Pressures of Experimental and Control Groups

Intervals	Absol. Pres.	Rel. Pres.
Overall	**.0528**	**.2453**
Pre-exer.	.1714	.6985
Post-exer. 0	.0528	.2453
Post-exer. 1	.0507	.2453
Post-exer. 2	.1213	.6985
Post-exer. 3	.1213	.2453
Post-exer. 4	.1714	.1713
Post-exer. 5	.2453	.2453
Post-exer. 10	.1213	.4386
Post-exer. 15	.0786	.2453
Post-exer. 20	.0786	.1213
Post-exer. 25	.0786	.1714
Post-exer. 30	.1079	.2279

The p-values for the respective intervals in the absolute pressure comparison follow a general trend. The values representing the intervals at post-ex.-0 min. to post-ex.-1 min. are both near the .05 level of significance. At these intervals, the largest differences between the groups are present. From interval post-2 min. to post-30 min. the p-values become larger, representing a smaller difference between the two groups. The data then reveals an increase in the difference between the groups towards the end of the recovery period with smaller p-values. This explains why the majority of criteria set for the determination of compartment syndromes focusses on the immediate exercise completion and post-exercise 20-30 minute pressures. The difference between groups is greatest at these times. The pressure readings immediately following exercise completion are the most critical and also the most difficult to record.

This data is represented in two graphs. Figure 10 is the overall plot of the absolute pressures of each subject in both groups. Interval means comparing experimental and control groups reveal the obvious difference between the groups as seen in Figure 11.

Figure 10

OVERALL PLOT OF ABSOLUTE COMPARTMENT PRESSURES OF CONTROL (N=2) AND EXPERIMENTAL (N=5)

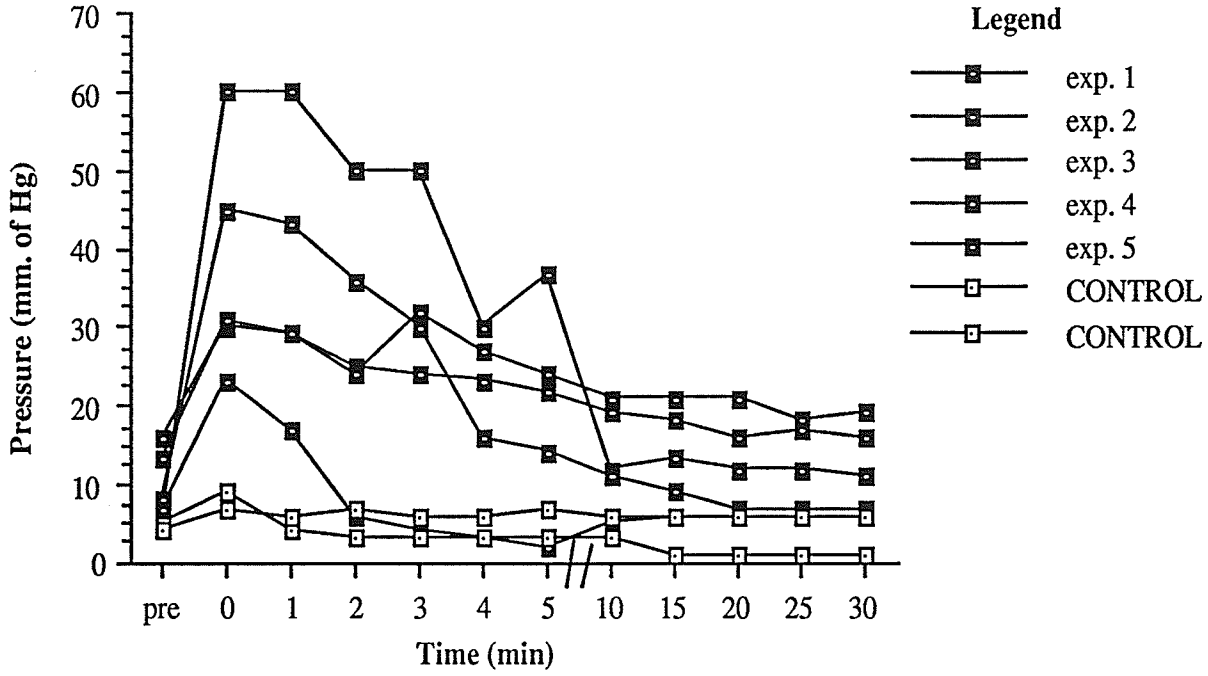
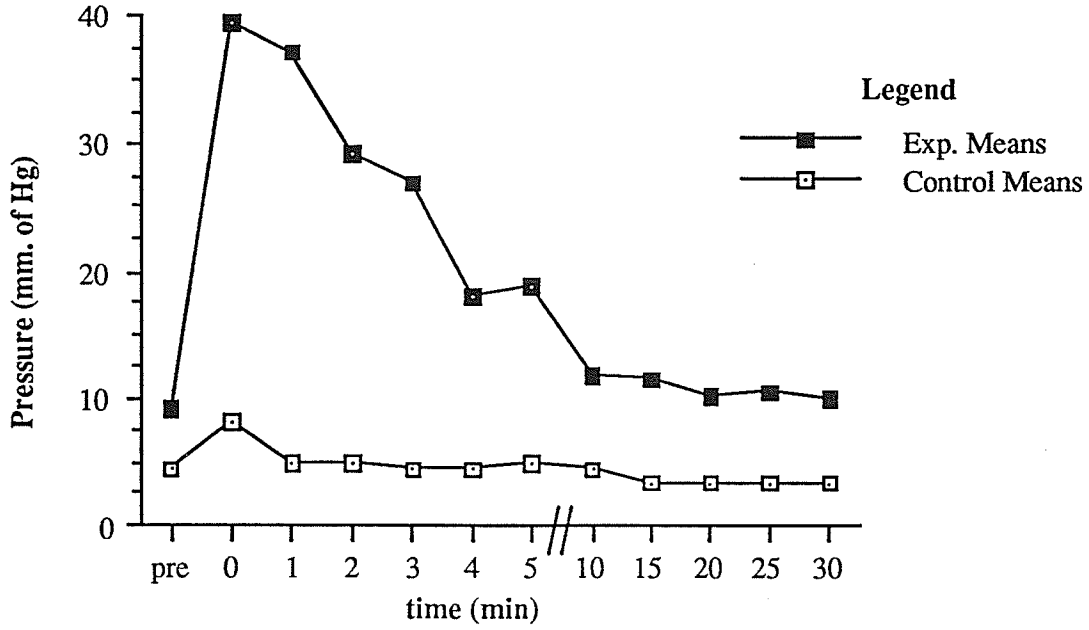


Figure 11

COMPARISON OF MEAN ABSOLUTE COMPARTMENTAL PRESSURES BETWEEN CONTROL (N=2) AND EXPERIMENTAL (N=5)



A comparison between the groups with respect to the relative pressures reveals no statistical significant difference. The p-values range from .1714 to .6985 throughout the intervals. The two groups fall within one range when compared on the bases of relative pressures. This can be seen in figure 12, where all subjects are plotted individually on one graph. There is no distinction between groups. However, when interval means are used to represent the groups, the two groups are seen as distinctly different populations (figure 13).

Figure 12

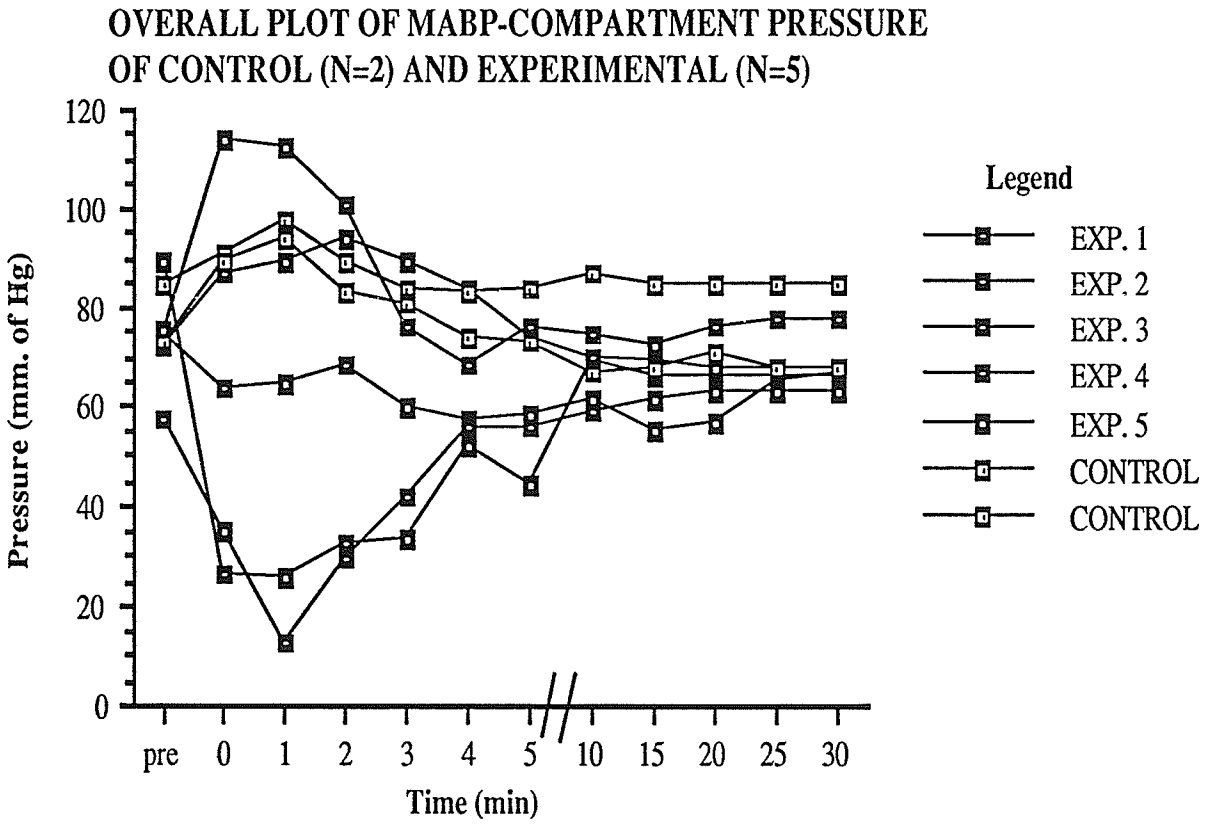
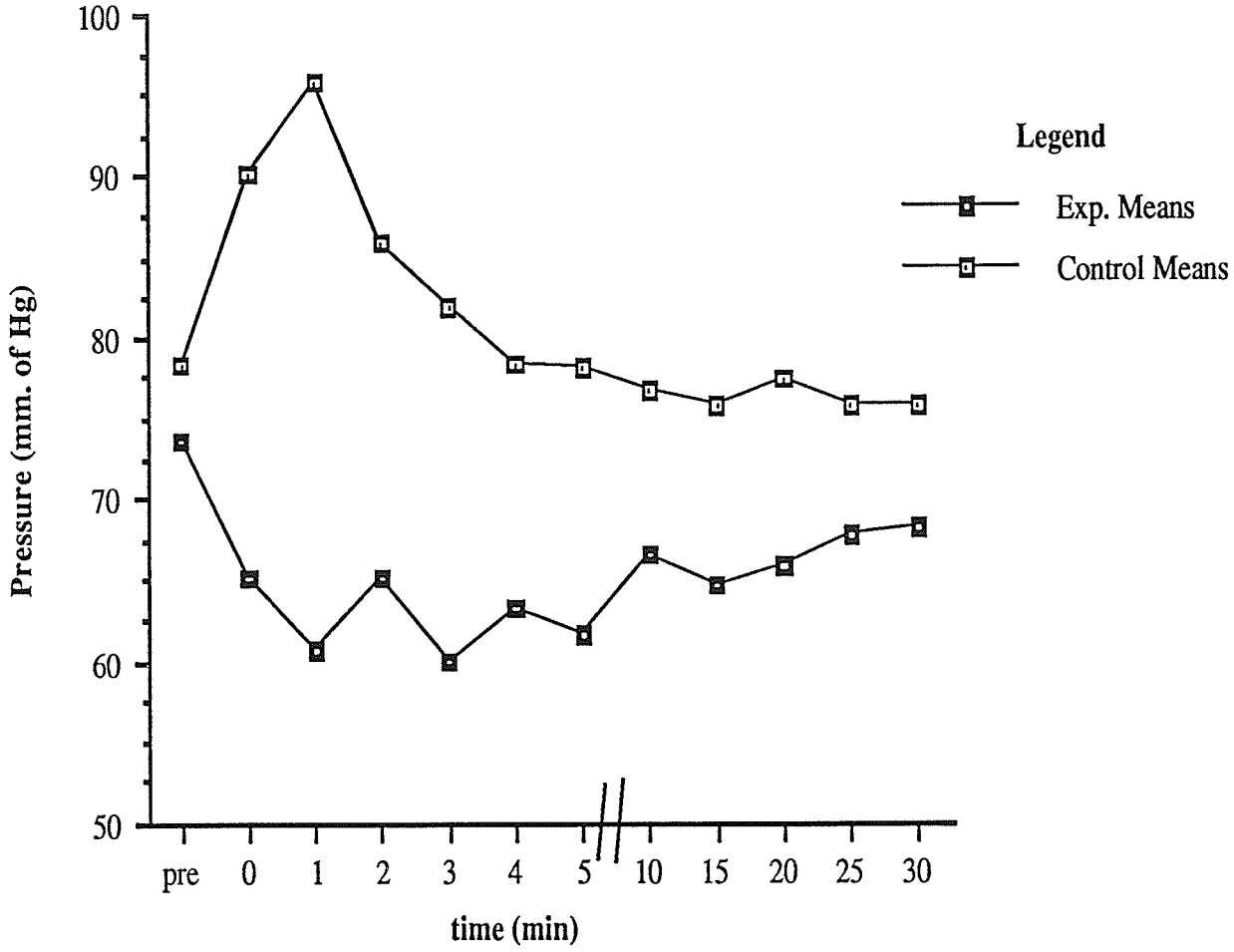


Figure13

COMPARISON OF MEAN ARTERIOLE BLOOD PRESSURE - COMPARTMENT PRESSURE BETWEEN CONTROL (N=2) AND EXPERIMENTAL (N=5)



Comparison to Literature Criteria

The remainder of the discussion in this chapter will be based on the assumption that medial tibial syndrome and tibialis posterior compartment syndrome correlate. From the previously discussed findings of this study, it is assumed that medial tibial syndrome is associated with tibialis posterior compartment syndrome.

Recent literature has produced a variety of criteria to determine whether or not a compartment syndrome is present. Most of these criteria focus on the compartment pressure levels as their main determining factor. Some of the criteria present conflict with other criteria. The criteria found in literature were administered to the pressure data points of the seven subjects and the results are plotted on contingency tables (Table 8). These tables produce the percentage of proper diagnosis of the seven subjects.

Table 8 Contingency Tables Comparing Literature Criteria Values to the Seven Subject's Pressures

Mubarak (1987)
(rest > 10 and post ex. 5 min > 25

	+	-
Exp.	1	4
Con.	0	2

3/7 = 43%

Styf (1987)
(rest > 35 and 20 min to normalize)

	+	-
Exp.	0	5
Con.	0	2

2/7=29%

Rorabeck (1986)
(post ex. 20 min >10)

	+	-
Exp.	3	2
Con.	0	2

5/7 = 71%

Allen & Barnes (1986)
(post ex. 0 > 40)

	+	-
Exp.	2	3
Con.	0	2

4/7 = 57%

Detmer (1986)
(anytime > 30)

	+	-
Exp.	4	1
Con.	0	2

6/7 = 86%

Melberg (1989)
(pre ex. >30, post ex. 0 > 30
6 min to normalize)

	+	-
Exp.	0	5
Con.	0	2

2/7 = 29%

Heppenstal (1988)
(MABP-CP < 40 anytime)

	+	-
Exp.	2	3
Con.	0	2

5/7 = 57%

Andrish (1984)
(> 40 deep post compart)

	+	-
Exp.	2	3
Con.	0	2

4/7 = 57%

By exposing the subjects results to the various criteria, it was found that some criteria had a higher percentage of diagnosing the subjects correctly than did others. Of the eight criteria being tested, Detmer's (1980) protocol has most accurately diagnosed the seven subjects with 86% accuracy. Only one of the five experimental group subjects was diagnosed as negative. Both control subjects were diagnosed negative, as could be expected. Rorabeck's (1986) criteria were the second most accurate according to the data, where 71% of the subjects were diagnosed properly. Two of the five experimental group subjects were diagnosed as false negatives.

The least accurate in diagnosing the data were Styf's (1987) and Melberg's (1989) criteria, where only 29% of the subjects were accurately diagnosed. None of the experimental group was diagnosed correctly. The high resting pressure of > 35 mmHg and > 30 mmHg respectively required to test positive, ruled out all of the experimental subjects. Other criteria within Styf's (1987) and Melberg's (1989) diagnostic protocol assessed many of the experimental subjects as testing positive. However, both researchers' criteria state that all conditions must be met in order to test positive.

None of the test criteria that were assessed diagnosed the control subjects as being positive, although many of the tests did fail to accurately diagnose the experimental group as positive. Some of the literature criteria seem to have set critical values too high to effectively descriminate between the normal population and compartment syndrome patients.

Literature's criterion to diagnose compartment syndromes has not accurately evaluated the experimental subjects as testing positive. However, the p-value of .0528 combined with the small sample sizes must elicit some curiosity. The inconsistency of the recent literature's criteria in testing experimental subjects as positive may not be the result of an accepted null hypothesis, where compartment syndrome therefore is not the cause of medial tibial syndrome, but rather inflated critical values in literature criteria. These inflated values guard against false positive diagnoses but may lead to increased occurrences of false negatives. More research is needed in the development of setting critical values for the determination of compartment syndromes.

CHAPTER 5

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Summary

Pain in the lower limb has been a controversial issue in past and present research. The term "shinsplints" is commonly used to define many of the lower leg pains. The purpose of this study was to try to associate a location of lower leg pain to a particular mechanism that produces pain. More specifically, the purpose of this study was to determine if a compartment syndrome in the tibialis posterior correlated with medial tibial syndrome.

Fifteen subjects were tested for compartment syndromes with a catheter placement into the tibailis posterior muscle compartment via an anterior approach. Eight subjects were patients who presented with medial tibial syndrome. Seven subjects represented the normal population. From the data collected, only seven subjects' results were analysed. Seven subjects were eliminated from the study because of incomplete data. Catheter plugging was a problem throughout the study. One subject was eliminated from the study because of a lack of confidence in the instrumentation. Statistical analysis was performed with a experimental group of five and a control group of two.

Compartment pressures were analyzed in both the absolute and relative form. Absolute pressures were recorded directly off the monitor and analyzed. Relative pressures were recorded as mean arteriole blood pressure minus compartment pressure. Comparison of absolute and relative pressures

between groups were performed using the Mann-Whitney U test statistic. Comparison to various literature cited criteria was also performed.

Conclusions

Based on the results of this study, the following conclusions appear to be justified:

1) A difference in pressure in the tibialis posterior muscle compartment was evident, though not statistically significant, when compartment pressures of normal population and patients presenting with medial tibial pain were compared.

2) The Stryker STIC monitoring system used to invade the tibialis posterior muscle compartment via an anterior approach, does not produce consistent pressure recordings as the procedure has a success rate of just over 50%. Exercising with a catheter placed in the tibialis posterior via an anterior approach causes bleeding and in turn catheter plugging.

3) The critical values cited in the literature, according to the data from this study, seem to be set too high. Although they safe-guard against a false-positive test, they may produce false-negative tests.

4) Clinical staff responsible for the diagnosis of potential injuries and possible treatments should be aware of the potential of compartment syndromes causing pain. On the basis of these results, compartment pressures in the tibialis posterior muscle compartment should be suspected when patients present with medial tibial syndrome.

Recommendations:

1) With an effective pressure recording rate of slightly over 50%, consideration of the instrumentation and procedure are necessary. A medial approach to the tibialis posterior will reduce the amount of soft tissue penetration and possibly reduce the amount of bleeding. Increasing the heparin-saline concentration in the catheter solution may also aid in the prevention of catheter clotting.

2) More research is needed to produce accurate critical values for determining the criteria used to diagnose compartment syndromes. A high critical value guards against false-positives but lends itself to more false negatives.

3) A continuation of data collection to add to this pool of data to improve the statistical significance of this comparison is recommended, even though the technique is questionable.

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APPENDICES

Appendix A

An Example of the Informed Consent Form

Informed Consent and Release

Explanation of tests

The following tests will consist of:
- -insertion of a pressure recording catheter into the deep posterior compartment of the injured leg.
- -after insertion is completed, an exercise session will be performed on a treadmill following the Bruce protocol.
- - pressure measurements will be recorded at five different times (pre-test, test, post-test (5min), post-test (10min) and post-test (15min).

The tests will be administered by a licensed physician. All results from the tests will be kept confidential.

Risks

I have read the above and understand that I may request that the testing be stopped or delayed at any time if I so desire. The testing may also be terminated if the tester observes any symptoms of distress. The risk of injury is minimal, with a slight chance of low grade infection and slight bruising of the muscle and subcutaneous tissue. A qualified physician will be there at all times to monitor the testing.

Waiver and Release

I understand that in participating in this study, I do so at my own risk and I undertake to indemnify the University of Manitoba and Pan Am Sports Medicine Center, its agents, officers and employees against any and all liability that may arise as a result of my participating.

_____ _____

Signature date

_____ _____

Witness signature date

Appendix B

An Example of the Lower Leg Pain Questionnaire

Name _____ Phone Number (home)_____
last given

(bus.)_____

Address _____
city/province

Postal Code _____ Birthdate _____/_____/_____ Sex _____
day month year

Height_____inches Weight_____pounds

Treating Physician _____

(I) **PAIN HISTORY**

1) When did this pain begin? Year:_____ Month:_____

2) Did this pain begin: gradually?_____ Suddenly?_____

3) How did this pain begin? [Mark (X) beside your response. An (X) may be placed beside one or more response.]

i) accident at work ____

ii) accident at home ____

iii) following an illness ____

iv) following surgery ____

v) following increased training levels ____

vi) following training startup after an inactive break ____

vii) pain just began ____

viii) other (eg. car accident) _____

4) Were there changes in your lifestyle during the 12 months before this pain began? (eg. new job, increased activity) Yes___ No___

If "Yes" describe._____

_____

_____

If "No" describe any regular activity you were participating in before the pain began._____

_____

_____

5) Is the pain the same now as it was when it began? Yes ___ No ___
If "No" how is it different? _____

_____

_____

(II) PAIN DESCRIPTION

6) Does the pain feel as if it is located in: [please mark (X)]
(more than one choice may be made)

bone_____ joint_____ muscle_____ nerve_____ skin_____ other_____

7) Choose one word group that most accurately defines your pain.

_____ continuous, steady, constant.

_____ rythmic, periodic, intermittent.

_____ brief, momentary, transient.

8) The following words represent pain of increasing intensity:

1.Mild, 2.Discomforting, 3.Distressing, 4.Horrible, 5.Excruciating,

Choose the word that best describes:

i) your pain right now _____

ii) your pain at its worst _____

iii) your pain at its least _____

iv) the worst toothache you ever had _____

v) the worst stomachache you ever had _____

vi) the worst headache you ever had _____

9) Using the body map on the following page:

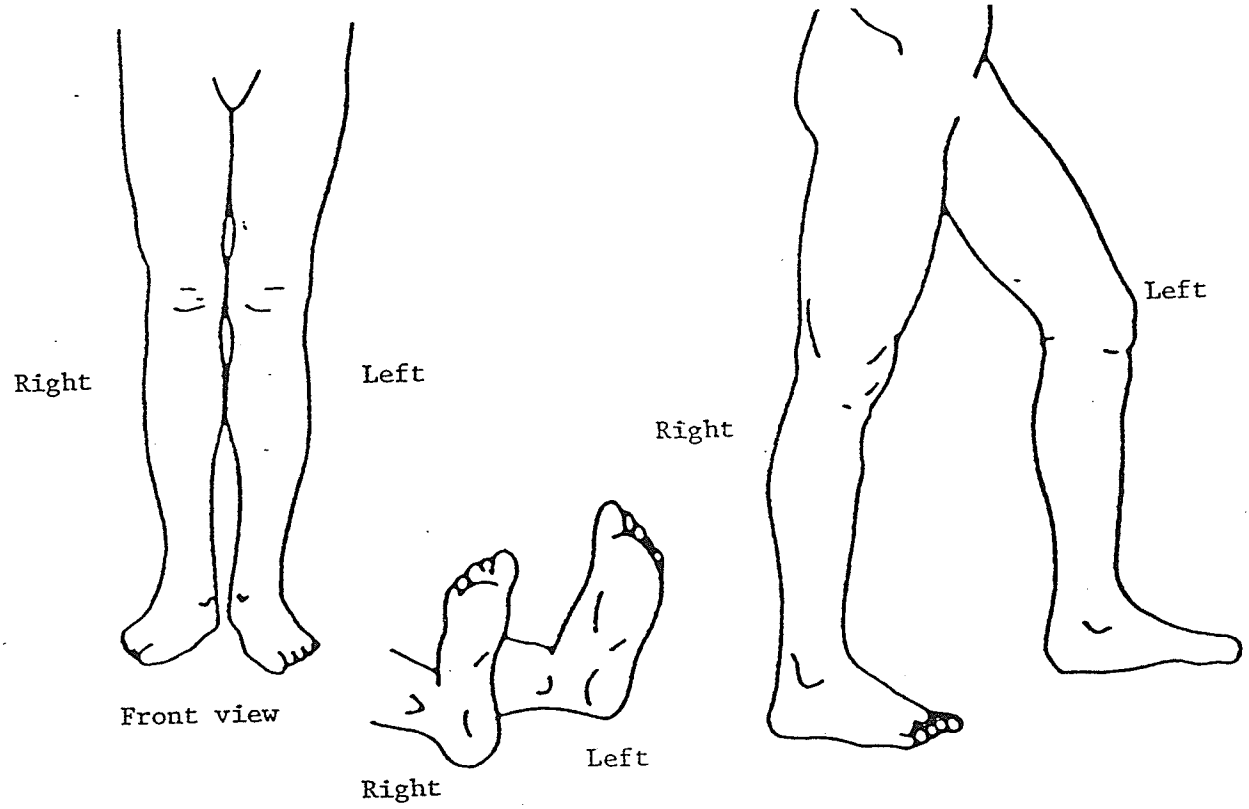
i) Mark the place where the pain is the greatest with a (X). If a whole area hurts, shade in that area with light pencil strokes (////).

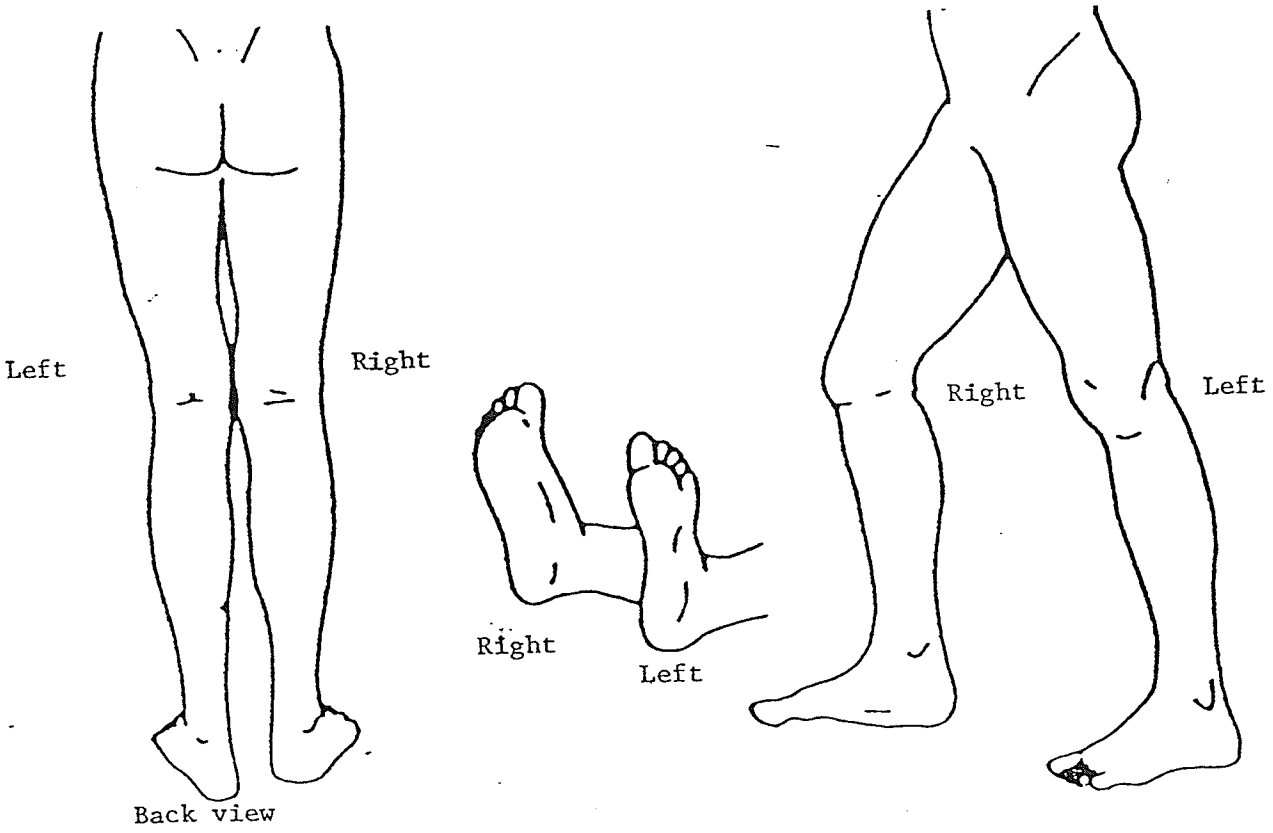
ii) If the pain radiates anywhere else use a dotted line and arrow to show how this usually occurs (------>).

iii) If the pain is internal (inside the body) put "I" beside the spot or area. If the pain is external (surface of the body) put "E" beside the spot or area. If both put "IE".

iv) Any areas where touch or applied pressure increases the pain can be indicated by the letter "T".

BODY MAP


Right
Left
Front view
Right
Left
Right
Left


Left
Right
Back view
Right
Left
Right
Left

(III) TIME PATTERN OF PAIN

11) Do you have the pain immediately on waking? Yes____ No____.
If "Yes" go to question 12.
If "No" when does it begin? (eg. afternoon) _____.

12) i) Does the pain change during the day? Yes____ No____.

ii) If "Yes" what part of the day is worse? _____.
If "No" go to question 12) iv).

iii) What part of the day is the pain better? _____.

iv) Does exercise affect the pain? Yes____ No____.
[If "Yes" answer question 13. If "No" you are finished. Thank You.]

13) a) Do you experience pain during intense exercise? (eg. training for competitive distance running) Yes ____ No ____.
[If "Yes" answer 13) b). If "No" go to question 14) a).]
b) Score the pain at its worst level.

1	2	3	4	5
mild		moderate		severe

How long does the pain continue after exercise has stopped? _____ hours. [Please continue to 14) a).]

14) a) Do you experience pain during moderate exercise? (eg. a jog in the park, cycling with a friend) Yes ____ No ____.
[If "Yes" answer 14) b). If "No" go to question 15 a).]
b) Score the pain at its worst level.

1	2	3	4	5
mild		moderate		severe

How long does the pain continue after exercise has stopped? _____ hours. [Please continue to 15) a).]

15) a) Do you experience pain during mild exercise? (eg. walking the dog, climbing stairs) Yes ____ No ____. [If "Yes" answer 15) a). If "No" you are finished. Thank You]
b) Score the pain at its worst level.

1	2	3	4	5
mild		moderate		severe

How long does the pain continue after exercise has stopped? _____ hours.

You have completed the survey. Thank You for your time.

Appendix C

Approval from Committee on Research Involving Human Subjects

FACULTY OF PHYSICAL EDUCATION AND RECREATION STUDIES

COMMITTEE ON RESEARCH INVOLVING HUMAN SUBJECTS

TITLE OF PROPOSAL:

Increased Tibialis Posterior Compartmental Pressure: A Possible Cause of Medial Tibial Syndrome.

PRINCIPAL INVESTIGATOR:

Pat Ilchena
Drs. W. Dahlgren, J. Irving, W. Hildahl

The Committee on Research Involving Human Subjects (Faculty of Physical Education and Recreation Studies) has evaluated the above proposal according to the criteria of the University of Manitoba Committee on Research Involving Human Subjects and finds it to be:

X	acceptable
_____	not acceptable

All conditions have been met.

April 5, 1989

D. Hrycaiko, Chair

Appendix D

Operating Procedures for Stryker STIC Monitoring System

Operating Instructions

QUICK S.T.I.C.

FOLLOW STERILE PROCEDURES

1. Open disposable pouch and remove contents.
2. Place needle firmly on tapered chamber stem. See figure 1.
3. Remove blue cap on pre-filled syringe and screw onto remaining chamber stem. See figure 1. Be careful not to contaminate the fluid pathway.
4. Open cover of monitor. Place chamber in well (black surface down) and push gently until disposable seats. See figure 2.
5. Snap cover closed — **Do Not Force.** Make sure latch has 'snapped'.
6. Pull clear end cap off syringe and attach plunger rod.
7. Hold the needle at approximately 45° up from horizontal. Slowly force fluid through the disposable to purge it of air.
   CAUTION: Do Not allow saline to roll down the needle into the transducer well.
8. Turn unit on now. Unit should read between 0 and 9 mm/Hg.
9. Approximate the intended angle of insertion of the needle into the skin. Press the 'zero' button. The display will read "00". See figure 3.
   NOTE: Display must read "00" before continuing. If it does not, please see troubleshooting section.
10. Insert needle into body. Slowly inject less than 3/10 cc of saline into the compartment to equilibrate with interstitial fluids.
11. Wait for the display to reach equilibrium
12. Read pressure.
13. **For Additional Measurements** — Turn the unit off and repeat steps 8-12. Make sure unit is rezeroed.
    NOTE: Pre-filled disposable syringe (each cc. contains: Sodium Chloride USP 0.9% and water for injection USP q.s.). A vehicle, solvent or diluent for parenterals.

CAUTION

Do not use pre-filled syringe if solution is discolored or contains a precipitate.

Disposable syringe is for single use only. Discard unused portion.

Federal law (U.S.A.) prohibits dispensing contents of syringe without a prescription.

Syringe solution and fluid path sterile, if protective caps are in place and package is intact.

Keep syringe at controlled room temperature. Keep from freezing.

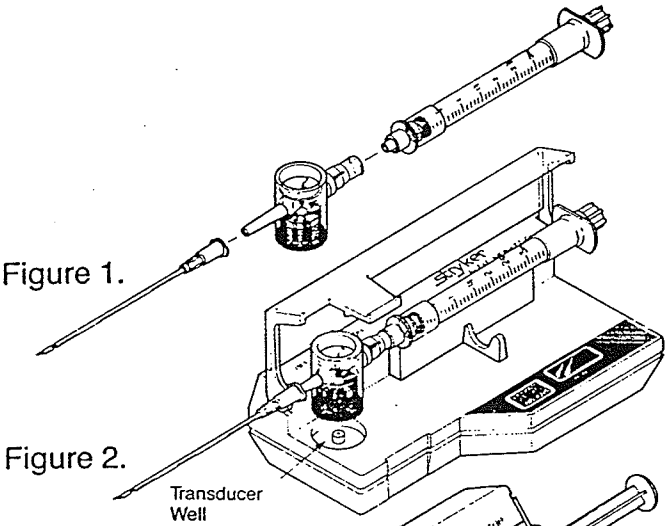


Figure 1.

Figure 2.

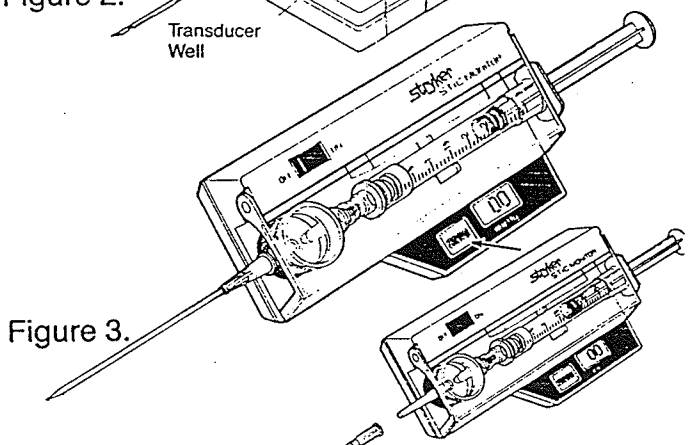


Figure 3.

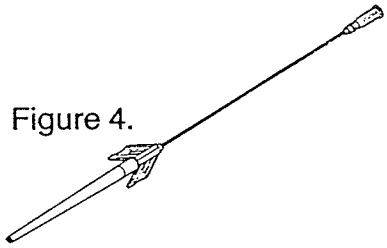


Figure 4.

Operating Instructions

INDWELLING SLIT CATHETER

FOLLOW STERILE PROCEDURES

NOTE: ALWAYS start procedure with a full syringe.

1. Remove the needle from the monitor.
2. Attach the slit catheter to the tapered chamber stem. Make sure it is a very snug fit. See figure 4. (Page 4)

   NOTE: Optional extension tubing may be used between the slit catheter and the monitor if added length is desired.
3. Remove the needle guard from the break-away needle and push the catheter in until it is just visible at the end of the needle.

CAUTION

Do Not let the catheter protrude from the needle as it may be cut by the needle upon insertion.

4. Hold the needle at approximately 45° up from horizontal. Slowly force fluid through the disposable to purge it of air.
5. Bring the catheter tip (slit end) to the same height as the diaphragm chamber. See figure 5.
6. Turn on the monitor and press the 'zero' button.

   NOTE: Display must read "00" before continuing. If it does not, please see troubleshooting section.
7. Hold the catheter and the needle together and push into the compartment. Make sure that the catheter's tip is protected inside the needle.
8. Push the catheter forward while pulling back on the needle. See figure 6. Tape the catheter in place after the needle is out of the skin.
9. Remove the break-away needle.

   NOTE: Pinch the green fins on the needle together; then, pull them apart and split the needle away. See figure 7.
10. Keep the chamber in the monitor at the same height as the estimated height of the slit tip of the catheter in the body. See Suggested Set-Up for the S.T.I.C. Pressure Monitor with Indwelling Slit Catheter Disposal Set. (Page 9).

    NOTE: Monitor may be secured to patient with monitor shield and tape.
11. Inject less than 3/10 cc of saline into the compartment to equilibrate with interstitial fluids.
12. Wait for the display to reach equilibrium.
13. Record the pressure readings at the appropriate intervals. Repeat step 11 to insure patency if necessary.

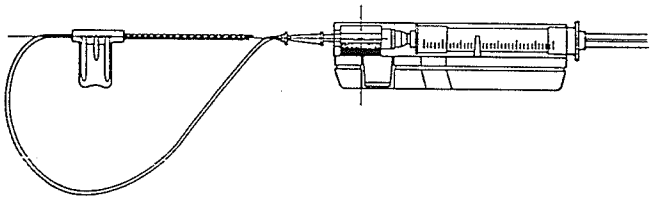


Figure 5.

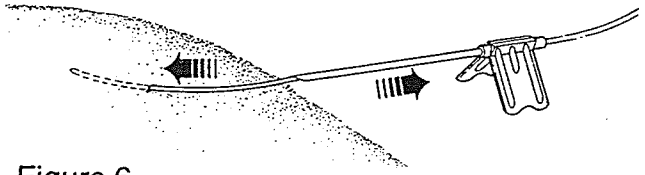


Figure 6.

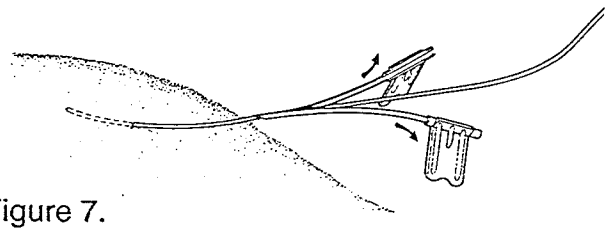


Figure 7.

Appendix E

Subject Data Sheet

SUBJECT DATA SHEET

Personal Data

Name_____ Phone # (home)_____
last given

(work)_____

Address_____ Postal code_____

Birthdate _____/_____/_____ Sex_____ Height_____(cm)

Weight_____(kg)

Subject test scores

Treadmill Score Time_____

	Compartment Pressure	(Blood Pressure) Systolic	Diastolic	MABP	MABP-CP
Pre-ex	_____	_____	_____	_____	_____
Ex-completion	_____	_____	_____	_____	_____
Post-ex (1 min)	_____	_____	_____	_____	_____
Post-ex (2 min)	_____	_____	_____	_____	_____
Post-ex (3 min)	_____	_____	_____	_____	_____
Post-ex (4 min)	_____	_____	_____	_____	_____
Post-ex (5 min)	_____	_____	_____	_____	_____
Post-ex (10 min)	_____	_____	_____	_____	_____
Post-ex (15 min)	_____	_____	_____	_____	_____
Post-ex (20 min)	_____	_____	_____	_____	_____
Post-ex (25 min)	_____	_____	_____	_____	_____
Post-ex (30 min)	_____	_____	_____	_____	_____

Appendix F

Subject Data Table

	A	B	C	D	E	F	G	H	I	J
1	COMPARTMENT PRESSURE ABSOLUTE (EXP=7 AND CONTROL=4)									
2										
3			EXP. 1	EXP. 2	EXP. 3	EXP. 4	EXP. 5	EXP. 6	EXP. 7	MEAN
4										
5	PRE-EX		16	6	7	11	16	8	13	11.00
6	EX-COMPLETION		30	60	23	16	40	45	31	35.00
7	POST-EX 1		29	60	17			43	29	35.60
8	POST-EX 2		25	50	6	8		36	24	24.83
9	POST-EX 3		24	50	4		10	30	32	25.00
10	POST-EX 4		23	30	3	2	10	16	27	15.86
11	POST-EX 5		22	37	2	4	10	14	24	16.14
12	POST-EX 10		19	12	5	6	9	11	21	11.86
13	POST-EX 15		18	13	6	9	9	9	21	12.14
14	POST-EX 20		16	12	6	8	13	7	21	11.86
15	POST-EX 25		17	12	6	8	10	7	18	11.14
16	POST-EX 30		16	11	6	7	10	7	19	10.86
17	Subject Means		21.25	29.42	7.58	7.90	13.70	19.42	23.33	18.44
18										
19										
20			CONTROL 1	CONTROL 2	CONTROL 3	CONTROL 4	MEAN			
21										
22	PRE-EX		8	5	3	4	5.00			
23	EX-COMPLETION			9	70	7	28.67			
24	POST-EX 1			4	56	6	22.00			
25	POST-EX 2		13	3	41	7	16.00			
26	POST-EX 3		12	3	23	6	11.00			
27	POST-EX 4		9	3	13	6	7.75			
28	POST-EX 5		5	3	8	7	5.75			
29	POST-EX 10		3	3	3	6	3.75			
30	POST-EX 15			1	4	6	3.67			
31	POST-EX 20			1	4	6	3.67			
32	POST-EX 25			1	5	6	4.00			
33	POST-EX 30			1	4	6	3.67			
34	Subject Means		8.33	3.08	19.50	6.08	9.58			

	A	B	C	D	E	F	G	H	I	J
1	MEAN ARTERIOLE BLOOD PRESSURE - COMPARTMENT PRESSURE (EXP=7 AND CONTROL=4)									
2										
3			EXP. 1	EXP. 2	EXP. 3	EXP. 4	EXP. 5	EXP. 6	EXP. 7	MEAN
4										
5	PRE-EX		74.0	89.3	71.7	61.0	64.3	57.3	75.1	70.39
6	EX-COMPLETION		63.3	26.7	87.0	87.3	28.7	35.0	114.0	63.14
7	POST-EX 1		64.3	25.3	89.3			12.3	112.0	60.64
8	POST-EX 2		68.3	32.7	93.7	88.7		29.3	100.9	68.93
9	POST-EX 3		59.3	33.3	89.3		58.6	41.7	75.5	59.62
10	POST-EX 4		57.0	52.0	83.4	82.7	58.6	55.7	68.5	65.41
11	POST-EX 5		58.0	44.3	73.7	72.7	57.3	56.0	76.0	62.57
12	POST-EX 10		61.0	68.7	69.7	65.3	54.3	59.0	74.5	64.64
13	POST-EX 15		55.3	66.0	68.7	81.0	53.7	61.0	71.8	65.36
14	POST-EX 20		56.7	66.0	67.3	82.0	49.0	63.0	75.8	65.69
15	POST-EX 25		65.0	66.0	67.3	82.0	52.0	63.0	77.5	67.54
16	POST-EX 30		66.7	65.7	67.3	83.0	52.0	63.0	77.8	67.93
17	Subject Means		62.41	53.00	77.37	78.57	52.85	49.69	83.28	65.16
18										
19										
20			CONTROL 1	CONTROL 2	CONTROL 3	CONTROL 4	MEAN			
21										
22	PRE-EX		84.5	84.1	77.5	72.6	79.68			
23	EX-COMPLETION			90.7	32.2	89.3	70.73			
24	POST-EX 1			97.7	37.0	93.7	76.13			
25	POST-EX 2		93.3	88.8	52.0	82.7	79.20			
26	POST-EX 3		87.7	83.4	64.7	80.4	79.05			
27	POST-EX 4		90.7	82.8	67.5	73.8	78.69			
28	POST-EX 5		93.1	83.5	73.1	72.8	80.63			
29	POST-EX 10		93.1	86.8	77.5	66.6	81.00			
30	POST-EX 15			84.2	65.9	67.2	72.43			
31	POST-EX 20			84.2	65.9	70.5	73.53			
32	POST-EX 25			84.2	65.9	67.2	72.43			
33	POST-EX 30			84.2	65.9	67.2	72.43			
34	Subject Means		90.39	86.22	62.09	75.33	76.33			

	A	B	C	D	E	F	G	H
1	COMPARTMENT PRESSURE ABSOLUTE (EXP=5 AND CONTROL=2)							
2								
3			EXP. 1	EXP. 2	EXP. 3	EXP. 4	EXP. 5	MEAN
4								
5	PRE-EX		16	6	7	8	13	9.25
6	EX-COMPLETION		30	60	23	45	31	39.50
7	POST-EX 1		29	60	17	43	29	37.25
8	POST-EX 2		25	50	6	36	24	29.25
9	POST-EX 3		24	50	4	30	32	27.00
10	POST-EX 4		23	30	3	16	27	18.00
11	POST-EX 5		22	37	2	14	24	18.75
12	POST-EX 10		19	12	5	11	21	11.75
13	POST-EX 15		18	13	6	9	21	11.50
14	POST-EX 20		16	12	6	7	21	10.25
15	POST-EX 25		17	12	6	7	18	10.50
16	POST-EX 30		16	11	6	7	19	10.00
17	Subject Mean		21.25	29.42	7.58	19.42	23.33	19.42
18								
19								
20			CONTROL 1	CONTROL 2	MEAN			
21								
22	PRE-EX		5	4	4.5			
23	EX-COMPLETION		9	7	8			
24	POST-EX 1		4	6	5			
25	POST-EX 2		3	7	5			
26	POST-EX 3		3	6	4.5			
27	POST-EX 4		3	6	4.5			
28	POST-EX 5		3	7	5			
29	POST-EX 10		3	6	4.5			
30	POST-EX 15		1	6	3.5			
31	POST-EX 20		1	6	3.5			
32	POST-EX 25		1	6	3.5			
33	POST-EX 30		1	6	3.5			
34	Subject Mean		3.08	6.08	4.58			

	A	B	C	D	E	F	G	H
1	MEAN ARTERIOLE BLOOD PRESSURE - COMPARTMENT PRESSURE (EXP=5 AND CONTROL=2)							
2								
3			EXP. 1	EXP. 2	EXP. 3	EXP. 4	EXP. 5	MEAN
4								
5	PRE-EX		74.0	89.3	71.7	57.3	75.1	73.48
6	EX-COMPLETION		63.3	26.7	87.0	35.0	114.0	65.20
7	POST-EX 1		64.3	25.3	89.3	12.3	112.0	60.64
8	POST-EX 2		68.3	32.7	93.7	29.3	100.9	64.98
9	POST-EX 3		59.3	33.3	89.3	41.7	75.5	59.82
10	POST-EX 4		57.0	52.0	83.4	55.7	68.5	63.32
11	POST-EX 5		58.0	44.3	73.7	56.0	76.0	61.60
12	POST-EX 10		61.0	68.7	69.7	59.0	74.5	66.58
13	POST-EX 15		55.3	66.0	68.7	61.0	71.8	64.56
14	POST-EX 20		56.7	66.0	67.3	63.0	75.8	65.76
15	POST-EX 25		65.0	66.0	67.3	63.0	77.5	67.76
16	POST-EX 30		66.7	65.7	67.3	63.0	77.8	68.10
17	Subject Means		62.41	53.00	77.37	49.69	83.28	65.15
18								
19								
20			CONTROL 1	CONTROL 2	MEAN			
21								
22	PRE-EX		84.1	72.6	78.35			
23	EX-COMPLETION		90.7	89.3	90.00			
24	POST-EX 1		97.7	93.7	95.70			
25	POST-EX 2		88.8	82.7	85.75			
26	POST-EX 3		83.4	80.4	81.90			
27	POST-EX 4		82.8	73.8	78.30			
28	POST-EX 5		83.5	72.8	78.15			
29	POST-EX 10		86.8	66.6	76.70			
30	POST-EX 15		84.2	67.2	75.70			
31	POST-EX 20		84.2	70.5	77.35			
32	POST-EX 25		84.2	67.2	75.70			
33	POST-EX 30		84.2	67.2	75.70			
34	Subject Means		86.22	75.33	80.78			