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Effect of External Counterpulsation (ECP) on Delayed Onset Muscle Soreness (DOMS) in Long Distance Runners

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CARLY RAE CATANESE

ABSTRACT

External counterpulsation (ECP) has previously been used in treating cardiac patients. Compression of the lower extremities during diastole increases venous return and coronary perfusion.

Purpose: To determine if ECP affects delayed onset muscle soreness (DOMS) and markers of muscle inflammation and skeletal muscle damage.

Methods: Ten trained runners, 5 males and 5 females, aged 30.5 ± 12.8 years ran 20 miles at 70% of their VO_2 max on a pre-determined course on two different occasions, once under control conditions and once under ECP treatment conditions. Conditions were randomly assigned to eliminate order effect. Perceived leg pain and blood markers (creatine kinase, CK; lactate dehydrogenase, LDH; and C-reactive protein, CRP were measured pre-run, immediate post-run and for 5 consecutive days post-control and ECP treatment runs. During the treatment condition, subjects received 60 minutes of ECP post-run and everyday for 5 days post-run. To control for effects of ECP 5 additional subjects aged 25.6 ± 2.1 years received 5 days of ECP treatment while remaining inactive. Repeated measures ANOVA was used to assess treatment effects. Protected t-tests were used to assess serial differences. Correlations were obtained to assess

relationships between dependent variables. Repeated measures ANOVA were also used to assess gender differences in the control run and treatment run.

Results: There was no significant difference in mean weight loss, fluid intake, exercise heart rate, percentage of maximum heart rate or maximal oxygen consumption (VO_2) achieved, or running time between the control and ECP treatment runs. All indicators of muscle damage (CK, LDH, CRP, and pain) increased over time for the two runs, although there were no significant changes in the control group receiving ECP treatment only. CRP, pain, and LDH significantly decreased by two, one, and one day(s) post-ECP treatment, respectively, compared to the control condition. CK remained significantly elevated longer (3 days) in the ECP treatment run group. Positive correlations were found between CRP and CK (Day 3, 4, and 5), DOMS and LDH (immediately post-run), DOMS and CRP (Day 2), and CK and LDH (Day 5) in the control run only. Only females experienced a significant difference in CRP and CK, a 20% increase in CRP and a significant difference in CK immediately post-runs. A 20% reduction in pain was observed for both males and females as a result of treatment, though females experienced pain for one additional day. Females experienced a 20% increase in LDH. Males experienced a significant change Day 4 of the control run and immediately post-ECP treatment run, though difference between LDH significance may be attributed to the subject variability response.

Conclusions: ECP treatment after prolonged running decreased inflammation**,** muscle pain, and LDH elevation. CK levels remained elevated significantly in the ECP treatment run when compared to the control run. ECP alone did not cause significant increases in CK suggesting other factors contributed to this. Relationships exist between

indicators of muscle damage and inflammation. Gender differences were found in response to the ECP treatment run and control run.

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CHAPTER I

INTRODUCTION

Background

Enhanced external counterpulsation (EECP*) was originally designed as a mode of treatment for people diagnosed with coronary artery disease (CAD). While Europeans have used it for treatment for many years, not until recently did the United States use it for the purpose of treating cardiac patients. EECP has been mainly used for patients experiencing angina. The purpose of EECP is to increase perfusion of blood through the heart, therefore increasing oxygen supply to the myocardium. It has been documented that ECP* treatments over time increases the growth of collateral blood vessels.[1] These collateral vessels provide a means of greater oxygen supply to the myocardium.

ECP uses pneumatic compression cuffs that are placed on a patient's calves, thighs, and buttocks, increasing blood flow to the heart. Inflation of the cuffs increases blood flow to the heart. This causes a change in blood volume and pressure in the aorta during diastole, which increases perfusion of the coronary arteries. When the compression cuffs deflate, it results in blood flowing back through the buttocks, thighs,

and calves, normalizing aortic cavity pressure.[1] The timing of the deflation in the early phase of systole can cause a reduction in pressure and workload of the heart. Therefore, less cardiac work lowers the oxygen demand.[1,2]

According to Wilmer et al.[3], EECP treatment allowed patients to increase their tolerance for exercise as a result of increased blood flow, which helped to improve the quality of their lives. Patients had experienced less fatigue during daily routine activities and increased their activity level and duration of those activities.[2]

Previous research conducted on myocardial muscle damage[3] and on skeletal muscle damage from long distance running,[4] have entertained the idea that myocardial and skeletal muscle damage are not all that different. Lezhen[5] found that athletes treated with ECP increased lactic acid clearance of the blood and recovery from high intensity exercise. This was likely due to increased venous return from the lower extremities.

Delayed onset of muscle soreness (DOMS) is an effect of performing an exercise that the body is not accustomed to or that places a great deal of strain on the skeletal muscles, especially subsequent eccentric exercise.[6] McArdle and Katch[7] have proposed a DOMS Model which includes a series of events occurring from DOMS to recovery. It is proposed that DOMS causes damage to the sarcolemma, the muscle cell membrane. This damage results in the release of biochemical markers of muscle damage (e.g. creatine kinase (CK), lactate dehydrogenase (LDH), and myoglobin (Mb)). Unaccustomed exercise can also cause damage to the sarcoplasmic reticulum, the primary regulator of calcium, an essential metabolite for muscle contraction. Neural stimulation causes intracellular calcium to be released, allowing for muscle contraction. When neural stimulation ceases, muscle action is inhibited as calcium returns for storage in the

sarcoplasmic reticulum. When structure and function of the sarcoplasmic reticulum are influenced by unaccustomed exercise it can result in inefficient calcium release and absorption and an increased concentration of free moving calcium into the damaged muscle fibers. The free floating calcium in the damaged muscle fibers causes a decreased ability to exert force, increased cell damage, therefore, causing inefficient muscle action resulting in muscle soreness. The inflammation process begins as a precursor to full recovery of damaged muscle fibers. Inflammation, indicated by blood marker C-reactive protein (CRP), denotes the onset of the healing process.[7]

It is theorized that if ECP increases blood flow in cardiac patients and aids in recovery from high intense exercise it could have an effect on DOMS by increasing venous return and skeletal muscle perfusion.

Statement of the Problem

Research is needed to determine if ECP might be effective in reducing delayed onset of muscle soreness (DOMS) in long distance runners.

Purpose of the Study

The purpose of this study was to measure recovery from prolonged running with the use of ECP and its effect on reducing DOMS and biochemical markers of muscle damage.

Hypothesis

1. ECP treatment will reduce the delayed onset of muscle soreness (DOMS).
2. ECP treatment will reduce biochemical markers of muscle damage (C-reactive protein (CRP), creatine kinase (CK), and lactate dehydrogenase (LDH)).

3. There will be no difference between genders in response to the ECP treatment run and control run.

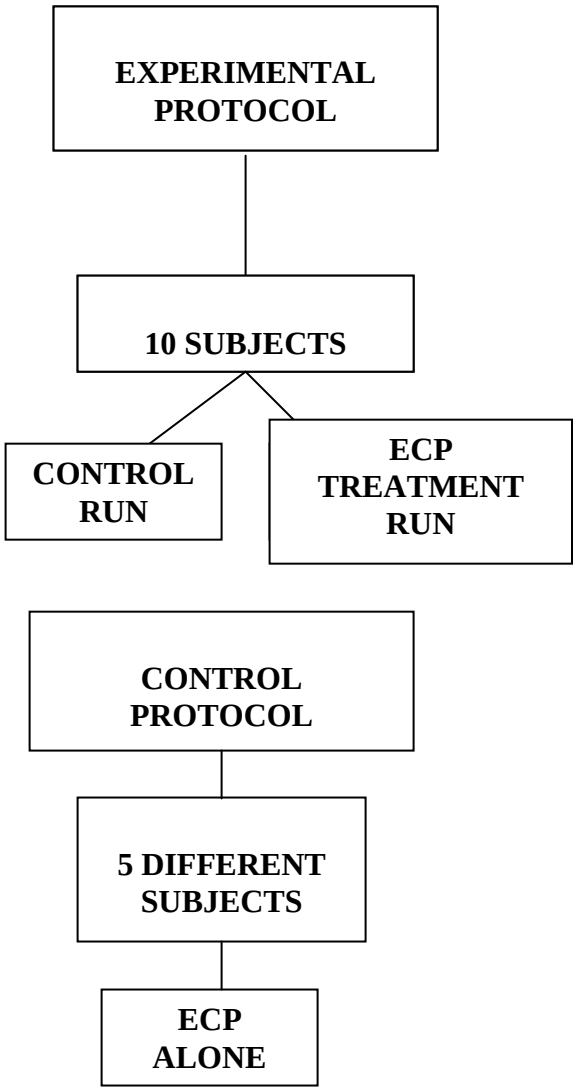
Definition of Terms

For the purpose of this study, several frequently used terms have been defined as follows[1]:

*Different manufacturers use different abbreviations; ECP (external counterpulsation) and EECP (enhanced external counterpulsation) are basically the same. For this study, ECP equipment from ScottCare Corporation (Cleveland, OH) was used.

- *DOMS:* Delayed onset muscle soreness or pain is experienced post-exercise due to skeletal muscle damage.
- *Diastolic Augmentation:* Diastolic pressure exceeds systolic pressure with a peak-to-peak ratio equal to 1.2 or greater, with 1.2 being therapeutic.
- *Peak-to-peak ratio:* The diastolic to systolic ratio (D/S).
- *End-diastolic pressure (EDP):* synonymous with preload. An increase in venous return causes an increase in EDP and ventricular filling during the heart's relaxation phase (diastole), which allows more blood to return to the heart during the contraction phase (systole).
- *Control run:* pertained to the ten subjects who ran 20 miles without ECP treatment.
- *ECP treatment run*: pertained to the ten subjects who ran 20 miles followed by 5 days of ECP treatment.

- *ECP alone:* pertains to 5 additional subjects, different from those involved in the two 20 mile runs, who received 5 days of ECP treatment only while remaining inactive.



CHAPTER II

LITERATURE REVIEW

The purpose of this study was to measure recovery from prolonged running with the use of ECP and its effect on reducing delayed onset of muscle soreness (DOMS). Literature relative to this study and its findings are summarized in the areas of EECP treatment and CAD, effect of various modes of exercise on muscle soreness and serum markers of DOMS, relationship of DOMS to inflammation, relationship between dehydration and DOMS, effect of ECP on recovery after prolonged exercise, and male and female responses to exercise induced muscle damage.

ECP and Cardiovascular Disease

According to Wilmer et al.,[3] coronary artery disease, myocardial ischemia, and refractory angina go hand-in-hand with arterial stiffness. Decreasing the elasticity of the arteries causes increased strain and pressure on the heart to work harder. Twenty patients, (16 men, 4 women) aged 48 to 70 years, served as subjects for this study. All patients exhibited cardiovascular risk factors, experienced prior cardiac problems and

treatments, and had chronic angina for at least 3 months due to myocardial ischemia. Drugs and medical procedures could not control the patients' problems and therefore they were prescribed EECP.

Each patient experienced 35, 1 hour daily EECP treatments for 7-8 weeks with an inflation pressure of 200-300 mmHg. Systolic, diastolic, and pulse pressure were measured during each treatment. Characteristics of the arterial walls and wave reflections were monitored and analyzed to determine if treatment improved these variables because both influence left ventricle afterload and myocardial oxygen demand.

Peripheral and central blood pressure, systolic blood pressure, pulse pressure, mean and diastolic pressure, and unaugmented pressure significantly decreased. As a result of EECP treatment, arterial stiffness was reduced and less energy was expended by the patients. Therefore, patients were able to increase their tolerance for exercise due to increased blood flow, which helped improve the quality of their lives. Patients experienced less fatigue doing daily routine activities and for that reason, they were able to increase their activity level and duration of those activities.

Urano et al.[8] demonstrated that ECP improved exercise tolerance, reduced exercise-induced myocardial ischemia, and improved diastolic filling in patients with CAD. Nine male and 3 female subjects, aged 51 to 78 years, participated in this study. All had CAD and documented ischemia, eight with effort angina and four with silent myocardial ischemia.

Each patient experienced sedentary or mild activity in a hospital setting, once with EECP treatment and once without. Before the subjects engaged in any activity, each subject had a medical history evaluation, a physical examination, and a stress test.

Systolic blood pressure was measured during each minute of rest, during exercise, and the first five minutes of the recovery phase. As soon as the subjects experienced any discomfort, such as fatigue or ischemia, exercise was terminated. The first phase, without EECP treatment, lasted 38 ± 9 days. Each patient underwent 35 hours of EECP treatment, each session lasting one hour, which was performed once or twice daily. This phase lasted 36 ± 6 days. Clinical examinations were performed before and after EECP treatment.

Cuffs were wrapped around the subjects' legs which exerted a pressure of 300 mmHg on the lower extremities in accordance with the subject's cardiac cycles. The results of the EECP treatment were determined by calculating the mean diastolic to systolic pressure ratio. From treatment, the mean diastolic to systolic pressure ratio was 1.1 ± 0.4 indicating that diastolic augmentation was achieved.

Exercise duration and exercise tolerance both significantly improved after EECP treatment. The results also showed that EECP treatment increased myocardial perfusion. There was a decrease in "the prevalence of reversible perfusion defects" and an increase in "the prevalence of normal perfusion" (p.96). It was concluded that exercise tolerance improved due to reduced episodes of myocardial ischemia.

Masuda et al.[9] found that ECP improved myocardial perfusion at rest as well as increased exercise tolerance and suggested that EECP treatment increased the production and use of collateral vessels. Eleven subjects, aged 21 to 81 years, who had been diagnosed with chronic stable angina participated in this study. They all performed a treadmill test, N-ammonia positron emission tomography, and had blood drawn within four weeks prior to treatment. The cuffs were placed on the calves, thighs and buttocks

and pressure was applied at the beginning of diastole. Once diastole had ended the pressure was released by deflating the cuffs. The amount of pressure used in this study was 250 mmHg and the treatment amounted to 35 hours, one to two sessions daily, taking approximately eighteen to thirty-five days.

The results revealed that myocardial perfusion at rest and perfusion in the anterior walls increased. There was also an increase in coronary flow reserve (maximal flow capacity) and exercise tolerance; however the latter was not significant.

The Effect of Various Modes of Exercise on DOMS and Serum Markers of Skeletal Muscle Damage

Schwane et al.[10] were the first researchers to study how running at different grades influence muscle soreness. They hypothesized that running down an incline, in which muscles are experiencing eccentric contractions, caused greater DOMS than running on level ground. Seven male subjects, aged 19 to 21 years, volunteered for this study. All were physically active on a regular basis, but for the duration of the study were asked to abstain from activity other than that performed during the experiment. Each subject performed three treadmill tests: a maximal oxygen consumption (VO_2 max) test, a run on level ground (0% grade), and a run at a 10% grade downhill, both for a 45 minute duration.

DOMS was assessed via plasma levels of creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) before exercise, 5 minutes, 24, 48, and 72 hours after the run at each grade. Subsequent tests were not performed immediately after the first. Six of the subjects waited six to seven days, and the seventh subject waited twenty-three days

between test one and two, and fourteen days between test two and three. Muscle soreness was also assessed using the Abraham scale.

There was significant muscle soreness in the gluteal, quadriceps, anterior leg, and posterior leg muscles, but not the hamstrings after the downhill run, while there was no significant muscle soreness after the run on level ground. There was no significant change in LDH levels, but there was a 351% increase of CPK within 24 hours of running downhill. The researchers also wanted to determine if there was a relationship between DOMS and inflammation, identifiable by the abundance of white blood cells found in the blood, but no significant relationship was found.

According to their results, Schwane et al.[10] found that running downhill perpetuated muscle soreness and caused CPK levels to significantly increase. They suggested that DOMS and increased levels of CPK were results of changes in muscle tissue structure due to the eccentric contractions performed. They also proposed that DOMS could have been the result of muscles being used in an unaccustomed way.

Koskinen et al.[11] conducted a study assessing proteins released into the bloodstream from the break-down of extracellular matrix components, after exercise-induced muscle damage. The researchers induced muscle damage in fourteen healthy, physically active male volunteers, aged 20-32 years. The men were separated into two groups, with both running at a 10% grade downhill on a treadmill for 45 minutes, but one group performed the run at room temperature (22 degrees Celsius) and the other in the cold (5 degrees Celsius). Each subject had venous blood samples taken pre-run, immediately post-run, and one, four, and seven days after the run.

The results showed that CK increased for both groups, each peaking after one day, some reaching four to six times the normal levels, although the participants who ran in the cold had significantly higher levels of CK. Therefore, those who performed in the cold temperature experienced more muscle damage. It was presumed that running in a cold environment decreased work capacity and therefore CK levels increased because it took increased work and involvement of additional muscle fibers. The authors felt this was more pertinent to the results than the temperature factor.

Kyrolainen et al.[12] examined metabolic measurements of seven triathletes including one woman and six men, aged 29 ± 5 years, who had volunteered to run a marathon. Blood samples were taken from each subject's ulnar vein before, during, and after the marathon to evaluate levels of serum creatine kinase (S-CK) and plasma skeletal troponin I (sTnI). Finger pricks were taken to determine blood lactate (B-La) levels.

The presence of CK in the blood reached its peak two to four days following intense exercise. By day six, CK had returned back to normal levels. STnI levels were increased which indicated a disruption in the skeletal muscle cells. It took approximately two days after the marathon for the sTnI levels to return to baseline. The onset of muscle soreness began during the run and did not cease until five days post-marathon. In summary, S-CK levels peaked at two days after the marathon, sTnI two hours after the marathon, and B-La remained relatively constant throughout the duration of the testing.

Muscle damage may be experienced if there is an increase in the levels of metabolites in the skeletal muscles or a mechanical disturbance of the muscle cell. The increased levels of S-CK and sTnI that the marathon runners' experienced was attributed to the latter.

Kobayashi et al.[13] was interested in the long term effects of marathon running performed by recreational runners. They wanted to determine how muscle damage affected muscle enzymes creatine kinase and lactate dehydrogenase. Fifteen healthy male volunteers took part in this study (mean age 43.5 years). All had participated in endurance training for an average of 50 km/week for 9 years. The study was performed in February at a temperature of between 8 and 10 degrees Celsius. The participants were not allowed to eat or drink the morning of the race. Before beginning the marathon (flat terrain), 10 mL venous blood samples of CK and LDH were taken from an antecubital vein to determine baseline levels. During the race the subjects had fluids available at all times. Ten milliliter blood samples were taken 5 minutes post-race. The baseline levels of serum CK and LDH were within the normal range, 163 U/L and 323 U/L, respectively. Subsequent samples were taken on days 1, 2, 3, 7, and 14. This method of sampling did not allow for maximal and minimal values of CK and LDH values during the recovery period.

CK levels tripled, on average. One day post-run the values had increased fifteen times baseline, then decreased, taking about one week to return to baseline. LDH levels doubled post-race and from there on decreased. It took about two weeks for LDH to return to normal levels. The subjects participated in very little running during the first week post-run due to muscle soreness. Kobayashi et al.[13] concluded that CK and LDH levels increased in recreational runners after running a marathon, therefore inferring that the run caused the muscle damage. Within two weeks the runners were back to baseline and normal training intensity.

Pachalis et al.[14] examined differences in muscle damage between high and low intensity eccentric exercise. The method of exercise was isokinetic quadriceps eccentric exercise. Twelve male subjects, aged 20 to 22 years, participated, all who had no prior resistance training experience. The extent of muscle damage experienced by each subject was assessed pre- and post-exercise, 24, 48, 72, and 96 hours after the exercise. Each subject performed two isokinetic quadriceps eccentric exercise sessions on each leg, with a 2 week rest period between sessions. This time period had previously been shown to recover muscle damage markers back to baseline. Each subject participated in high intensity (HI) and low intensity (LI) eccentric exercise, with equal work performed doing both HI and LI. Plasma CK and DOMS increased for both HI and LI eccentric exercise, however, there was only a significant difference between CK levels 24 hours post-exercise. The results indicated that HI and LI exercise had similar effects on muscle damage suggesting that muscle damage is caused more by the volume of exercise as opposed to the intensity of exercise.

Clarkson et al.[15] had 203 subjects aged 18 to 40 years perform two sets of 25 maximal eccentric contractions of the elbow flexor on a modified preacher curl bench. There was a five minute rest period between each set. Each contraction lasted for three seconds with a subsequent twelve second rest period. The subjects had to maximally contract their elbow flexors to resist the downward movement of a lever controlled by a tester. Subjects were encouraged to remain hydrated pre- and mid-exercise. Prior to the exercise and 4, 7, and 10 days post-exercise, blood samples were drawn to assess renal function and muscle damage until blood markers had returned to baseline levels.

Peak CK levels were averaged and reported to be 6420 UL^{-1} on day 4, 2100 UL^{-1} on day 7, and 311% above baseline on day 10. The normal CK range was reported to be 24-195 U/L. One-hundred eleven subjects had CK levels above 2000 UL^{-1} by day 4 which has been used to diagnose myositis (an inflammatory muscle disease due to a viral, bacterial, or parasitic infection).[16] Fifty-one subjects (25%) had greater than 10,000 UL^{-1} which has been associated with rhabdomyolysis (a condition in which there is excessive amount of myoglobin in the urine—due to muscle damage).[16] The normal range for LDH was reported to be 118-273 U/L with the greatest range being 86 - 1608 ± 292 U/L.

LDH levels increased significantly with a strong correlation existing between CK and LDH levels (r = .95). Renal function was not impaired as a result of the elevated blood markers. It was concluded that elevated CK levels, as a result of exercise induced muscle damage, did not influence renal function.

The purpose of Sayers et al.'s[17] study was to determine if activity affected the recovery of muscle of the elbow flexors after eccentric exercise. Twenty-six college aged men aged 20.8 $\pm$ 0.9 years who were not weight trained participated in this study. Each subject was randomly assigned to one of three groups: (1) immobilization—in which the subjects' non-dominant arm was placed in a cast immediately post-exercise for the duration of the treatment, (2) control, and (3) light exercise—which consisted of 50 bicep curls with 5 lb weights, therefore increasing activity level without causing additional muscle damage. The duration of the study was fifteen days. For three days baseline measurements of relaxed arm angle (RANG), flexed arm angle (FANG), maximal isometric force (MIF), and perceived muscle soreness (SOR), were taken. For four days subjects performed 50 maximal eccentric contractions of their non-dominant elbow flexor

using a modified preacher curl. The purpose of this exercise was to induce muscle damage. As the subject applied maximal resistance to perform a curl, an investigator used a lever to cause the elbow to extend. The subjects were asked to resist the lever, therefore resisting the action to extend their elbow. The fifty contractions were split into two sets with five minute rest periods, each contraction lasting approximately three seconds with a twelve second rest period between contractions. Treatment was followed by eight days of recovery measurements.

No significant difference was found among groups over time for baseline RANG, FANG, or MIF, though there was a significant main effect for time in RANG, FANG, and MIF from before to immediately after eccentric exercise. There was no significant group by time interaction in pre- and post-exercise RANG and MIF, but there was in exercise FANG. The immobilization group had a significantly greater average FANG than the control group immediately post exercise.

Subjects from all groups experienced an extended decrease in RANG, an increase in FANG and SOR, and a decrease in MIF in the days subsequent to exercise. Throughout the eight day recovery period no significant group by time interaction was found in RANG or FANG, though there was a significant group by time interaction for MIF and SOR. Activity or inactivity did not influence the recovery of the joint angle.

No significant difference was found in MIF among the groups until the fourth day of recovery, in which the light exercise group had a significantly greater MIF than the control group. From the fifth to the eighth day of recovery the immobilization and light exercise group experienced significantly greater MIF than the control group. No significant difference was found between the immobilization and light exercise group

throughout the duration of recovery. Immediately post treatment (day 5) the control group had the greatest recovery of baseline MIF of 76%, as opposed to the light exercise group (71%), and the immobilization group (68%). Within the next seven days the control group experienced a plateau in recovery, whereas the immobilization and light exercise group improved considerably. No group returned to baseline levels for MIF, but 90% improvement was experienced by the immobilization group, 88% for the light exercise group, and 82% for the control group.

A significant group main effect, time main effect, and group by time interaction was found in SOR throughout the eight day recovery period. The immobilization group had significantly greater SOR the last day of treatment and the first day of recovery than either the control or light exercise group. After day two there was no longer a significantly greater SOR. All groups had practically returned to baseline levels of SOR by the fourth day of recovery.

The researchers determined that activity level did influence muscle recovery after eccentric exercise of the elbow flexor, with immobilization and light exercise resulting in improved muscle function post exercise.

Hyatt[18] performed a study to analyze the release and clearance of CK in the muscles following two periods of eccentric exercise and to determine if the decrease in CK elevation from the first to the second bout of exercise was due to enhanced enzyme clearance. Eight college aged males performed two sets of 50 maximal eccentric exercise contractions on a modified preacher-curl bench. Each set was separated by 5 minutes of rest. Each contraction lasted about 3-5 seconds with a 10-12 second rest period in between contractions to prevent fatigue of the exercised muscles.

Subjects were randomly assigned to one of two groups, the control (CON) and experimental (EXP). The difference between the groups was that the EXP performed eccentric exercises on the opposite arm for the second bout of exercise, whereas the CON performed both bouts on the same arm. Subjects were assessed for 12 consecutive days. Blood was drawn for baseline levels on day 1, day 2 the exercise was performed, and day 3-6 blood samples and criterion measurements were taken, including: range of motion (ROM), circumferences (CIR), muscular strength (STR), maximal voluntary contraction (MVC), and muscle soreness (SOR), the particular measurement of interest, as it relates to the current study. Subjects were randomly assigned to the CON or EXP group on day 7, following the same procedure performed in the previous bout of exercise.

Soreness was measured using multiple means: a visual analog scale (VAS), self-palpation (SOR-PALP) and movement (SOR-MVT) by extending and flexing the exercised arm. The VAS scale ranged from 0 (not sure) to 100 (very, very sore). Soreness was evaluated pre- and immediately post-exercise and every day thereafter for a duration of 5 days per bout of exercise performed. Blood samples were taken 2, 6, 24, 48, 72, 96, and 120 hours post-exercise.

Similar SOR-MVT and SOR-PALP results were found after the first exercise bout and significantly increased following the first exercise in both groups, with no significant difference between the two. The EXP group, which used a different arm to perform the second bout of exercise, experienced significantly greater soreness than the CON after the second bout of exercise. The difference between each exercise bout for both groups was significant.

The second exercise bout had a similar effect on muscle damage as the first bout, though on the opposing arm. The CON groups second bout was not as influential as the first, indicating a repeated-bout effect. There was an additional effect on ROM, STR, CIR, and SOR, but it was not as drastic as the first.

The EXP group experienced a similar response post-exercise in ROM, STR, and CIR, though not in SOR. The suspected reasoning for the difference in SOR between EXP groups' bouts was an increased pain tolerance. The subjects gauged their pain score based on there perceived pain from the previous exercise.

The second bout of exercise for the CON group, performed within days of one another, did not further elevate total creatine kinase levels (TCK), though the EXP groups' TCK rose again. The study suggests that the TCK blunted response was to due to accelerated enzyme clearance by the reticulo-endothelial system.

The study reported that the majority of TCK (90%) is found in the muscle (MM) isoform, which is further divided into MM1, MM2, and MM3. Only MM1 is found within the muscle, the other two are by-products. The presence of MM1 in the blood indicates freshly released CK from the muscle. The early release of MMI post-exercise bout 1 corresponded with the penetration of neutrophils found within muscle tissue and circulating blood. It is suspected that the increase in the %MM1 could be due to the diffusion of neutrophils. As mentioned, there was a rise in the % of MM1 for the EXP and CON group, but less so in the CON group, possibly from muscle fibers that had not completely recovered from the first bout, or as a result of additional muscle fiber damage to fibers that were not influenced from the first exercise bout. Post-exercise bout two

there was an increase in MM1, a decrease in strength, ROM, and soreness, though less than that experienced from bout one.

In summary, CK cleared faster after the first bout of exercise and additional CK was released in both groups during the second bout of exercise when assessing MM isoform data.

The study by Nosaka et al.[19] examined the correlation between DOMS and indicative measures of muscle damage after performing bouts of eccentric exercises of the elbow flexor, similar to those described previously. One-hundred ten healthy male subjects aged 20.3 ± 2.3 years participated in this study. Fifty subjects were randomly selected to perform 12 maximal eccentric exercises (12ECC) of the non-dominant arm's elbow flexor whereas sixty subjects were chosen to perform 24 maximal eccentric exercises (24ECC). Fourteen of the 50 subjects who completed the 12ECC had to partake in 60ECC the following year because there were minimal changes in the indicative markers of muscle damage. The exercise required the use of a modified arm curl machine in which after 1 s of maximal isometric contraction an investigator manipulated the lever the arm was resting on, therefore causing the elbow at 90 degrees to extend to 180 degrees in 3 s. The subjects were asked to resist the force of the investigator, therefore resisting the alteration in range of motion. Fifteen seconds was allotted between contraction for each of the groups (12ECC, 24ECC, 60ECC).

A modified VAS was used to assess muscle soreness. The scale ranged from 0 = "no pain" to 50 = "unbearable pain". A palpation assessment (SOR-Pal) was performed to assess soreness via 3 s of applied pressure to deep tissue of the proximal, middle, and distal portion of the biceps brachii by the investigator's fingers. A flexion

and extension assessment (SOR-Flx and SOR-Ext, respectively) was performed by means of an investigator manually moving the subjects' forearm in a way to maximally flex or extend the elbow flexor, while the subjects relaxed their arm.

Indirect markers of muscle damage (MIF, RANG, FANG, CIR) were taken pre, immediately post, and 1, 2, 3, and 4 days after the eccentric exercises were performed. Variations in level of soreness existed among subjects even when similarities in markers of muscle damage were present. The greater the SOR values the greater the degree of recovery of MIF. Ultrasound pictures of the subjects pre, immediately post, and 4 days after exercise revealed that there was invariable peak muscle soreness levels though there were distinct differences between peak CK levels and the other markers of muscle damage. Therefore, it is suggested that DOMS and muscle damage are not directly related. Nosaka et al.[19] stated that there is minimal to no relationship between DOMS and indicative markers of muscle damage.

Nosaka et al.[19] suggests that subjectivity of the soreness reported could have been a resultant of the variability in responses to the eccentric exercise, especially since different subjects were used for the 12ECC and 24ECC groups. Nosaka et al.[19] stated that viable documentation is provided stating that other factors could play a role in the soreness response, such as past experiences, personal threshold for pain, mood, and health and hormonal status.

Relationship of DOMS to Inflammation

Simpson et al.[20] examined the biological and immune response to hill racing to determine the effects a competition had on muscle damage and the acute phase response to inflammation. Seven runners, six males and one female, aged 32 ± 0.8 years, served as

subjects. They all competed a 7 km run and had blood samples taken before the race, immediately post-race, and 48 hours post-race to assess skeletal muscle damage markers.

The majority of muscle soreness was experienced 48 hours post-race, the same time period that it took CK levels to reach their peak. There was no significant change in C-reactive protein (CRP). Although there was muscle damage and soreness, there was no change in acute-phase proteins, which are indicators of inflammation, CRP being one of many acute-phase reactants. No increase in neutrophils, white blood cells that are the first to react to inflammation, were found, although a characteristic of neutrophils is that they are short-lived and there was presence of immature neutrophils 48 hours post-race. It was concluded that the runners experienced muscle damage, but there was no acute phase inflammatory response.

MacIntyre et al.[21] examined the time-course and relationship between inflammation and DOMS following eccentric exercise in twelve healthy males, aged 21 to 29 years, who exercised less than six hours a week. At the onset of their participation, subjects had eccentric muscle strength tested on the right quadriceps and had 10 mL of blood drawn via venipuncture to assess the contractile proteins. Three hours prior to exercise, 100 mL of blood was taken and neutrophils were separated, labeled with a contractile protein, and infused back into the subjects prior to exercise. Each subject performed three hundred eccentric repetitions of the right quadriceps muscle using an isokinetic dynamometer. Ten milliters of blood was drawn from the subjects 0, 2, 4, 6, 20, 24, 48, 72 hours, 6 and 9 days post-exercise to assess DOMS.

DOMS was the highest immediate post-exercise until 48 hours when it started to decline; it disappeared by the ninth day. Increased levels of contractile protein labeled

neutrophils in the quadriceps muscles were present, even though neutrophils have a short lifespan as a result of them quickly being transported to the exercised muscle. The researchers found a significant relationship between muscle soreness and inflammation and claimed that they were the first to report this relationship.

In the study by Chleboun et al.,[22] 11 female subjects, aged 20.6 $\pm$ 1.4 years, volunteered to perform eccentric exercises of the elbow flexor. Muscle soreness, isometric strength, stiffness, and swelling measurements were recorded 3 days prior to the eccentric contractions, immediately post-exercise, everyday for 5 days, and every other day for the next week.

Muscle soreness was assessed using a five point scale, 0 = no pain, 1 = pain on palpation only, 2 = mild pain with full flexion or extension of the elbow, 3 = significant pain with full flexion or extension of the elbow, and 4 = constant pain. Stiffness was assessed by examining the angle and torque of the elbow flexor while performing extension exercises and examining relaxed arm angle. Swelling within the arm was determined by alterations in arm circumference and variations in the muscle volume of the biceps and brachialis, tricep, and humerus.

Peak muscle soreness was experienced 2-3 days post-exercise. Average pain was determined to be 2.9 on day 2 which indicated that the majority of pain was experienced when the muscle was being fully extended or flexed as opposed to relaxed. Minute swelling of the elbow flexor was observed immediately post-exercise, rose day 1 and 2, reached a plateau by day 3-5 and progressively decreased day 7-11. Muscle volume significantly increased from pre- to post-exercise (days 2-7) by 26.1 $\pm$ 4.3%. The whole arm also swelled up, but to a lesser extent. The volume of the elbow flexor muscles was

15.6% pre-exercise and 19.1% 3 days post-exercise. The volume of the subcutaneous tissue and cross-sectional area increased, though it was subsequent to the increased volume of the elbow flexor muscles. The significant increase in cross-sectional area size was seen post-exercise days 2-7 with peak swelling occurring post-exercise day 3 (24.2 $\pm$ 5.2%). Stiffness significantly increased by 59.9 $\pm$ 14.1% immediately post-exercise and remained prominent for 5 days post-exercise. Elbow angle was significantly less by day 3-7 post-exercise.

The study suggests that muscle swelling is not responsible for muscle stiffness experienced 48 hours post-exercise, though it could influence the succeeding stiffness experienced. Chleboun et al.[22] suggests that extracellular edema is related to tissue damage and the resulting inflammation and that intra- and extracellular edema were the reason for the swelling of the exercised muscle. The authors also reason that the delay in subcutaneous swelling could be a result of the muscle fluid moving from the exercise muscle to the tissue.

The Relationship of Dehydration to DOMS

Cleary et al.[23] performed a study to determine if thermoregulation in a heated environment negatively affected muscle damage and DOMS. Ten healthy male subjects, aged 18 to 35 years, volunteered. All subjects had not participated in any type of lower body training for 6 months prior to the study. For the duration of the study they were advised to refrain from any treatment that could reduce DOMS such as massage therapy, stretching exercises, or anti-inflammatory drugs. Four variables of DOMS were measured: perceived pain, muscle tenderness, passive range of motion, and isometric muscle strength.

Baseline information was collected 15 to 20 hours before the study began. The subjects were randomly assigned to two groups, one euhydrated/hyperthermic and the other dehydrated/hyperthermic. Hyperthermia was defined as maintaining an average core body temperature of greater than 38 degrees Celsius. The researchers aimed to get the dehydrated group's body mass down by 3 ± 1%.

All subjects participated in a heat stress trial where they walked on a motor-driven treadmill in an environmental chamber at a temperature of 40 ± 1.0 degree Celsius and a relative humidity of 75 ± 10%. The temperature and humidity were monitored often to verify that exercise was being performed in a hot and humid environment. The walking exercise was performed at a 0 degree grade for a total of 60 minutes, including a 5 minute warm-up at 2.5 mi/hr and exercise for 55 min at 3.0 mi/hr. The dehydrated group was restricted from drinking any fluids whereas the euhydrated group was permitted to drink cool water at their discretion.

At the completion of the 60 minute walk, subjects from both groups had to run downhill to induce muscle soreness. Each subject began the run within 15 minutes of the walking exercise to ensure they remained in the hyperthermic state. The total run time was 45 minutes at a negative 12 degree grade, including a 5 minute warm-up and 40 minutes of running at 5.0 ± 0.2 mi/hr at 50% of their average heart rate. The run was performed in a thermoneutral environment (22 degrees Celsius; relative humidity of 45%). Once again, the dehydrated group was not allowed to ingest any fluids whereas the euhydrated group could drink water as they wished.

The researchers concluded that there was no significant difference between the rectal temperature between both groups pre- and post-heat-stress trial. The dehydrated

group had a significant 3.3% decrease in body mass (-2.40 kg), a 44% higher perception of pain, a 6.9% increase in punctuate tenderness of the vastus lateralis, and a 6% increase in knee-flexion passive range of motion as compared to the euhydrated group. The researchers also concluded that DOMS was caused by downhill running and dehydration increased the symptoms associated with DOMS. The combination of dehydration and a hyperthermic condition intensified the degree of microdamage to the skeletal muscles. Increasing DOMS reduced the ability to generate force, therefore the researchers recommended rehydration to reduce the influence of DOMS.

Effect of ECP on Recovery After Exercise

Only one study has previously assessed the effect of ECP on exercise recovery. Lezhen[5] examined the effect of ECP on reducing lactic acid and thus fatigue, probably the most comparable study to the one being proposed. Fourteen men, aged 15 to 19 years, were subjects for this study. All subjects were athletes involved in track and field and swimming. After experiencing an unreported "sublimited load" 9 of the subjects received ECP treatment, while 5 served as controls. Blood lactic acid was measured at rest and five minutes after performing the "sublimited load." ECP treatment was administered for 50 minutes. Lactic acid was assessed at 15 and 30 minutes during treatment and 10 minutes after the treatment was completed.

There was a significant decrease in blood lactic acid after ECP treatment as compared to the control group. Lezhen[5] concluded that ECP increases the rate at which lactic acid is removed from the blood. The results suggest that ECP increases cardiac output, skeletal muscle perfusion, and improves skeletal muscle metabolic function. Based on this, it was concluded that ECP treatment may aid in decreasing fatigue.

Male and Female Responses to Exercise Induced Muscle Damage

The purpose of the study by Rinard et al.[24] was to determine if males and females responded differently to high-force eccentric exercise. Eighty-three women aged 24.8 $\pm$ 4.9 years and 82 males aged 24.7 $\pm$ 5.1 years participated in this study. They performed 70 maximal repetitions of the elbow flexors using a modified preacher bench machine which had two cushions keeping the arm from moving. The investigators had control of a mechanical lever which the subjects were to resist.

The objective was to produce maximal resistance of the elbow flexor. Isometric strength, resting elbow angle and muscle soreness were assessed prior, immediately post, and everyday post-exercise for one week, with muscle soreness being of most importance to the present study.

Two procedures were used to measure muscle soreness, lifting and palpation. The subjects lifted a 1 lb weight three times through the elbow's full-range of motion and then reported perceived soreness using a 100 mm visual analog scale, with 0 mm = no soreness and 100 mm = severe soreness. The same 100 mm scale was used for assessing soreness after palpating the exercised elbow flexor. The subjects' loss in strength and arm angle immediately post-exercise was similar in males and females, therefore experiencing similar exercise stress.

Both males and females experienced significant levels of muscle soreness, peaking at 32-48 hours post-exercise, with no significant difference between the genders. Males' and females' relative strength post-exercise and recovery time was the same. There was not a significant interaction between gender and time for any of the variables tested, even though the women experienced a 27% reduction in strength as opposed to

24% experienced by men. Based on this study's results, women do not have a lower response to eccentric exercise than men.

Thompson et al.[25] conducted a study among thirty-four 18 to 36 year aged women in order to determine the relationship between serum creatine kinase activity and ovulatory status in exercising and sedentary women. They found that exercise increased women's CK levels regardless of their menstrual and exercise status. Normally sedentary women's CK levels increased 33 $\pm$ 3.4 IU/l whereas the active women's CK rose from 47.7 $\pm$ 3.1 IU/l. The active women participated in at least 2 hours of activity a week for at least 12 months.

The women were divided into three groups, ovulating-sedentary, ovulating-active, and amenorrheic-active women. The amenorrheic-active women had significantly greater CK levels (54.4 $\pm$ 3.6 IU/l) than the ovulating-sedentary and ovulatory-active groups (33.0 $\pm$ 3.4 and 43.7 $\pm$ 4.1 IU/l, respectively). The ovulating-active group consisted of women with various hormone status, 7 with normal hormone environments and 8 who were luteal phase deficient, therefore they had reduced luteal phase progesterone levels. When the hormone status was evaluated in the ovulating-active women, those who were luteal phase deficient had significantly greater CK levels. Therefore, exercise was the primary influence on CK levels in women with normal hormone status as opposed to those who were progesterone deficient (51.9 $\pm$ 5.4 and 36.6 $\pm$ 5.2 IU/l, respectively).

Kostopoulos et al.[26] examined muscle soreness, damage, and physical performance after 10 minutes of intense basketball-simulated exercise. Forty-eight men served as subjects for this study, half with chronic compartment syndrome (CACS) and

the other half without CACS. The ones without CACS were aged 19.55 ± 1.04 years. (From this point on the subjects referred to are those without CACS).

The 10 minute stimulation was representative of a real basketball game's intensity. The drill included dribbling, shooting, jumping, and forward and backward running. This stimulation was performed by each subject 5 times in one week. The actual experimental procedure consisted of the subjects warming up with a light jog and stretching routine. The subjects caught a basketball from a chest pass thrown to them from a distance of 5 m from the three-point line. From that location the subjects ran and dribbled to the basket, stopped with both feet on the floor, parallel distance from the free-throw line, then made a jump shot. The subjects ran back to their starting position and repeated the drill. They continued with this drill for a duration of 10 minutes.

Muscle soreness was assessed using a modified ordinal scale which ranged from 1 = no soreness to 10 = very, very sore. CK and LDH blood markers were two means of measuring muscle damage. CK activity was significantly increased immediately post-exercise, remained elevated until 48 hours post exercise, and returned back to baseline 72 and 96 hours post-exercise. LDH activity was also significantly elevated post-exercise, reached a plateau day 2 post-exercise, and back to baseline by day 3 post-exercise. Significant increases in muscle soreness were experienced immediately post-exercise and returned back to baseline levels by day 3 post-exercise.

Therefore, Kostopoulos et al.[26] concluded that intense basketball causes skeletal muscle damage, a significant rise in CK and LDH activity, and muscle soreness.

Milias et al.[27] determined the role inflammation played in DOMS. They examined blood markers pre- and post-eccentric exercise of the elbow flexor of the

non-dominant arm using a motorized muscle dynamometer. Thirteen male subjects, aged 27.5 ± 3.78 years, participated in this study. Each subject performed 6 sets of 6 repetitions producing maximum force of the elbow flexor, starting at an elbow angle of 110 degrees and ending at 20 degrees. Ten seconds of rest was taken between each repetition and 1 minute of rest was taken between each set.

Venous blood samples were drawn pre-exercise and 2, 24, 48, 72, and 96 hours post-exercise to examine the levels of CK, LDH, and CRP. Degree of muscle soreness was assessed using a visual analog scale (VAS) and a present pain index (PPI), from 0 cm = no pain to 10 cm = very painful and 0 = no pain to 5 = excruciating, respectively. Soreness was assessed after palpation of the bicep.

CK and LDH levels were significantly different post-exercise, significantly increasing at 24 hours post-exercise and maxing at 96 hours post-exercise. No significant differences in CRP were seen throughout the duration of the study. No significant difference was seen between the two assessments of muscle soreness, though soreness was significant as compared to pre-exercise levels. Muscle soreness progressively increased until 48 hours post-exercise then gradually decreased. Milias et al.[27] suggests that there was no significant change in CRP because there was no endurance component to the exercise protocol and that significant levels of CK and LDH activity were a result of muscle damage due to maximal knee flexions. The changes in muscle soreness revealed progressive recovery 48 hours post-exercise.

Paschalis et al.[28] examined the effects eccentric exercise induced muscle damage had on gait biomechanics. To examine the effect on walking and running kinematics the

researchers induced muscle damage of the knee extensors of 10 male college students aged 20 ± 1 year performing 6 sets of 10 maximal knee flexions.

Muscle damage and DOMS was assessed pre- and 48 hours post-eccentric exercise (the researchers justified this time of testing based on previous researchers' findings that muscle damage appears 12-24 hours post-exercise and peaks at 24-72 hours post-exercise). CK and LDH blood markers were significantly elevated 48 hours post-exercise. Normal reference range was 45-130 IU and 120-240 IU, respectively. At 48 hours post-exercise CK and LDH values were 1370 ± 1527 IU and 268 ± 145 IU, respectively. DOMS was assessed after palpation of the muscle belly based on a perceived soreness scale ranging from 1 (normal) to 10 (very, very sore).

Jamurtas et al.[29] examined the differences between leg and arm exercises of the same relative intensity on indices of muscle damage. Three indices of particular interest were DOMS, CK, and LDH activity. Eleven males aged 21.2 ± 1.0 years participated in this study, each performing 2 sets of sub-maximal arm and leg exercises for 5 consecutive days, separated by at least 2 weeks, with 6 subjects' first set being the leg exercise and the other 5 subjects being the arm exercises. Exercises were performed on an isokinetic dynamometer. Six sets of 12 eccentric actions were performed, with 2 minutes of rest between sets. The intensity of exercise was determined to be 75.2 ± 4.1% and 76.6 ± 3.5% of eccentric peak torque of the legs and arms, respectively.

DOMS was determined by self-palpation of the leg muscle and the elbow flexor based on a perceived pain scale ranging from 1 = normal to 10 = very, very sore. Blood was drawn from the antecubital vein to assess CK and LDH activity. All determinants of muscle damage were assessed pre-exercise and 24, 48, 72, and 96 hours post-exercise.

The normal reference range for CK and LDH in this study was 45-130 IU L^{-1} and 120-240 IU L^{-1}, respectively.

DOMS from the leg exercise peaked at 48 hours post-exercise and still remained elevated by 96 hours post-exercise. DOMS of the arms was significantly greater than the legs at 72 and 96 hours post-exercise. Leg CK activity significantly increased 24 hours post-exercise, peaking at 96 hours post-exercise. Peak CK activity ranged from 79-1,787 IU L^{-1}. LDH, on the other hand, did not significantly increase. The peak CK in the arms was significantly greater, 135 - 14,995 IU L^{-1}, but LDH significantly increased 24 - 96 hours post-exercise for the arms. The arms had significantly greater increases in enzyme activities than the legs.

Muscle damage was significantly greater in the arms than the legs. Recovery time was significantly greater for the arm elbow flexors than the knee extensors.

CHAPTER III

METHODS

An experimental research design was used. The independent variable was ECP treatment vs. no ECP treatment. The dependent variables were measures of DOMS (i.e. pain, serum CK, LDH, and C-reactive protein levels). The ECP method used in this study was validated by ScottCare Incorporation (Cleveland, OH).

Subjects

Ten trained runners, 5 males and 5 females, aged 19 to 52 years from Cleveland State University and the Cleveland area served as subjects. Subjects reported to Cleveland State University's (CSU) Human Performance Laboratory for a pre-participation screening questionnaire to determine eligibility (Appendix A). Subjects were excluded if they had a history of musculoskeletal problems or any predisposed health problem that placed them at risk for injury. All subjects were trained and capable of completing a twenty mile run at 70% of their age-predicted maximal heart rate. After

determining eligibility, each subject signed an informed consent form approved by the Institutional Review Board at CSU (Appendix B). Subjects were asked to refrain from training forty-eight hours prior to their initial test.

Experimental Procedures

Each subject completed a pre-measured twenty mile course on relatively flat terrain, with a total elevation change of 579 ft (176 m) and a total climb of 256 ft (78 m). Venous blood samples were obtained from the antecubital vein prior to the run to establish baseline levels for creatine kinase (CK) and lactate dehydrogenase (LDH). CK and LDH analysis was performed by Saint Vincent Charity Hospital's Biochemistry Laboratory (Cleveland, OH). C-reactive protein (CRP) was measured to determine inflammation; a 50 µL sample was obtained with a finger stick. The sample was analyzed on the Cholestech LDX analyzer in the CSU Human Performance Laboratory. Weight was obtained pre- and post-run; heart rate and blood pressure were obtained before and after each ECP treatment; heart rate, pulse pressure, and oxygen saturation were obtained continuously during ECP treatment. Blood pressure was taken with a stethoscope and a sphygnomonmeter, resting heart rate was measured using ECG, pulse pressure and arterial oxygen saturation was measured via finger pulse oximeter, height was measured using a stadiometer, and weight was measured using a physician's beam scale.

Subjects then completed a twenty mile run at an intensity of at least 70% of their age-predicted maximal heart rate (220-age). Each subject wore a Polar™ heart rate monitor to determine running intensity. Subjects were provided Gatorade® Original Lemon-Lime sports beverage approximately every three miles to prevent dehydration.

Fluid-intake was monitored and recorded. At the conclusion of the twenty mile run, blood samples were obtained and the subject's vital signs were measured again. The subjects were randomly assigned to the treatment or control trial to avoid order effect; therefore half of the runners received the ECP treatment after the first run and half received the ECP treatment after the second run. Time between trials was approximately three to four weeks, based on subject and researcher availability. Regardless of trial, post-run weight was measured. During the control trial, subjects reported daily for blood samples without treatment until all blood levels had returned to baseline levels. During the treatment trial, subjects were treated with ECP for 5 consecutive days, at the same time of day, until all blood markers returned to baseline levels. For both conditions, muscle pain was assessed using the Management of Cancer Pain Scale[30,31] (Appendix C).

ECP Treatment

Prior to ECP treatment, each subject was sized for pressure cuffs to assure proper cuff inflation and treatment pants (i.e. tights) were worn to prevent irritation from the pressure cuffs.

The ECP treatment was performed in the Human Performance Laboratory at CSU. ECP treatment required subjects to lie supine on a table and have pneumatic compression cuffs placed around the calves, thighs, and buttocks. Electrodes were placed on the subject to monitor the electrocardiogram tracing (ECG) (Appendix D). Guidelines developed by ScottCare were followed for all ECP treatments.

When treatment began, both arms remained relaxed at the side of the body (Appendix E). The head of the bed was at no more than a 30 degree angle; preferably flat, to prevent occlusion of blood to the femoral arteries. The 60 minute ECP treatment

procedure allowed for a 5 minute warm-up at a low pressure (between 1 and 3 psi) in order to gradually increase the diastolic pressure, allowing the subject to become acclimated to the pressure. The cuffs inflated sequentially at 50 ms intervals, distal to proximal and deflated simultaneously (Figure 1).[31]

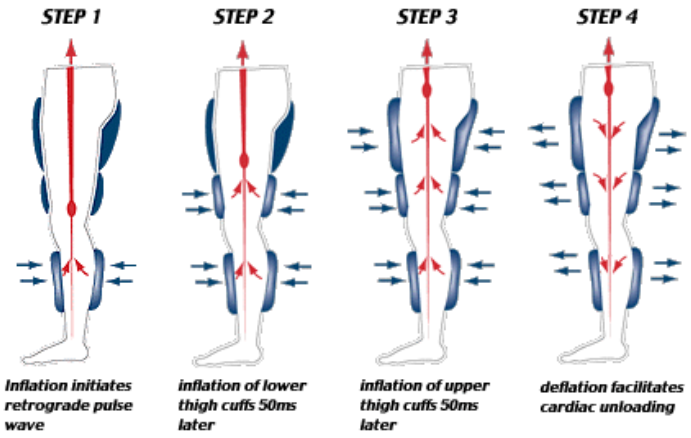


Figure 1. Sequential inflation and deflation of pneumatic cuffs during ECP treatment

After 5 minutes, psi was increased to a therapeutic peak-to-peak ratio (P/P ratio) of 1.2 or greater. The maximum pressure allowed was 6 psi, a safety regulation established by the manufacturers. The pressure intensity needed to be maintained at a comfortable and therapeutic level. Treatment ceased with any complaints of discomfort from the subject. Potential adverse side effects from ECP are skin irritation due the pneumatic compression cuffs, muscle aches, or headaches, though minimal and short-lived.[27]

The timing of the cuffs' inflation or deflation depended on whether the heart was in diastole or systole, working in conjunction with the ECG. The timing was important to assure proper timing of the treatment during the cardiac cycle. The cuffs were used to

apply varying amounts of pressure to the arteries and veins. Inflation causes an increase in venous return to the heart which results in an increased end diastolic volume, therefore stroke volume and cardiac output increase. The inflation of the cuffs causes a change in blood volume and pressure in the aorta which in turn increases the volume and pressure in the coronary arteries. This increase in diastolic pressure increases perfusion to the heart. The increase in arterial pressure causes diastolic augmentation. When the compression cuffs deflate, it results in blood flowing back through the buttocks, thighs, and calves to return the pressure in the aortic cavity back to normal. Timing this correctly was important to make sure that deflation occurred in the early phase of systole so that it decreased the force with which the heart had to work, therefore decreasing myocardial oxygen requirement.[1]

The researchers were present through the duration of the treatment to ensure the subject's comfort and safety, and to monitor data collection. Every 10 minutes, a manual P/P ratio was performed by the researcher to assure the proper timing of the inflation and deflation of the cuffs (Appendix F). Upon completion of each treatment, blood pressure and heart rate were recorded.

Regardless of experimental condition, subjects were asked to record all activities 5 days post-run and replicate those same activities for each of the 2 trials as a means of controlling extraneous variables.

Control Group Treatment

To determine if muscle damage and inflammation resulted from ECP treatment alone, void any form of exercise, five additional subjects received ECP treatment for 5 consecutive days. Subjects were asked to abstain from exercise prior to and throughout the duration of the treatment.

Maximal Oxygen Consumption (VO_2 max)

The ten trained runners performed a VO_2 max treadmill test subsequent to complete recovery from both 20 mile runs. Indirect calorimetry was used to measure VO_2 max in the Human Performance Laboratory at CSU using a metabolic cart (Physiodyne™) under the direct supervision of a trained professional. Measuring the subjects' aerobic capacity and maximal heart rate made it feasible to determine what percentage of maximal heart rate and maximal oxygen consumption was achieved during the runs. These values were calculated using the speed and time of each subject's run using the following equation: VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$) = speed (m/min) x .2 + 3.5 ml.[7]

Statistical Analysis

Descriptive statistics were obtained on all measures. A repeated measures ANOVA was used to assess treatment differences due to the independent variable, ECP treatment vs. no ECP treatment, on the dependent variables, pain, CK, LDH, and C-reactive protein blood markers. Protected t-tests were used to determine significant differences across serial markers. Correlations were run among the dependent variables to determine if any significant relationships existed. SPSS-PC (version 14.0) was used for all analyses with 0.05 used as the level of significance.

CHAPTER IV

RESULTS & DISCUSSION

Control Group (ECP only)

In order to validate that running was responsible for the changes in DOMS and inflammation, as opposed to the ECP treatment itself, a control group, which included 5 additional subjects, underwent 5 days of ECP treatment.

Table 1. Control group subjects' characteristics.

Control Group (ECP only)	Males (N=3)	Females (N=2)	Total Group (N=5)
Height (cm)	187.1 ± 14.0	168.3 ± 8.1	179.6 ± 13.3
Weight (kg)	92.4 ± 19.9	65.0 ± 3.2	81.5 ± 20.6
Age (yrs)	26.3 ± 0.6	24.5 ± 3.5	25.6 ± 2.1

Values are mean ± standard deviation

The control group abstained from exercise prior to and throughout the duration of the treatment to avoid experiencing any acute muscle damage. No significant difference was observed among any of the biochemical blood markers from pre- to post-treatment days (Table 2, Table 3, Table 4).

C-reactive protein (CRP)

The control group's CRP remained within the normal range throughout the 5 day treatment period, with no significant difference in inflammation from pre- to Day 5 post-treatment (Table 2). The normal range for CRP is between 1.00 and 3.00 mg/L.

Table 2. CRP results with ECP treatment in the control group.

Condition (N=5)	Mean	Std. Deviation	p-value
Pre-treatment	1.70	.91	-----
Day 1	1.96	1.05	.612
Day 2	1.83	1.40	.852
Day 3	1.61	1.24	.901
Day 4	1.48	1.05	.726
Day 5	1.42	1.17	.671

Normal = 1.00-3.00 mg/L

Lactate Dehydrogenase (LDH)

The control group's LDH remained within normal range throughout the 5 day treatment period, with no significant difference from pre- to Day 5 post-treatment (Table 3).

Table 3. LDH results with ECP treatment in the control group.

Condition (N=5)	Mean	Std. Deviation	p-value
Pre-treatment	164.8	29.0	-----
Day 1	164.2	27.5	.946
Day 2	165.6	33.9	.960
Day 3	168.0	32.7	.670
Day 4	149.4	26.0	.213
Day 5	154.6	28.1	.280

Normal =75-190 (U/L)

Creatine Kinase (CK)

The control group's CK increased beyond the normal range peaking at Day 2 of treatment (Table 4). However, these differences were not significant.

Table 4. CK results with ECP treatment in the control group.

Condition (N=5)	Mean	Std. Deviation	p-value
Pre-treatment	193.0	192.7	-----
Day 1	264.4	154.3	.343
Day 2	316.0	264.0	.374
Day 3	256.4	196.2	.411
Day 4	223.6	97.5	.735
Day 5	188.2	73.8	.938

Normal = 21-232 (U/L)

Experimental Protocol

The experimental group consisted of the ten subjects who ran 20 miles under control conditions and ECP treatment conditions. Subject characteristics for the experimental group (N = 10) are presented in Table 5.

Table 5. Experimental groups subjects' characteristics.

Experimental Group (Control Run vs. ECP Treatment Run)	Males (N=5)	Females (N=5)	Total Group (N=10)
Height (cm)	186.3 ± 12.8	164.1 ± 4.6	179.1 ± 12.5
Weight (kg)	71.1 ± 8.3	58.8 ± 3.1	64.9 ± 8.7
Age (yrs)	34.2 ± 11.5	26.8 ± 14.1	30.5 ± 12.8
VO_2 max ($ml \cdot kg^{-1} \cdot min^{-1}$)	57.5 ± 8.2	48.3 ± 5.6	52.9 ± 8.2

Values are mean ± standard deviation

Males were found to be significantly taller and weigh significantly more than the females in the experimental group. No significant difference in age or VO_2max was seen between genders (Table 6).

Table 6. Gender comparison of height, weight, age, and VO_2 max in the experimental group.

Condition (N = 10)	Mean ± SD	p-value
Male—female height	13.208 ± 5.507	.006 *
Male—female weight	12.294 ± 5.464	.007 *
Male—female age	7.400 ± 15.307	.341
Male—female VO_2 max	9.212 ± 8.194	.066

**Significant difference ($p < .05$)*

There was no significant difference in mean weight loss, fluid intake, heart rate, percentage of maximum heart rate, percentage of VO_2 max, or running time during the run for the two trials ($p \geq .05$) (Table 7).

Table 7. Comparison between ECP treatment run and control run for the experimental group.

Condition (N=10)	Control	Treatment	Mean Difference	p-value
Weight loss (kg)	1.7 ± 1.0	1.1 ± 0.7	0.5 ± 0.8	.062
Fluid intake (ml)	1650.0 ± 807.7	1787.0 ± 650.2	136.9 ± 470.7	.382
HR (bpm)	162.8 ± 15.6	160.0 ± 14.2	2.8 ± 9.7	.383
% Max HR	87.4 ± 5.7	86.0 ± 7.1	1.3 ± 4.9	.404
%VO2 Max	72.8 ± 6.1	71.3 ± 9.2	1.4 ± 4.9	.380
Running time (min)	189.3 ± 30.6	194.3 ± 27.8	5.0 ± 17.7	.400

Values are mean ± standard deviation

Indicators of Muscle Damage and Inflammation

C-reactive protein, pain, CK, and LDH variables were analyzed using a repeated measures ANOVA. Multiple comparisons were determined using protected t-tests to control for Type I error inflation.

C-reactive protein (CRP)

According to the literature, a change in acute-phase proteins is indicated by the significant elevation in CRP[19], as supported by the results (Table 8). C-reactive protein elevation peaked Day 1 post-control and ECP treatment runs. Significantly elevated CRP was present from Day 1 to Day 3 post-control run, but only Day 1 of ECP treatment run.

Normal CRP range was obtained by Day 4 post-control and post-treatment runs. Therefore, treatment reduced CRP elevation by 40% (Table 8).

Table 8. CRP results for the control run and ECP treatment run.

Condition (N=10)	Mean	Std. Deviation	p-value
Pre - Control Run	1.18	0.92	----
Post - Control Run	1.13	0.77	.405
Day 1 Control Run	6.88	3.22	.000 *
Day 2 Control Run	4.20	2.26	.001 *
Day 3 Control Run	3.01	1.70	.005 *
Day 4 Control Run	1.73	0.84	.135
Day 5 Control Run	1.66	1.11	.292
Pre - ECP Treatment Run	1.86	2.18	----
Post - ECP Treatment Run	2.05	2.51	.167
Day 1 ECP Treatment Run	7.23	3.27	.001 *
Day 2 ECP Treatment Run	5.01	2.67	.011
Day 3 ECP Treatment Run	3.42	2.51	.167
Day 4 ECP Treatment Run	2.80	3.61	.505
Day 5 ECP Treatment Run	2.64	4.28	.626

Normal = 1.00-3.00 mg/L

**Significant difference from Pre- (protected t-test, $p < .01$)*

Simpson et al[20] showed no significant difference in CRP, even though muscle damage was present. It can be suggested that muscle damage does not equate to inflammation. CRP has been associated with the endurance component of exercise (i.e. eccentric cycling until exhaustion, downhill running)[27] , which would aid in explaining the significantly elevated CRP levels observed in the ten subjects following each run.

Pain

Perceived pain was recorded immediately post to Day 5 post-runs. The perceived pain scale ranged from 0 – 10, with 0 = no pain and 10 = unbearable pain. Subjects' pain peaked immediately post-runs (Table 9). Pain was still present Day 5 post-control run, but had diminished by Day 4 of the ECP treatment run. A 20% reduction in significant

pain elevation was experienced from ECP treatment (Table 9). Therefore hypothesis one was accepted; ECP treatment reduced the delayed onset of muscle soreness (DOMS).

Table 9. Pain results for the control run and ECP treatment run.

Condition (N=10)	Mean	Std. Deviation	p-value
Pre - Control Run	0.0	----	----
Post - Control Run	6.6	2.4	.000 *
Day 1 Control Run	4.9	2.4	.000 *
Day 2 Control Run	2.8	2.5	.006 *
Day 3 Control Run	1.4	1.4	.013
Day 4 Control Run	0.4	0.8	.168
Day 5 Control Run	0.3	0.7	.193
Pre - ECP Treatment Run	0.0	----	----
Post - ECP Treatment Run	5.7	2.2	.000 *
Day 1 ECP Treatment Run	3.3	2.7	.004 *
Day 2 ECP Treatment Run	1.2	1.7	.051
Day 3 ECP Treatment Run	0.3	0.5	.177
Day 4 ECP Treatment Run	0.0	----	----
Day 5 ECP Treatment Run	0.0	----	----

Scale 0 (no pain) - 10 (unbearable pain)
**Significant difference from Pre- (protected t-test, $p < .01$)*

It was foreseen that subjects would likely gauge pain of the second run based on pain experienced during the first run. It was important to account for these potential discrepancies by having 5 subjects experience treatment the first run and 5 subjects experience the non-treatment condition the first run, therefore accounting for order effect. Nosaka et al.[19] found variations in levels of muscle soreness even when similarities in markers of muscle damage were present and suggested that other factors can influence pain perception such as past experiences, pain threshold, mood, health and hormonal status. Coincidentally, subjects in the present study verbalized their thought process

about pain perceived, stating how the pain from the second run differed or compared to the first run.

Although there were no objective means of measuring exercise tolerance, the perceived pain (via pain scale) decreased faster while undergoing ECP, as compared to the control condition. This suggests that the intensity and duration of exercise post-treatment could return to that achieved prior to the run quicker, hence increasing exercise tolerance. Other studies have shown that an increase in myocardial perfusion increased exercise tolerance for CAD patients.[3,8,9] The same may be true for trained runners.

Schwane et al.[10] found no significant measurement of muscle soreness after a 45 minute run on level ground. Even though the ground in the current study was fairly level, there was a slight variation in the elevation (579 feet), whereas on the treadmill in the study by Schwane et al.[10], a 0% grade remained constant. Therefore, some of the muscle soreness experienced by the runners may have been due to the slight variation in elevation in addition to the greater volume of exercise.

The subjects in the current study reported the highest degree of pain immediately post-run, which agrees with MacIntyre et al.[21] who reported peak muscle soreness immediately post-marathon until 48 hours after eccentric exercise of the quadriceps muscle.

Lactate Dehydrogenase (LDH)

Lactate dehydrogenase elevation peaked post-runs and returned to normal range (75-190 U/L) by Day 1 for the experimental group (Table 10). Lactate dehydrogenase was significantly elevated immediately post to Day 2 post-control run. Significantly

elevated LDH levels decreased by 20% due to the ECP treatment, subsiding by Day 1 post-run (Table 10).

Table 10. LDH results for the control run and ECP treatment run.

Condition (N=10)	Mean	Std. Deviation	p-value
Pre - Control Run	151.4	12.4	----
Post - Control Run	244.1	36.0	.000 *
Day 1 Control Run	177.3	18.9	.003 *
Day 2 Control Run	178.5	21.7	.003 *
Day 3 Control Run	175.6	36.4	.054
Day 4 Control Run	164.4	23.8	.076
Day 5 Control Run	161.2	22.9	.146
Pre - ECP Treatment Run	157.8	20.5	----
Post - ECP Treatment Run	234.1	37.7	.000 *
Day 1 ECP Treatment Run	186.4	17.8	.000 *
Day 2 ECP Treatment Run	180.6	19.2	.012
Day 3 ECP Treatment Run	177.1	23.7	.011
Day 4 ECP Treatment Run	169.1	22.1	.069
Day 5 ECP Treatment Run	169.3	22.5	.061

Normal =75-190 U/L

**Significant difference from Pre- (protected t-test, $p < .01$)*

In the study by Schwane et al.[10] no significant change in LDH was found post-race. It could be speculated that the longer duration of the present study (mean duration ~192 minutes), as opposed to the 45 minutes of running experienced by subjects in the study by Schwane et al.[10], could have influenced the significant rise in LDH. Schwane et al.[10] suggested that DOMS was a result of the changes in muscle structure caused by the eccentric contractions performed throughout the run. Muscle damage may be experienced if there is an increase in the levels of metabolites in the skeletal muscles or a mechanical disturbance of the muscle cell which can occur from running, especially of long duration.[12] Pachalis et al.[14] suggested that muscle damage is due more to volume as opposed to intensity of exercise.

Creatine Kinase (CK)

Creatine kinase elevation peaked Day 1 post-runs (Table 11). Significantly elevated CK levels were present immediately post and Day 1 post-control run, whereas significantly elevated CK levels post-ECP treatment run were present immediately post and Day 4 post-run. Normal CK range (21-232 U/L) was obtained by Day 3 post-control run and Day 4 post-ECP treatment run (Table 11).

Table 11. CK results for the control run and ECP treatment run.

Condition (N=10)	Mean	Std. Deviation	p-value
Pre - Control Run	150.2	104.4	----
Post - Control Run	322.0	140.7	.000 *
Day 1 Control Run	574.8	326.5	.004 *
Day 2 Control Run	330.0	158.0	.011
Day 3 Control Run	216.5	120.9	.142
Day 4 Control Run	201.9	164.9	.405
Day 5 Control Run	174.5	109.1	.596
Pre - ECP Treatment Run	126.1	85.1	----
Post - ECP Treatment Run	269.9	131.4	.000 *
Day 1 ECP Treatment Run	602.90	373.5	.003 *
Day 2 ECP Treatment Run	363.3	163.2	.001 *
Day 3 ECP Treatment Run	287.30	100.6	.002 *
Day 4 ECP Treatment Run	224.3	84.6	.004 *
Day 5 ECP Treatment Run	192.1	61.3	.043

Normal = 21-132 (U/L)
**Significant difference from Pre- (protected t-test, $p < .01$)*

This study agrees with previous research by Schwane et al.[10] that found a significant increase in CK after running for 45 minutes at a 10% grade downhill. The increased level of CK was attributed to changes in muscle structure caused by the eccentric contractions performed throughout the run. It also agrees with Jamurtas et al.,[29] who studied the differences between eccentric exercises of the elbow flexors and legs at the same relative intensity, found CK activity to significantly increase 24 hours post-exercise, the same time period that CK elevation peaked in the present study.

Hypothesis two of this study stated that ECP treatment would reduce the biochemical markers of muscle damage (CRP, LDH, and CK). The hypothesis is accepted for CRP and LDH, but rejected for CK, suggesting that other factors could be responsible for these differences. Some of these differences could be due to variations in environmental conditions. Research has shown that running in colder temperatures results in higher levels of CK, therefore suggesting an increase in muscle damage.[11] It is presumed that running in a colder environment decreases an individual's ability to perform work, causing the recruitment of more muscle fibers to complete the run.[11] In this study the two runs were scheduled periodically beginning in March and ending in late August of 2007. Even though each of the runners' two runs was scheduled within 3 to 4 weeks of each other to minimize the variations in temperature, the two runs were not performed under identical conditions.

Comparisons between DOMS, CK, LDH, and CRP

Numerous studies have examined the effects of eccentric exercise using the elbow flexors on DOMS, CK, LDH, and CRP. Though the indices of muscle damage are the same, variations have been shown to exist between eccentric exercise performed on the arms and legs (as mentioned previously). Arm exercises have been shown to elicit greater DOMS, CK, and LDH activity than the legs. Overall, muscle damage is significantly greater in the arms, resulting in greater recovery time. It is suggested that changes and trends in CK, LDH, and DOMS are situational, based on the exercise protocol administered. It is noteworthy to recognize that studies reporting indices of muscle damage of the elbow flexors may not accurately represent the trends in blood markers and DOMS following eccentric exercise of the lower extremities.

Literature reviewed throughout this study often mentioned peak levels of CK, LDH, CRP, and DOMS that differed from the present study, though they did not account for all the days post-exercise to recovery, therefore not providing the most ideal means of comparison.[15,20,21,27,28,29] The greatest elevation among the dependent variables in the present study was observed immediately post or Day 1 post-exercise. It is recommended that future research studies observing the effects of eccentric exercise on CK, LDH, CRP, and DOMS should be measured from the time the exercise is performed until recovery.

Relationships between Indicators of Muscle Damage and Inflammation

Correlations were obtained to determine if relationships existed between DOMS and inflammation, DOMS and markers of muscle damage, inflammation and markers of muscle damage, and between markers of muscle damage (Table 12). All significant relationships were found post-control run, but none post-ECP treatment run. The results do not exactly coincide with the literature. Though as mentioned previously, various factors can play a role in the elevation of particular enzymes and pain tolerance.

Table 12. Relationships between control run variables.

Variables	r	r^2	p-value
CRP--CK Day 3	.699	.489	.025*
CRP--CK Day 4	.647	.419	.043*
CRP--CK Day 5	.803	.645	.005*
pain--LDH post-run	.767	.588	.010*
CK--LDH Day 5	.693	.480	.026*
CRP—pain Day 2	.855	.731	.002*

**Significant difference ($p<.05$)*

Relationships between CRP and CK

No literature reviewed looked at the relationship between CRP and CK though a strong relationship was observed Day 3 and Day 4 (r = .699 and r = .647, respectively) and a very strong relationship Day 5 of the control run (r = .803). C-reactive protein and CK were both found to peak Day 1 post-run. The present researchers claim they are the first to report this relationship.

Relationships between DOMS and LDH

Nosaka et al.[19] found minimal to no relationship between DOMS and markers of muscle damage after performing eccentric exercises of the elbow flexors, whereas the present study found a significant relationship between LDH and pain post-control run (r = .767). Both variables were also found to peak immediately post-control run. Jamurtas et al.[29] reasoned that influences of eccentric contractions of the elbow and knee flexors are not synonymous. This could account for this discrepancy.

Relationships between DOMS and CRP

Schwane et al.[10] found no significant relationship between DOMS and inflammation after running at different grades. However, a strong correlation was observed between pain and CRP Day 2 of the control run (r = .855) in this study. This finding agrees with MacIntyre et al.[21] who claims to have been the first to report a relationship between DOMS and inflammation. This finding also agrees with Chelboun et al.[22] who has suggested that intra and extracellular edema were a result of tissue damage and inflammation, respectively. It could be argued that the delay in peak CRP, as compared to pain, was due to muscle fluid transitioning from the exercised muscle to the tissue.[22]

Relationship between CK and LDH

There is little research reported for the correlation between CK and LDH. The present study and a study by Clarkson et al.[15] shows a strong correlation between CK and LDH ($r = .693$ and $r = .95$, respectively). More research is needed to clarify this relationship.

Gender Differences in Biochemical Markers of Muscle Damage and Inflammation

To account for gender differences in biochemical markers of muscle damage and inflammation, the analyses were run separately for each gender.

C-reactive protein (CRP)

Both males and females reached peak CRP levels on Day 1 post-runs (Table 13). Males experienced elevated, though not significant, levels of CRP until Day 3 post-control and ECP treatment runs. Therefore there were no significant differences among males' CRP levels post-control and ECP treatment runs. Females, on the other hand, experienced a 20% increase in CRP from the control to the ECP treatment runs, remaining significantly elevated Day 1 and Day 2 post-treatment. Normal CRP range was obtained by Day 4 post-control run, but was still above normal range by Day 5 post-ECP treatment run. The additional CRP elevation observed in the females may be attributed to the run itself.

Table 13. Gender results for CRP.

	Males (N=5)		Females (N=5)	
Condition	**Mean ± SD**	**p-value**	**Mean ± SD**	**p-value**
Pre - Control Run	1.34 ± 1.26	----	1.02 ± 0.51	----
Post - Control Run	1.30 ± 1.00	.765	0.95 ± 0.50	.162
Day 1 Control Run	6.26 ± 4.18	.043	7.49 ± 2.22	.002 *
Day 2 Control Run	3.87 ± 2.70	.066	4.53 ± 1.98	.012
Day 3 Control Run	2.80 ± 1.54	.056	3.21 ± 2.01	.063
Day 4 Control Run	1.63 ± 0.86	.667	1.84 ± 0.89	.060
Day 5 Control Run	1.34 ± 0.66	1.000	1.97 ± 1.44	.191
Pre - ECP Treatment Run	2.65 ± 2.92	----	1.07 ± 0.76	----
Post - ECP Treatment Run	2.90 ± 3.41	.364	1.19 ± 0.85	.091
Day 1 ECP Treatment Run	6.47 ± 4.52	.157	7.99 ± 1.49	.000 *
Day 2 ECP Treatment Run	3.89 ± 2.31	.379	6.12 ± 2.74	.006 *
Day 3 ECP Treatment Run	2.27 ± 1.31	.776	4.57 ± 3.02	.045
Day 4 ECP Treatment Run	1.55 ± 1.01	.440	4.04 ± 4.94	.234
Day 5 ECP Treatment Run	1.29 ± 0.61	.348	3.98 ± 6.02	.328

Normal = 1.00-3.00 mg/L
**Significant difference from Pre- (protected t-test, p < .01)*

Pain

Pain was experienced post-control run for the females and post-ECP treatment run for the males and females, peaking immediately post-runs (Table 14). Both males and females experienced a 20% reduction in pain due to treatment, though females experienced pain one day longer post-control and ECP treatment runs, as compared to the males. It was clearly evident that males had a higher pressure tolerance exuded by the compression cuffs than the females. The females often requested the pressure to be decreased whereas the males, more times than not, requested the pressure to be as high as they could tolerate.

Table 14. Gender results for pain.

	Males (N=5)		Females (N=5)	
Condition	**Mean ± SD**	**p-value**	**Mean ± SD**	**p-value**
Pre - Control Run	0.0	----	0.0	----
Post - Control Run	5.7 ± 3.0	.013	7.5 ± 1.6	.000 *
Day 1 Control Run	4.5 ± 2.5	.016	5.2 ± 2.6	.011
Day 2 Control Run	2.4 ± 1.0	.080	3.2 ± 2.9	.067
Day 3 Control Run	1.0 ± 1.0	.089	1.8 ± 1.8	.088
Day 4 Control Run	0.0	----	.8 ± 1.1	.178
Day 5 Control Run	0.0	----	.6 ± .9	.208
Pre - ECP Treatment Run	0.0	----	0.0	----
Post - ECP Treatment Run	4.3 ± 2.1	.010 *	7.0 ± 1.4	.000*
Day 1 ECP Treatment Run	1.6 ± 1.3	.056	5.0 ± 2.6	.013
Day 2 ECP Treatment Run	0.2 ± 0.4	.374	2.2 ± 1.9	.063
Day 3 ECP Treatment Run	0.0	----	0.5 ± 0.7	.189
Day 4 ECP Treatment Run	0.0	----	0.0	----
Day 5 ECP Treatment Run	0.0	----	0.0	----

Scale 0 (no pain) –10 (unbearable pain)
**Significant difference from Pre- (protected t-test, p < .01)*

Lactate Dehydrogenase (LDH)

Both males and females reached peak LDH levels post-runs (Table 15). Males experienced significantly elevated LDH levels post-treatment run and females post- treatment run and Day 1. During the control run only females experienced a significant change post-run. They experienced a 20% increase in LDH elevation post-ECP treatment run which is likely attributed to the run itself. Males experienced a significant change on Day 4 of the control run. It is unclear as to what attributed to this occurrence. It may be due to subject variability response.

Table 15. Gender results for LDH.

	Males (N=5)		Females (N=5)	
Condition	**Mean ± SD**	**p-value**	**Mean ± SD**	**p-value**
Pre - Control Run	151.2 ± 13.5	----	151.6 ± 12.8	----
Post - Control Run	233.8 ± 46.4	.019	254.4 ± 22.4	.000 *
Day 1 Control Run	176.2 ± 15.5	.084	178.4 ± 23.6	.029
Day 2 Control Run	181.0 ± 19.6	.057	176.0 ± 25.8	.049
Day 3 Control Run	191.4 ± 43.0	.100	159.8 ± 22.6	.324
Day 4 Control Run	173.0 ± 10.9	.010 *	155.8 ± 31.1	.730
Day 5 Control Run	163.6 ± 13.8	.190	158.8 ± 31.1	.522
Pre - ECP Treatment Run	132.2 ± 17.4	----	153.4 ± 24.3	----
Post - ECP Treatment Run	243.2 ± 38.3	.007 *	225.0 ± 40.9	.005 *
Day 1 ECP Treatment Run	192.0 ± 16.2	.025	180.8 ± 19.3	.004 *
Day 2 ECP Treatment Run	183.0 ± 26.2	.180	178.2 ± 11.3	.041
Day 3 ECP Treatment Run	182.8 ± 24.3	.095	171.4 ± 24.4	.106
Day 4 ECP Treatment Run	177.0 ± 25.8	.124	161.2 ± 16.8	.407
Day 5 ECP Treatment Run	178.0 ± 24.4	.106	160.6 ± 18.7	.414

Normal =75-190 U/L

**Significant difference from Pre- (protected t-test, p < .01)*

Creatine Kinase (CK)

Both males and females reached peak CK levels Day 1 post-runs (Table 16). Normal ranges for CK varies based on gender. Males' normal range is 35-232 U/L and females' normal range is 21-215 U/L. Only females experienced significantly elevated CK after both runs. Though, not statistically significant, the males' CK elevation took two additional days to return to normal range from the control run to the ECP treatment run, while the females' CK elevation took one day less to return to normal range from the control run to the ECP treatment run. The responses of both males and females to CK changes in this study agrees with Kyrolainen et al.[12] who reported 4-6 days for CK to return to baseline levels after running a marathon. This also agrees with Pachalis et al.[14] that exercise volume influences the extent of muscle damage.

Table 16. Gender results for CK.

	Males (N=5)		Females (N=5)	
Condition	**Mean ± SD**	**p-value**	**Mean ± SD**	**p-value**
Pre - Control Run	117.2 ± 79.8	----	183.2 ± 124.3	----
Post - Control Run	308.4 ± 130.3	.011	335.6 ± 164.6	.003 *
Day 1 Control Run	629.6 ± 437.8	.069	520.0 ± 201.7	.019
Day 2 Control Run	344.8 ± 180.6	.072	315.2 ± 151.6	.116
Day 3 Control Run	193.2 ± 50.3	.112	239.8 ± 170.2	.512
Day 4 Control Run	163.4 ± 38.5	.079	240.4 ± 236.7	.668
Day 5 Control Run	136.8 ± 57.3	.597	212.2 ± 141.2	.756
Pre - ECP Treatment Run	153.6 ± 113.9	----	98.6 ± 37.9	----
Post - ECP Treatment Run	316.4 ± 168.0	.014	223.4 ± 72.3	.003 *
Day 1 ECP Treatment Run	675.8 ± 493.6	.071	530.0 ± 238.7	.019
Day 2 ECP Treatment Run	388.2 ± 212.7	.048	338.4 ± 114.5	.021
Day 3 ECP Treatment Run	307.2 ± 89.3	.033	267.4 ± 117.5	.048
Day 4 ECP Treatment Run	258.2 ± 96.6	.049	190.4 ± 62.5	.080
Day 5 ECP Treatment Run	230.8 ± 57.5	.205	153.4 ± 37.4	.139

Normal = 21-215 U/L (females)
35-232 U/L (males)
**Significant difference from Pre- (protected t-test, p < .01)*

Rinard et al.[24] conducted research on males and females to determine whether responses to high-force eccentric exercises of the elbow flexors are influenced by gender. Males and females were found to experience significant levels of muscle soreness, peaking at 32 and 48 hours post-exercise, with no difference between genders. Thompson et al.[25] examined the relationship between CK levels and ovulatory status in exercising and sedentary women. They found that CK activity increased with exercise, in both ovulating groups, with the active women's CK response being greater. Therefore, degree of activity may have played a factor in levels of CK reached, as well as the5 hormonal level. Amenorrheic exercising women have been found to have greater CK elevation than sedentary and exercising normally ovulating women.[25] Therefore, exercise

was the primary influence on CK levels in women with normal hormonal status. No hormonal tests were administered in the current study though all female subjects reported no menstrual problems, irregularities, or use of birth control. It would be beneficial to examine the role gender plays on the effects of ECP treatment, taking into account the many extraneous variables that could influence differences in gender (i.e. hormonal status, nutritional status, activity level, etc.).

The current study did show differences between males and females responses after the control and treatment runs, therefore, hypothesis three is rejected.

CHAPTER V

SUMMARY & CONCLUSION

Ten trained runners ran two 20 mile runs on a pre-determined course in order to induce muscle soreness (experimental group—control run and ECP treatment run). Each subject participated in the control run and ECP treatment run. Trials were randomly counterbalanced to ensure no order effect.

Five additional subjects underwent 5 days of ECP treatment with no exercise intervention (control group--ECP alone). The purpose was to determine if ECP treatment alone caused muscle damage. Muscle damage was observed by measuring biochemical markers of CRP, CK, and LDH. No significant difference was noted for any of the markers in the control group. It was concluded that ECP treatment alone did not cause muscle damage. However, CK levels were increased; it can be argued that ECP treatment alone stimulates CK release while the run and other extraneous variables

contributed to the additional significant elevation in CK observed post-treatment as opposed to post-control condition.

The experimental group experienced a 20% reduction in DOMS, as measured via pain scale, therefore hypothesis one was accepted. ECP treatment reduced the delayed onset of muscle soreness (DOMS). The experimental group also experienced a 40% reduction in CRP and a 20% reduction in LDH due to treatment. Though treatment did not reduce CK elevation; CK returned to normal range one day sooner from the control run as opposed to the ECP treatment run. Therefore, hypothesis two, which stated that ECP treatment would reduce biochemical markers of muscle damage (CRP, LDH, and CK), was accepted for CRP and LDH, but rejected for CK.

Correlations were obtained to determine if relationships existed between DOMS and inflammation, DOMS and markers of muscle damage, inflammation and markers of muscle damage, and between markers of muscle damage. All significant relationships were found post-control run, but none post-ECP treatment run. Positive relationships were found between CRP and CK (Day 3, 4, and 5), DOMS and LDH (immediately post-run), DOMS and CRP (Day 2), and CK and LDH (Day 5).

To account for gender differences in biochemical markers of muscle damage and inflammation, analyses were run separately for each gender. Only females experienced a significant difference in CRP and CK. Females experienced a 20% increase in CRP from the control run to the ECP treatment run, which may be attributed to the run itself. Creatine kinase was significantly elevated immediately post-runs for the females, taking one less day to return to normal CK range from treatment. Males, though not significant, took two additional days to return to normal CK range from the control run to the ECP

treatment run. There was a 20% reduction in pain for both males and females due to treatment, though females experienced pain for one additional day. Females experienced a 20% increase in LDH, which may be attributed to the run itself. Males experienced a significant change Day 4 of the control run and immediately post-ECP treatment run, this difference between LDH significance may be attributed to the subject variability response. Differences were found between genders in response to the control run and ECP treatment run, therefore hypothesis three was rejected. Overall, it can be concluded that ECP treatment was effective in decreasing inflammation, DOMS, and LDH, but was not effective in decreasing CK levels.

Application

The results of this study could be applied to professional and non-professional athletes who spend a great deal of time training, especially those who perform long duration eccentric exercises. ECP treatment can aid in decreasing recovery time so that training can continue sooner.

Limitations

1. Running weather conditions varied; very cool or warm temperatures affect heart rate, running time, exercise performance, hydration status, and therefore DOMS. Subjects' runs were scheduled within 3 to 4 weeks of each other to try and control for those differences.
2. There was a subjective measurement of pain and a variation in pressure augmentation based on subjects' request.
3. A limited sample size was used in this study.

4. Extraneous variables: training history, muscle fiber constituents (i.e. fast to slow twitch fiber ratio), past experiences (i.e. injuries), hormonal status, all may influence enzyme activity.

Future Research Recommendations

1. Based on the results of this study, and previous studies supporting ECP's effect on enhancing blood perfusion to the heart, it is recommended that ECP treatment be studied on patients with peripheral vascular disease, such as diabetes. Diabetics suffer from reduced peripheral blood flow, therefore, ECP treatment may increase peripheral blood flow and perfusion in diabetic patients as well.

2. Future research is needed to determine if a non-exercising training effect exists with the use ECP treatment.

3. Future research is needed to determine if relationships exist between CK and LDH and CK and CRP, since little to no research has been documented.

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APPENDIX A

NAME _____ DATE _____

AHA/ACSM Preparticipation Screening Questionnaire (AHA/ACSM, 1998)[1]

ssess Your Health Needs by Marking All *true* Statements

History

You have had:

- ☐ A heart attack
- ☐ Heart surgery
- ☐ Cardiac catheterization
- ☐ Coronary angioplasty (PTCA)
- ☐ Pacemaker/implantable cardiac
- ☐ Defibrillator/rhythm disturbance
- ☐ Heart valve disease
- ☐ Heart failure
- ☐ Heart transplantation
- ☐ Congenital heart disease

Other health issues:

- ☐ You have musculoskeletal problems. Specify on back*
- ☐ You have concerns about the safety of exercise. Specify on back*
- ☐ You take prescription medication(s). Specify on back*
- ☐ You are pregnant.

Symptoms

- ☐ You experience chest discomfort with exertion.
- ☐ You experience unreasonable breathlessness.
- ☐ You experience dizziness, fainting, blackouts.
- ☐ You take heart medications.

Recommendations

If you marked any of the statements in this section, consult your healthcare provider before engaging in exercise. You may need to use a facility with a **medically qualified staff.**

Cardiovascular risk factors

- ☐ You are a man older than 45 years.
- ☐ You are a woman older than 55 years or you have had a hysterectomy or you are postmenopausal.
- ☐ You smoke.
- ☐ Your blood pressure is greater than 140/90 mm Hg.
- ☐ You don't know your blood pressure.
- ☐ You take blood pressure medication.
- ☐ Your blood cholesterol level is > 240 mg/dl.
- ☐ You don't know your cholesterol level.
- ☐ You have a blood relative who had a heart attack before age 55 (father/brother) or 65 (mother/sister).
- ☐ You are diabetic or take medicine to control your blood sugar.
- ☐ You are physically inactive (i.e., you get less than 30 minutes of physical activity on at least 3 days/week).
- ☐ You are more than 20 pounds overweight.
- ☐ None of the above is true.

If you marked two or more of the statements in this section, you should consult your healthcare provider before engaging in exercise. You might benefit by using a facility with a **professionally qualified exercise staff** to guide your exercise program.

You should be able to exercise safely without consulting your healthcare provider in almost any facility that meets your exercise program needs.

* Proceed with test if musculoskeletal problems are minor, concerns about safety of exercise are normal, and prescription medications are not for cardiac, pulmonary, or metabolic disease

..SK STATUS(Low; Moderate; High): _____

APPENDIX B

INFORMED CONSENT FOR PARTICIPATION
Effect of Counterpulsation on Delayed Onset of Muscle Soreness

Introduction

You have been asked to participate in a research study to be conducted in the Human Performance Laboratory at Cleveland State University. It is the purpose of this study to measure the recovery from prolonged running with the use of External Counterpulsation (ECP) and its affect on reducing delayed onset of muscle soreness.

Previous studies have documented changes in skeletal muscle enzymes resulting from the physical demands of long-distance running. Serum markers can be used to assess muscle damage, these include creatine kinase (CK), and lactate dehydrogenate (LDH). C-reactive protein is also a marker for the presence of inflammation. Inflammation occurs during the healing of tissue and may be the cause of muscle soreness after exercise. External counterpulsation (ECP) is a device, commonly used in cardiac patients, that increases vascular profusion therefore decreasing the effect of coronary artery disease. This device uses pneumatic pressure cuffs worn on the legs that inflate rhythmically to increase blood flow. Previous studies have documented that ECP increases myocardial blood flow in cardiac patients therefore increasing exercise tolerance.

Procedures

You will be asked to refrain from hard training 48 hours prior to the initial testing. Blood samples will be taken prior to the prolonged run and measured for CK, LDH, and C-reactive protein. You will then be instructed to complete a twenty mile run. Blood samples will be drawn immediately after the run and each day after until baseline levels are again achieved.

You will complete the same run, two times. After one of the runs you will be treated with ECP (Approximately 45 minutes) daily until blood markers return to normal. The second run will be the control where only the blood markers will be measured and no ECP treatment will be given.

RISKS AND DISCOMFORTS

Risk associated with this study include mild muscle soreness resulting from the run. Also, discomfort experienced from giving venous blood sample. The risk associated with the run would be the same as experienced from your normal training regimen. Every effort will be made to minimize these risks . A Clinical Nurse Specialist or qualified technician will be responsible for obtaining blood samples and a Nurse or trained technician will be responsible for administering the ECP treatment. Every effort will be made to minimize these risks.

BENEFITS

The benefits of this study are numerous. If ECP treatment can improve blood flow and reduce time for recovery during training, it would be of great importance for you and competitive athletes in any sport. It would also minimize muscle soreness associated with hard training.

CONFIDENTIALITY

To protect your privacy, your name will not be used in any documentation of the project. The information, however, may be used for a statistical or scientific purposes with your right of privacy retained.

Participation

I understand that participation in this project is voluntary and that I have the right to withdraw at any time with no consequences. I understand that if I have any questions about my rights as a participant, I can contact Cleveland State University's Review Board at (216)687-3630.

I attest and verify that I have no known health problems that could prevent me from successfully participating in the sub-maximal graded exercise test.

Inquiries

Any questions about the procedures used in this project are welcome. If you have any doubts or questions, please ask us for further explanations or call Dr. Kenneth Sparks at (216) 687-4831 or Dr. Kathleen Little at (216) 687-4877.

Patient Acknowledgement

The procedure, purposes, known discomforts and risks, possible benefits to me and to others have been explained to me. I have read the consent form or it has been read to me, and I understand it.

I agree to participate in this program. I have been given a copy of this consent form.

Signature: _____ **Date:** _____

Witness: _____ **Date:** _____

APPENDIX C

Pain should be reassessed at least every 24 hours

- Key questions to ask patients

PAIN INTENSITY – Describe the pain (sharp, aching, dull, throbbing, etc)

LOCATION – Where on your body is the pain?

ONSET – When did the pain start?

DURATION – Is the pain always there or does it come and go?

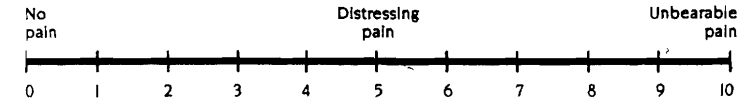
VARIATION – What makes the pain worse?
What makes the pain better?

QUALITY – Does the pain affect activities like sleeping, eating, walking, etc?

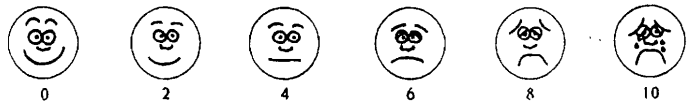
- Watch for behavior, expressions, movements and activities that may indicate pain, eg:

NONVERBAL INDICATORS
- Facial wrinkling, blinking eyes, grimacing
- Guarding an area of the body
- Crying, moaning
- Decrease in social interaction/routines
- Aggression, eg, hitting/biting
- Increase in body movements
- Irritability; increased confusion

Pain Assessment Scales

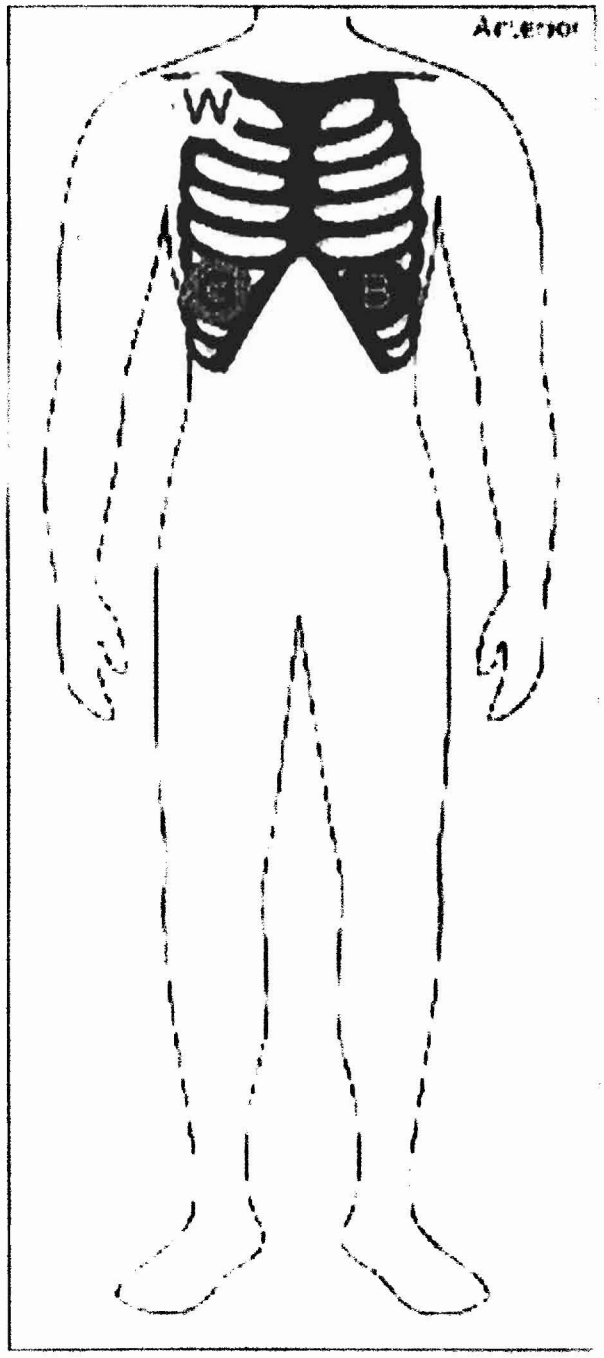


From Iacco A, Carr DB, Payne R, et al. Clinical Practice Guideline Number 9: Management of Cancer Pain. Rockville, Md: US Dept of Health and Human Services, Agency for Health Care Policy and Research; 1994. AHCPR publication 94-0592.

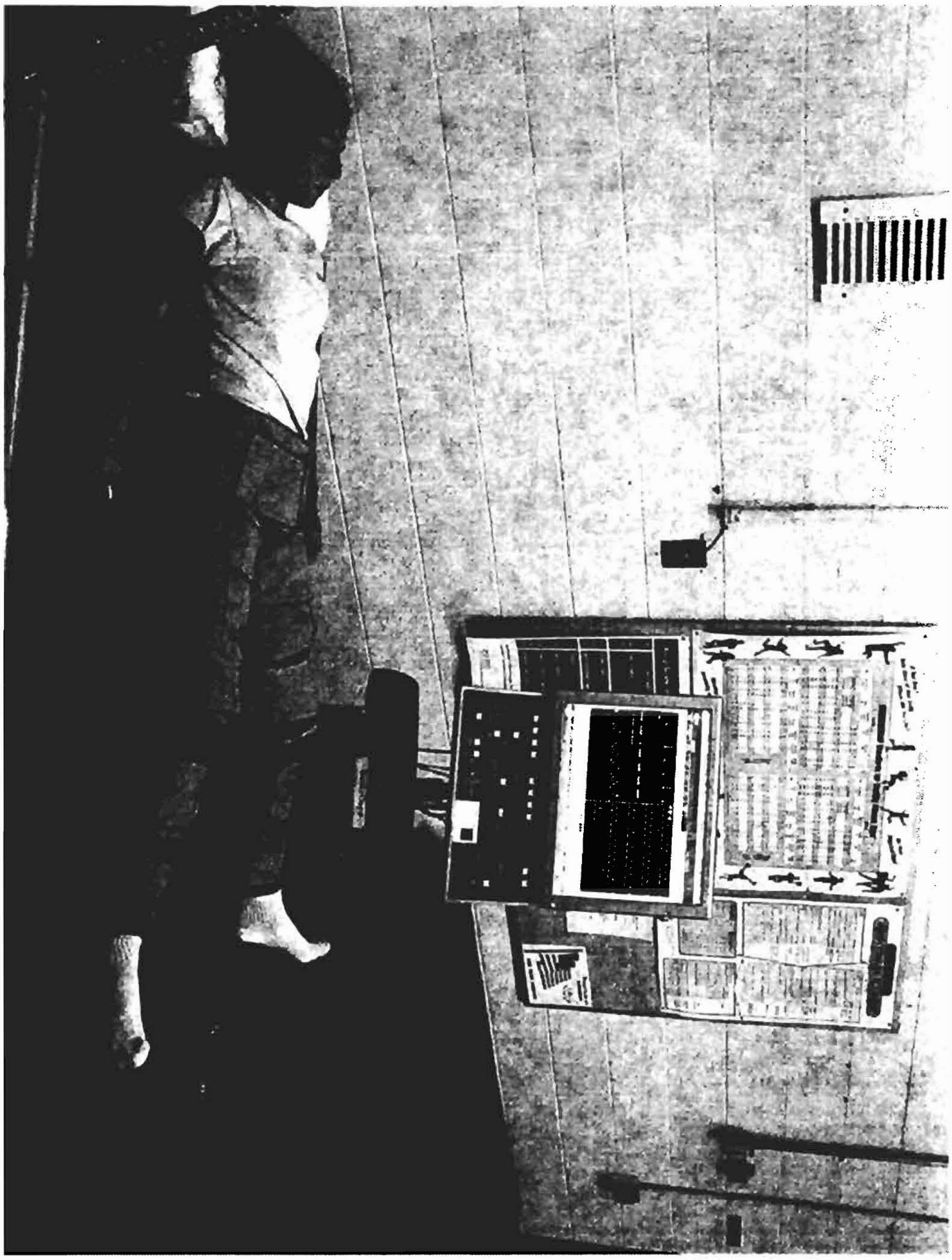


APPENDIX D

Electrode Placement for ECP



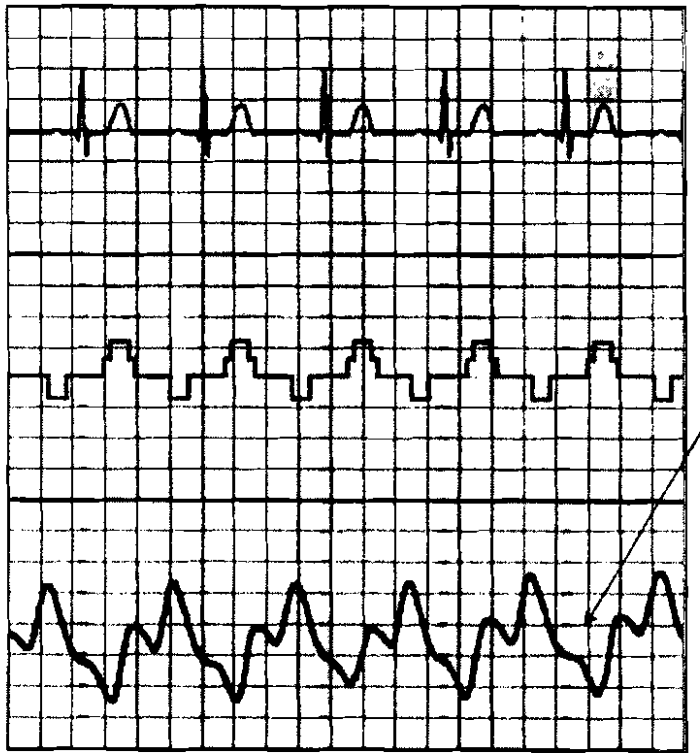
APPENDIX E



APPENDIX F

ECP Timing

Good Deflation



Goal of Deflation:

1) **Decrease vascular resistance**
2) **Afterload reduction**
3) **Decrease cardiac workload**

Indicator:

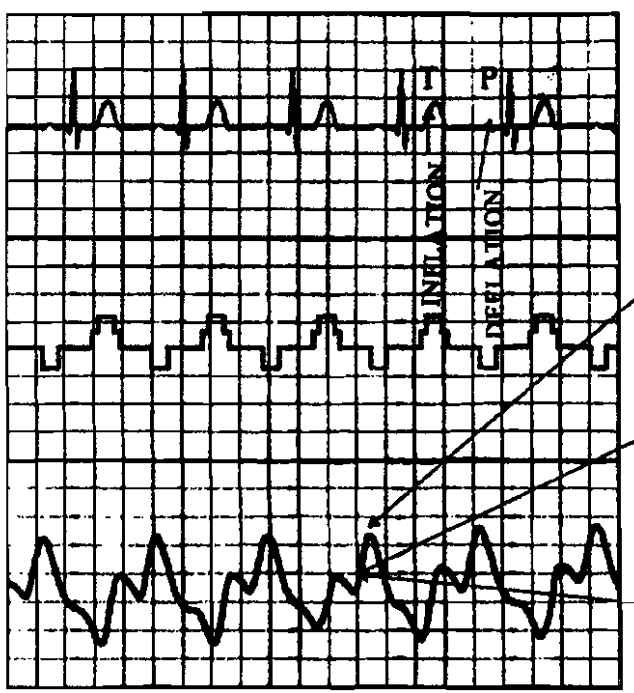
End-diastolic pressure (EDP) area

Characteristic:

Gradual sloping in EDP

ECP Timing

Good Inflation



Inflation should occur at dicrotic notch/ aortic valve closure

Goal of Inflation:

Augmentation of diastolic pressure

Indicator:

Transition from systole to diastole

Characteristic:

1) Check mark appearance
2) Begins 1/3-1/2 of systolic peak

TIMING ERRORS