A COMPARISON OF ANTERIOR COMPARTMENT PRESSURES IN COMPETITIVE RUNNERS AND CYCLISTS

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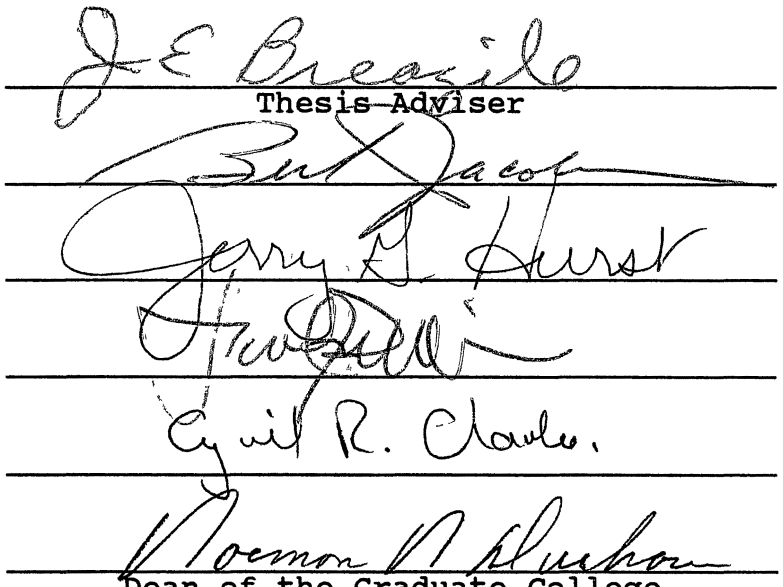

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CHAPTER I

INTRODUCTION

Chronic Anterior Compartment Syndrome

During the 1970's Americans experienced a renewed interest in health and exercise. This interest lead to the "running boom", which permanently changed the lives of many. Although interest in running has plateaued, it is still a popular sport as evidenced by the number of local and national road races. As more fitness enthusiasts began exercising, sports medicine emerged as a medical specialty.

Running injuries, in particular, were studied with much fervor as injured runners began filling physicians' offices. A study (17) conducted at the Institute for Aerobics Research in Dallas showed that injury rates were minimal in runners who ran less than 20 miles per week. However, injury rates increased exponentially in runners exceeding 20 miles per week. Many of the studies which have investigated running injuries have reported that the majority are overuse injuries caused by running too many miles or increasing mileage too rapidly. In fact, many world-class marathoners have drastically reduced their weekly training mileage in the last decade, emphasizing quality over quantity.

A common injury to running athletes is "shin splints" which is characterized by pain in the front of the lower leg. With shin splints, pain is induced by weight bearing activity and relieved by cessation of activity. Shin splints is a general term as it is commonly used to describe the same symptoms produced by four different mechanisms of injury. The four causes of shin splints (78) are: 1) strain to the posterior tibialis muscle; 2) tibial periostitis; 3) stress fracture of the tibia; and 4) interruption of the blood supply to the anterior compartment. Strain to the posterior tibialis muscle, caused by overuse, accounts for approximately 75% of all cases of shin splints (78). A less common, but more puzzling cause of shin splints involves fluid accumulation in the anterior compartment. Fluid accumulation elevates compartment pressure and decreases local blood flow. This condition is termed chronic anterior compartment syndrome.

Chronic anterior compartment syndrome has afflicted world-class runners such as Mary Decker-Slaney and Dick Quax. Its etiology has puzzled researchers and physicians. The anterior compartment is surrounded by a relatively inelastic fascial covering and contains the tibialis anterior, extensor digitorum longus and extensor hallucis longus muscles. These muscles, particularly the tibialis anterior, are extensively utilized during running and may be damaged, with disruption of the sarcolemma. Mechanical damage to myofibers increases the osmolality of

interstitial fluid (50). Increased osmolality increases capillary filtration which further increases intracompartmental pressure. Eventually, venous outflow is compromised, resulting in ischemia.

Currently, the only treatment is surgical opening of the fascia which surrounds the compartment (fasciotomy). Post surgical reports suggest that 60 to 100% of patients' symptoms are relieved by fasciotomy (72). In some patients, anterior compartment syndrome reoccurs or peroneal nerve entrapment results in continued pain after surgery.

To date, a review of literature reveals no cases of anterior compartment syndrome in bicyclists. Cycling is a non-weight bearing activity which requires the tibialis anterior muscle to contract concentrically. During weight bearing activities which involve running, the muscles of the anterior compartment demonstrate concentric as well as eccentric muscle contractions (61). The eccentric muscle contraction characteristic of weight bearing activity may contribute to increases in compartment pressure. This suggests that anterior compartment pressures may be lower during cycling than running. Research involving anterior compartment pressures in cyclists may provide scientists with clues to the etiology of compartment syndrome in runners. Furthermore, if pressures are lower during cycling than running, cycling may provide a valuable therapeutic alternative to surgical fasciotomy.

Statement of the Problem

This study investigated anterior compartment pressures in competitive runners and cyclists at equivalent workloads. To date, compartment pressures have not been quantified during cycling or at equivalent workloads as determined by oxygen consumption during running. Cycling, which involves only concentric contractions of the musculature of the anterior compartment may have a different effect on compartment pressure than running. During running, the musculature of the anterior compartment must utilize both eccentric and concentric muscle contractions to control foot placement. The eccentric contraction utilized during running may elevate compartment pressure in runners. Hence, measurement of anterior compartment pressures at equivalent workloads in competitive runners and cyclists would yield valuable information about anterior compartment pressure during two different types of exercise. This information may be useful in prescribing alternative activities for persons with chronic anterior compartment syndrome.

Null Hypotheses

1. Anterior compartment pressures measured at rest in competitive runners and cyclists will not be significantly different.
2. Anterior compartment pressures measured in competitiverunners immediately after completion of a 20 minute run on a treadmill at 80% of maximal oxygen

consumption will not be significantly different than pressures measured in competitive cyclists immediately after completion of 20 minutes of cycling at 80% of maximal oxygen consumption.

3. Compartment pressures measured in competitive runners immediately following a maximal treadmill test will not be significantly different than pressures measured in competitive cyclists immediately after a maximal cycling test.

4. The change in CPK enzyme levels measured before and after 20 minutes of exercise at 80% of VO2 max in competitive runners will not be significantly different than the change in CPK enzyme levels measured in competitive cyclists.

Limitations of the Study

1. All subjects are volunteers, eliminating a true random sampling procedure.

Delimitations of the Study

1. The subjects were limited to 10 competitive male distance runners and 10 competitive male cyclists age 18 to 38 years of age who met participant guidelines and who volunteered to participate in the study.

Assumptions

1. No medical or other problems occurred between

measurement periods to affect any of the physiological parameters measured.

2. No training effects were expected to occur between the two test periods.

3. Heart rate has a direct linear relationship to oxygen consumption.

Definition of Terms

Conceptual

Anaerobic Threshold. The level of oxygen consumption above which metabolic acidosis and associated changes in ventilation and gas exchange occur.

Anterior Compartment. Located on the anterior, lateral aspect of the lower leg, this compartment contains the tibialis anterior, extensor digitorum longus and extensor hallucis muscles.

Chronic Anterior Compartment Syndrome (CACS). A condition characterized by fluid accumulation in the anterior compartment which decreases local blood flow resulting in ischemia.

Concentric Muscle Contraction. A type of muscle contraction which involves muscle shortening and produces movement as the internal force of contraction exceeds the external force.

Eccentric Muscle Contraction. A type of muscle contraction which involves muscle lengthening and produces

controlled movement when external force exceeds the force of muscular contraction.

Electrocardiogram (EKG). A recording of the changes in electrical potential of the heart as detected through surface electrodes (8).

Maximal Oxygen Consumption (VO_2 Max). Maximal volume of oxygen consumed per kilogram of body weight per minute (ml/kg/min). The highest value for oxygen consumption obtained during an exercise test of gradually increasing intensity, VO_2 max is an indicator of one's level of fitness (21).

Maximal Symptom-Limited Stress Test. An exercise test performed on an ergometer which utilizes incremental increases in workload until the subject is exhausted or signs or symptoms necessitate test termination (56).

Mean Arterial Blood Pressure. Diastolic blood pressure + 1/3 pulse pressure.

Mean Muscle Pressure (MMP). Calculated during exercise by adding muscle relaxation pressure to half of the pressure amplitude (muscle relaxation pressure + 1/2 muscle contraction pressure) (82).

Muscle Contraction Pressure (MCP). The maximal value for intramuscular pressure measured during muscle contraction (82).

Muscle Relaxation Pressure (MRP). The lowest value obtained for intramuscular pressure between two consecutive muscle contractions (82).

Submaximal Workload. Work or exercise at less than maximal intensity.

Functional

Competitive Cyclist. Cyclist licensed by the United States Cycling Federation or who competes an average of 2 times per month in citizen's races, and has been competing for 1 year or more in endurance competitions and averages a minimum of 150 miles per week.

Competitive Runner. Distance runner who competes at a collegiate or national level, averages a minimum of 40 miles per week and has been competing consistently for 1 year or more in endurance competitions.

Endurance Competitions. Endurance races which require runners to complete a distance of 8 kilometers or more and cyclists to complete a distance of 25 miles or more in one race.

Description of Instruments

Indoor Trainer. A device which supports a bicycle for use indoors and applies variable resistance to the back wheel.

Intra Compartmental Pressure Monitor. A solid state transducer designed to measure tissue fluid pressures in mm Hg.

Metabolic Cart. A portable machine designed to measure oxygen consumed, CO_2 produced and ventilation at rest or during exercise.

Pressure Cuff. A cuff placed around the subject's upper arm which is inflated and deflated to obtain blood pressure measurements.

Motorized Treadmill. A machine with a movable belt which can be adjusted to operate at different speeds and inclines.

Sphygmomanometer. A column of mercury with a scale for measuring blood pressure.

Stethoscope. An instrument used to detect heart sounds when placed over the brachial artery during cuff deflation.

Surface Electrodes. Sensors attached to the skin which transmit electrical impulses of the heart into an EKG machine for a written recording.

CHAPTER II

A SELECTED REVIEW OF LITERATURE

Chronic Anterior Compartment Syndrome

Chronic anterior compartment syndrome is a condition characterized by exercise-induced pain over the anterior aspect of the lower leg. Swelling due to increased compartment volume as well as impaired nerve and muscle function may accompany the exercise-induced pain which is relieved by rest. Styf (83) stated that 30 to 50% of patients with chronic compartment syndrome report tenderness during palpation of the anterior margin of the tibia.

The anterior compartment, one of the four compartments in the lower leg of humans, is surrounded by fascia. Located between the tibia and fibula, the anterior compartment contains the tibialis anterior, extensor digitorum longus, extensor hallucis longus muscles, the deep peroneal nerve and the anterior tibial artery and vein. The location of the anterior compartment between the tibia and fibula increases compartment rigidity. The strong, relatively impermeable fascial covering (65) of the anterior compartment may also contribute to the elevated intramuscular pressures which are characteristic of chronic anterior compartment syndrome, often termed CACS. Elevated

intramuscular pressures impede blood flow to the exercising muscles and result in ischemic pain (83). In 20-60% of cases (65, 67), muscle hernias were reported, suggesting that elevated compartment pressures were present for a long time.

Pathophysiology of Anterior Compartment Syndrome

Since perfusion of contracting skeletal muscle occurs between contractions (24), muscle relaxation pressures have been targeted for study. Styf and Korner (82) investigated intramuscular pressures and muscle blood flow during exercise on an ankle dorsiflexion/plantar flexion ergometer in nine patients with chronic anterior compartment syndrome. Utilizing the 133-xenon clearance technique (48), they measured muscle blood flow in the anterior tibialis muscle, muscle blood flow, muscle relaxation pressure (MRP), muscle contraction pressure (MCP) and mean muscle pressure (MMP) in symptomatic and asymptomatic legs were compared. Mean muscle pressure was calculated using the following formula: MMP = [(MCP - MRP)/2] + MRP. Muscle relaxation pressure increased in both asymptomatic and symptomatic legs but was significantly higher in the symptomatic legs. Styf and Korner (80) reported a 15-25 mm Hg increase in muscle relaxation pressure in healthy individuals. Furthermore, the development of pain and impaired muscle function during exercise correlated with decreased muscle blood flow and

increased muscle relaxation pressure. Changes in muscle blood flow, however, were not related to significant changes in mean muscle pressure or muscle contraction pressure. They concluded that muscle blood flow was impeded when intramuscular relaxation pressures exceeded 35 mm Hg. Also, the onset of symptoms correlated with a decreased difference between mean arterial blood pressure and muscle relaxation pressure.

In a similar study, Styf and Korner (81) measured muscle relaxation pressure during and after ankle ergometer exercise. They reported elevated muscle relaxation pressure during and immediately after exercise. Time required for normalization of pressure after exercise was prolonged. They also reported that elevated values for MRP were associated with the development of pain, swelling and impaired muscle function (muscle weakness with active dorsiflexion). Since MRP was not correlated with impaired muscle function after exercise, Styf and Korner concluded that MRP was dependent on the volume of fluid within the anterior compartment and not the development of muscle dysfunction. Furthermore, MMP was not significantly different in symptomatic and asymptomatic legs and hence, would not be useful in diagnosing chronic anterior compartment syndrome. This was explained by the fact that muscle contraction pressure, which increased as the force of muscle contraction increased, decreased with the development of pain. Therefore, the decrease in muscle contraction

pressure that accompanied pain would result in a lower pressure amplitude which would decrease rather than increase mean muscle pressure.

Qvarfordt et. al. (65) studied 15 patients with lower leg pain of unknown cause. Researchers measured intramuscular pressures, systolic pressure of the great toe, muscle blood flow and venous emptying at rest, during exercise and after exercise utilizing an ankle ergometer. Muscle biopsy specimens were also extracted from the tibialis anterior muscle prior to exercise and immediately following exercise at a maximal workload. Muscle biopsies were analyzed for lactate, water content, adenosine triphosphate (ATP) and phosphocreatine (CP). Intramuscular pressures were elevated at rest, during exercise and after exercise for a period of 40 minutes compared to normal subjects. The water content in the tibialis anterior of symptomatic patients was also elevated after exercise. This supports the hypotheses of other researchers that increased transudation of fluid from the capillaries into surrounding tissues during exercise increases intramuscular pressure (36, 43, 71). Lactate levels measured in muscle biopsies immediately after exercise were also elevated. This is consistent with the reduction in blood flow which occurs during exercise in patients with chronic anterior compartment syndrome. Pre-exercise ATP and CP concentrations were normal in biopsies taken before and after fasciotomy. Concentrations of ATP and CP in muscle

biopsies extracted after exercise both before and after fasciotomy were significantly lower. Exercise-induced depletions of ATP and CP were normal.

Other studies report decreased muscle perfusion when intramuscular relaxation pressures exceed 30 to 50 mm Hg (1, 6, 36, 76). These observations are consistent with the fact that capillary blood pressure in muscle ranges from 20-30 mm Hg. Intracompartmental pressures exceeding 30 mm Hg also probably obstruct lymphatic flow further limiting fluid drainage (67).

Etiology of Chronic Anterior Compartment Syndrome

The etiology of compartment syndrome is unknown; however, factors such as exertion, trauma and venous diseases have been proposed as contributing factors. Since many patients diagnosed with CACS report high levels of physical activity, muscular hypertrophy which exceeds the elasticity of the surrounding fascia has also been suggested as a cause of CACS (83). A large percentage of patients with CACS are distance runners, sprinters, or athletes who compete in weight-bearing sports such as skiing, football, basketball or dancing, with some reports in swimmers. The literature reports only a few cases of CACS in bodybuilders and powerlifters; therefore, microtrauma and excessive stress to capillary beds and lymphatics in muscle tissue due

to strenuous exercise have been suggested as more plausible mechanisms for CACS then hypertrophy (83).

Exertion. During high intensity exercise in asymptomatic, healthy individuals, blood flow to the exercising muscles increases, blood vessels dilate and muscle contraction and relaxation pressure increases with increasing force of contraction (44, 75). Mean muscle pressure investigated at submaximal workloads in healthy individuals has been reported to remain constant with increased workload (41). During exercise in healthy individuals, muscle volume increased by 20%, contributing to the increase in intracompartmental pressure (71). The increase in compartmental volume with exercise occurs as a result of increased capillary filtration due to increased capillary hydrostatic pressure and capillary surface area (67). This transudation of fluid is not compensated for by accelerated lymphatic drainage (67). Increases in anterior compartment pressure with exercise also depend on the size and compliance of the surrounding fascia (67).

Styf (83) suggests that microtrauma to muscle tissue and excessive stress to the capillary bed and lymphatics of the muscle tissue may occur during strenuous exercise. These changes may lead to myositis and inflammatory reactions which result in increased capillary filtration. Rippe et al. (69) reported a 3 to 5 fold increase in capillary filtration pressure following myositis and inflammatory reactions. This results in an increase in

total intramuscular pressure. Eventually, blood flow is compromised by elevated compartment pressure and exercise must be terminated or the intensity of exercise must be decreased.

In addition to the inflammatory reactions associated with strenuous exercise, eccentric muscle contractions have been shown to damage the contractile apparatus, resulting in delayed onset of muscle soreness, swelling and tenderness when palpated (5, 30). Group IV sensory neurons which carry dull, diffuse pain (14) terminate in free nerve endings in the muscle connective tissue between fibers (57). Friden et al (31) suggest that these free nerve endings are activated by increased compartment pressure that develops during eccentric exercise.

Friden et al. (31) suggests that during eccentric contractions, the muscle, as well as the fascia, is stretched as the muscle resists external forces. This stretching of the compartment results in a narrowing of the anterior compartment which further reduces compliance. Friden et al. (29) found that eccentric exercise was accompanied by disturbances in myofibrillar cross-striated band patterns such as Z line streaming. Armstrong et al. (4) also demonstrated exercise-induced skeletal muscle injury following eccentric exercise in rats. In addition to these findings, Friden et al. (31) reported that the musculotendinous junction was the site of maximal point tenderness after eccentric contraction of the tibialis

anterior. They suggested that since muscle fibers are oriented most obliquely in the area just proximal to the musculotendinous junction, the ability of these fibers to tolerate high levels of tension produced during eccentric muscle contractions may be reduced. Eccentric contractions also increase muscle water content more than concentric contractions (32).

Intramuscular pressures recorded during eccentric exercise and afterwards for a period of 2 days were significantly higher than after concentric exercise (31). A study by Friden et al. (31) which investigated dynamic and resting intramuscular pressure in 8 healthy males during eccentric and concentric exercise reported that average peak intramuscular pressure measured during eccentric ankle exercise (236 mm Hg) was significantly higher than during concentric exercise (157 mm Hg). However, since none of the subjects reported experiencing any ischemic pain during either the concentric or eccentric exercise, they concluded that the high peak pressures were not fused and were followed by low muscle relaxation pressures (<10 mm Hg). Hence, pressure was not continuously elevated and would not lead to any sustained disruptions of flow in the muscle microvascular circulation. These findings in healthy individuals emphasize the role of elevated muscle relaxation pressures in the etiology of chronic anterior compartment syndrome.

Plasma levels of creatine phosphokinase (CPK), a primary cytoplasmic enzyme of muscle, are often used as an index of muscle damage (62). Page et al. (62) reported that total plasma CPK levels were elevated after eccentric exercise for a period of 6 days. Wolf et al. (86) also reported significant increases in total CPK levels in international-class, medium-distance runners within 1 hour after a 5-km run at maximal effort.

CPK is present as three major isoforms: CPK MM, CPK MB, and CPK BB. CPK MM is the predominant isoform of CPK in skeletal muscle and cardiac muscle; CPK MB is found primarily in cardiac muscle (62) and in smaller concentrations in skeletal muscle. CPK MB comprises 10-25% of total CPK activity in myocardium (3). In healthy, sedentary persons, serum CPK MB levels are undetectable by electrophoresis (86). However, studies by Apple et al. (3) revealed CPK MB levels in gastrocnemius muscle of runners comprised up to 10.5 % of total CPK activity. Furthermore, CPK MB levels were elevated in women after completion of a marathon. CPK BB is the dominant isoform of CPK in the brain.

Serum CPK MB levels have been utilized to differentiate cardiac from skeletal muscle damage (19). However, in trained athletes, the enrichment of skeletal muscle with CPK MB precludes the use of CPK MB as an index of myocardial damage. Electron microscopy studies (38, 85) of muscle biopsies from gastrocnemius muscle report

myofibrillar damage and fibrosis following a marathon. Based on these observations, researchers concluded that the source of CPK and CPK MB following exercise in trained athletes was most likely skeletal rather than cardiac muscle (3). This suggests that elevations in total CPK after eccentric exercise are related to skeletal muscle damage rather than cardiac muscle.

A study by Kosano et al. (45) provides further support for this conclusion. Kosano et al. studied changes in myogenic serum components during 93 hours of strenuous physical exercise. They reported that total CPK and CPK MB activity steadily increased throughout the period of exercise. In addition to CPK activities, they measured Lactate Dehydrogenase (LD) activities. LD is present in cardiac and skeletal muscle in 5 isoforms. An increase in the ratio of LD1/LD2 isoform is indicative of myocardial damage. Although, LD activity increased after exercise, the LD1/LD2 ratio decreased suggesting that the source of CPK and LD was skeletal muscle rather than myocardium.

A study by Cummins et al. (19) further confirmed these findings. After completing a study of male marathon runners, they reported that although serum CPK MB levels were elevated, the levels of cardiac troponin-I were within the normal range. Cardiac troponin-I levels were determined by post-precipitation double antibody competition radioimmunoassay.

Activities, such as running, require the tibialis anterior to contract eccentrically during foot-strike to prevent foot-slap in rearfoot strikers. It has been estimated that approximately 80% of distance runners are rearfoot strikers (70). During foot-strike the lower extremity must absorb shock equivalent to 200-250% of body weight (61) as the body continues in forward propulsion. The repetitive eccentric contractions associated with weight bearing sports damage the contractile apparatus and alter the structural integrity of the muscle cells. Friden et al. (29) suggest that muscle damage caused by eccentric muscle contractions results in the formation of protein components and is accompanied by the release of protein-bound ions. Globular proteins and damaged Z-proteins which are degraded by lysosomal enzymes in response to muscle damage may bind ions in the cytosol before their release into the interstitial fluid. Another study by Friden et al. (27) reported disruption of Z-bands following bouts of running downstairs. Hikida et al. (38) examined human muscle biopsies extracted before a marathon and at intervals for 7 days following the marathon. They reported disruption of the sarcolemma with an intact basal lamina in post marathon biopsies as compared to pre-marathon biopsies. Disruption of the sarcolemma resulted in the release of mitochondrial proteins and to a lesser extent, myofibrillar proteins into the extracellular space. Erythrocytes and macrophages were also found within the interstitial compartment. The presence

of erythrocytes in the interstitial fluid suggests that mechanical trauma to muscle may result in the disruption of blood vessels in the microcirculation during marathon running (38).

Eccentric exercise triggers an inflammatory response including the phagocytosis of damaged tissue by macrophages and neutrophils. During phagocytosis, oxygen consumption is increased; this leads to the generation of free radicals by mitochondrial enzymes, lysosomes, peroxisomes and cell surface, nuclear and endoplasmic membranes (12). Free radicals are directly utilized during intracellular phagocytosis or converted to reduced oxygen species. Free radicals and other reduced oxygen species are also secreted into the extracellular fluid, enhancing the inflammatory process. The deleterious effects of free radicals are, however, limited by antioxidant enzymes, many of which are essential nutrients. Antioxidants and essential components of antioxidants include vitamin C and E, beta-carotene, zinc, selenium, copper, iron, manganese, and taurine (13).

A study by Brady et al. (11) which investigated the antioxidant effects of vitamin E in rat liver showed that vitamin E reduced exercise-induced lipid peroxidation. Davies et al. (20) demonstrated that vitamin E deficiency increased tissue levels of free radicals in rats. They reported alterations in the sarcoplasmic and endoplasmic reticulum, in addition to lipid peroxidation. Vitamin E deficient rats also exhibited a reduction in exercise

endurance. This research suggests that a deficiency in the essential nutrients which are antioxidants or components of antioxidants may exacerbate tissue damage caused by eccentric muscle contractions.

Chronically, elevated levels of tissue damage related to inadequate nutrition may predispose an athlete to compartment syndrome by increasing the interstitial fluid osmolality. Increases in interstitial osmolality would then increase compartment pressure. Many athletes develop compartment syndrome after many years of participation in the activity which precipitated the compartment syndrome. These athletes may slowly develop a deficiency in one or more of the essential nutrients necessary for adequate antioxidant levels. A deficiency of antioxidants would enhance tissue damage and impede tissue recovery after an exercise bout. Hence, investigation of levels of antioxidants and analysis of dietary intake in persons with chronic compartment syndromes may warrant further investigation.

The presence of muscle proteins, phagocytized erythrocytes, ions and muscle enzymes in the interstitial fluid increases the osmolality of interstitial fluid and enhances filtration. Hargens et al. (36), Lundvall (50) and Rorabeck and Macnab (71) propose that tissue hyperosmolarity increases interstitial fluid volume which increases intramuscular pressure. Studies by Lundvall (50) suggest that work-induced tissue hyperosmolarity accounts for 75% of

the fluid accumulation during exercise. Increases in perfusion pressure which also facilitate filtration account for the remainder of the interstitial fluid accumulation in working musculature.

Increased intramuscular pressure then results in decreased blood flow to the working musculature. Different opinions exist concerning the cause of the ischemia associated with anterior compartment syndrome. Compression of arterioles, capillaries and venules by elevated compartment pressure have been proposed in the literature as mechanisms for reduced blood flow in CACS. Since hydrostatic pressure is lower in the venules (<15 mm Hg), these vessels are more easily compressed than arterioles or capillaries. Experiments conducted by Brooks (13) which involved ligation of arteries and veins demonstrate that venous obstruction produces significantly different effects than arterial obstruction. Occlusion of primary arteries for several hours yielded no demonstratable physiologic or anatomic changes after removal of the obstruction. After 17 hours of occlusion, scattered areas of muscle necrosis were present. A very slow developing inflammatory reaction was noted. Edema was present only after the reestablishment of circulation in damaged tissues. With venous ligation, Brooks reported hemorrhage, edema, acute inflammation and fibrosis. Fibrosis was associated with contracture and decreased force of contraction in the injured muscle.

Edema and inflammation characterize the chronic form of anterior compartment syndrome, while the acute form is characterized by muscle necrosis. Sheridan and Matsen (76) suggest that these differing histologic patterns are a function of tissue pressure. At pressures above 70 mm Hg, they found ischemic necrosis of muscle in the anterior compartment; at pressures below 50 mm Hg, venous congestion, edema and inflammation were present. Inflammation and edema have also been reported after heavy workloads of eccentric exercise (84). These findings are consistent with the hypothesis of eccentric muscle damage as a cause of CACS in athletes.

Several researchers (43, 54, 68, 74) have measured venous pressure in response to increases in externally applied pressure to the human foot, calf muscle of cats, rabbit tenuissmus muscle and in vitro studies of the internal mammary and common iliac veins. They report that when externally applied pressure reached mean venous pressure, further increases in externally applied pressure yielded equivalent increases in venous pressure. Increases in venous pressure must be accompanied by increases in hydrostatic pressure at the venule end of the capillary. Elevated venous pressures were reported in the presence of dilated arterioles (68). Elevation of capillary hydrostatic pressure near the venous end may occur as a result of increases in plasma oncotic pressure, decreases in interstitial osmolality and increases in interstitial

pressure. Jacobsson and Kjellmer (40) report that increased plasma oncotic pressure and dilution of proteins in the interstitial fluid increase the colloid osmotic pressure gradient across the capillary wall by 3-4 mm Hg, at most. They concluded that the most important factor limiting capillary filtration was tissue pressure. Increases in interstitial osmolarity facilitate the movement of fluid from the arterial end of capillaries into the interstitium. This increases the hydrostatic pressure of the interstitium and increases plasma osmolality. Both of these factors enhance fluid reabsorption at the venous end of capillaries and increase capillary hydrostatic pressure.

During exercise, muscle blood flow is regulated at the local level by the production of metabolites and other vasodilators such as bradykinin and adenosine which dilate arterioles. Local blood flow in the exercising muscles is determined by the local arterial and venous pressures and the local vascular resistance. The following formula (25) expresses the relationship between arteriovenous gradient and local blood flow:

$$\text{Local Blood Flow} = \frac{P_A - P_V}{R},$$

where PA is local arterial pressure, PV is local venous pressure and R is the local vascular resistance. In exercising muscle, small decreases in the perfusion pressure are compensated for by changes in local vascular resistance. Maintenance of local blood flow over a range of perfusion

pressures is termed autoregulation (8). However, when local vascular resistance is minimal as evidenced when arterioles are maximally vasodilated, autoregulation becomes ineffective and local blood flow is determined primarily by the arteriovenous pressure gradient.

Matsen et al. (54) measured the effect of externally applied pressure and limb position on arteriovenous gradient. As externally applied pressure was increased, the arteriovenous pressure gradient decreased (Figure 1, Appendix). These findings are consistent with another study by Matsen et al. (52) which examined the effect of increased externally applied pressure on muscle blood flow and muscle PO2. The partial pressure of oxygen and argon were measured with a Teflon catheter-mass spectrometer system. Argon washout rate was utilized to calculate muscle blood flow. Figure 2 (Appendix) illustrates the decline in muscle blood flow and PO2 with increases in externally applied pressure. These observations lead to the arteriovenous gradient theory as a mechanism for the development of chronic anterior compartment syndrome.

In patients with anterior compartment syndrome, the increased interstitial osmolarity due to muscle damage facilitates movement of fluid out of the vascular system into the compartment. Hargens et al. (36) report that when compartment pressure exceeds 30 mm Hg, lymphatic vessels are probably unable to drain the interstitial fluid. This is substantiated by the fact that lymphatic flow is obstructed

at low external pressures. As interstitial pressure and capillary oncotic pressure continue to rise toward the venous end of the capillary bed, reabsorption is enhanced. This occurs in the presence of an increase in mean capillary pressures of 5-15 mm Hg during exercise (40). A study by Jacobsson and Kjellmer (40) concluded that the primary factor which limits outward filtration of fluid into tissue is interstitial pressure. When interstitial pressure exceeds capillary pressure, subsequent increases in compartment pressure yield equivalent increases in venular end capillary hydrostatica pressure (68, 74).

The increase in capillary pressure at the venous end decreases the arteriovenous pressure gradient and results in a decrease in local blood flow (16). Decreased flow reduces oxygen tension and leads to the accumulation of lactic acid, increased PCO_2, increased hydrogen ion concentration and elevated concentrations of other vasodilators which enhance capillary filtration pressure through arteriolar dilatation and increased interstitial osmolality. Elevated compartment pressure and metabolites stimulate localized pain receptors and increase capillary permeability (18). In patients with chronic anterior compartment syndrome, the onset of pain forces the patient to stop exercising or to decrease the intensity of exercise. The onset of pain limits the amount of ischemia which is tolerable; this explains why muscle necrosis is rarely observed in muscle biopsies taken in patients with the chronic form of compartment syndrome.

The relationship between muscle blood flow, arteriovenous gradient and function are outlined in Figure 3 (Appendix). Initial declines in arteriovenous gradient are compensated for by changes in local vascular resistance via autoregulation (53). When autoregulation is no longer effective (point A), local muscle blood flow begins to decline significantly. Eventually, muscle blood flow is inadequate to meet the metabolic needs of the tissue (point B), resulting in functional abnormalities and the development of a compartment syndrome. Point C in Figure 3 represents a total loss of function; this point is rarely reached in the chronic form of anterior compartment syndrome due to the onset of ischemia which limits activity and further increases in compartment pressure. This model demonstrates why pain is often relieved by decreasing the intensity of exercise in patients with chronic anterior compartment syndrome. Decreasing the intensity of exercise lowers the metabolic requirements of the tissue, which decreases the muscle blood flow requirements.

This model also explains other clinical findings in patients with chronic anterior compartment syndrome. A study by Carter et al. (15) of 5 patients with chronic anterior compartment syndrome revealed normal femoral arteriograms at 8 weeks after the onset of symptoms. Researchers also report the presence of distal pulses in patients with chronic anterior compartment syndrome (28). Both of these clinical findings may be explained by this

local venous hypertension model (64) for chronic compartment syndromes. Since the artery is not obstructed as it passes through the compartment, distal pulses are intact and arteriograms are normal. Figure 4 (Appendix) depicts the relationship of peripheral flow to the effected compartment. Digital veins empty into veins with normal low pressures; therefore, arteriovenous pressure gradient in the digital circulation is normal even in the presence of anterior compartment syndrome.

Hargens et al. (36) report that only gradual increases in compartmental volume are observed up to 25 mm Hg. However, at pressures exceeding 33 mm Hg, compartment compliance sharply declines; above this critical pressure small increases in compartment volume yield substantial increases in compartment pressure. Hargens et al. (35) suggest that a compartment pressure of 33 mm Hg represents a limit to which fascia may be stretched. Matsen (53), however, reported that pressure tolerance varied greatly in a study of 42 patients at risk of compartmental syndromes. No patients with tissue pressures below 45 mm Hg developed a clinical compartment syndrome. All patients with pressures of 60 mm Hg or more developed clinical compartmental syndromes. Five of 7 patients with compartment pressures between 45 and 60 mm Hg developed compartment syndrome; two did not. This suggests that tissue pressure tolerance varies among individuals. In studies performed by Hargens et al. (35) and Matsen (53),

pressures were measured in a variety of compartments. Data was examined collectively and statistical analysis of individual compartments was not performed. Hence, variability may have been caused by differences in elasticity between fascia of the different compartments.

An unpublished study (31) of the effect of eccentric and concentric exercise in the superficial posterior compartment which surrounds the gastrocnemius, revealed that eccentric exercise increased muscle volume but was not associated with an increase in intramuscular pressure. This suggests that fascial compliance in the anterior compartment is reduced compared to the posterior compartment in the lower leg and explains the high incidence of compartment syndromes in the anterior compartment over other compartments in the lower leg.

Venous Disease. Another hypothesis proposed to explain the etiology of CACS is venous disease. Venous insufficiency caused by inadequate closure of venous valves decreases venous return and is often characterized by edema. Patients with venous insufficiency demonstrate elevated muscle relaxation pressures during exercise and a prolonged time for normalization of compartment pressures after exercise (79). Qvarfordt et al (65) found that venous function estimated by plethysmography and venous emptying was subnormal in 13% of patients with CACS. This small percentage suggests that venous disease is not a major cause of chronic anterior compartment syndrome.

Trauma. Styf and Korner (81) reported CACS following major trauma to the lower leg in 11 of 80 patients studied. However, none of these patients exhibited signs of acute anterior compartment syndrome or ischemic contracture. Trauma to the lower leg could result in damage to the musculature which would increase compartmental pressure in a mechanism similar to that proposed for exertion.

Techniques for Measurement of Anterior Compartment Pressure

Clinical signs alone are not sufficient to diagnose chronic anterior compartment syndrome; compartment pressures must be used to differentiate CACS from other causes of lower leg pain. Three methods have been used to measure compartment pressures in the lower leg.

Wick Catheter

The Wick catheter method (47) utilizes a wettable material such as cotton or polyglycolic acid suture to increase the surface area in contact with the interstitium. Filled with heparinized saline, the wick catheter is inserted into the compartment through a larger cannula which is withdrawn. Pressures are recorded using a multichannel recorder which is connected to a pressure transducer.

The primary problem with the wick catheter method is the slow response time. Styf and Korner (80) reported a mean delay of 4 seconds per single muscle contraction when

using the wick catheter to measure muscle pressures. Furthermore, muscle pressure never reached an equilibrium before the next contraction started. Pressure recordings using the wick catheter also showed very small amplitudes which varied with the frequency of contraction when muscle load was held constant. The slow response time associated with the wick catheter method makes it impossible to record muscle relaxation pressure during dynamic exercise (80). Hence, the wick catheter may be used to accurately measure resting pressures but due to its slow response time is unsuitable for recording pressures during exercise (80). Also, if large injections of heparinized saline are required to insure catheter patency, local bleeding may result. Local bleeding would most likely affect compartment pressure measurements.

Needle Injection Technique

Another method utilized to measure resting pressures is the needle injection technique. Approximately 0.1 cc of saline is injected into the compartment. A pressure transducer which is attached to a saline-filled syringe measures the pressure necessary to inject a small amount of saline into the tissue through the needle (67). The pressure transducer with digital readout then records compartmental pressure. Originally, this method was shown to be less accurate than other techniques (47); however, the addition of side holes at the tip of the needle prevent

occlusion and increase the accuracy of the needle injection method (83). It is now considered an acceptable technique for measurement of resting compartment pressure. The needle injection technique may be used with a catheter to measure compartment pressure during exercise. It is rarely used, however, because it must be flushed repeatedly to prevent occlusion (83). Occlusion of the needle or catheter would yield artificially high compartment pressures.

Infusion Technique

Constant infusion techniques were developed to prevent catheter or needle occlusion during exercise. This technique utilizes a solid state transducer and an intracompartmental catheter with a constant infusion rate of 0.1 to 3.0 ml of saline per hour to prevent occlusion. Studies by Styf and Korner (80) showed that infusion rates above 3 ml/hr significantly elevated compartment pressure readings. Infusion rates below 0.1 ml/hour resulted in occlusion of the needle and a decline in the dynamic properties of the pressure recording system. Styf and Korner (80) do not recommend flow rates above 1.5 ml/hour because no further improvement in the dynamic properties of the pressure recording system occurs at rates above this. Hence, they suggest that an infusion rate of 0.1-1.5 ml/hour is ideal for measuring muscle pressure during sustained contractions (80). The infusion technique may be utilized

to record accurate resting or exercise compartment pressures.

Compartment Pressures in Healthy Individuals and CACS Patients

Several studies have utilized infusion techniques to measure compartment pressures at rest and during exercise to establish criteria for diagnosis of CACS. Resting compartment pressure alone is not sufficient to diagnose CACS because the variability of data is too large. Normal resting values are approximately 5-15 mm Hg (65, 73, 80).

Gershuni et al. (34) examined the effect of knee and ankle position on resting anterior compartment pressures and reported that knee position did not significantly affect compartment pressures but that 30 degrees of relaxed ankle plantar flexion yielded the lowest compartment pressures. Most of the studies which have investigated compartment pressures did not do so at constant ankle positions, which would account for the large variation in resting compartment pressures. The depth of catheter insertion has also been shown to affect compartment pressure readings (42, 75). Mean muscle pressure and mean contraction pressure both depend on the depth of the pressure catheter in the muscle. This is another reason why measurement of muscle relaxation pressure is preferred as a diagnostic criterion over MMP and MCP.

Rorabeck et al. (73) reported resting pressures of 18.1 ± 2.9 mm Hg in patients diagnosed with CACS. Studies by Puranen and Alavaikko (64) however, found no significant elevation in resting pressures in patients with CACS over controls.

Compartment pressures return to pre-exercise levels within 5 to 15 minutes after exercise in subjects tested without CACS (72, 80). Rorabeck et al. (73) reported average immediate post exercise pressures of 37.3 ± 8.4 mm Hg in patients with diagnosed CACS as compared with 18.1 ± 2.0 mm Hg in healthy controls. During the exercise protocol, subjects ran at a speed of 6 mph on a treadmill. Pressures taken at 15 minutes post exercise showed pressures were still elevated in patients with CACS with average values of 19.9 ± 1.8 mm Hg compared with 10.5 ± 1.1 mm Hg in controls. The results of studies by Puranen and Alavaikko (64) confirm Rorabeck's results that compartment pressures measured immediately after exercise, and for a period of 15 minutes post exercise, are most helpful in the diagnosis of patients with CACS. Generally, the criteria used to diagnose CACS are (1) compartment pressures which exceed 30 to 35 mm Hg immediately following exercise to a level which elicits pain and (2) pressures measured 15 minutes after exercise which exceed pre-exercise compartment pressures (81, 83).

Since compartment pressures measured immediately following exercise were equivalent to those measured just

prior to cessation of exercise, continuous monitoring during exercise is rarely utilized. Also, the catheter position cannot be well controlled during weight-bearing or other exercise which requires considerable range of motion at the knee joint. These forms of exercise make it difficult to keep the catheter in place, which is necessary to obtain accurate pressure measurements. For this reason, many studies have utilized an ankle ergometer for pressure studies (80, 82). In these studies, the patient performs ankle plantar flexion and dorsiflexion from a supine position which decreases movement artifact and the position of the catheter is more easily maintained.

Treatment of Chronic Anterior Compartment Syndrome

The preferred method of treatment for patients with CACS is fasciotomy. Non-surgical treatments such as diuretics and anti-inflammatory agents do not relieve the symptoms (66). Fasciotomy involves decompression of the compartment through a longitudinal incision of the fascia. Allen and Barnes (1) reported that resting and exercise pressures returned to normal in 51 of 60 patients treated with fasciotomy. They also reported that all but 3 patients improved and returned to full activity within 6-8 weeks after surgery. Fronek et al. (33) also reported good success rates with fasciotomy. Eleven of 12 patients treated with fasciotomy demonstrated increased exercise

tolerance and reported relief of pain. Studies by Qvarfordt et al. (65) and Styf et al. (82) showed that fasciotomy relieved the pain associated with CACS and normalized muscle blood flow, intramuscular pressures and skeletal muscle metabolism. Qvarfordt et al. (65) further reported that water content of the tibialis anterior muscle decreased after fasciotomy. Follow-up reports of patients after fasciotomy suggest that 60 to 100 % of patients' symptoms are relieved by fasciotomy (72, 83, 84). Reneman (67) found that 9 patients diagnosed with anterior compartment syndrome continued to be symptomatic for at least one year.

Mozan and Keagy (60) report that fascial release decreases muscle force in animals. However, when performed in humans, a successful fasciotomy is usually followed by an increase in functional capacity. National class athletes, such as Mary Decker-Slaney and Dick Quax, have set records and won Olympic medals after undergoing fasciotomy to relieve CACS (78). Styf and Korner (81) suggest that normalization of muscle relaxation pressures after fasciotomy relieves pain which permits the athletes to increase their functional capacity.

In some patients CACS reoccurs or peroneal nerve entrapment results in continued pain after surgery. Bell (7) suggests that in unsuccessful fasciotomy, new fibrous tissue may grow too rapidly and strongly between the two fascial edges, decreasing the size of the compartment. Also, the patient may rest for too long of a period after

fasciotomy resulting in muscle atrophy. When activity is finally resumed, the compartment is often unable to accommodate the resultant muscle hypertrophy (7). Puranen and Alavaikko (64) reported that all postoperative relapses were due to insufficient fascial release. Hence, they recommend release of the fascia along its full length.

Athletic Participation and the Occurrence of Chronic Anterior Compartment Syndrome

Chronic anterior compartment syndrome primarily afflicts athletes participating in weight bearing types of sports which require prolonged periods of running (78). A study by Fronek et al. (33) which included 18 patients diagnosed with chronic anterior compartment syndrome revealed that 15 of these patients were runners. The other 3 patients participated in soccer, cricket or tennis. Reports of CACS in the literature show no cases of CACS in cyclists. Cycling is a nonweight bearing activity which does not utilize the muscles of the anterior compartment to the extent required during weight-bearing activities. This suggests that compartment pressures during cycling are lower than during running or other weight- bearing activities. If anterior compartment pressures are lower during cycling, this may provide a successful nonsurgical treatment option for patients with CACS who wish to maintain high activity levels. Cycling may also prove valuable in cases of unsuccessful fasciotomy; patients may be able replace

running with cycling as a form of aerobic exercise. Hence, quantification of compartment pressures during cycling may provide valuable information for the treatment of patients with chronic anterior compartment syndrome.

CHAPTER III

METHODS AND PROCEDURES

This study investigated anterior compartment pressures in competitive runners and cyclists after equivalent workloads to determine if pressures in runners are significantly different than those measured in cyclists. CPK enzyme levels were also measured after equivalent submaximal workloads in runners and cyclists.

Selection of Subjects

The subjects consisted of 10 competitive male runners and 10 competitive male cyclists who volunteered to participate in the study. Athletes ages ranged from 19 to 38 years. All subjects were involved almost exclusively in running or cycling competitions on a regular basis. Each subject was paid $50.00 for his full participation in the study. Permission to conduct research involving human subjects was obtained from the Oklahoma State University and University of Oklahoma Institutional Review Boards.

Test Procedure

Each subject participated in 2 testing sessions, approximately one week apart. Two different workloads based

on oxygen consumption were administered at each testing session. Subjects were instructed not to exercise before either of the two testing sessions. During the first session, each subject completed a medical questionnaire and signed a consent form (Appendix). Information about each subject's level of training and competition was also obtained. Subjects were screened for medical conditions or major cardiovascular risk factors which would preclude their participation in a maximal symptoms limited stress test as outlined in the American College of Sports Medicine's Guidelines for Exercise Testing and Prescription (2). Height, weight and resting blood pressure measurements were recorded for each subject prior to testing. All blood pressures were measured with a sphygmomanometer and stethoscope.

During the first test session, resting compartment pressure measurements were made on all subjects prior to their participation in a maximal symptoms limited stress test on the treadmill or bicycle. Techniques for measurement of compartment pressures are outlined later in the procedure section. During the maximal symptoms limited stress test, oxygen consumption, heart rate, EKG, and blood pressure were measured. A metabolic cart (Horizon model) located in the Pulmonary Laboratory was utilized to collect respiratory gases during the stress tests for the cyclists. A Sensor Medics metabolic cart was used to measure VO_2 max on the runners.

Runners were tested using a protocol designed by the researcher. The protocol included a 5 minute warm-up at 7 mph on the treadmill. Many of the runners had never trained on a treadmill before and needed this time to become comfortable with treadmill running. After the warm-up, treadmill speed was increased to 8 mph. At 2 minute intervals, treadmill speed was increased .5 mph until a speed of 9.5 mph was reached. This was the maximum attainable speed on the Quinton 2000 treadmill used for testing. During subsequent 2 minute intervals, speed was maintained at 9.5 mph and grade was increased in increments of 2.5% until the subject wanted to stop or oxygen consumption began to decrease.

Cyclists were tested on their own racing bicycles which were set up on a stand called a trainer. Toe-clips or Look pedals which fix the cyclists shoe to the pedal were utilized by all cyclists during both testing sessions. A protocol similar to the one used for runners was used for the cyclists. Each cyclist began pedaling in his large chain ring and shifted into a smaller toothed cog every 2 minutes until he was exhausted or oxygen consumption had started to decline. Cyclists were monitored as outlined above for the runners. Maximal exercise tests were designed such that both cyclists and runners would reach a maximal level of exertion in approximately 10 to 15 minutes. All subjects were encouraged to exercise to exhaustion to insure

that VO_2 max was reached if oxygen consumption had not leveled off or declined with increasing workload.

Immediately following the maximal exercise test, anterior compartment pressures were measured on runners and cyclists. Pressure measurements were taken again at fifteen minutes post exercise to determine if pressures had returned to pre-exercise values. During this time, blood pressure was monitored.

Approximately one week after the first visit, each subject returned for the second testing session. Prior to the second exercise test, approximately 3 milliliters of blood was drawn using the venipuncture method. These samples were analyzed for Total Creatine Phosphokinase (CPK) enzyme levels. Subjects were then exercised at a heart rate equivalent to 80% of maximal oxygen consumption as determined by their VO_2 max test performed one week earlier. Runners exercised on the treadmill and cyclists on their own racing bicycles. Heart rate, measured by EKG recording, was used to monitor each subject's workload during the test. Subjects exercised for 20 minutes immediately after which pressure measurements in the anterior compartment were made. Another pressure was taken 15 minutes post exercise. Three milliliters of blood was again drawn to determine post exercise levels of CPK. Blood pressures were measured using a stethoscope and mercury sphygmomanometer and recorded each time compartment pressure was measured.

Compartment Pressure Measurements

Each subject assumed a supine position for measurement of anterior compartment pressures. In order to insure accuracy, ankle position was limited to a relaxed position of approximately 30 degrees of plantar flexion. Each subject's right knee was supported by a pillow to insure that no compressional forces caused by leg placement would affect compartment pressures.

Utilizing sterile surgical gloves, the skin which lies over the anterior compartment was cleaned with betadine solution. Thereafter, the skin and subcutaneous tissue 2-3 cm lateral to the tibial crest and 10-12 cm below the tibial tuberosity was anesthetized with 1-2 cc of 1% Lidocaine. This permitted the subjects to relax during measurement of compartment pressures, increasing the accuracy of measurements and relieving any mild discomfort associated with a brief needle stick.

The Quick S.T.I.C. (Model 295-1) method for pressure measurement designed by Stryker was utilized to measure compartment pressures. This procedure is analogous to the needle injection measurements outlined earlier. Quick S.T.I.C. utilizes a pressure transducer, saline filled syringe and an 18 gauge needle to measure compartment pressures. Prior to measurement, the system was purged of air and calibrated. For calibration, the unit was held at an angle of 20 degrees below horizontal and calibrated to

zero. The needle was then inserted into the anterior compartment perpendicular to the longitudinal axis of the compartment. A point 10-12 centimeters distal to the tibial tuberosity and approximately 2-3 centimeters lateral to the tibial crest (80) was chosen for insertion. After penetrating the skin and subcutaneous tissue, the needle was advanced through the fascia and 2-3 mm into the anterior compartment. The texture of the fascia made it possible to accurately determine the location of the needle. After insertion, less than .1 cc of saline was slowly injected into the anterior compartment. A stable pressure reading indicated that saline had equilibrated with interstitial fluid. After equilibration, pressure was read in mm Hg from the digital readout. All pressure readings were rechecked for accuracy. The same portal was used for both pressure measurements taken post exercise. The skin over the anterior compartment was cleaned with betadine again and wiped with sterile alcohol pads. The area over the perforations was then covered with a bandage.

Analysis of CPK

Analysis of Total CPK was performed at the medical laboratory at Oklahoma Memorial Hospital in Oklahoma City. Blood samples were delivered to the laboratory within one hour of collection and centrifuged. Eleven microliters of serum was deposited on a Kodak Ektachem slide with N-acetylcysteine activated reagents. Reflection density at

670 nm was measured during a 5 minute incubation period at 37 degrees Centigrade. Two of the blood samples collected from runners number 19 and 20 were left in the centrifuge for 8 hours prior to analysis. Both of these samples yielded elevated levels of CPK activity compared to other subjects. Hence, these samples were not included in the statistical analysis for Total CPK levels.

Analysis of Data

Initially, an F test was utilized to determine if the 2 independent samples of runners and cyclists had equivalent variances for compartment pressures, CPK enzyme levels, and other descriptive variables at an alpha level of five percent. The results of this test were used to calculate the variance utilized in the t-test. An unpaired t-test (79) was used to test the following parameters in competitive runners as compared to competitive cyclists at an alpha level of five percent.

1. Resting anterior compartment pressures
2. Anterior compartment pressures measured after a submaximal workload at 80% of maximal oxygen consumption
3. Anterior compartment pressures measured after a maximal workload
4. Anterior compartment pressures measured 15 minutes after completion of 20 minutes of exercise at a submaximal workload at 80% of maximal oxygen consumption

5. Anterior compartment pressures measured 15 minutes after completion of a maximal workload

6. Pre-exercise levels of CPK enzymes

7. Post exercise levels of CPK enzymes

8. Change in the level of CPK enzyme after 20 minutes of exercise at a submaximal workload

A paired t-test at an alpha level of five percent was also utilized to determine if the following pressure differences were significant within each group.

1. Resting compartment pressures and compartment pressures after a workload at 80% of VO_2 max

2. Resting compartment pressures and compartment pressures after a VO_2 max test

3. Compartment pressures after a workload at 80% of VO_2 max and compartment pressures after a VO_2 max test

4. Compartment pressures at rest and compartment pressure at 15 minutes after 20 minutes of exercise at 80% of VO_2 max

5. Compartment pressures at rest and compartment pressure at 15 minutes after a VO_2 max

6. Compartment pressures after a VO_2 max test and at 15 minutes after a VO_2 max test

7. Compartment pressures after 20 minutes of exercise at 80% of VO_2 max and at 15 minutes after exercise at 80% of VO_2 max

CHAPTER IV

RESULTS AND DISCUSSION

Ten competitive runners and cyclists participated in a study to determine the effect of both types of exercise on pressure in the anterior compartment. Compartment pressures were obtained at rest, after a VO_2 max test and after 20 minutes of exercise at 80% of VO_2 max.

Descriptive Data

Table I lists the descriptive data for the twenty subjects participating in the study. The average age of the twenty subjects was 29 years with a range of 19-38 years. Participants had competed in either running or cycling races for 1-17 years with a mean of approximately 7 years of competition. The study group participated in an average of 21 races per year with a range of 10-40 competitions. Mean VO_2 max was 62.15 ml/kg/min $\pm$ 7.06 with a range of 53.1-78.4 ml/kg/min.

Runners

Mean values for descriptive data specific to the 10 runners are listed in Table II. The average age for runners

TABLE I

DESCRIPTIVE DATA FOR SUBJECTS

Subject	Age (Yrs.)	Competitions Per Year	Years of Competition	VO_2 max (ml/kg/min)
B1	36	15	1	60.2
B2	19	20	1	60.0
B3	22	15	1	53.1
B4	22	25	3	60.6
B5	27	20	7	55.7
B6	24	20	2	56.2
B7	24	30	3	56.2
B8	30	20	1	66.0
B9	29	15	5	53.7
B10	33	40	4	53.4
R1	33	25	12	63.9
R2	32	20	6	60.3
R3	29	20	5	65.7
R4	29	25	17	78.4
R5	33	12	7	60.2
R6	32	17	14	63.6
R7	29	13	3	66.8
R8	38	10	10	63.7
R9	28	30	15	77.4
R10	28	25	15	67.9
X	28.9	20.6	6.6	62.15

TABLE II

DESCRIPTIVE DATA FOR RUNNERS

Variable	Range	Mean	Standard Deviation
Age (years)	28-38	31.1	3.14
Mileage (mi/wk)	30-90	56.0	18.38
Competition Distance (mi)	6-26	16.0	8.69
Race Pace (min/mi)	4:54-6:00	5:28	0:22
Years of Competition	3-17	10.4	4.90
Competitions per Year	10-30	19.7	6.63

was 31 years with a range of 28-38 years. With 3-17 years of competition experience, the runners showed an average of 10 years of consistent competition. Runners participated in an average of 20 competitions per year with a range of 10-30 races yearly. Average weekly mileage was 56 miles; weekly mileage, however, varied from 30-90 miles. The runners recorded maximum competition distances of 6-26 miles with a mean of 16 miles. Average race pace varied with race distance, but was averaged for a distance of 10 kilometer (6.2 miles). Mean race pace was 5:28 minutes per mile with a range of 4:54 to 6:00 per mile. A 6:00 minute per mile pace was the slowest accepted pace for subject participation. The mean value for VO_2 max in runners was 66.8 ml/kg/min with a range of 60.2-78.4 ml/kg/min.

Cyclists

Table III lists mean values for descriptive data obtained on the 10 cyclists. The average age was 27 years with a range of 19-36 years. The cyclists reported from 1-7 years of competition experience with a mean of 3 years. With an average of 22 competitions per year, this group participated in 15-40 competitions yearly. The cyclists averaged 255 training miles per week with a range of 200-300 miles weekly. Competition distances ranged from 45-100 miles with a mean competition distance of 73 miles. The average pace for cyclists was 24.9 miles per hour with a reported range of 22-27 miles per hour. Cyclists yielded a

TABLE III

DESCRIPTIVE DATA FOR CYCLISTS

Variable	Range	Mean	Standard Deviation
Age (years)	19-36	31.1	5.38
Mileage (mi/wk)	200-300	254.5	42.72
Competition Distance (mi)	45-100	73.4	20.42
Race Pace (mph)	22-27	24.9	1.29
Years of Competition	1-7	2.8	2.04
Competitions per Year	15-40	22.0	7.89

lower mean value for oxygen consumption than runners; the mean VO_2 max for cyclists was 57.5 ml/kg/min with values ranging from 53.1-66.0 ml/kg/min.

Unpaired T-Test for
Descriptive Data

An unpaired t-test at an alpha level of 0.05 was utilized to determine if the runners and cyclists differed with respect to years of competition experience and number of races entered yearly. The results of the t-test are outlined in Table IV. The runners differed significantly from the cyclists with respect to years of competition experience. The observed significance level for this variable was 0.0007. The number of competitions participated in on a yearly basis, however, was not significantly different. VO_2 max was significantly greater in runners as compared to cyclists. Generally, VO_2 max is lower in competitive cyclists as compared to distance runners (51). The higher VO_2 max values in runners reflect the increased utilization of oxygen by musculature in the arms during running (49). McArdle et al. (56) report that VO_2max values are 6.4-11.2% higher in runners as compared to cyclists. After VO_2max values for cyclists were adjusted upward by 9.3% (51), average VO_2max values remained significantly higher in the runners; the observed significance level was 0.045.

TABLE IV

UNPAIRED T-TEST FOR DESCRIPTIVE DATA
IN RUNNERS AND CYCLISTS

Variable	Variances	t	Probability
Years of Competition	Unequal	-4.2540**	0.0007
Competitions per Year	Equal	0.7057	0.4894
VO_2 max (ml/kg/min)	Equal	-3.8755**	0.0011
Adjusted VO_2 max (ml/kg/min)	Equal	-2.1559*	0.0449

* = P<0.05

** = P<0.01

Compartment Pressure Measurements

Compartment pressure measurements in runners and cyclists were obtained at rest, after a VO_2 max test, after 20 minutes at a workload equivalent to 80% of VO_2 max, and at 15 minutes following both exercise sessions. An unpaired t-test was used to determine if pressures were significantly different between the two groups. Table V list the results of these t-tests in addition to the means and standard errors obtained from both groups.

Resting Compartment Pressures

Mean resting compartment pressures were 14.5 $\pm$ 1.24 and 11.1 $\pm$ 1.26 mm Hg for runners and cyclists, respectively. The unpaired t-test revealed that resting compartment pressures were not significantly different in runners and cyclists at an alpha level of 0.05.

Compartment Pressures With Exercise

Compartment pressures were measured immediately following 2 different exercise workloads to determine if compartment pressures were significantly higher in runners than cyclists at each workload. An unpaired t-test at an alpha level of 0.05 was utilized to determine if any differences existed.

TABLE V

RESULTS OF UNPAIRED T-TEST FOR COMPARTMENT PRESSURE IN RUNNERS AND CYCLISTS

Condition	Group	Mean (mm Hg)	Standard Error	t	Probability
Rest	Runners	14.5	1.24	-1.92	0.0705
	Cyclists	11.1	1.26		
80% of VO_2 max	Runners	12.2	1.10	-0.37	0.7190
	Cyclists	11.5	1.57		
VO_2 max * = P<0.05	Runners	19.1	2.18	-2.67*	0.0160
	Cyclists	12.2	1.41		
Recovery 80% VO_2 max	Runners	12.7	1.14	-0.96	0.3502
	Cyclists	10.9	1.50		
Recovery VO_2 max	Runners	14.8	1.26	-1.15	0.2649
	Cyclists	12.7	1.32		

Anterior compartment pressures measured immediately after 20 minutes of exercise at a workload equivalent to 80% of maximal oxygen consumption were not significantly different in runners and cyclists. Mean compartment pressures of 12.2 $\pm$ 1.10 mm Hg and 11.5 $\pm$ 1.56 mm Hg were measured in runners and cyclists, respectively.

Mean compartment pressure measured after a VO_2max test was 19.1 $\pm$ 1.41 mm Hg in runners; cyclists demonstrated a lower mean pressure of 12.2 $\pm$ 2.18 mm Hg. The unpaired t-test revealed that compartment pressures were significantly higher in runners as compared to cyclists. The observed significance level ($P<0.05$) for this observation was 0.016.

Post Exercise Compartment Pressures

Anterior compartment pressures were measured 15 minutes after cessation of exercise at both of the reported workloads. An unpaired t-test at an alpha level of 0.05 was utilized to determine if post exercise compartment pressures remained elevated in the runners as compared to the cyclists.

Mean compartment pressure in runners measured 15 minutes after completion of 20 minutes of exercise at a workload equivalent to 80% of VO_2 max was 12.7 $\pm$ 1.14 mm Hg. Cyclists yielded a mean pressure of 10.9 $\pm$ 1.49 mm Hg. The unpaired t-test revealed that these recovery pressures were not significantly different ($P\geq0.05$).

Results of the unpaired t-test suggest that anterior compartment pressures were not significantly different in runners and cyclists at 15 minutes after completion of a VO_2 max test. Recovery compartment pressures were 14.8 ± 1.26 mm Hg for runners and 12.7 ± 1.32 mm Hg for cyclists.

CPK Enzyme Levels

Serum levels of CPK enzymes were compared in runners and cyclists before and after 20 minutes of exercise at a workload equivalent to 80% of VO_2 max. The difference in CPK levels obtained before and after exercise at this workload was also analyzed using an unpaired t-test at an alpha level of 0.05. Mean CPK levels and standard errors are outlined in Table VI.

Average pre-exercise CPK levels were 255.6 ± 40.83 units/liter (u/l) in runners and 173.8 ± 19.16 u/l in cyclists. These values were not significantly different with an observed significance level of 0.07.

Serum CPK levels after 20 minutes of exercise at 80% of VO_2 max were 284.8 ± 46.90 u/l and 177.1 ± 20.41 u/l in runners and cyclists, respectively. These post exercise CPK levels were not significantly different ($P > 0.05$.

The mean differences in CPK levels measured before and after exercise at 80% of VO_2 max were significantly different with an observed significance level of 0.01. The average difference for runners and cyclists was 29.13 ± 7.43 and 3.3 ± 2.17 u/l, respectively.

TABLE VI

RESULTS OF UNPAIRED T-TEST FOR CPK VALUES
IN RUNNERS AND CYCLISTS

CPK Sample	Group	Mean	Standard Error	t	Probability
Pre-Exercise	Runners	255.63	40.83	-1.94	0.0701
	Cyclists	173.8	19.16		
Post Exerise	Runners	284.75	46.09	-2.10	0.0627
	Cyclists	177.1	20.41		
Difference	Runners	29.13	7.43	-3.34**	0.0100
	Cyclists	3.30	2.17		

* = $P \leq 0.05$

A Comparison of Compartment Pressures and CPK Levels in Runners

Anterior compartment pressures measured at rest, after exercise at 80% of VO_2max, and after a maximal workload were compared in runners. A paired t-test was utilized for this comparison. Data and results of the paired t-test are outlined in Table VII. Pre and post exercise CPK levels were also compared among runners.

Anterior Compartment Pressures in Runners

Anterior compartment pressures measured at rest and after exercise at 80% of VO_2 max were 14.5 and 12.2 mm Hg, respectively. The paired t-test revealed that compartment pressures measured after exercise at 80% of VO_2 max were significantly less than at rest. The observed significance level was 0.008.

Average resting compartment pressure in runners was 14.5 mm Hg as compared to 19.1 mm Hg after a maximal workload. Compartment pressures were significantly higher after the maximal workload with an observed significance level of 0.022.

Anterior compartment pressures measured after a maximal workload (19.1 mm Hg) were also significantly greater than those measured after exercise at 80% of VO_2 max (12.2 mm Hg).

TABLE VII

RESULTS OF PAIRED T-TESTS FOR COMPARTMENT PRESSURE AND CPK IN RUNNERS

Comp. Pr./CPK	Mean	Standard Error	t	Probability
Resting (mm Hg)	14.5	0.79	2.91**	0.0087
80% VO_2 max	12.2			
Resting (mm Hg)	14.5	1.93	-2.39	0.0225
VO_2 max	19.1			
80% VO2 max (mm Hg)	12.2	1.63	-4.24**	0.0029
VO_2 max	19.1			
Resting (mm Hg)	14.5	1.12	1.12	0.1500
Recovery 80% VO_2 max	12.7			
Resting (mm Hg)	14.5	1.16	0.26	>0.2500
Recovery VO_2 max	14.8			

TABLE VII (Continued)

Comp. Pr./CPK	Mean	Standard Error	t	Probability
80% VO_2max (mm Hg)	12.2	1.16	-0.43	>0.2500
Recovery 80% VO_2max	12.7			
VO_2 max (mm Hg)	19.1	2.38	1.98*	0.0437
Recovery VO_2max	14.8			
Pre-Exercise CPK (Units/liter)	255.6	7.45	-3.92**	0.0050
Post Exercise CPK	284.8			

* = P<0.05

** = P<0.01

The observed significance level for this difference was 0.0029.

Neither of the recovery compartment pressures measured 15 minutes after exercise at a maximal workload (14.8 mm Hg) or after exercise at 80% of VO_2 max (12.7 mm Hg) were significantly different than resting compartment pressures in runners. This comparison is useful in determining if recovery was complete.

Anterior compartment pressures measured after exercise at 80% of VO_2 max and after 15 minutes of recovery were not significantly different. Differences in compartment pressure measured immediately following a maximal workload and after a 15 minute recovery were significant. The observed significance level was 0.04375.

CPK Levels in Runners

The difference in CPK levels before (255.6 u/l) and after (284.8 u/l) 20 minutes of exercise at 80% of VO_2max was significant. Post exercise CPK levels were significantly higher in the runners with an observed significance level of 0.006.

A Comparison of Compartment Pressures and CPK Levels in Cyclists

Anterior compartment pressures measured at rest, after exercise at 80% of VO_2max, and after a maximal workload were compared in the cyclists. A paired t-test at an alpha level

of 0.05 was utilized to determine if pressures were significantly different. The same procedure was used to determine if CPK values obtained before and after exercise at 80% of VO_2 max were significantly different. Results of this data are outlined in Table VIII.

Anterior Compartment Pressures in Cyclists

Anterior compartment pressures measured at rest (11.1 mm Hg) were not significantly different than pressures measured after exercise at 80% of VO_2max (11.5 mm Hg) or after maximal exercise (12.2 mm Hg). Compartment pressures measured 15 minutes after both exercise workloads were also not significantly different from resting pressures.

CPK Levels in Cyclists

CPK levels measured before (173.8 u/l) and after exercise at 80% of VO_2 max (177.1 u/l) were not statistically different.

Discussion of Results

Anterior compartment pressures have been measured during running in healthy individuals and patients with chronic anterior compartment syndrome (41, 72). However, pressures have never been measured during cycling, in a select group of competitive runners or at equivalent workloads as determined by oxygen consumption. The muscular

TABLE VIII

RESULTS OF PAIRED T-TESTS FOR COMPARTMENT PRESSURE AND CPK IN CYCLISTS

Comp. Pr./CPK	Mean	Standard Error	t	Probability
Resting (mm Hg)	11.1	1.83	-0.22	>0.2500
80% VO_2 max	11.5			
Resting (mm Hg)	11.1	1.14	-0.97	0.1750
VO_2 max	12.2			
80% VO2 max (mm Hg)	11.5	1.61	-0.31	>0.2500
VO_2 max	12.2			
Resting (mm Hg)	11.1	1.55	0.13	>0.2500
Recovery 80% VO_2 max	10.9			
Resting (mm Hg)	11.1	1.23	-1.30	0.100
Recovery VO_2 max	12.7			

TABLE VIII (Continued)

Comp. Pr./CPK	Mean	Standard Error	t	Probability
80% VO_2max (mm Hg)	11.5	1.11	0.45	>0.2500
Recovery 80% VO_2max	10.9			
VO_2 max (mm Hg)	12.2	2.38	1.98*	0.0437
Recovery VO_2max	12.7			
Pre-Exercise CPK (Units/liter)	173.8	2.17	-1.52	0.075
Post Exercise CPK	177.1			

* = P<0.05

contractions involved in cycling are primarily concentric (46, 63) with the possible exception of minimal eccentric work by the ankle plantar flexors (23). However, during running, the tibialis anterior must contract eccentrically during foot-strike in rearfoot landers to prevent foot-slap (61). Contraction of the tibialis anterior during the swing phase as the lead leg moves forward is concentric to allow the foot to clear the ground. Hence, the tibialis anterior contracts concentrically during cycling and both concentrically and eccentrically during running. Eccentric contraction of the tibialis anterior during running, but not cycling may contribute to elevations in anterior compartment pressure during running.

Eccentric exercise has been demonstrated to cause muscular soreness, myofibrillar damage (29) and significantly elevate compartment pressure as compared to concentric work (31). Friden et al. (27) suggest that the greater tension per active motor unit during eccentric exercise as compared to concentric exercise may increase the risk of mechanical damage to the contractile apparatus. This increased tension present during eccentric work may damage the myofibers. Since the tibialis anterior performs only concentric contractions during cycling, the eccentric work characteristic of running may elevate anterior compartment pressure in runners as compared to cyclists.

Anterior Compartment Pressures in Runners and Cyclists

This study suggests that resting anterior compartment pressures are not significantly different in competitive runners than cyclists. The observed significance level for resting pressures was 0.0705, which was not significant at an alpha level of 0.05.

The normal range for resting anterior compartment pressure in healthy individuals is 5-15 mm Hg (65, 73, 80). Average resting compartment pressures measured in this study for both cyclists and runners were within this normal range for asymptomatic individuals. Resting compartment pressures ranged from 6-17 mm Hg in cyclists and 5-19 mm Hg in runners. All of the subjects in this study were asymptomatic with recovery pressures equivalent to resting pressure measurements. The results of this study further support the utilization of post exercise and recovery pressures in the diagnosis of compartment syndrome.

Compartment pressures measured after exercise at 80% of VO_2 max were not significantly different in runners and cyclists. Figure 5 illustrates the resting and exercise compartment pressures obtained in runners and cyclists. Exercise at 80% of VO_2 max was below the anaerobic threshold of all but two of the subjects as demonstrated by anaerobic threshold measurements. The range for anaerobic threshold (Appendix) was 65.5-93% of VO_2 max as determined by minute

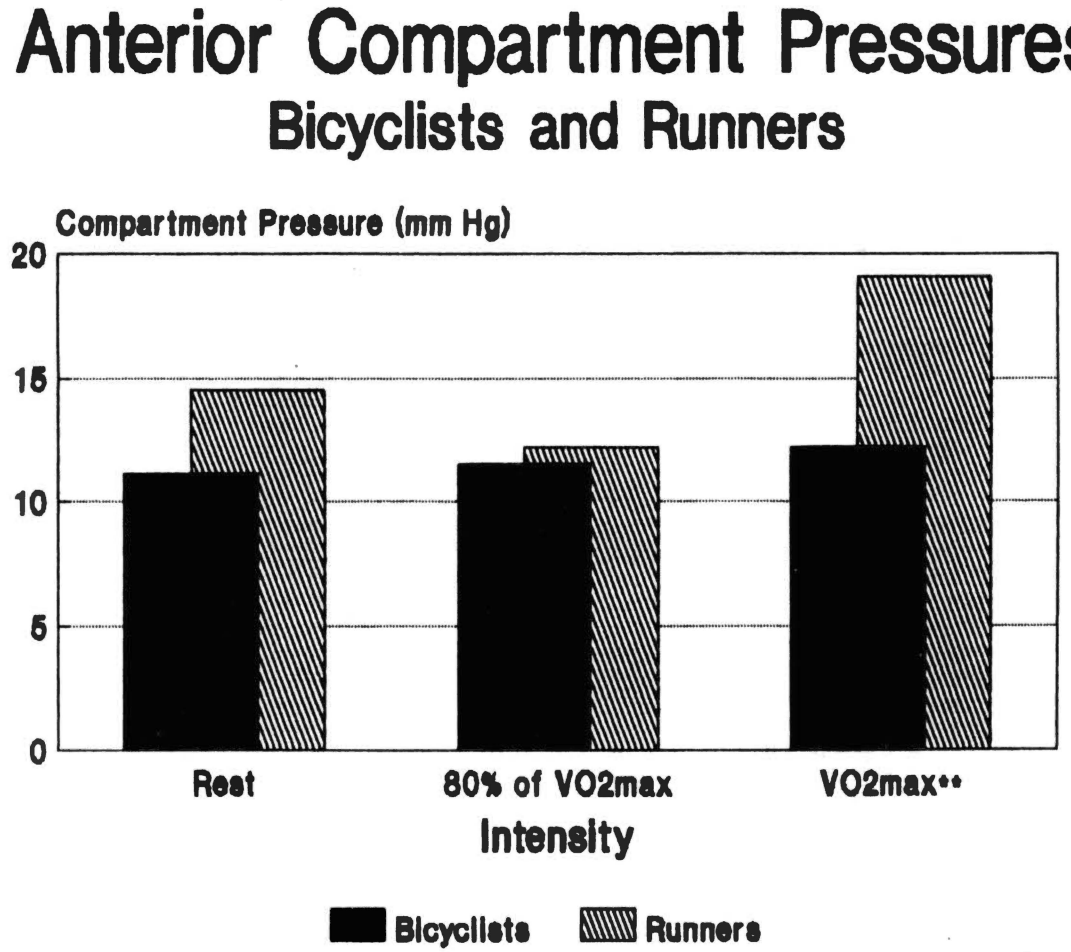

Figure 5. Anterior Compartment Pressures in Runners and Cyclists Measured at Rest and Immediately after Exercise at 80% of VO_2 Max and Maximal Exercise

ventilation, O_2 consumption and CO_2 production measured during the VO_2 max test. The fact that compartment pressures measured after this submaximal, aerobic workload in cyclists and runners were not significantly different, suggests that this workload was not sufficient to elevate compartment pressure in either group.

Anterior compartment pressures during maximal exercise were significantly greater ($P<0.05$) in runners as compared to cyclists. One of the major functions of the tibialis anterior during foot contact is impact absorption (22, 59). Eccentric contraction of the quadriceps, dorsiflexors and other musculature which controls pronation during foot-strike serves to dissipate the foot impact forces (58). Harrison et al. (37) reported peak forces in the dorsiflexors of 0.5 times body weight during foot-strike.

The observed increase in anterior compartment pressure after maximal exercise in runners as compared to cyclists, but not at 80% of VO_2 max suggests that a critical workload exists above which compartment pressure increases. During cycling, this critical workload which marks an increase in compartment pressure is absent. Elevation of compartment pressure during running at maximal workloads may be related to eccentric contraction of the tibialis anterior during running and/or differences in the frequency and force of contraction of the musculature of the anterior compartment. Muscle contraction and relaxation pressure have been shown to increase with increasing force of contraction (44, 75).

The frequency or force of muscular contraction was not quantified in this study. However, studies performed by other researchers provide information with respect to these variables.

Studies (44, 75) which measured compartment pressure in response to changes in the force of muscular contraction, demonstrate that muscle contraction and relaxation pressure increase with increasing force of contraction. Isometric muscle contractions and isotonic weight training types of exercise were used in these studies. An increase in the force of muscular contraction during dynamic exercise which utilizes a large portion of total body musculature may yield different results as the muscle pump may facilitate removal of fluid from the anterior compartment. During cycling (23) as well as running (58, 77), the force of muscular contraction in the tibialis anterior increases with increasing exercise intensity. As running speed increases, the frequency and rate of impact forces as measured by electromyography and force-plate studies increases (58, 77). During cycling, toe-clips have been demonstrated to double the muscular activity in the tibialis anterior as compared to cycling without toe-clips (23). Toe-clips were utilized in this study to permit maximal muscular activity by the tibialis anterior. Studies which compare the amount of force generated in the tibialis anterior during cycling and running have not been conducted. Hence, a greater increase in the force of muscle contraction during running as

compared to cycling may have contributed to increases in compartment pressure. However, the low incidence of anterior compartment syndrome in body builders and power lifters as compared to runners suggests that force of muscular contraction alone would not explain the elevation of compartment pressures during running as compared to cycling.

Differences in frequency of muscular contraction during running and cycling may contribute to elevations in compartment pressure in runners as compared to cyclists. Studies investigating the effect of frequency of muscular contraction on compartment pressure are not available. As running speed increases, the frequency of muscular contraction of the tibialis anterior increases (58, 77). During cycling, pedal speed measured in revolutions per minute is relatively consistent over a variety of resistances in trained cyclists (23). Hence, frequency of contraction would not be expected to increase as a direct function of speed or exercise intensity as occurs during running. This suggests that frequency of contraction may have increased with increasing workload in runners but not cyclists. However, in this study treadmill speed was not increased above 9.5 mph; after this speed, workload was increased by elevation of the treadmill. All of the runners ran at a speed of 9.0-9.5 mph during exercise at 80% of VO_2 max as compared to 9.5 mph during maximal exercise. Hence, frequency of muscular contraction during exercise at 80% of

VO_2 max and maximal exercise was similar. This suggests that frequency of contraction of the tibialis anterior did not increase at the maximal workload as compared to exercise at 80% of VO_2 max.

Of the factors which may contribute to differences in compartment pressure during maximal exercise (type, frequency and force of muscle contraction) in runners as compared to cyclists, the utilization of eccentric muscular contractions of the tibialis anterior during running but not cycling may be the primary factor in elevating compartment pressure. Muscle relaxation pressures measured by Friden et al. (31) after eccentric ankle ergometer exercise were significantly greater than after concentric contractions at equivalent workloads. Furthermore, adaptations to eccentric exercise which occur with training may explain why compartment pressures in runners during exercise at 80% of VO_2 max were not significantly greater than resting values.

Adaptations to repetitive eccentric contractions are known to occur with training. Adaptations result in decreased myofibrillar damage and muscular soreness (28). Other researchers (39, 55) have reported reductions in CPK following an exercise training program. However, research investigating the effects of training on anterior compartment pressure in runners has not been conducted. A reduction in myofibrillar damage with training would decrease interstitial fluid osmolality. A decrease in osmolality of interstitial fluid would, in turn, decrease

transudation of fluid from capillaries into the interstitium which would reduce anterior compartment pressure. Training adaptations may have contributed to the finding of no significant difference in anterior compartment pressure during submaximal exercise in competitive runners and cyclists.

The results of this study suggest that even maximal workloads of cycling are not sufficient to elicit an increase in compartment pressure as seen in runners. Hence, patients with chronic anterior compartment syndrome may be able to cycle at any workload without eliciting an increase in compartment pressure. Generally, patients with compartment syndrome exhibit average resting pressures of 18 mm Hg (73). However, if resting pressures do not exceed 35-50 mm Hg, the critical compartment pressure range which elicits ischemia and pain in many patients, cycling may provide a nonsurgical treatment option. This would allow the patient to maintain a level of cardiovascular fitness consistent with a decreased risk of cardiovascular disease (10). Future research should investigate compartment pressures during cycling in patients with chronic anterior compartment syndrome.

Recovery pressures taken 15 minutes after exercise at 80% of VO_2 max and after maximal exercise were not significantly different in runners and cyclists (Figure 6). This is the expected result since resting pressures were not significantly different at an alpha level of 0.05 and

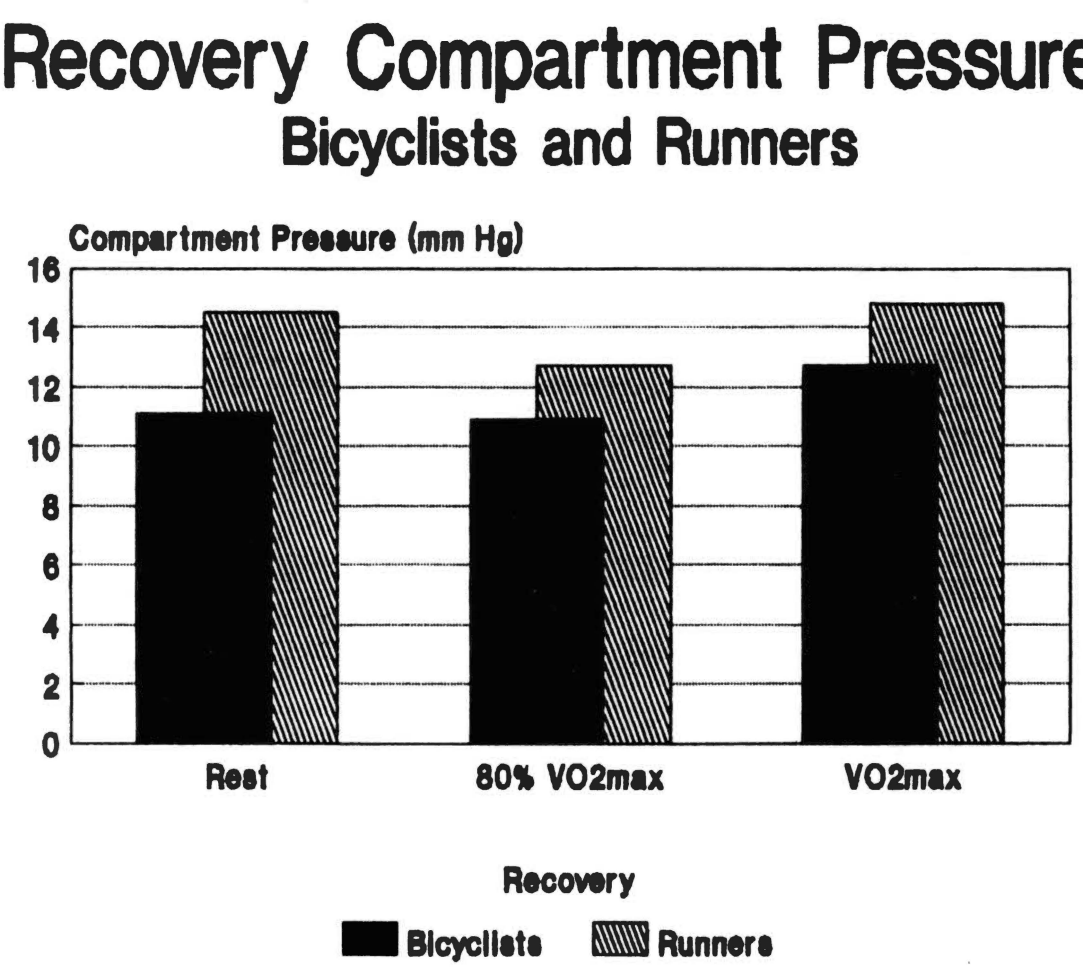

Figure 6. Resting and Recovery Compartment Pressures Measured after Exercise at 80% of VO_2 Max and Maximal Exercise

pressures should return to normal within 5-15 minutes following an exercise bout (73, 81).

CPK Levels in Runners and Cyclists

Figure 7 outlines pre and post exercise CPK levels in runners and cyclists. The assumption was made that plasma volume changes were not significantly different in cyclists and runners. If plasma volume changes in runners and cyclists were significantly different, direct comparison of CPK levels would yield erroneous results. Room temperature, length and intensity of the exercise bout were consistent for both groups; hence, this assumption is not unreasonable. Pre-exercise CPK levels were not significantly different between runners and cyclists. The normal range for CPK is 55-170 u/l (86). The CPK levels observed for the runners in this study are consistent with values reported by Wolf et al. (86). With an observed significance level of 0.0996, the small sample size and large standard error made it difficult to achieve significance. Furthermore, the loss of 2 degrees of freedom in the sample of runners due to inadequate processing of CPK samples contributed to the finding of no significant difference.

Post exercise CPK levels were also not significantly different between runners and cyclists. The observed significance level was 0.0627. Again, the loss of 2

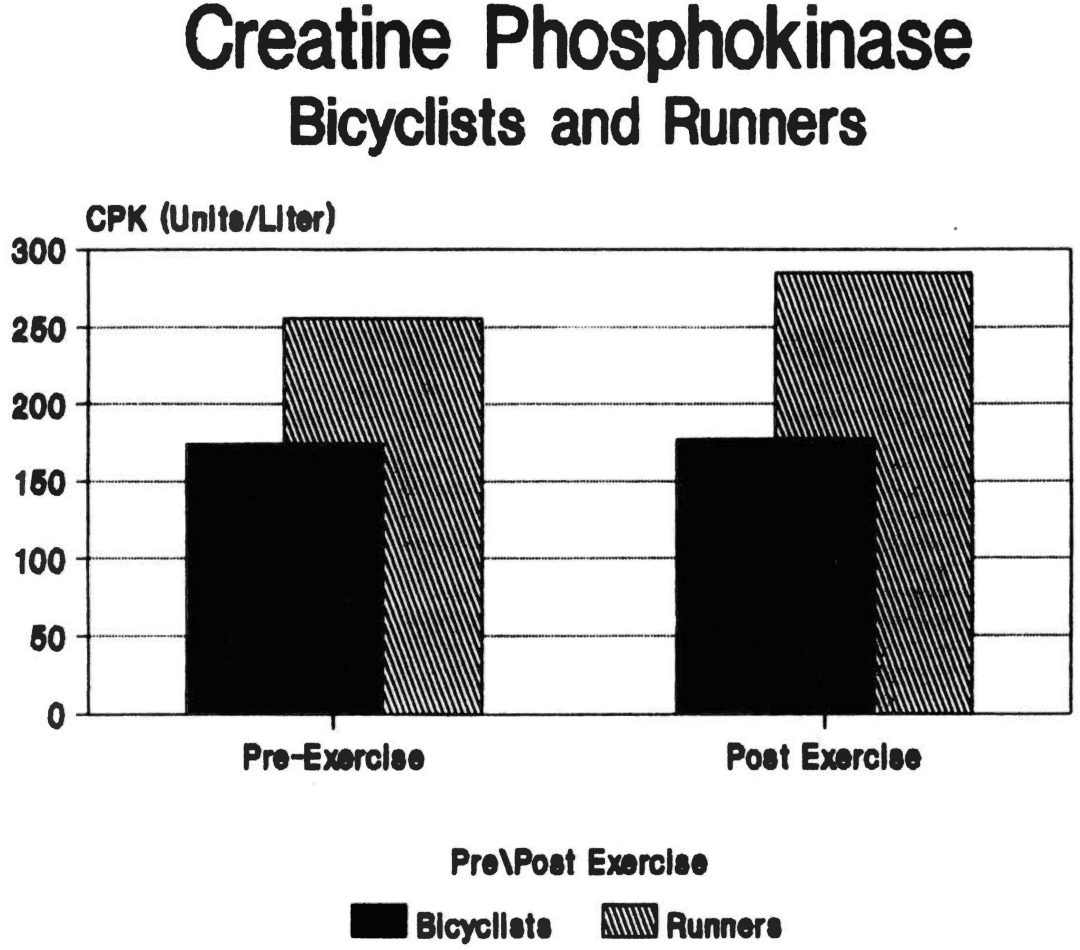

Figure 7. Total Creatine Phosphokinase Levels Measured in Runners and Cyclists before and after Exercise at 80% of VO_2 Max

samples, the small sample size and large standard error contributed to the finding of no significant difference.

The change in CPK values in runners as compared to cyclists, however, was significant at 0.0100. Differences in CPK levels were greater by a factor of 10 in the runners as compared to cyclists. Figure 8 depicts this large change in CPK. These findings are consistent with other studies which have examined the effect of running on CPK level (38, 86).

Assuming that skeletal muscle is the source of this elevated CPK, the data suggest that running may damage muscle tissue. Eccentric, but not concentric exercise has been demonstrated to damage muscle fibers (27). Running elicits eccentric contractions in a large portion of the musculature of the lower leg, unlike cycling (23). Hence, it could not be concluded that the increase in CPK during exercise in runners represented only CPK from muscles of the anterior compartment. However, it does support the hypothesis of eccentric muscle damage as a possible mechanism for the development of chronic anterior compartment syndrome.

Comparison of Descriptive Data for Runners and Cyclists

Runners and cyclists did not differ with respect to the number of competitions they participated in during the last year. However, cyclists had a lower aerobic capacity

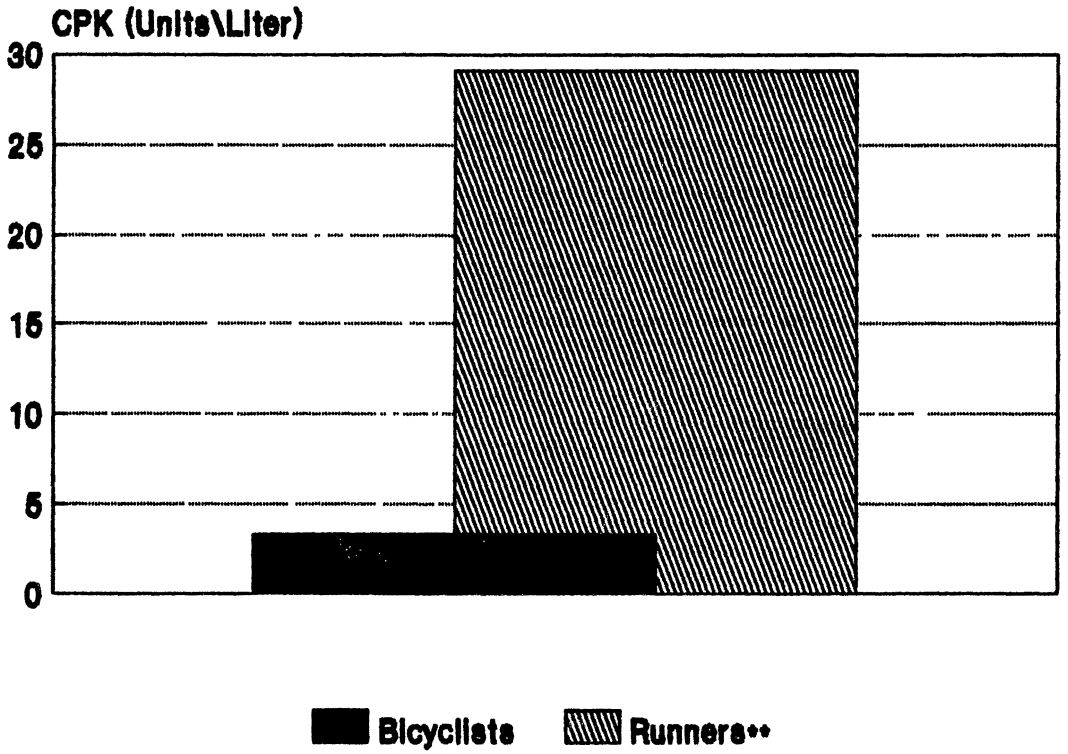

Figure 8. Change in Creatine Phosphokinase Levels in Runners and Cyclists after Exercise at 80% of VO_2 Max

than the runners; this was evident by the difference in VO_2 max values. Cyclists had only competed for an average of 2.8 years compared to 10.4 years for the runners. Both of these findings reflect the novelty of the sport of cycling as compared to running. The running boom of the last 2 decades lead to increased utilization of running as a means to improve health and fitness. Only recently, within the last 6 years, has participation in cycling increased and opportunities for local competition have been available.

Although runners were more fit, the cyclists would be considered very fit compared to standards for the general population. Since the cyclists had competed regularly for an average of 2.8 years, both groups would be considered, highly trained competitive athletes. Hence, both groups should demonstrate training adaptations specific to a given sport. This study did compare 2 groups of highly trained and fit cyclists and runners. Furthermore, since workloads were adjusted as a percent of VO_2 max, this research eliminated problems associated with other studies (41, 80). Studies (41, 80) which utilized subjects with a variety of fitness levels and who participated in different sports have made the interpretation of results difficult. Training adaptations are specific to the sport of participation. Furthermore, many studies (65, 80) utilized ankle ergometer exercise rather than dynamic total body exercise such as running or cycling. This limits the generalization of their results. Ankle ergometer exercise utilizes a very small

amount of total body musculature and would be similar to weight training. During weight training, systolic as well as diastolic blood pressure can increase due to extensive vasoconstriction in the nonworking musculature. This would tend to enhance the transudation of fluid into the anterior compartment and then increase compartment pressure at a more rapid rate. During aerobic exercise which utilizes large muscle groups, diastolic blood pressure often decreases due to the extensive vasodilation. A decline in diastolic blood pressure would tend to enhance reabsorption during muscle relaxation.

Anterior Compartment Pressures
in Runners

Anterior compartment pressures in runners were compared to determine if pressures were significantly different at different workloads. Compartment pressures at rest, after exercise at 80% of VO_2 max and after maximal exercise were significantly different in runners. Other studies (80), (82) report that muscle relaxation pressure in healthy individuals increases 15-25 mm Hg during running.

In this study, anterior compartment pressure decreased in runners after exercise at 80% of VO_2 max as compared to rest ($P<0.0087$). This was not reported in other studies probably because they utilized subjects with a variety of fitness levels and many used ankle ergometer exercise. During exercise, local muscle pressure is determined by a

balance of forces. An increase in mean arterial pressure as well as force of muscle contraction tends to increase compartment pressure while the venous pump which enhances venous outflow tends to lower compartment pressure during muscle relaxation (24, 53). During dynamic exercise which utilizes a large portion of total body musculature, the venous pump may function to decrease muscle relaxation pressures in response to moderate increases in the force of muscular contraction. At some critical point, increases in mean arterial pressure and interstitial fluid osmolality override the enhanced venous return of the muscle pump and pressure within the compartment begins to rise.

In sedentary individuals, increases in compartment pressure would be expected to occur at very low workloads as they would not demonstrate adaptations to eccentric exercise or cardiovascular exercise. Hence, any decrease in compartment pressure in fit individuals would be cancelled by the increased pressures measured in unfit individuals who participated in the same study. Adaptations to eccentric exercise, increased vascularization and other cardiovascular changes known to occur with exercise training may be a factor in lowering compartment pressure during submaximal exercise in runners. The venous pump may function more efficiently such that enhanced venous outflow lowers compartment pressure. The decrease in muscle relaxation pressure in runners at submaximal workloads, was not observed in the cyclists. This is not surprising as less of

the total body musculature is utilized during cycling suggesting that vasodilation would be less extensive. Hence, the venous pump would not be expected to lower anterior compartment pressure significantly during cycling as compared to running which utilizes upper as well as lower extremity musculature.

Compartment pressure measured after maximal exercise was significantly greater as compared to rest ($P<0.0225$) and after exercise at 80% of VO_2 max ($P<0.0020$) in runners. Compartment pressure increased by 4.6 mm Hg during maximal exercise as compared to rest. Muscle relaxation pressure is reported in the literature (80, 82) to increase by 15-25 mm Hg. The use of highly trained runners in this study may explain the smaller increase in compartment pressure as compared to other studies. Adaptations to training may yield smaller increases in compartment pressure.

The average increase in compartment pressure after maximal exercise may have been greater had all runners been rearfoot strikers. Midfoot strikers utilize an eccentric contraction in the plantar flexors to absorb the shock of foot impact rather than the dorsiflexors. Hence, the muscles of the anterior compartment would not be subjected to as much eccentric work in midfoot landers. For this reason, lower anterior compartment pressures would be expected in midfoot landers. A review of the literature revealed no studies which have compared anterior compartment pressures in midfoot and rearfoot landers.

Another factor which may also have suppressed the increase in compartment pressure after maximal exercise was the VO_2 max protocol. Since treadmill speed could not exceed 9.5 mph, elevation had to be increased to achieve a maximal workload in the runners. Increasing treadmill elevation would tend to shift rearfoot strikers to more of a midfoot landing pattern. This may have shifted eccentric muscle activity from the dorsiflexors to the plantar flexors. Compartment pressure would not be expected to rise as much in this case.

An examination of anterior compartment pressures in runners after exercise at 80% of VO_2 max and after maximal exercise revealed that every runner exhibited an increase in compartment pressure with maximal exercise. This leaves no doubt that compartment pressure increases at some critical workload, but the magnitude of this increase may have been affected by factors such as the exercise protocol and foot-strike pattern.

Force-plate studies combined with anterior compartment pressure measurements are necessary to determine if a relationship exists between foot-strike pattern and compartment pressure. The results of such a study may also provide useful information in the treatment of patients with chronic anterior compartment syndrome. Perhaps, in some patients, alteration of foot-strike pattern would lower compartment pressures sufficiently to permit asymptomatic training. Unfortunately, other factors such as degree of

pronation further complicate foot-strike pattern. Athletes who pronate, regardless of whether they exhibit a rearfoot or midfoot landing pattern, would utilize an eccentric contraction of the tibialis anterior to control pronation at foot-strike. Hence, degree of supination or pronation may also be a factor in determining anterior compartment pressure.

Resting anterior compartment pressure in runners was not significantly different than recovery pressures measured 15 minutes after exercise at 80% of VO_2 max and after a maximal workload. This finding is consistent with the literature; in healthy individuals, compartment pressure returns to pre-exercise values within 5-15 minutes after cessation of exercise (73, 81).

An examination of average anterior compartment recovery pressures measured 15 minutes after exercise at 80% of VO_2 max revealed that restoration of compartment pressure in runners was slower than after maximal exercise. Post exercise compartment pressure was 12.2 mm Hg with a 15 minute recovery pressure of 12.7 mm Hg measured 15 minutes after exercise at 80% of VO_2 max. This suggests that recovery is more rapid after maximal exercise as compared to submaximal exercise. Post exercise and recovery pressures after maximal exercise were 19.1 and 14.8 mm Hg, respectively.

Blood pressure was measured after each anterior compartment pressure measurement (Appendix). A comparison

of mean arterial pressure immediately after exercise at 80% of VO_2 max (87.4 mm Hg) and after maximal exercise (77.6 mm Hg) in runners revealed that mean arterial pressure was significantly lower after maximal exercise ($P<0.0100$). This was confirmed by observation of the subjects after completion of a maximal workload. Subjects were light-headed, showed signs of pallor and a few reported being nauseous. The significantly lower mean arterial pressure after maximal exercise probably resulted from the fact that maximal vasodilation had been achieved but not at the submaximal workload. According to Bevegard and Shepherd (9), blood flow to contracting muscle is not occluded until force of contraction exceeeds 70% of the maximal voluntary contraction. A reduction in mean arterial pressure would result in a larger pressure gradient between the compartment and capillary hydrostatic pressure. This would enhance fluid reabsorption, restoring compartment pressure to resting values more rapidly. After exercise at 80% of VO_2 max, an intensity below anaerobic threshold for all but 2 of the cyclists, vasodilation would not be as significant. Hence, the pressure difference between the compartment and capillary hydrostatic pressure would not be as large; therefore, restoration of resting compartment pressure would occur more slowly.

Anterior Compartment Pressures
in Cyclists

Anterior compartment pressures measured in cyclists at rest, after exercise at 80% of VO_2 max and after maximal exercise were not significantly different. This suggests that cycling is not accompanied by an increase in anterior compartment pressure even at maximal workloads. The absence of eccentric muscle contractions by the musculature of the anterior compartment may have contributed to this finding in cyclists as compared to runners. Increases in the force of contraction of the tibialis anterior during maximal exercise as compared to exericse at 80% of VO_2 max did not elevate compartment pressure in cyclists. Since a cyclist pedals at a rate of 80-100 revolutions/minute, the dorsiflexors are contracting concentrically at a rate of 80-100 contractions/minute. Pedal rate which does not increase significantly with increases in intensity (23), would not be expected to affect anterior compartment pressure measured at different workloads. The absence of eccentric contractions by the tibialis anterior or an insufficient increase in the force of muscular contraction during cycling may contribute to the finding of no significant increase in anterior compartment pressure during cycling. Regardless of the factors which contribute to elevations in anterior compartment pressure, the results of this study suggest that cycling may serve as an alternate activity for patients with

chronic anterior compartment syndrome who are unable to participate in weight bearing activities without pain in the lower leg.

A paired t-test revealed that none of the compartment pressures measured during cycling were significantly different, including pressures measured 15 minutes after exercise at 80% and 100% of VO_2 max.

CPK Levels in Runners

A paired t-test was utilized to determine if pre (255.6 u/l) and post (284.8 u/l) exercise CPK levels were significantly different in runners. This difference was significant with an observed significance level of 0.005. This finding is consistent with studies by Wolf et al. (86) and Apple et al. (3), suggesting that running imposes a significant stress upon skeletal muscle.

CPK Levels in Cyclists

Pre and post exercise CPK levels were not significantly different in the cyclists ($P<0.075$). In fact, average pre and post CPK levels for cyclists were at the upper end of the normal range (55-170 u/l). This suggests that the musculoskeletal demands of cycling do not damage skeletal muscle significantly.

The results of this study suggest that anterior compartment pressure increases significantly at some critical workload in runners with no workload related

increase in cyclists. This critical workload probably varies between individuals and is likely a function of training level. The presence of elevated CPK levels in the runners as compared to the cyclists is consistent with the hypothesis of eccentric muscle damage as a mechanism for chronic anterior compartment syndrome in runners as compared to cyclists. These findings of no significant increase in anterior compartment pressure during cycling suggest that patients with chronic anterior compartment syndrome may be able to tolerate cycling if they opt to avoid surgery or have an unsuccessful fasciotomy. The use of cycling as a pain free form of exercise in patients with chronic anterior compartment syndrome should be tested in a group of patients to determine its efficacy.

CHAPTER V

CONCLUSIONS AND RECOMMENDATIONS

Anterior compartment pressures have never been measured in response to cycling. This study measured anterior compartment pressure in response to a submaximal and a maximal workload in runners and cyclists. The findings of this study suggest that cycling does not elevate muscle relaxation pressure in the anterior compartment above resting values even after maximal exercise; running, however, yielded significantly greater compartment pressures after maximal exercise. At some critical workload, compartment pressures begin to rise during running. This critical point probably reflects decreased blood flow and is related to the eccentric muscle contraction utilized during running and/or the force of muscular contraction. Other factors such as exercise protocol, foot-strike pattern, and degree of pronation may also influence anterior compartment pressure during running.

The significant increase in total CPK activity after running for 20 minutes at 80% of VO_2 max suggests that running may damage skeletal muscle. Assuming, total CPK reflects skeletal and not cardiac muscle damage, the findings of this study support the hypothesis of eccentric

muscle damage as a mechanism for elevation of compartment pressure during exercise. However, other factors must be investigated to determine why chronic anterior compartment syndrome suddenly develops in an athlete who has competed for years.

Based on the results of this study, cycling may prove beneficial for the maintenance of activity in patients with chronic anterior compartment syndrome. Patients with resting pressures below 35-50 mm Hg may benefit most as pressures exceeding this range may readily elicit pain (36, 51). Next, anterior compartment pressure should be measured in response to cycling in patients with chronic anterior compartment syndrome. These measurements may yield useful information regarding the mechanism for chronic anterior compartment syndrome.

Conclusions

Based on the hypotheses stated and the limitations of this study, the following conclusions were made:

1. There was no significant difference in anterior compartment pressures measured at rest between competitive runners and cyclists.

2. There was no significant difference in anterior compartment pressures between competitive runners and cyclists after completion of 20 minutes of exercise at 80% of VO_2 max.

3. There was a significant increase in anterior compartment pressures after completion of a VO_2 max test in runners as compared to cyclists.

4. There was a significant increase in the difference in total CPK measured before and after 20 minutes of exercise at 80% of VO_2 max in runners as compared to cyclists.

Recommendations

This investigation included only healthy, competitive runners and cyclists. Therefore, these results cannot be extrapolated to those with chronic anterior compartment syndrome. A study investigating the effect of cycling on anterior compartment pressures in people with compartment syndrome would be the next step. This would demonstrate whether cycling could be utilized as a nonsurgical treatment option to allow patients to remain active without elevating compartment pressure.

Another area which should be investigated is the effect of foot-strike pattern on anterior compartment pressure. Initially, a force-plate analysis should be conducted on each subject to determine which are rearfoot and which are midfoot strikers. Next, compartment pressure could be measured at 2 or 3 equivalent workloads defined as a percentage of each subject's VO_2 max. If compartment pressures are higher in rearfoot as compared to midfoot strikers, alteration of foot-strike pattern in patients,

correction of pronation with orthotics, or both may reduce compartment pressure to a level which does not elicit pain. Elevation of the treadmill should be avoided in the treadmill protocol to eliminate any effect it may have on compartment pressure. The utilization of trained and untrained individuals would permit a comparison of the critical workload at which compartment pressure increases in both groups.

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APPENDIX

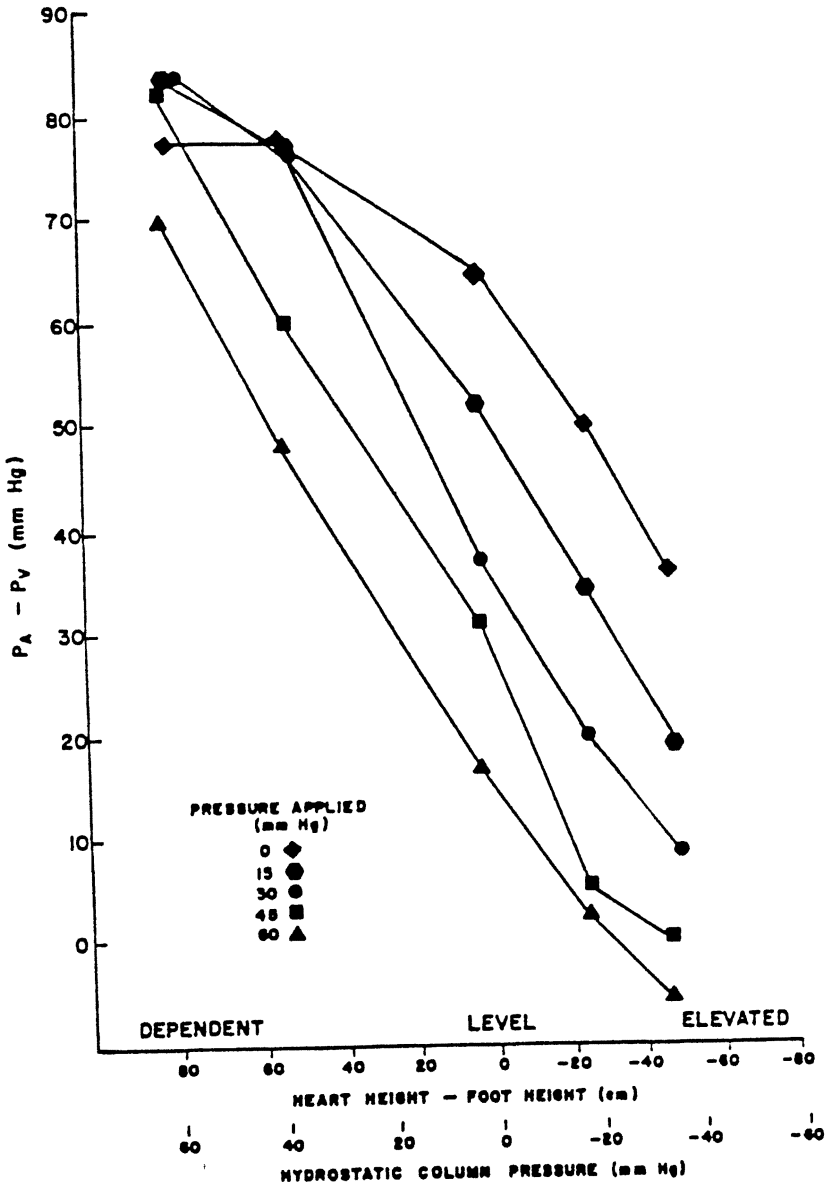

Figure 1. The Effect of Limb Position and Increased Tissue Pressure on Local Arteriovenous Gradient (54)

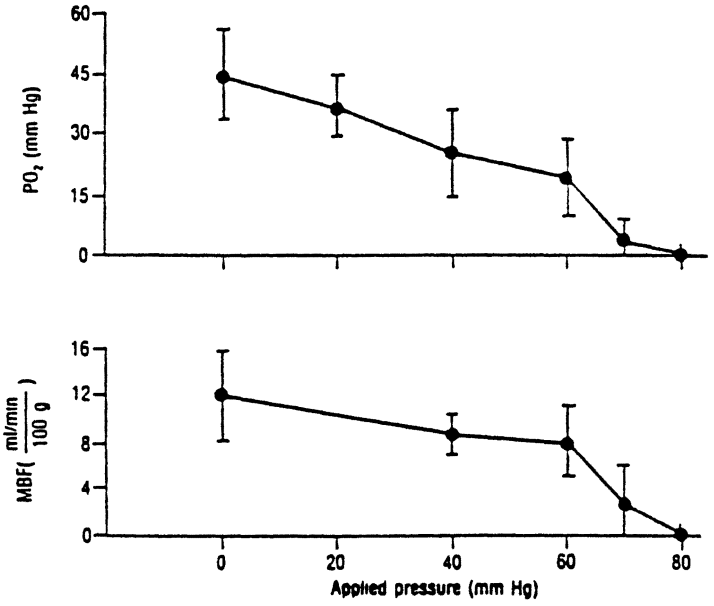

Figure 2. Muscle PO_2 and Blood Flow in the Anterior Compartments of Rabbits (52)

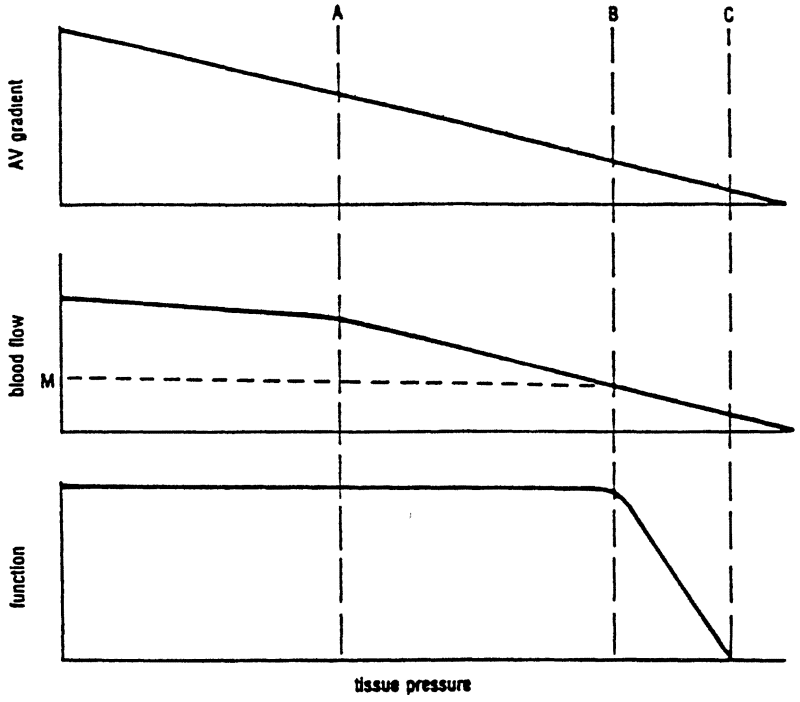

Figure 3. Relationships of Local Arteriovenous Gradient, Blood Flow, Tissue Function and Local Tissue Pressure (53)

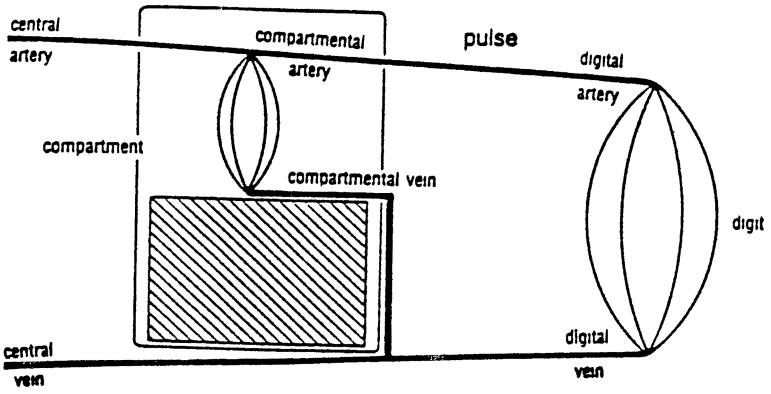

Figure 4. Comparison of Local and Distal Effects of Increased Tissue Pressure (53)

Runner or Cyclist (Circle One)

Name_____Age_____

Address_____City_____Zip_____

Average number of miles/week that you ride or run_____

Years of consistent competition_____

At what distances do you compete?_____

What is your average race pace?_____min/mile

On the average, how many competitions do you participate in on a yearly basis?_____

Do you have or have you ever had any of the following medical problems?

YES	NO	
_____	_____	High Blood Pressure
_____	_____	Any History of Cardiovascular Problems
_____	_____	Rapid or Abnormal Heart Rate
_____	_____	Do you smoke?
_____	_____	High Cholesterol
_____	_____	Abnormal EKG (Electrocardiogram)
_____	_____	Family History of Cardiovascular Disease prior to age 50
_____	_____	A history of Hemophilia (Excessive bleeding)
_____	_____	Injury or Trauma to the Lower Leg, if so, explain_____
_____	_____	Diabetes Mellitus
_____	_____	A History of Fainting Episodes or Dizziness
_____	_____	Do you take any over-the-counter or prescription medications?, If yes, list_____
_____	_____	A History of Chest Pain
_____	_____	Narrowing or abnormal functioning of a heart valve. If yes, explain_____

Name _____ Date _____

Time	BP	HR	RPM	GEARS SPEED/GRADE
Pre-X	_____	_____		
2:00	_____	_____	_____	_____
4:00	_____	_____	_____	_____
6:00	_____	_____	_____	_____
8:00	_____	_____	_____	_____
10:00	_____	_____	_____	_____
12:00	_____	_____	_____	_____
14:00	_____	_____	_____	_____
16:00	_____	_____	_____	_____
Post-X	_____	_____		

Compartment Pressures

Pre _____ Post-X _____ 15 Min Post _____

BP 15 Min Post _____

VO_2 Max _____ml/kg/min 80% VO_2 Max _____ml/dg/min

Anaerobic Threshold _____%VO_2 Max HR_____

_____

Date _____ Workload _____

Pre-X CPK _____ Post-X CPK _____

Dif CPK _____ 15 Min X HR _____ BP_____

Compartment pressures

Post-X _____ 15 Min Post _____

Post-X BP _____ 15 Min Post _____

RAW DATA FOR RUNNERS AND CYCLISTS

OBS	GROUP	ACTVTY	SUBJ	WKMILE	COMPDIST	AVEPACE	AGE	YRSCOMP	COMPSTYR	RESTPR	COMP8OX	COMPR8AX	POST8AX	POSTMAVX	VO2MAX	ADJVO2	PREPCPK	POSTCPK	DIFFCPK
1	B	BIKE	1	240	63	25.0	36	1	15	8	6	12	4	15	60.2	64.4	261	271	10
2	B	BIKE	2	280	56	22.0	19	1	20	10	12	7	17	16	60.0	64.2	173	179	6
3	B	BIKE	3	200	45	26.0	22	1	15	6	14	13	14	9	53.1	56.8	106	103	-3
4	B	BIKE	4	275	100	25.0	22	3	25	13	8	9	9	13	60.6	64.8	130	122	-8
5	B	BIKE	5	300	100	27.0	27	7	20	15	20	19	16	16	55.7	59.6	178	172	-6
6	B	BIKE	6	300	80	25.0	24	2	20	17	12	17	13	16	56.2	60.1	106	111	5
7	B	BIKE	7	300	70	25.0	24	3	30	16	9	18	11	14	56.2	60.1	112	119	7
8	B	BIKE	8	200	60	25.0	30	1	20	10	19	11	14	16	66.0	70.6	230	242	12
9	B	BIKE	9	200	60	24.0	29	5	15	6	9	9	4	7	53.7	57.5	183	185	2
10	B	BIKE	10	250	100	25.0	33	4	40	10	6	7	7	5	53.4	57.1	259	267	8
11	R	RUN	11	50	10	5.7	33	12	25	16	12	15	12	18	63.9	63.9	81	104	23
12	R	RUN	12	45	26	5.8	32	6	20	15	10	17	8	13	60.3	60.3	171	185	14
13	R	RUN	13	65	10	5.6	29	5	20	14	10	17	18	19	65.7	65.7	237	249	12
14	R	RUN	14	60	10	4.9	29	17	25	5	5	9	7	8	78.4	78.4	391	449	58
15	R	RUN	15	30	10	6.0	33	7	12	17	14	25	13	21	60.2	60.2	182	201	19
16	R	RUN	16	55	6	5.6	32	14	17	19	16	24	14	12	63.6	63.6	237	251	14
17	R	RUN	17	30	26	5.5	29	3	13	12	14	17	10	14	66.8	66.8	330	357	27
18	R	RUN	18	65	26	5.6	38	10	10	18	16	17	17	17	63.7	63.7	416	482	66
19	R	RUN	19	70	10	5.0	28	15	30	15	10	16	15	15	77.4	77.4	.	.	.
20	R	RUN	20	90	26	5.0	28	15	25	14	15	34	13	11	67.9	67.9	.	.	.

ANAEROBIC THRESHOLD FOR RUNNERS
AND CYCLISTS

Subject	Anaerobic Threshold (% of VO_2 max)
B1	85.0
B2	83.0
B3	82.0
B4	65.5
B5	91.0
B6	68.0
B7	87.5
B8	92.0
B9	85.0
B10	85.0
R1	89.0
R2	89.0
R3	93.0
R4	90.4
R5	-
R6	90.0
R7	92.0
R8	90.0
R9	87.0
R10	91.0

MEAN ARTERIAL PRESSURE IN RUNNERS
AFTER TWO EXERCISE
WORKLOADS

Subject	MAP at 80% VO_2 max (mm Hg)	MAP at Max (mm Hg)
R1	83	73
R2	86	85
R3	81	73
R4	87	73
R5	91	76
R6	78	-
R7	89	74
R8	93	90
R9	97	77
R10	89	-
$\overline{X}$	87.4	77.6

$t = 4.679$ OSL < 0.005**

MEAN ARTERIAL PRESSURE IN CYCLISTS
AFTER TWO EXERCISE
WORKLOADS

Subject	MAP 80% VO_2 max (mm Hg)	MAP at Max (mm Hg)
B1	92	107
B2	92	99
B3	90	89
B4	93	97
B5	94	94
B6	102	107
B7	79	99
B8	85	92
B9	86	66
B10	83	93
$\bar{X}$	89.6	94.3

$t = -1.37$ OSL > 0.10

PARTICIPANT RELEASE FORM
INFORMED CONSENT

Procedure:

Each participant will participate in two testing sessions, approximately one week apart. During the first session, height, weight, and resting blood pressure will be measured. A questionnaire regarding each participant's medical history and level of training will also be secured. The researcher will then measure lower leg pressures in the area next to the shin bone with the participant lying down. Local anesthesia will be administered to the surface tissue where lower leg pressures will be measured. A needle and pressure recording device will be used to accurately measure the lower leg pressure in the right leg. Next, the participant will participate in an exercise test of gradually increasing intensity on the bicycle or treadmill to the point of fatigue. The test will be terminated at the participant's request or if signs or symptoms necessitate. Heart rate, EKG, oxygen consumption and blood pressure will ne monitored throughout the test. Immediately following the exercise test, lower leg pressures will be taken and after 15 minutes. This first session will take approximately 70 minutes. During the second visit, 5 milliliters of blood will be drawn to test for levels of an enzyme called CPK. Next, each participant will repeat his previous test but at a lower intensity. This exercise test will last for 20 minutes and heart rate, EKG and blood pressure will be monitored. Following the test, the researcher will measure lower leg pressures as in the first test session. Five milliliters of blood will be drawn to measure enzyme levels after exercise. This second session will take approximately 45 minutes.

Although minor complications of these procedures are infection and some lower leg soreness, neither is expected. Any signs of infection should be reported to the researcher immediately. In such case medical attention will be provided at no cost to the participant. A very unlikely complication of the exercise test to fatigue is heart attack. The researcher has been trained to respond appropriately. Medical help will also be provided immediately at reasonable cost to the participant. All records will be strictly confidential and available only to the researcher and her research advisor. Records will be kept in a locked file for 1 year when they will be destroyed. The results of this study will be used to determine if bicycling is a plausible nonsurgical treatment option for chronic anterior compartment syndrome in athletes and active individuals.

Participants will be paid $50.00 for participation in all procedures related to both testing sessions. Reimbursement will be by mail approximately 4 weeks after completion of testing.

To the best of my knowledge I am not suffering from any of the following and am in excellent physical condition.

1. heart attack
2. heart failure
3. problems related to clotting of the blood
4. abnormal heart rhythm
5. narrowing or abnormal functioning of a heart valve
6. hypertension (high blood pressure)
7. metabolic disease such as diabetes
8. circulatory problems

To the best of my knowledge, my physical condition is adequate for my participation in this study. I understand that my participation is voluntary, that there is no penalty for refusal to participate, and that I am free to withdraw my consent and participation in this project at any time after notifying the project director. I understand that I was chosen for this study because I am a competitive cyclist or runner age 18-34 years.

I may contact Terry Maciula, University Research Services, 001 Life Sciences East, Oklahoma State University, Stillwater, OK 74078; Telephone 405-744-5700 or Dr. Breazile vitain Veterinary Medicine at 405-744-8089 should I like further information about the research.

A copy of this consent form has been given to me.

Date_____Participant_____

"I certify that I have personally explained all elements of this form to the subject or his/her representative before requesting the subject or his/her representative to sign it."

_____,Researcher

This is done as part of the investigation entitled

A Comparison of Anterior Compartment Pressures In Competitive Runners and Competitive Cyclists

CONSENT FORM

I, _____ have been asked to voluntarily participate in this study entitled: A Comparison of Anterior Compartment Pressures in Competitive Runners and Cyclists as part of the research required for Sue Beckham to complete her Ph.D. in Physiology, under the supervision of William A. Grana, M.D.

Purpose: I understand that the purpose of this study is to learn more about pressures in the lower leg during cycling and running. The results of this study may yield useful information in the treatment of patients with elevated lower leg pressures which prevent them from exercising or performing heavy physical labor.

Status of Device Used to Measure Lower Leg Pressures: I understand that the device used to measure lower leg pressures before and after exercise is an approved standard method for measuring lower leg pressures.

Description of Study: I understand that I have volunteered to participate in 2 testing sessions, approximately one week apart. During the first session, my height, weight and resting blood pressure will be measured. I will also be asked to complete a questionnaire about my medical history and level of training and competition which will take about 5 minutes to complete. In order to participate in the study, I must complete the medical questionnaire which evaluates whether my level of conditioning and health status is appropriate for the study. Next, the pressures in the lower part of my right leg next to my shin bone will be measured while I am lying down. Local anesthesia will be used to deaden the skin over the area where lower leg pressures will be measured. I know that a needle and pressure recording device will then be used to measure the pressure on the outside part of my right lower leg. A few teaspoons of saline solution will be injected into my lower leg through the needle in order to measure the pressure. The needle will remain in the leg for 5 to 10 seconds. The needle will then be removed. Next, I will participate in an exercise test of gradually increasing intensity on the bicycle or treadmill to the point of fatigue. I understand that the researcher will stop the test if I request or if any abnormal signs or symptoms are present at any time. My heart rate, electrocardiogram (measures the electrical activity of the heart), oxygen consumption and blood pressure will be monitored during the test. I will also wear a mask over my mouth which I will breathe into during the test. The exercise test will take approximately 15 minutes. Immediately after the test, the pressure in my right lower leg will be measured again and after 15 minutes.

The first testing session will last approximately 70 minutes.

In approximately one week, I will return for a second testing session. I understand that 5 teaspoons of blood will be drawn to test for levels of a muscle protein. Next, I will repeat my previous test on the bike or running on the treadmill, but at a lower intensity. This exercise test will last for 20 minutes and my heart rate, EKG and blood pressure will be monitored. During this test the intensity of the exercise will not change. I understand that I may stop the exercise test at any time and that the researcher will stop the test if abnormal signs or symptoms are present. Immediately following the exercise test and after 15 minutes, the pressure in my lower leg will be measured using the same method as in the first testing session. After the exercise test is completed, 5 teaspoons of blood will be drawn by the researcher to be tested for muscle protein again. The second session will take approximately 50 minutes.

Benefits: I may learn what my fitness level is by participating in this study. I will receive $50.00 for participating in all parts of both testing sessions. I understand that this will be sent to me by mail in approximately 4 weeks after I complete the study. I may also be helping doctors gain valuable medical information which can be used to treat athletes and other physically active persons with elevated lower leg pressures.

Possible Risks: I recognize that an unlikely risk is the possibility of heart attack during or after the exercise tests. I understand that the researcher has evaluated my medical history and that medical treatment will be provided for me at a reasonable cost in the event of a heart attack. Another unlikely risk is local infection over the area where lower leg pressure was measured. I will report any redness or swelling of this area to the researcher immediately so that medical treatment can be provided for me at a reasonable cost. I understand that the use of local anesthesia over the area where pressures will be measured will minimize the discomfort associated with a needle stick. I may have some soreness over the area where compartment pressures were measured for about 24 hours, but this is normal. Also, I might get a bruise, redness or infection from the needle stick when blood is drawn. I will report any signs of infection to the researcher immediately so that medical treatment can be provided for me at a reasonable cost.

In The Event Of Injury, Information Concerning Medical Treatment and Compensation: It is clear to me that no compensations will be available to me from the State of Oklahoma Teaching Hospitals or their employees unless I

otherwise qualify for the Hospital's health insurance or for other employee benefits. I understand that if I am so injured, medical facilities and treatment will be available to me. However, I will be required to pay a reasonable fee for such care. This does not mean that I could not receive medical benefits if otherwise entitled. I understand that if I have any questions or desire further information concerning the availability of compensation or medical care, I may contact the Chief of Staff for OMH, at 271-6323.

Assurances: When I sign this paper, it means that I am willing to be in this study. I have not given up any of my legal rights. I can stop being in this study at any time. If I do stop, it will not have any effect on my ability to get needed medical care. I understand that no one will be able to tell who I am from any reports or magazine articles that come out of this study. My answers to medical and other questions will be grouped in with other people's. I will be given a number for any blood tests which will be analyzed in a laboratory.

If I have questions or need to report any adverse effect about the research procedures, I will contact the principal investigator, Dr. William A. Grana at 232-1208 during the workday or at 341-9410, or Sue Beckham at 271-8177 during a workday or at 495-3799 on weekends.

If I have any questions about the study or my rights as a person in a research study, I can all Ms. Jan Trice, the Director of Research at the University of Oklahoma Health Sciences Center, Room 121, Library Building, at 405-271-2090.

Signatures:

I have read this informed consent document. I understand its contents and I freely consent to participate in this study under the conditions described in this document. I understand that I will receive a copy of this signed consent form.

Date	Signature of the Research Subject
Date	Signature of the Witness
Date	Signature of the Principal Investigator
Date	Sign. of Co-Principal Investigator

VITA

Susan Gay Beckham

Candidate for the Degree of

Doctor of Philosophy

Thesis: A COMPARISON OF ANTERIOR COMPARTMENT PRESSURES IN COMPETITIVE RUNNERS AND CYCLISTS

Major Field: Physiological Sciences

Biographical:

Personal Data: Born in Wichita, Kansas, March 27, 1956, the daughter of Mr. and Mrs. Rex Beckham.

Education: Graduated from Broken Arrow High School, Broken Arrow, Oklahoma, in May, 1974; received Bachelor of Science Degree in Geology from Southwest Missouri State University in May, 1980; completed Master of Science degree at Oklahoma State University in Health, Physical Education and Recreation in December, 1984; completed requirements for the Doctor of Philosophy degree at Oklahoma State University, August, 1989 through May, 1991.

Professional Experience: October 1990 to Present, Fitness, Inc., Oklahoma City, Oklahoma. Owner; consultant to St. Anthony Hospital for corporate marketing and educational programs.

June, 1985 to October, 1990, Oklahoma Center for Athletes, Oklahoma City, Oklahoma. Fitness Director; Supervised OCA fitness facility, budgeting, hiring of staff, conducted health evaluations including stress tests, exercise prescription and patient counseling for apparently healthy and high risk individuals. Developed a variety of workshops and certifications for fitness professionals. Marketed corporate wellness programs and supervised clinical internship programs for students at OSU, OU, OBU and CSU. Host and site director for American

College of Sports Medicine Workshops. Co-founder and Advisory Board member for Oklahoma Aerobic Teachers Association.

April, 1985 to June, 1985, St. Francis Hospital Wellness Center, Tulsa, Oklahoma. Exercise Technician: Exercise testing and prescription, supervised fitness center, developed weight training program for Reading and Bates satellite fitness facility.

August, 1983 to December, 1984, Oklahoma State University, Stillwater, Oklahoma. Student research assistant for the OSU Health and Fitness Center; stress testing, cardiac rehabilitation and exercise prescription.

August, 1983 to April, 1985, Tulsa Racquetball Aerobics Club, Tulsa, Oklahoma. Fitness Instructor.

April, 1982 - July, 1984, Jim Winnek, Inc., Tulsa, Oklahoma. Geologist; coal, hazardous waste and water wells.

Professional Organizations and Affiliations: American College of Sports Medicine, Oklahoma Aerobic Teachers Association, Oklahoma Fitness Professionals Association, Association for Fitness in Business.

Certifications: American College of Sports Medicine Program Director, American College of Sports Medicine Exercise Test Technologist.