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Effects of Intermittent Pneumatic Compression on Delayed Onset Muscle Soreness (DOMS) in Long Distance Runners

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EFFECTS OF INTERMITTENT PNEUMATIC COMPRESSION ON DELAYED
ONSET MUSCLE SORENESS (DOMS) IN LONG DISTANCE RUNNERS

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Bachelor of Science in Recreation Management and Youth Leadership

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December 2011

Submitted in partial fulfillment of requirements for the degree

MASTER OF EDUCATION

at the

CLEVELAND STATE UNIVERISTY

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ABSTRACT

Purpose: The purpose was to measure the effects of intermittent pneumatic compression (NormaTec, NT) on muscle inflammation after long distance running.

Methods: Ten long distance runners, five males and five females, ages 18-55 years performed two, 20 mile runs at 70% VO_2 max. The runs were followed by either no treatment (control) or NT treatment for five consecutive days. For the NT run, subjects were treated for one hour immediately following the run and daily for five days after. For the control run, subjects did not receive any treatment. Serum C – reactive protein (CRP), a marker of muscle inflammation, was measured pre and post run and daily thereafter for five days for both trials. Repeated measures ANOVA and two-way ANOVA were used to assess treatment differences. **Results:** The results indicated no significant difference ($P > 0.05$) between the control and treatment runs in CRP levels. There was also no gender differences or order effect of runs. Subjective pain ratings indicated no significant difference in pain between the control and treatment runs except when comparing the first run compared to the second run (regardless of whether the first run was a treatment or control run); there was a significant difference in which the subjects experienced less pain on the second run. There was no significant difference in mean weight loss, fluid intake, sweat rate, heart rate, percentage of maximum heart rate, or percentage of maximum VO_2 max. There was, however, a significant difference ($P = 0.038$) in running

time when comparing the control run (196.2 minutes) against the treatment run (204.8 minutes). **Conclusions:** Although the test subjects recovered one day earlier when using the NT device (Day 4) compared to the control (Day 5), this difference was not significant.

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CHAPTER I

INTRODUCTION

Background

Delayed onset of muscle soreness (DOMS) is an effect of performing an exercise that places a great deal of strain on the skeletal muscles, especially eccentric exercise (1).

Armstrong (2) has 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. lactate dehydrogenase (LDH), creatine kinase (CK), and myoglobin (Mb)). The inflammation process begins as a precursor to full recovery of damaged muscle fibers. Inflammation, indicated by the blood marker C-reactive protein (CRP), denotes the onset of the healing process (2).

In 2002, the NormaTec Pneumatic Compression Device (NTPCD) was introduced to the world of external compression. It was backed as an FDA cleared medical device which yielded highly effective results for patients who had been diagnosed with peripheral vascular diseases (PVD) (3). The NTPCD differentiates from other external compression devices because of its patented peristaltic pulse pneumatic waveform. This peristaltic pulse was a major advancement that went beyond the existing and already

established inflation and deflation strategies (non-sequential, sequential, and sequential gradient) used in the 1960s to 1980s (3).

In 2007, NT extended its technology from clinical medicine to sports medicine as a recovery tool for athletes. The sequential pulse technology compression (peristaltic pulse pneumatic waveform, in the sports medicine jargon) is based on normal physiology and combines three distinctive massage techniques to speed the body's natural recovery process (3). The first technique is pulsing. Sequential pulse technology uses pulsing, or dynamic compression, to more effectively mimic the muscle pump of the legs and arms. This dynamic compression greatly enhances the movement of fluid and metabolites out of the muscles after intense workouts. The second technique is gradients. Sequential pulse technology uses gradient hold pressures to prevent body fluids from being forced down to the feet by the pulsing actions described previously. The gradient hold allows maximum pressure to be delivered throughout the entire limb and the effectiveness of the pulsing action is not curtailed at the top of the limb. The third technique is distal release. Extended static pressure can not only be detrimental to the body's natural circulatory flow, but to the limb where the pressure is applied as well. The sequential pulse technology, however, releases the held pressures placed on the limb once they are not needed to prevent backflow of blood. In releasing the held pressure in each zone as quickly as possible, each portion of the limb gains maximal recovery time without a significant pause between compression cycles (4, Appendix A).

Statement of the Problem

Research is needed in order to determine if the NT recovery unit is effective in the reduction of muscle inflammation after long distance running. If the NT recovery unit can speed recovery as well as reduce the effects of DOMS on athletes, this may allow for athletes to enhance their training by helping them recover quicker and by allowing them to train harder.

Purpose of the Study

The purpose of this study was to measure the effects of intermittent pneumatic compression (NT) on the muscle inflammation marker C-reactive protein (CRP) and DOMS in trained athletes after long distance running.

Hypothesis

NT treatment will reduce inflammation, as indicated by the blood marker C-reactive protein (CRP), and reduce delayed onset muscle soreness (DOMS).

CHAPTER II

LITERATURE REVIEW

The literature review discusses DOMS and exercise-induced muscle damage, potential effects of massage and other recovery treatments (relatively older forms of treatment), and intermittent pneumatic compression (relatively newer form of treatment) on DOMS and exercise-induced muscle damage.

DOMS and Exercise Induced Muscle Damage

Miliias et al. (5) sought to determine if a relationship existed between platelet activator factor (PAF) and eccentric exercise induced muscle damage. The subjects consisted of 13 healthy, active males who had not participated in any kind of resistance training for a minimum of six months before participating in the study.

Subjects performed six sets of six repetitions (maximum eccentric actions of the elbow flexors) with their non-dominant arm using a motorized muscle dynamometer with a one minute rest period between sets. Venous blood samples were collected from the dominant arm immediately prior to, and following the non-dominant arm exercise. Additional blood samples were taken 2, 24, 48, 72, and 96 hours following the exercise. PAF, C-reactive protein(CRP), creatine kinase (CK), fibrinogen, lactate dehydrogenase

(LDH), complement C3, and whole blood level leukocytes (including their subsets) were determined.

The results indicated CK, LDH, leukocytes, and PAF were the only biochemical markers which elevated and showed statistically significant differences following exercise. The authors concluded a possible role of PAF in the mechanism of exercise induced muscle injury and suggested future research investigate the exact features of PAF in its role as a mechanism of exercise induced muscle injury, and whether it could be utilized as a new marker of muscle damage (5).

Nosaka et al. (6) investigated DOMS and enzymes indicating muscle damage following eccentric exercise. They enrolled 110 healthy males, mean age 23 years, who had not been involved in a resistance exercise training program. Subjects were placed in one of two groups based on the number of eccentric exercises they were able to perform. The first group (N=50) performed 12 eccentric exercise actions. The second group (N=60) performed 24 eccentric exercise actions.

Muscle soreness was measured with a visual analog scale indicating “no pain” to “unbearable pain” based on three assessments: palpating over the elbow flexors, and flexing and extending the elbow joint in a full range of motion. Soreness developed on day one following exercise and peaked two to three days after exercise for both groups. Time course changes in soreness were similar in palpitation, flexion, and extension. However, peak soreness values were significantly smaller for flexion compared to palpitation or extension. There were no significant differences between the first group and second group for palpitation and flexion soreness, but the second group had significantly higher extension soreness values three and four days after exercise

compared to the first group. Peak soreness values were similar for the first and second group for palpitation and flexion soreness. However, the second group showed significantly higher peak extension soreness values compared to the first group. A Palpitation soreness did not correlate significantly with any indicators, however, extension and flexion soreness revealed a weak ($r < 0.32$) but significant correlation with the other indicators.

Maximal isometric force, relaxed and flexed elbow joint angle (RANG and FANG), upper arm circumference, and CK were measured before, immediately after, and every day after for four consecutive days following the exercise. The results indicated no significant difference in the pre-exercise maximal isometric force between the first group (12 eccentric exercise actions) and second group (24 eccentric exercise actions). Maximal isometric force dropped about 58.1% for the first group and about 47.1% for the second group immediately after exercise. The force deficit was significantly larger for the second group compared to the first group. Four days after exercise, maximal isometric force had recovered to about 73.1% of the pre-exercise value for the first group which was significantly larger than the second group which was about 52.5% of the pre-exercise value at the same time point.

The results for the elbow joint angles for RANG showed a significantly larger mean decrease immediately following exercise in the second group compared to the first group. Four days post-exercise, RANG was found to still be significantly smaller than the pre-exercise level for both the first and second groups. However, the amount of decrease from baseline was significantly larger for the second group compared to the first group. The mean increase in FANG following the second group was significantly larger

compared to the mean increase in the first group. Four days post-exercise, FANG was found to be still significantly larger than pre-exercise levels for both the first and second groups. However, the amount of increase from baseline was significantly larger in the second group compared to the first group.

Upper arm circumference significantly increased immediately following exercise for both the first and second groups. The upper arm circumference continued to increase and peaked three to four days post-exercise in both groups but the greatest value in the second group was significantly ($P < 0.01$) larger compared to the first group.

Plasma CK activity significantly increased and peaked three to four days post-exercise in both groups. However, CK in the second group was significantly higher compared to the first group.

It appeared the degree of DOMS associated with eccentric exercise was not affected by the severity of muscle damage. Likewise, levels of muscle soreness either have little or no correlation with other commonly used muscle damage indicators. Thus, preventative or treatment measures for DOMS are not the same as preventative or treatment measures for muscle degeneration and regeneration. The authors further suggested that finding preventative or treatment measures which reduce strength loss or plasma CK responses are not necessarily effective in treating DOMS and that in the absence of DOMS, muscle damage and the loss of muscle function will persist. So using DOMS to judge the degree of muscle damage is not recommended. However, when used jointly with other indicators of muscle damage, DOMS can provide further evidence of changes within the muscle (6).

Lee, Clarkson (7) investigated 60 untrained healthy males and females ages 18 to 30 years who had not previously been involved in a weight training program for six months for the determination of total resting glutathione levels. The first 30 test subjects had three baseline blood draws measured for total glutathione, CK, and myoglobin (Mb). Maximal voluntary isometric contraction (MVC) and relaxed arm angle (RANG) were also measured. From the initial 60 test subjects, only 17 subjects were included in the study's inclusion criteria of total glutathione (<2.5 or $>3.8 \mu\text{mol}\cdot\text{L}^{-1}$). The subjects who were included in the <2.5 range were put into a low glutathione group (LG, N=8) while the subjects in the >3.8 range (HG, N= 9) were put into a high glutathione group.

The exercise protocol consisted of the subjects performing one set of 50 maximal eccentric contractions, three second duration with 12 second rest in between, on a modified preacher curl machine. Using a strain gauge attached to the modified preacher curl machine, MVC was assessed with three trials. Using a goniometer to measure elbow joint angles, RANG was measured with the subject's arm hanging at his or her side passively with the wrist in a neutral position. The arm was marked at the wrist, elbow, and shoulder with a skin marker to ensure day to day measurement reproducibility. RANG was determined by taking the mean of the three trials.

Baseline measures determined no difference in resting plasma glutathione and CK levels over the two baseline days. The intraclass R-value over the two days for CK was 0.80 for the 17 subjects. A significant group, time, and group by time interaction was found for MVC. The RANG results revealed no significant group effect however, there was a significant main effect across time and in the group by time interaction.

There was no significant group effect; but there was a significant time effect and group by time interaction in CK response. There was a significant group, time, and group by time interaction in Mb levels. There was a significant group, time and group by time interaction for Glutathione. There was also no significant relationship between Glutathione and CK in LG or HG.

The authors concluded that subjects with low plasma Glutathione levels had a smaller plasma CK and Mb response and recovered faster from eccentric exercise than subjects with high plasma Glutathione levels. It was suggested that subjects with low plasma Glutathione had a lowered inflammatory response (7).

Chleboun et al. (8) enrolled 11 females, ages 19-22 years, who had not been involved in a resistance exercise program which included the elbow flexors for 6 months before the study. Isometric MVC, were determined using the elbow flexors from three separate repetitions on three different days and the subject rested two minutes between each contraction. Muscle soreness, isometric strength, stiffness and swelling measurements were taken three times before exercise, post exercise and daily for five days and every other day during the 2nd week following the exercise bout. The authors used 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; 4 = constant pain) to measure muscle soreness.

The authors measured stiffness by using an apparatus which moved by a stepper motor in four degree steps. The motor moved from a resting position of 90 degrees to full elbow extension and it was programmed to record angle as well as torque 12 seconds after each of the 4 degree steps. This allowed for 80-90% of stress relaxation.

A second measure of stiffness was measured by using relaxed arm angle. The amount of swelling in the upper arm was measured by using arm circumference and muscle compartmental volume by cross-sectional ultrasound imaging. The average peak soreness resulted in a score of 2.9 on day two. MVC decreased immediately after exercise and reached its lowest point on day one post exercise. Strength remained significantly decreased 11 days post exercise. The volume of the flexor muscles exhibited a significant increase over pre-exercise volumes. Stiffness increased immediately after exercise and remained elevated for the next five subsequent days.

It was determined that delayed onset of swelling did not account for the immediate rise in muscle stiffness. It was also determined that the increased stiffness was related to a change in calcium homeostasis from muscle injury. This suggests stiffness coincides with the flexor muscles returning to normal volume, which further indicates that the return to normal values from stiffness may be a result of muscle edema (8).

Nosaka, Clarkson (9) enrolled 14 males, 20-24 years of age, who had never been involved in a weight training program. Six of the 14 subjects were chosen at random to be scanned using an MRI. The subjects performed 24 maximal eccentric actions utilizing the elbow flexors of their non-dominant arm on a modified arm curl machine. The subjects were evaluated for maximal isometric force (MIF), range of motion (ROM), circumference (CIR), soreness (SOR), ultrasound images (USG), MRI, CK, aspartate aminotransferase (AST), lactate dehydrogenase (LDH), interleukin - 1 α (IL-1 α), interleukin - 1 β (IL-1 β), interleukin - 2 (IL-2), interleukin - 6 (IL-6), tumor necrosis factor - α (TNF- α), CRP, cortisol and zinc. Measurements were taken immediately

before and after exercise and for five days post- exercise, except the MRI images were taken on day 1, 3, 6 pre-exercise and day 10, 23, 31, and 58 post-exercise.

MIF was measured on an arm curl machine using a transducer which was connected to a digital indicator. The MIF was measured at an elbow joint angle of 90 degrees. Using a goniometer, the elbow joint angle ROM was evaluated using flexed (FANG) and relaxed (RANG) at the elbow joint. After obtaining FANG and RANG measurements, ROM was calculated by subtracting FANG from RANG. Muscle soreness was assessed by using an analog scale, based on the subject's perception, of no pain to very painful. CIR was assessed using the upper arm at four different sites (4, 6, 8, 10 cm) above the elbow joint with the subject's arm hanging down by their side.

All subjects were assessed using USG (7.5 MHz linear probe) on the same four sites used in the CIR measurements to obtain transverse scans of the brachialis and biceps brachii. MRIs were taken from the upper arm using the elbow joint as a landmark for consistent scanning. Three circular areas of interest, which indicated an increase in signal intensity, were set in the transverse section of the biceps brachii or brachialis. The mean value from the three ROI was used as a reference during measurement, and was placed in the center of the triceps brachii.

There was a significant drop in MIF (approximately 55%) in pre-exercise levels immediately post exercise. Five days post exercise, MIF was still 70% below pre-exercise levels. There was also a significant decrease in ROM immediately following exercise with no indication of recovery three days after exercise with a gradual recovery following. There was no significant difference in SOR levels between palpation and extension of the elbow joint. There were also no significant differences in all four upper

arm circumference measurements. In eight of 14 subjects, USG showed an increase in echo intensity in the brachialis three to five days post exercise. Increased echo intensity was shown by six subjects in both the brachialis and biceps brachii. USG also revealed an increase (0.6 cm – 2.2 cm) in muscle thickness with a maximum increase three to four days post exercise which remained unchanged for five days after exercise.

The elbow flexor muscles showed enlargement in the cross sectional area with profound increases in signal intensity at three days post exercise. CK, AST, and LDH revealed no significant increase as the changes over time among all the enzymes were similar. No significant changes were observed in CRP, cortisol and zinc following exercise. The authors concluded that in spite of similar responses in exercise induced muscle damage and inflammatory responses accompanying infection or tissue injury, there are identifiable differences overall from the classical observance of typical tissue damage/repair processes (9).

Hosoi et al. (10) enlisted 15 male recreational runners, 41-46 years old, in good health with predominantly daily sedentary employment to run a marathon. Following the marathon, participants adhered to their normal daily routine; however, due to muscle soreness, participants did little running in the first week. The subjects were unable to resume their normal training activity for at least two weeks after the marathon.

Two weeks before subjects participated in the marathon, each was required to perform a maximal exercise test and body impedance was used to determine body fat percentage. Venous blood samples were obtained to establish baseline levels on the morning of the marathon. The first post race blood sample was drawn within five minutes of each subject's finish and subsequent samples were taken on days 1, 2, 3, 7, and 14 after the

marathon. Measures included: total cholesterol (TC), triglycerides (TG), HDL-C, LDL-C, free fatty acids (FFA), lactate dehydrogenase (LDH) and hematocrit (Hct).

Hematocrit increased significantly from baseline (43.1%) to post race (44.0%). In days one, two, and three, hematocrit levels were within 0.3% of baseline. Plasma TG levels significantly elevated immediately following the marathon (7%); but, after day one and two of recovery, levels fell significantly (28%) below baseline. After one week, TG levels returned to baseline. LDL-C levels were not significantly affected by the marathon, but gradually fell significantly below baseline levels for three days, returning to baseline after one week. HDL-C levels increased 14% the first day following the marathon and remained significantly elevated for three days, gradually decreasing to baseline. TC increased slightly following the marathon and then significantly fell below baseline after two days, returning back to baseline within one week. TC/HDL-C ratio reduced 9 % immediately following the marathon and the ratio declined 16% below baseline for three days, reaching baseline levels after two weeks.

Average CK levels post race tripled from baseline with a continual increase reaching a 15-fold peak after the first day, falling to baseline levels after one week. LDH levels doubled from baseline post race and declined linearly in a two week period towards baseline. Post race CK and LDH levels were significantly correlated over time (average $r=+.71$). FFA levels rose by a factor of 10 immediately post race with a rapid decline after the first day. With the decreasing values, FFA remained elevated, when compared to baseline, for two days.

It was determined, through this study, that prolonged strenuous exercise in recreational runners produces beneficial changes in lipid blood profiles. However, these

changes were only significant for three days. Further, evidence of exercise-induced muscle damage occurring during the marathon returned to baseline levels within two weeks (10).

Schwane et al. (11) enrolled seven healthy males ages 19-21 years who were active in aerobic activities but none were highly trained. The subjects performed three treadmill exercises in a specific order: VO_2 max test, level run, and downhill run. Six subjects performed the three outlined consecutive tests separated by six to seven days. The seventh subject had a 23 day separation between the first two sets and a 14 day separation between the second and third tests.

Following the VO_2 max, all subjects had a short period of recovery. The subjects ran on the treadmill at 0% incline during which their VO_2 was measured. The speed attained from the recovery run was used on the level and downhill tests. The level test was run at 0% incline and the downhill test was run at -10%. Subjects ran intermittently for 45 minutes with two minute rest periods interspersed between five minute runs. During the last minute, within each 5 minute bout in the level and downhill tests, VO_2 was measured. The oxygen requirement for each subject within the exercise was calculated using the average of the last minute within each running bout.

Blood was drawn from the antecubital vein immediately prior to exercise and five minutes 24 hours, 48 hours and 72 hours after the level and downhill runs to determine creatine, phosphokinase (CPK) and lactate dehydrogenase (LDH). Additionally, total and differential hematocrit (Hct) and white blood cell (WBC) counts were obtained. Muscle soreness was evaluated based on a four point scale (0 = complete absence of soreness; 1 = light pain felt only on palpation; 2 = moderate pain with some stiffness and/or

weakness during movement; 3 = severe pain that limits range of motion). The ratings were based specifically on the anterior and posterior leg muscle groups (gluteal, hamstring, quadriceps).

During the level run, VO_2 and heart rate were significantly ($P < 0.001$) higher compared to the downhill run; however, there was no difference in stride frequency between the two tests. Also, during the level run, muscle soreness was only reported in isolated muscle groups. However, it was found through the duration of post exercise, that there was no significant increase in muscle soreness found in any muscle group of the anterior and posterior part of the leg.

Muscle soreness after downhill running was experienced in several different muscle groups. Post exercise muscle soreness ratings had a significant increase compared to pre-exercise for the anterior and posterior part of the leg. Plasma CPK was not significant following the level run; but during the downhill run, 24 hours post exercise CPK values were significantly elevated compared to pre-exercise values. At 48 hours post exercise, CPK values were not significantly different from 24 hours post exercise, nor from pre-exercise values. The association ($r = 0.53$) between plasma CPK and muscle soreness over time was significant.

There was no significant change from pre to post exercise at any point during the study between the level and downhill run, nor was there a correlation ($p = 0.05$) between muscle soreness and LDH values. Additionally, there were no changes observed in pre-exercise LDH values in either level or downhill running compared to post exercise LDH values.

There was only one significant change in WBC from pre-exercise to post exercise immediately following the level run and only one significant change in WBC count during the downhill run in which there was a decrease in monocytes 48 hours post exercise. The largest change in Hct occurred in one subject 24 hours post exercise during the downhill run, -18% pre-exercise value. Changes in Hct levels were observed to be fairly stable in the other six subjects.

It was determined that delayed onset muscle soreness (DOMS) caused by downhill running did not increase WBC indicative of inflammation. It was further determined structural changes occurring in muscle tissue as a result of eccentric muscle contractions produce plasma enzyme activities and DOMS (11).

Millar et al. (12) recruited 21 subjects who were divided into a control group and an exercise intervention group, matched by age and gender, to determine the effects of aerobic exercise on aspartate aminotransferase (AST), pain, strength, alanine aminotransferase (LT), creatine kinase (CK), and range of motion (ROM). The test subjects all performed eccentric hamstring contractions using a Biodex isokinetic dynamometer consisting of five sets of 10 repetitions. Active knee extension, pain ratings, AST, isometric hamstring strength, ROM, AST, and CK were all assessed before exercise as well as 24, 48, and 72 hours following exercise. Those in the exercise group walked on a motorized treadmill at 50% of their maximum heart rate for 20 minutes at 3, 24, and 48 hours following exercise.

No significant differences were found between the control and exercise intervention groups. A trend analysis was run and significant differences were found in the response to CK, ROM, and strength. The exercise intervention group continually lost ROM until

day three with less ROM compared to the control group. The exercise intervention group also experienced a more rapid rise in CK compared to the control group. The authors concluded that aerobic exercise used as an intervention did not alleviate the symptoms of DOMS and based on the trend analysis, it may even slow down the normal healing process (12).

Effects of Massage on DOMS

Zainnuddin et al. (13) tested the hypothesis that if massage was applied after eccentric exercise, DOMS would be effectively alleviated without affecting muscle function. They enrolled 10 healthy subjects, five women and five men, ages 22-24 years, who had no history of upper arm injury nor did they have any experience in resistance training. An arm to arm comparison model was used in which one arm served as the control and the other served as the treatment condition. Dependent variables measured on the exercised arm were maximal isometric and isokinetic voluntary strength, range of motion, upper arm circumference, plasma CK, and muscle soreness (VAS, 0 indicating no pain and 100 indicating extremely painful).

The subjects performed 10 sets of six maximal isokinetic eccentric actions involving the elbow with each arm with a two week separation between tests. A standard sports massage was administered by a professional massage therapist who had been working with the Australian football club for several years. One arm was massaged for 10 minutes three hours after the eccentric exercise was performed, while the other arm received no treatment. Massage techniques used were deeply applied clearing techniques with palmar (petrissage) and finger (effleurage) pressure and strokes to the muscle.

The results indicated no significant difference between baseline maximal isometric and isokinetic strength between the massage and control arms. Similarly, no significant difference existed between peak torque and total work during eccentric exercise. No significant differences were observed between maximal isometric torque at an elbow angle of 90 degrees and 30 degrees between the massage and control arms. The isometric torque values decreased about 60% from the pre-exercise values immediately post exercise and remained at these decreased values for two days; torque returned to the pre-exercise level by 10 days post-exercise. There was no significant difference between the massage and control arms for changes in isometric torque over time.

The changes in maximal voluntary isokinetic torque were similar to those found in the isometric torque throughout the post-exercise period. No significant difference was evident between the massage and control arms for any of the velocities tested. Additionally, the isokinetic torque values returned to pre-exercise levels by day 10 for both conditions. No significant differences were evident in the pre-exercise ROM values between the massage and control arms. Immediately post-exercise, ROM decreased significantly by about 30% from the baseline and did not recover over the next four days.

The upper arm circumference significantly increased during the post-exercise period in both conditions, however, the massaged arm showed a significantly smaller increase as compared to the control arm. While plasma CK activity significantly increased in both conditions post-exercise, there were significantly smaller CK increases in the massaged arm as compared to the control arm. Additionally, the massage condition peak value was 36% lower compared to the control condition.

Peak soreness occurred one to three days post-exercise, whereas peak soreness occurred four days post-exercise for the elbow joint extension and all reports of soreness were resolved seven days post-exercise. Significant differences existed between the massage and control conditions in regard to peak soreness with palpitation and elbow joint extension. Massage resulted in a 20% to 40% decrease in muscle soreness compared to the control. The authors concluded that massage was effective in reducing the magnitude of DOMS, swelling, and plasma creatine kinase. However, massage seemingly had no positive effects on muscle strength or ROM (13).

Moraska (14) enrolled 317 finishers in a 10 km running race. A questionnaire recorded the following information: race finish time, demographic information and perceived exertion during the run. The participants rated their muscle soreness on VAS with 0 being absence of muscle soreness and 10 indicated extreme muscle soreness.

Immediately following the race, subjects were randomly assigned a student massage therapist and were blinded to their educational training of their massage therapist. Massage students with 450, 700 or 950 hours in didactic massage therapy training administered massage in all sessions. Massage was administered to test subjects after the 10 km race for 12 to 15 minutes, within 15 to 60 minutes after completing the race. Student therapists were not bound to a specific protocol and were able to use massage strokes and techniques deemed appropriate for each subject's needs. However, the primary technique used was effleurage, which consists of applying deep flushing strokes to the areas of concern to the race participant.

It was determined that there were no significant differences among age ($P = 0.90$), time and perceived exertion among the treatment groups. Average muscle soreness was

rated a 4.4 among all treatment groups. Participants who received massage from student therapists who accumulated 950 hours of training reported significantly greater improvement in muscle soreness throughout the study compared with those receiving massage with student therapists accumulating 700 or 450 hours of training.

Gender had no affect on muscle soreness throughout the study. In addition, at the 24 hour post race time, muscle soreness for the participants receiving massage from the student therapists who accumulated 950 hours (2.4 pain rating) of training continued to report significantly lower muscle soreness ratings as compared to the subjects receiving massage from the student therapists with 700 (3.7 pain rating) or 450 (3.6 pain rating) accumulated training hours.

Post hoc analysis at the 48 hour post race point in the study, revealed a trend for a lower muscle soreness rating in the group of student therapists with 950 massage training hours. Results indicated a significant improvement in muscle soreness immediately after the 12-15 minute massage compared with pre-massage values. Immediately following post massage, average muscle soreness reported for all three treatment groups was 3.1. It was determined that the amount of therapist training impacted the effectiveness of using massage as a post race recovery tool. The greatest reduction in muscle soreness was achieved by the student therapists who had accumulated 950 hours of training compared with those with 700 or 450 accumulated training hours (14).

Wiltshire et al. (15) sought to determine if sport massage aided muscle recovery following exercise by increasing blood flow in order to improve lactic acid removal by recruiting 12 healthy males, ages 23-26 years. Subjects were in the supine position throughout the duration of the experiment with the forearm positioned at heart level in

each of the three testing conditions. Time allowed between trials for all variables being measured to return to original baseline levels was about 15 to 20 minutes before the initiation of the next trial.

The three experimental protocols were arranged such that 11 of the 12 test subjects performed all three protocols on the same day. The three experimental conditions were counterbalanced between subjects. Subjects were required to perform three preliminary maximal isometric hand grip (IHG) contractions by using a calibrated hand grip dynamometer with a one minute recovery between contractions. Maximum voluntary contraction (MVC) was the highest of the three trials. Once MVC was established, subjects squeezed IHG continuously for two minutes at 40% of their MVC at the beginning of the testing protocol. Following IHG exercise there were two types of recovery: passive and active. In the passive recovery protocol, the subject laid quietly following the two minute IHG with their forearm at rest for 10.5 minutes. The active recovery protocol consisted of subjects laying quietly for 30 seconds with their forearm at rest. Following the 30 second rest, subjects began rhythmic forearm contractions at 10% of their MVC for 10 minutes. The subjects also had a duty cycle of 1:2 contraction relaxation coordinated with a metronome.

For the post IHG massage protocol, following the subject's two minutes of IHG, the subject rested for 30 seconds lying quietly with their forearm at rest in which forearm massage started. Massage was administered by a single registered massage therapist with 13 years of sport massage experience. Massage consisted of effleurage strokes during the first and last 2.5 minutes of therapy with petrissage strokes in the five interim minutes of therapy.

Before initiating the experimental protocols all test subjects had their right hand heated using a hydrocollator heating pad for 10 minutes. Following the hand heating, a blood sample was obtained for arterial lactic acid and hemoglobin during the experimental protocol.

Heart rate was measured using standard CM₅ ECG placement and mean arterial pressure (MAP) was measured using finger photoplethysmography, both measured beat to beat. The brachial artery mean blood velocity (MBV) measurements were obtained beat by beat on the experimental arm. A pulse Doppler ultrasound probe was used at an operating frequency of four MHz.

The results for MBV indicated an uninterrupted pulsatile flow during passive rest. Active rest results indicated a negative brachial artery blood velocity. Petrissage and effleurage massage strokes at first decreased blood velocity followed by an increase in blood flow velocity during the brief pauses by the massage therapist.

IHG strength declined over time in the subject's ability to maintain 40% of MVC. No difference was detected between MAP or HR. The MAP increased more in the passive recovery compared to massage and active recovery, by the end of the IHG. During active recovery, MAP remained higher than massage during the first 2.5 minutes of post exercise achieving statistical significance compared to passive recovery.

Immediately following IHG, VO₂ was noted to be greater in passive recovery than both active and massage recovery. Active recovery was greater than massage recovery. The VO₂ was substantially greater in active recovery compared to both passive and massage recovery. Following IHG, there were no significant differences in recovery conditions after four and a half minutes. Total forearm blood flow following post IHG

was significantly reduced in massage recovery compared to passive recovery. However, it was not significant compared to active recovery. Post IHG active recovery indicated no difference from passive recovery. Lactic acid post IHG was found to be about 17% lower in active recovery compared to passive recovery but was not statistically significant. Hydrogen did not differ between conditions.

It was determined that severe impairment of blood flow occurs during the massage stroke, resulting in a decrease of muscle blood flow in the recovery period following strenuous exercise. Active recovery showed similar effects. The authors stated the decreased blood flow is responsible for impaired lactic acid removal from the exercised muscle. It appears that active recovery does not improve muscle blood flow following exercise; but, it does appear to increase lactic acid uptake by the muscle, improving lactate removal from muscle tissue. The authors noted that sports massage would not be optimal in situations where acute bouts of repeated exercise are occurring and where lactic acid builds up (15).

Hinds et al. (16) compared a control condition against massage to determine if massage affected femoral artery blood flow (FABF), muscle temperature (MT), skin temperature, and skin blood flow (SKBF) following quadriceps exercise. The subjects consisted of 13 males, ages 20-22 years, who participated in an exercise bout consisting of three, two minute concentric quadriceps exercises followed by either three, six minute deep effleurage and petrissage massages or a 12 minute rest period (control). Blood lactate concentration (BLa), FABF, MT, heart rate, blood pressure, skin temperature, and SKBF were taken at baseline, immediately following exercise, and at the midpoint as well as the end of the massage or rest periods.

The results indicated significant differences for all the variables over time due the exercise compared to baseline. Massage was found to not significantly elevate heart rate, BLa, FABF, blood pressure, or MT compared to the control. Skin temperature and SKBF significantly elevated after massage compared to the control. The authors concluded that any increases in SKBF could potentially redirect blood flow away from recovering muscle tissue, thus questioning the ability of massage to aid in post exercise recovery (16).

Haas et al. (17) examined the effectiveness of using immediate compared to delayed compressive massage-like loading on inflammatory cell infiltration as well as peak isometric torque recovery following eccentric type exercises. White New Zealand rabbits (N=18), which were skeletally mature, were used in the study by adapting peroneal nerve cuffs to the hind limb in order to stimulate the tibialis anterior muscles. Following a bout of eccentric exercise the rabbits were randomly assigned to one of the three massage-like compressive loading protocols for 15 minutes for four consecutive days. The first protocol started immediately following exercise, the second started 48 hours following exercise, and the third protocol consisted of no massage-like compressive loading to act as a control. A relationship between torque angle was gathered from 21 joint angles before and after eccentric exercise as well as following four consecutive days of either massage-like compressive loading or no massage-like compressive loading. Following the final treatments immunohistochemical sections as well as muscle wet weights were gathered.

The results indicated an average decrease in peak isometric torque output following eccentric exercise of about 38-64%. Immediate application of massage-like compressive

loading was determined to have the most considerable peak torque recovery. Differences were found in torque recovery among delayed massage-like compressive loading and control, immediate and delayed massage-like compressive loading, and immediate massage-like compressive loading and control. An immunohistochemical analysis was performed and indicated a 39.3% increase in the amount of RPN3/57 (antibody which detects uncharacterized antigens found on T lymphocytes, neutrophils, platelets, and thymocytes) and a 366.0% increase in CD11b (an antibody used mainly to identify neutrophils but it can also identify blood monocytes, macrophages, bone marrow cells, and other granulocytes) positive cells among the immediate and delayed massage-like compressive loading. The torque angle indicated a difference for immediate and delayed massage-like compressive loading compared to the control. Peak isometric torque angle was determined and indicated a rightward shift (9.8-10.2 degrees) following exercise compared to the pre-exercise angle. The control, immediate, and delayed massage-like compressive loading indicated a leftward shift in peak isometric torque angle compared to the pre-exercise angle. The authors concluded that immediate massage-like compressive loading following exercise was more beneficial than delayed massage-like compressive loading in balancing inflammatory cell infiltration and in recovering muscle function. The authors found that by delaying the administration of massage-like compressive loading following eccentric exercise there was a greater amount of infiltration of immune cells as well as a greater amount of edema (17).

Weerapong et al. (18) conducted a meta-analysis and evaluated three different aspects of massage: types of massage, possible mechanisms for effect, and effects on performance, recovery, or muscular injury prevention. They concluded more research is

needed to determine if the effects of massage are beneficial. Furthermore, the authors concluded that the different types of massage, the appropriate timing of massage on performance, recovery from injury, or as an injury prevention method all need to be further examined. From their research, Weerapong et al. posed the following seven questions to initiate further research (18):

1. “Can massage increase blood flow, muscle temperature, neuromuscular excitability or muscle flexibility?”
2. “Can massage increase performance in sprinting, jumping, or endurance athletic events?”
3. “What type of massage can produce benefits?”
4. “How long should massage be applied?”
5. “When should athletes receive massage?”
6. “Are the effects of massage universal or are they specific to each massage therapist?”
7. “Is the cost and time for massage appropriate when a warm-up or cool-down may be as, or more, effective?”

Weerapong et al. (18) suggested an additional six points on which future research studies on massage should consider to overcome limitations:

1. “An appropriate control group should be provided.”
2. “The ideal control group for a massage study should be a passive therapy modality. This is where the participants receive the same attention in terms of time from the therapists as the massage group.”
3. “The therapists should not apply any pressure on the muscle.”

4. “Some physiotherapy equipment might be appropriate to incorporate in the study such as a sham shortwave diathermy.”
5. “Studies should utilize a counterbalance design in order to minimize the difference responses of individual participants.”
6. “Appropriate outcome measures and massage techniques should be used in the study.”

Alternative Recovery Treatments on DOMS

Mekjavic et al. (19) recruited 24 healthy males, 20-35 years, to investigate if hyperbaric oxygen therapy (HBOT) would speed muscle recovery following exercise-induced injury to the muscle. Subjects were assigned randomly to a HBOT group or a placebo group. Before performing the workout each subject completed a maximal isometric strength test on the right elbow flexor. Following the maximal isometric strength test the subjects then performed the workout which induced DOMS. The workout consisted of a high force eccentric workout using the elbow flexor muscle group. Following the workout the subjects were exposed to either a normoxic (0.2 ATA) mixture or a hyperoxic (2.5 ATA) mixture for an hour over seven continuous days. During one of their oxygen therapies, each subject had a transcutaneous PO₂ electrode which was attached over the biceps brachii via the skin in order to determine the amount of tissue oxygenation by the oxygen therapy. The subjects used a VAS to rate their perceived muscle soreness from “no pain at all” to “worst pain I could possibly feel.”

The results indicated a significant decrease in isometric strength from pre-exercise to the post-exercise in both the placebo and HBOT groups. Over the recovery period no significant difference was found in the rate of muscle strength recovery between the

groups. No significant difference between the groups was found in perceived soreness with soreness levels peaking 48 hours post exercise. Additionally, increases in arm circumference caused by exercise were similar between groups. The authors concluded that HBOT was not effective in the treatment of DOMS and the use of this therapy is not warranted and athletes should be discouraged from using it (19).

Pointon et al. (20) examined the effects of cold water immersion (CWI) on recovery following simulated collision sport exercise. The study consisted of 10 male rugby players, ages 19-23 years, who performed three sessions including two, 30 minute intermittent sprint exercises (ISE) consisting of 15 meter sprints every minute followed by self-paced exercise bouts of walking, hard running, or jogging making up the rest of the minute. Randomly incorporated into the ISE were three testing conditions: no tackling with passive recovery (the control), tackling with passive recovery, and tackling with a 20 minute CWI recovery. During every sixth rotation, the test subjects received shoulder led tackles to the lower body while performing five, 10 meter sprints on each exertion. The variables recorded in the study were sprint times and distances covered in the ISE, electromyogram, voluntary activation, maximal voluntary contraction, and perceived soreness. Venous and capillary blood markers were measured before and after exercise to establish baseline measures, immediately following recovery as well as two and 24 hours post recovery for metabolites indicating muscle damage.

Results indicated tackling with CWI significantly increased voluntary action, electromyogram, and maximal voluntary contraction immediately following recovery. Tackling with CWI had no effect on the elevation of DOMS markers but blood lactate was reduced significantly following recovery compared to tackling with passive

recovery. The results further indicated CWI reduced perceived muscle soreness two hours following recovery compared to tackling with passive recovery. The authors concluded that with bodily collision, the ability to perform exercises decreases, CWI aids in an accelerated recovery of voluntary action, electromyogram, and maximal voluntary contraction, as well as promotes an improved perception of muscle soreness and contraction following exercise which is collision-based (20).

Mawhinney et al. (21) sought to determine if cold (eight degrees Celsius) and cool (22 degrees Celsius) water immersion affects cutaneous and femoral artery blood flow following exercise. The study consisted of 12 men, age 21-30 years, who completed a cycle test at 70% of the subject VO_2 max until the desired core body temperature of 38 degrees Celsius was achieved. Once the desired core body temperature was achieved, the subjects were immersed into either cold or cool water while semi-reclined up to the iliac crest for 10 minutes or participated in a seated rest (control). The variables measured in the study before immersion, and up to 30 minutes following immersion were thigh skin and rectal temperature, superficial and deep muscle temperature, blood flow to the calf and thigh using laser Doppler flowmetry, as well as blood flow of the superficial femoral artery using duplex ultrasound. Calculated variables during the study included vascular conductance indices such as flux and blood flow, heart rate, and mean arterial pressure.

Results of the study indicated similar rectal temperature reductions (a range of 0.6 degrees to 0.7 degrees Celsius) in all three of the trials. During recovery following exercise, the average skin temperature of the thigh was 25.4 degrees for the cold water trial, 28.2 degrees for the cool water trial, and 33.8 degrees for the control trial. Similarly, the lowest recorded recovery muscle temperature during the study was found in the cold

water trial. Conductance of the femoral artery 30 minutes following immersion in both the cold and cool water trials was lower in temperature (about 55%) compared to the control trial. The study also revealed greater cutaneous vasoconstriction in both the calf and thigh during and following cold, as well as cool water immersion trials compared to the control. The authors concluded that colder water temperature could perhaps be more effective in treating damage caused by exercise and in rehabilitating injuries due to a greater reduction in muscle temperature, not muscle blood flow (21).

Wheeler and Jacobson (22) assessed the effects of whole body vibration (WBV) on delayed onset muscle soreness (DOMS), explosive power, and flexibility following induced DOMS. The study included 10 men and 10 women, ages 19-23 years, randomly assigned to either a control or experimental group. Subjects performed baseline measurements prior to DOMS-induced exercise for explosive power, VAS, hamstring flexibility, and lower back flexibility. These same variables were measured immediately following exercise and again immediately following treatment. Following the initial testing session, the subjects reported back to the lab for four consecutive days at the same time of day for additional data collection. These sessions included the subjects participating in a VAS, vertical jump assessment, and a sit and reach. Following the completion of the assessments, the subjects in the treatment group then spent 10 minutes on the WBV machines while those in the control group spent 10 minutes walking on a treadmill. Following the ten minutes of either WBV or treadmill walking VAS, vertical jump, and the sit and reach assessments were again repeated and recorded.

When comparing pre and post-assessments of explosive power, DOMS, and flexibility between groups or within groups, no significant difference was found. When comparing

light exercise to WBV, no significant difference was found for explosive power, DOMS, or flexibility. The authors concluded that WBV is as equally effective as light exercise as a treatment modality for lowering the symptoms of DOMS, qualifying it as a recovery option to be used in addition to other current DOMS treatments (22).

Cantanese (23) sought to determine if skeletal muscle damage, DOMS, as well as markers of muscle inflammation were affected by external counterpulsation (ECP). Prior to this study, ECP had been used primarily in treating cardiac patients by compressing the patient's lower extremities during diastole increasing coronary perfusion and aiding venous return. Five males and five females, ages 18-43 years, participated in two 20 mile runs completed at 70% of their VO_2 max, once under a treatment trial and once under a control trial. Variables measured were lactate dehydrogenase (LDH), C- reactive protein (CRP), creatine kinase (CK), and perceived leg pain. The variables were measured before the run, immediately following the run, and daily for five consecutive days on both the control and treatment conditions. Additionally, in order to control for effects of ECP five more subjects, ages 24-28 years, remained inactive while receiving five consecutive days of ECP treatment.

The results indicated no significant changes between the 10 subjects and the control group receiving ECP treatment only in pain, CRP, CK, and LDH. Subject's pain, LDH, and CRP significantly decreased following ECP treatment compared to the control. The author found that CK remained significantly elevated three days longer in the ECP treatment group compared to the control. Females experienced a significant difference in CK and CRP whereas the males did not. In both males and females a 20% reduction in pain occurred as a result of the ECP treatment, however, females did experience pain for

one additional day compared to the males. Females also experienced a 20% increase in LDH compared to the males. The males experienced a significant change in LDH on day four of the control run, and immediately following the ECP treatment run.

The author concluded ECP treatment decreased inflammation, LDH elevation, and leg pain in runners following a 20 mile run. When comparing the treatment run to the control CK levels were found to remain significantly elevated however, it was determined ECP alone did not cause a significant increase in CK (23).

Effects of Intermittent Pneumatic Compression (IPC) Treatments

Konstantinos and Knaggs (24) examined three intermittent pneumatic compression (IPC) sequences and their effects on patients with peripheral arterial disease (PAD). The three IPC sequences examined consisted of separated IPC deliveries to the foot and calf, and simultaneous compressions on both the foot and calf. These three sequences were used to determine duration and amplitude decay of acute leg inflow enhancement. The experimental design was a cross sectional study which included patients with PAD and intermittent claudication resulting from either superficial femoral artery occlusion or severe stenosis. The control group consisted of 20 subjects, seven females and 13 males, ages 44-78 years, with no medical history of cardiovascular disease, claudication, and with normal circulation. The experimental group consisted of 22 subjects, nine females and 13 males, ages 50-70 years, diagnosed with intermittent claudication. The experimental and control groups were matched according to age and gender and were treated with IPC.

The baseline volume flow was measured and was determined to be higher in the experimental group without reaching significance. The peak volume flow measured with any IPC mode applied was found to be not significantly different between the experimental and control groups. Additionally, the pulsatility index was found to be lower in the limbs in the experimental group than in the control. It was also found to be lower in the experimental group at the baseline level, five seconds after the delivery of IPC, and throughout the 5 to 50 seconds of its determination with IPC.

Flow enhancement was longer in the simultaneous IPC on the foot and calf versus the foot only in both the experimental and control groups. Experimental and control group decay rates of volume flow enhancement indicated a significant decline. Conclusions drawn from the study indicated that the arteriovenous pressure gradient, nitric oxide, and peripheral sympathetic auto-regulation are likely to be active following IPC administration (between 0 and 20 seconds). During the mid-phase (between 20 and 35 seconds) the arteriovenous pressure gradient and peripheral sympathetic auto-regulation were the only active factors, nitric oxide being excluded. The authors concluded that the arteriovenous pressure gradient is perhaps the most effective flow enhancer found in the late phase of flow decay (between 35 and 50 seconds) (24).

Moseley et al. (25) undertook a broad systematic review of common and conservative therapies to treat secondary arm lymphoedema which included complex physical therapy, limb exercises and limb elevation, oral pharmaceuticals, manual lymphatic drainage, compression bandaging and garments, low level laser therapy, and pneumatic pumps. The study included participants who had a formal diagnosis of chemotherapy-induced and/or, radiotherapy-induced, secondary arm lymphoedema following breast cancer surgery

regardless of whether the surgery was a total or partial mastectomy. The study excluded participants who had primary lymphoedema or recurrent cancer.

Of the studies being reviewed for pneumatic pump therapy, the authors found better results in volume reduction occurred when the pneumatic pump was combined with other therapies such as self-massage, manual lymphatic drainage, and compression garments compared to only using the pneumatic pump as therapy. Additionally, in the reviewed studies, the pneumatic pump demonstrated the ability to maintain initial volume reductions in the patients who continued with pneumatic pump therapy (25).

Partsch (26) discussed different therapeutic modalities used in reducing edema and aiding in venous leg ulcer healing. He emphasized the value intermittent pneumatic compression (IPC) has in the treatment of limb edema and the associated skin changes frequently seen on immobile patients' legs. Partsch discussed how commonplace compression stockings and compression bandages are not as effective in the treatment of immobile patients. This is because compression stockings and bandages are not as efficient in supporting the impaired veno-lymphatic pump but however do impede the leg from swelling. Comparatively, IPC actively compresses the immobile patient's limb producing cyclical pressure waves which mimic the resting and working pressures which are applied by compression bandages. Partsch further stated that IPC not only reinforced the veno-lymphatic pump that is critical in restoring damaged microcirculation of the skin but also reduces limb swelling. Partsch makes the clarion call that using IPC as a treatment modality is underused, particularly in patients who are immobile and/or bound to a wheelchair (26).

Avery et al. (27) presented a case study involving a five month old male diagnosed with congenital lymphedema in the right lower limb. The five month old patient had been successfully treated using intermittent pneumatic compression (IPC) therapy. The authors discussed various treatment options and the pros and cons of each individual treatment in which IPC is discussed. One of the authors, Dr. Laura Jacobs, defined pneumatic medicine as using an airtight sleeve made of fabric to apply pressure to an extremity. The compression device having been calibrated by a physician for both time and pressure is then used to inflate the fabric sleeve in a rhythmic fashion. Moderate external pressure compresses the limb and creates a mechanical force which in turn, decongests the tissues and improves not only lymphatic return but venous return as well.

The authors referenced a study which claimed IPC therapy to be a proven, safe, and an effective modality in the treatment of lymphedema in patients of all ages (28). In comparing IPC to complex physical therapy (CPT), there are several advantages in using IPC over CPT. One advantage being the decongesting of the limb through the sequential pumping style of distal to proximal compared to the static nature of CPT treatment methods. The second advantage of using IPC over CPT is the pumping action, duration, and pressure of the sequential pneumatic compression is both consistent and reproducible whereas CPT is subject to human inconsistency. The third advantage is IPC treatments can easily be self-administered on a daily basis and at home whereas CPT requires a specially trained therapist to administer the therapeutic treatment. The authors cautioned to rule out deep vein thrombosis by using a duplex ultrasound prior to the initiation lower limb therapy. The reason being that compression coupled with a deep vein thrombosis could lead to a pulmonary embolism. The authors concluded that IPC is effective, safe,

and underutilized in reducing and treating children diagnosed with congenital lymphedema (27).

Labropoulos et al. (29) sought to determine if the use of intermittent pneumatic compression (IPC) in patients with critical limb ischemia would increase skin, gastrocnemial, collateral arterial, and popliteal blood flow. All patients included in the study had at least two-level disease diagnosed by angiography. The average age of the study participants was 74 years. Using laser Doppler fluxmetry and duplex ultrasound scans, 20 limbs which had been diagnosed with critical limb ischemia in 20 patients were assessed in a semi-erect position before, during, and following IPC treatments. Of the 20 limbs, 14 of the limbs had been identified as inoperable, and six had been identified as marginal for reconstruction. One pneumatic cuff was placed on the foot and the second on the calf using a maximum inflation pressure of 120 mmHg with the compression cycle set at three seconds with three cycles per minute. The flow volumes measured in the study consisted of the popliteal, genicular collateral artery and the medial gastrocnemial with the skin blood flow being measured at the same time on the dorsum of the foot.

The results indicated a significant increase in the flow of all three arteries compared to baseline levels with the highest change found in the popliteal, with the gastrocnemial and the collateral artery following suit respectively. Following the discontinuance of the IPC, blood flow returned to baseline values which can be attributed to the diameter of the arteries not changing. The increase in skin blood flow was also significant. The results also indicated of the 20 limbs measured there were two which did not increase in arterial or in skin blood flow. This was attributed to a significant popliteal vein reflux and both of the two limbs were amputated not long after the completion of the study. The authors

concluded that IPC augments skin, muscular, axial, and collateral blood flow in patients diagnosed with critical limb ischemia. However, patients with significant venous reflux may not benefit from using IPC. The authors further concluded that IPC may prove beneficial for patients who may not be candidates for revascularization and that IPC enhances blood flow through augmenting the arterio-venous pressure gradient (29).

CHAPTER III

METHODS

An experimental design was used to test the effectiveness of the NT recovery unit. The independent variable was the NT recovery treatment vs. the control condition. The dependent variables were the biochemical marker associated with inflammation; CRP; and DOMS. The study was delimited to trained long distance runners capable of running 20 miles at 70% or above of their maximum heart rate.

Subjects

Ten subjects, five trained males and five trained females, age 20 to 53 years, who attended Cleveland State University (CSU) or lived in the surrounding Cleveland, OH area were enrolled. The subjects were obtained through a convenience sample, by recruiting volunteers from local running and triathlon clubs, and individuals from the CSU women's cross country team. The subjects consisted of marathoners, ultra marathoners, ironman triathletes, and collegiate athletes. All potential subjects completed an AHA/ACSM Health/Fitness Facility Pre-participation Screening Questionnaire (30, Appendix B) to measure potential health risk factors for people engaging in an exercise program. Subjects needed to be in the "Low Risk" category after completing the screening questionnaire. Subjects included in the study were trained and capable of

running 20 miles at 70% of their VO_2 max. Subjects were excluded from participating in the study if they had a history of musculoskeletal, cardiovascular, circulatory, or any other health problems which may have put them at risk if they participated in the study.

All subjects were asked to refrain from other physical activity, flu shots or other immunizations, and other recovery treatments such as stretching, ice baths, or consuming anti-inflammatory medications such as NSAIDS during the six days they participated in the study, as well as 48 hours before the start of the six days. Subjects were also not permitted to participate in the study if they had a cold, and had to wait until they had completely recovered from their cold as inflammation levels would not be a valid indicator of exercise inflammation.

Procedures

Subjects who qualified to participate in the study signed an informed consent form (Appendix C) approved by the Institutional Review Board at CSU (Appendix D).

Subjects who qualified for the study were required to perform a graded VO_2 max test to establish running intensity (70 % VO_2 max) for the running trial. A cross-over design was implemented and subjects were randomly assigned to either the treatment run or the control run for their first 20 mile run to avoid order effect. The second run was completed three to four weeks after completing the first run. Test participants were asked to refrain from training 48 hours prior to their 20 mile run. Procedures for this study were based on a previous study conducted in the human performance lab at CSU (23).

Immediately before each participant started their 20 mile run, baseline data was collected. Nude weights were taken immediately before and after the run using a physician's beam scale, and height using a stadiometer. CRP blood samples were

obtained via venipuncture using SST BD Vacutainer serum collection tubes (3.5 mL).

Blood samples sat for 30 minutes to coagulate then were spun down using a centrifuge at 2900 RPM for 10 minutes.

Once the baseline measurements were acquired, the run started on a premeasured course. The test subjects completed a 20 mile run at an intensity of 70% or higher of their VO_2 max. Each runner wore a Polar heart rate monitor to monitor the running intensity (heart rate corresponding to 70% VO_2 max obtained during the max test). Subjects were provided water, or their preferred liquid, to drink during competition or training, according to each participant's specific hydration routine to prevent dehydration. Fluid intake was monitored and recorded for each runner. Immediately after the 20 mile run, another venous blood sample was taken from the antecubital vein for CRP. Perceived pain was assessed using the Management of Cancer Pain Scale Test (31, Appendix E); body weight was again obtained to determine fluid loss from dehydration and to calculate sweat rate. Subjects who completed the treatment run received treatment using the NT recovery system immediately following blood sampling.

All treatment subjects received the same treatment, one hour of NT at an intensity setting of 10 (90mmHg for cell 1 and cell 5 and 100 mmHg for cells 2-4) with a compression duration of 30 seconds. The NT recovery system is a boot which is divided into five sections and in each of these five sections there are inflatable bladders or cells. Cell one is located on the foot and each cell continues up the leg to the top of the thigh where cell five is located. Each bladder or cell inflates or compresses the limb for 30 seconds. NT recommends that for athletes, the intensity setting be at 10 (intensity settings range from 1-10) to ensure the best results for recovery. The amount of pressure exerted on the limb at an intensity setting of 10 in each of the cells is as follows: cell one and cell

five - 90mmHg; cells two, three, and four - 100mmHg. This inflation is synchronized to mimic a massaging action and to increase blood flow. Each leg was treated simultaneously.

For five consecutive days after the run, blood samples were obtained for CRP. Perceived pain was recorded and subjects used the NT device for five consecutive days. Subjects who did not receive treatment after the 20 mile run will undergo the same protocol except for the NT treatment, and were able to leave the lab after CRP and pain were measured.

The second 20 mile run was held approximately three to four weeks after the first 20 mile run to ensure complete recovery. Subjects who were previously in the treatment trial participated in the control trial, while those who were previously in the control trial participated in the treatment trial. Procedures for the second 20 mile run were exactly the same as those in the first 20 mile run.

Data Analysis

Descriptive statistics were acquired on all measures. A repeated measures ANOVA was used to assess recovery within a treatment group, whereas a two-way ANOVA was used to assess treatment differences due to NT recovery treatment vs. control on CRP and subjective pain across the five days. SPSS (version 18) was used for all analyses with 0.05 used as the level of significance. Paired sample t-tests were used to specify differences across the five days.

CHAPTER IV

RESULTS & DISCUSSION

Results

Ten subjects, five males and five females participated in the study to determine the effectiveness of the NT compression system for recovery after a 20 mile run. As expected, the males were taller, heavier, and had a higher VO₂ max than the female runners (Table 1).

Table 1. Characteristics of the subjects.

Characteristics	Males (N=5) Mean ± SD	Females (N=5) Mean ± SD	Total Group (N=10) Mean ± SD
Age (years)	41.2 ± 10.6	36.2 ± 12.4	38.7 ± 11.2
Height (cm)	176.7 ± 4.5	164.6 ± 5.2	170.7 ± 7.8
Weight (kg)	74.1 ± 4.5	60.5 ± 5.2	67.3 ± 8.5
VO ₂ Max (ml·min·kg)	54.1 ± 4.5	48.6 ± 6.0	51.4 ± 5.8

Males were found to be significantly taller and weigh significantly more than the female runners. No significant difference was found in age or VO₂ max between genders (Table 2).

Table 2. Gender comparison of age, height, weight, and VO₂ max of the subjects.

Condition (N=10)	Mean ± SD	P - value
Male vs. Female Age	41.2 ± 10.6 vs. 36.2 ± 12.4	.608
Male vs. Female Height	176.7 ± 4.5 vs. 164.6 ± 5.2	.046*
Male vs. Female Weight	74.1 ± 4.5 vs. 60.5 ± 5.2	.013*
Male vs. Female VO ₂ Max	54.1 ± 4.5 vs. 48.6 ± 6.0	.254

* *Indicates significance*

There was no significant difference in mean weight loss, fluid intake, sweat rate, heart rate, percentage of maximum heart rate, or percentage of maximum VO₂ max between runs (Table 3). There was, however, a significant difference (P = .038) in running time when comparing the control run against the treatment run. This was likely due to differences in weather conditions.

Table 3. Comparison between the control run and the NT treatment run.

Condition (N=10)	Control (Mean ± SD)	Treatment (Mean ± SD)	P - value
Weight Loss (kg)	1.17 ± .74	1.32 ± .72	.370
Fluid Intake (ml)	1087.3 ± 505.7	1307.8 ± 728.2	.308
Sweat Rate (L/hr)	.37 ± .28	.40 ± .23	.649
Heart Rate (bpm)	143.9 ± 10.8	147.8 ± 12.8	.213
% Max Heart Rate (bpm)	79.8 ± 6.5	81.8 ± 5.6	.222
% VO ₂ Max (ml/min/kg)**	72.3 ± 9.0	69.8 ± 10.9	.082
Running Time (min)	196.2 ± 28.7	204.8 ± 31.1	.038*

* *Indicates significance*

** *Estimated from American College of Sports Medicine (ACSM) formula*

To determine if the first run had an effect on the second run, a repeated measures ANOVA was used comparing all of the first runs with all of the second runs. The results

indicated no significant difference ($P > 0.05$) in any variables except for in one of the pain ratings. When comparing the pain experienced immediately following the first run versus second run, runners indicated they experienced more pain following the second run (4.9 on VAS) compared to the first run (3.7 on VAS). This slight increase in pain on the second run occurred regardless of whether the run was a treatment run or a control run.

CRP

No significant difference between the control run and the treatment run was found for CRP (Table 4). The subjects recovered by day four while using the NT device whereas the control had still not recovered by day five. However, this difference was not significant.

Table 4. Comparing baseline CRP values to post run CRP values.

Condition (N=10)	Mean \pm SD	P - value	Condition (N=10)	Mean \pm SD	P - value
Control Post Run	2.90 \pm 0.00	----	Treatment Post Run	2.90 \pm 0.00	----
Control Day 1	7.06 \pm 3.39	.004*	Treatment Day 1	7.95 \pm 4.08	.004*
Control Day 2	5.92 \pm 4.20	.049*	Treatment Day 2	4.85 \pm 1.95	.012*
Control Day 3	4.80 \pm 4.90	.251	Treatment Day 3	3.19 \pm 0.72	.234
Control Day 4	3.68 \pm 2.33	.317	Treatment Day 4	2.94 \pm 0.13	.343
Control Day 5	3.17 \pm 0.85	.343	Treatment Day 5	2.90 \pm 0.00	----

Normal = 2.9 mg/L

* *Indicates significant difference from baseline*

The results indicated no significant difference in CRP levels between the control run and treatment run. However, the subjects recovered one day earlier when using the NT device compared to the control. Statistically this may not be significant, but a full day recovery for a professional athlete is important (Figure 2).

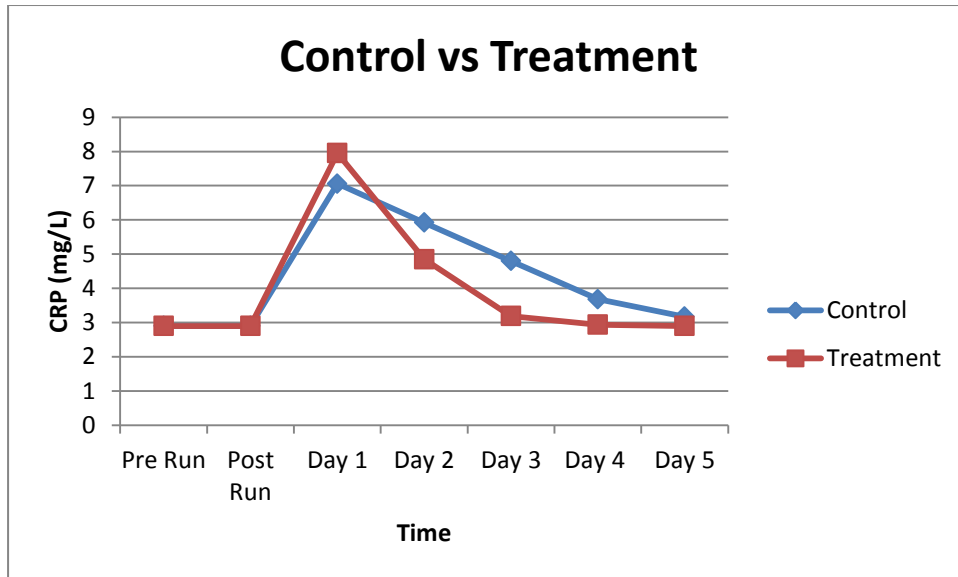


Figure 2. Control Run vs Treatment Run.

CRP Gender Comparisons

When the males used the NT device, there were significant increases in CRP from baseline on days one and two. The results indicated the males had recovered by day three, compared to the control, who had still not recovered by day five (Table 5). However, this difference was not significant.

Table 5. Comparing male baseline CRP values to each recovery day.

Condition (N=5)	Mean ± SD	P - value	Condition (N=5)	Mean ± SD	P -value
Control Post Run	2.90 ± 0.00	----	Treatment Post Run	2.90 ± 0.00	----
Control Day 1	7.00 ± 3.72	.069	Treatment Day 1	7.76 ± 3.53	.037*
Control Day 2	6.98 ± 5.60	.178	Treatment Day 2	4.42 ± 1.11	.038*
Control Day 3	6.32 ± 6.90	.329	Treatment Day 3	2.94 ± 0.09	.374
Control Day 4	4.38 ± 3.31	.374	Treatment Day 4	2.90 ± 0.00	----
Control Day 5	3.44 ± 1.21	.374	Treatment Day 5	2.90 ± 0.00	----

Normal = 2.9 mg/L

* Indicates significant difference from baseline

No significant difference was determined from baseline in either the control run or the treatment run among the female runners. Additionally, the females recovered in the same amount of time (by day four) regardless if they were using the NT device (Table 6).

Table 6. Comparing female baseline CRP values to each recovery day.

Condition (N=5)	Mean ± SD	P - value	Condition (N=5)	Mean ± SD	P -value
Control Post Run	2.90 ± 0.00	----	Treatment Post Run	2.90 ± 0.00	----
Control Day 1	7.12 ± 3.48	.053	Treatment Day 1	8.14 ± 4.99	.078
Control Day 2	4.86 ± 2.37	.138	Treatment Day 2	5.28 ± 2.62	.112
Control Day 3	3.28 ± 0.85	.374	Treatment Day 3	3.44 ± 1.00	.293
Control Day 4	2.98 ± 0.18	.374	Treatment Day 4	2.98 ± 0.18	.374
Control Day 5	2.90 ± 0.00	----	Treatment Day 5	2.90 ± 0.00	----

Normal = 2.9 mg/L

* Indicates significant difference from baseline

Pain

A significant difference from the baseline pain rating occurred from the time of the post run until day three for both the control run and treatment run. During the treatment run trial however, the test subjects had no pain on day five while using the NT device compared to the control in which the subjects still had some pain on day five (Table 7).

Table 7. Comparing baseline pain ratings to post run pain ratings.

Condition (N=10)	Mean ± SD	P - value	Condition (N=10)	Mean ± SD	P -value
Control Post Run	4.10 ± 2.42	.000*	Treatment Post Run	4.70 ± 1.42	.000*
Control Day 1	2.90 ± 1.51	.000*	Treatment Day 1	3.00 ± 1.76	.000*
Control Day 2	2.35 ± 1.38	.000*	Treatment Day 2	2.15 ± 1.86	.005*
Control Day 3	0.95 ± 1.26	.041*	Treatment Day 3	1.00 ± 1.05	.015*
Control Day 4	0.40 ± 0.84	.168	Treatment Day 4	0.20 ± 0.48	.223
Control Day 5	0.10 ± 0.32	.343	Treatment Day 5	0.00 ± 0.00	----

Scale 0 (no pain) - 10 (worst pain possible)

* Indicates significant difference from baseline (no pain)

Pain Gender Comparisons

In males, the pain ratings during the control trial were significantly elevated from baseline until day three while the treatment trial ratings were only significantly elevated until day one. This indicates that the males had significant pain through day two on the control run compared to significant pain only through day one on the treatment run when using the NT device (Table 8).

Table 8. Comparing male baseline pain ratings to post run pain ratings.

Condition (N=5)	Mean ± SD	P - value	Condition (N=5)	Mean ± SD	P -value
Control Post Run	3.60 ± 2.88	.049*	Treatment Post Run	4.40 ± 1.82	.006*
Control Day 1	3.30 ± 1.92	.019*	Treatment Day 1	2.60 ± 1.95	.041*
Control Day 2	2.50 ± 2.00	.049*	Treatment Day 2	2.10 ± 2.25	.105
Control Day 3	1.10 ± 1.67	.216	Treatment Day 3	1.00 ± 1.41	.189
Control Day 4	0.40 ± 0.89	.374	Treatment Day 4	0.30 ± 0.67	.374
Control Day 5	0.00 ± 0.00	----	Treatment Day 5	0.00 ± 0.00	----

Scale 0 (no pain) - 10 (worst pain possible)

* *Indicates significant difference from baseline (no pain)*

The female pain ratings indicated significantly elevated pain ratings from baseline in the treatment trial until day three compared to the control trial where pain ratings were only significantly elevated from baseline until day two. This indicates that the females had higher amounts of pain while using the NT device (Table 9).

Table 9. Comparing female baseline pain ratings to post run pain ratings.

Condition (N=5)	Mean ± SD	P - value	Condition (N=5)	Mean ± SD	P -value
Control Post Run	4.60 ± 2.07	.008*	Treatment Post Run	5.00 ± 1.00	.000*
Control Day 1	2.50 ± 1.00	.005*	Treatment Day 1	3.40 ± 1.67	.010*
Control Day 2	2.20 ± 0.45	.000*	Treatment Day 2	2.20 ± 1.64	.040*
Control Day 3	0.80 ± 0.84	.099	Treatment Day 3	1.00 ± 0.71	.034*
Control Day 4	0.40 ± 0.89	.374	Treatment Day 4	0.10 ± 0.22	.374
Control Day 5	0.20 ± 0.45	.374	Treatment Day 5	0.00 ± 0.00	----

Scale 0 (no pain) - 10 (worst pain possible)

* *Indicates significant difference from baseline (no pain)*

Temperature Conditions During the 20 Mile Runs

The temperature of the first run was compared to the temperature of the second run to determine if a difference occurred. There was a significant difference ($P = .030$) between the temperatures of the first run ($6.4\text{ }^{\circ}\text{C}$) compared to the second run ($2.8\text{ }^{\circ}\text{C}$). The temperature of the control run ($4.8\text{ }^{\circ}\text{C}$) was compared to the temperature of the treatment run ($4.4\text{ }^{\circ}\text{C}$) to determine if a difference occurred. No significant difference ($P = .855$) was found in temperature between the control and treatment runs.

Discussion

C- reactive protein levels peaked on Day 1 for both the control and treatment runs and then gradually decreased until they returned to baseline by day 4 (NT) and day 5 (control). This is consistent with the research performed by Fallon (32) who stated that the acute phase response has been suggested to relate to skeletal muscle damage when associated with exercise. The acute phase response is a response of certain proteins in the blood plasma whose concentrations either increase or decrease due to inflammation. C- reactive protein is reported to be the most plentiful of the acute phase proteins and has been reported to be elevated following exercise, especially when muscle damage has

occurred (32, 33). C- reactive protein remained higher than baseline until Day 5 for the control run but remained higher than baseline only until day 4 for the treatment run.

These results are consistent with the literature (32, 33).

A study performed by Miliadis et al. (5) found no significant differences in the inflammatory markers CRP, complement C3, and fibrinogen following eccentric exercise. Simpson et al. (34) also found no significant difference in CRP even after a hill race although muscle damage was present. This suggests that inflammation may not correspond to muscle damage. The results of the present study support this.

The results in the present study conflict with those found in a previous study by Cantanese (23) in which the same experimental protocol was used, except external counter pulsation (ECP) was the treatment modality. In that study, ECP helped the runners recover two days faster than in the control trial which was statistically significant. Additionally, only the females in the ECP study experienced a significant difference in CRP whereas in the current study, it was the males who responded to the treatment. The major differences between treating the runners with ECP compared to NT were the cuff pressure and compression duration. The maximum ECP cuff pressure was 6 psi and the maximum NT cuff pressure was 2.1 psi. The ECP compression duration only lasted during diastole (about a second) whereas the NT compression lasted about one minute. The difference in cuff pressure and compression may have contributed to why we did not obtain the same results with the NT device as Cantanese (23) did with the ECP study.

When comparing the first run against the second run for both males and females, CRP peaked on day one for both runs but the peak on the second run was 1.4 mg/L less than

the peak on the first run. This is also consistent among gender with the second run 1.7 mg/L less CRP among males and 1.0 mg/L less CRP among females compared to the first run, suggesting rapid adaptation. In a study by Clarkson et al. (35), less muscle damage was produced when the same exercise was performed several months later due to adaptation. Rapid adaptation could potentially explain smaller responses seen after downhill running due to preadaptation. The exact etiology of this adaptation is not known. In the study by Clarkson et al. (35), one group exercised six weeks apart and after the second bout of exercise DOMS, and flexed arm angle responses were found to be significantly smaller. This may explain the blunted peak CRP response on the second run for both males and females, and suggests a longer span of time needed between the treatment and control runs (six weeks or longer) to mitigate long-term adaptations.

Concerning gender, the males recovered about two days faster using the NT device compared to control but the females did not recover faster using the NT device compared to control. When comparing the male CRP values to baseline, the males had a significant difference from baseline on day one and two when using NT compared to the control (no significant difference from baseline) suggesting NT made the inflammation worse. The CRP values compared to baseline among females revealed no significant difference from baseline suggesting there was no benefit in using NT. Regarding gender differences, there has been some debate as to the existence of sex differences in exercise-induced muscle damage and repair based on an assumption that estrogen levels exert a response that may enhance the recovery of skeletal muscle to damaging exercise. These assumptions are based on estrogen's potential ability to stabilize the cells of skeletal muscle during exercise due to estrogen's antioxidant properties (36). Studies that assess DOMS or pain

usually do not find sex differences (37, 38, 39) and those that do site differences between men and women are inconsistent (36). Another study demonstrated that there were no differences in sex when comparing untrained men and women in muscle fatigue or other changes in muscle function parameters during eccentric exercise (40). The results from the present study, however, indicate a difference in gender response to the NT recovery device adding to the inconsistency of whether gender differences exist.

Previous physical conditioning is the most effective technique to speed up the recovery process after strenuous exercise (41). The majority of subjects involved in the study were trained marathoners, ultramarathoners, triathletes, and collegiate athletes. Two subjects (one male and one female) did not elicit a CRP response following the protocol on either the control run or treatment run. The level of training (perhaps due to interval training) and running efficiency of the subjects may possibly have contributed to the lack of an inflammatory response, limiting the effectiveness of the NT recovery device, which could influence the results of the study.

Pain

When comparing pain ratings to baseline, the results indicated no significant difference between NT and control. The male pain ratings were significant from baseline until day one in the treatment trial compared to the control which was significant from baseline until day two, indicating NT helped ease the pain one day sooner for the males. The females exhibited the opposite. The female pain ratings were significant from baseline until day two in the control trial compared to the treatment which was significant from baseline until day three, indicating the females had more pain while using NT.

Perceived pain in all 10 subjects while using the NT device was lower compared to the control. This is consistent with a study by Hilbert et al. (42) in which they tested the physiological and psychological effects of massage on DOMS. They concluded massage may provide psychological benefits as it reduces the intensity of soreness which is commonly associated with DOMS (42). Many subjects, both male and female, stated that while using the NT device they kept waiting for the pain associated with DOMS to come a day or two after the run and they stated it never came. These statements are subjective and based on self-reported data.

When comparing the first run to the second run for pain there was a significant difference between the two runs. The subjects in the second run experienced less pain compared to the first run. However, when comparing the first run to the second run in terms of gender, no significant difference was found. For males, by day two the second run recorded lower pain levels compared to the first run while the females recorded a higher level of pain in the second run compared to the first run.

Temperature

Temperature and weather may have played a role in the present study. Testing started on October 21, 2013 and ended January 18, 2014. Subjects started the study in ideal running conditions but as the study progressed subjects ran in cooler conditions. A significant difference between the temperatures of the first run compared to the second run was found. This may suggest why the second run was significantly slower compared to the first in Table 3. No significant difference was found in temperature between the control and treatment runs.

CHAPTER V

SUMMARY & CONCLUSION

Ten trained runners ran two 20 mile runs and each subject participated in the control and treatment runs. Trails were randomly counterbalanced to provide a safeguard against order effect. Muscle soreness was induced on both runs to elicit a CRP response and subjects were asked to rate their pain immediately following the run and each day after for five days.

The subjects recovered by day four while using the NT device whereas the control had still not recovered by day five. However, this difference was not significant. The results indicated the males had recovered by day three, compared to the control, who had still not recovered by day five. However, this difference was not significant. No significant difference was determined from baseline in either the control run or the treatment run among the female runners and the females recovered in the same amount of time (by day four) regardless if they were using the NT device.

During the treatment run trial, the test subjects had no pain on day five while using the NT device compared to the control in which the subjects still had some pain on day five. The males had significant pain through day two on the control run compared to

significant pain only through day one on the treatment run when using the NT device.

The female pain ratings indicated significantly elevated pain ratings from baseline in the treatment trial until day three compared to the control trial where pain ratings were only significantly elevated from baseline until day two, indicating that the females had higher amounts of pain while using the NT device.

In conclusion, the results indicated statistically there was no significant difference in CRP or pain levels between the control run and treatment run therefore, my hypothesis was rejected. However upon closer inspection, the data suggests the test subjects recovered one day earlier when using the NT device compared to the control. Statistically this may not be significant, but a full day recovery for a professional athlete is very important. Gender differences were found in response to the control and treatment run. It appears based on the present study that males respond better to being treated with NT compared to the females in reducing inflammation following a 20 mile run.

Application

There are many devices used to help athletes recover faster. Unfortunately, many of them do not work as advertised, and many have not undergone sufficient scientific research to support or reject their claims. Coaches, athletic trainers, and athletes should seek scientific support of therapeutic interventions which claim to help reduce the effects of muscle damage, DOMS, and speed of recovery from exercise and athletic endeavors.

Limitations

Limitations of the study were identified as follows:

1. Running weather conditions varied. Warmer and cooler temperatures can influence cardiac drift and level of hydration which can affect heart rate, exercise performance, and DOMS (thereby affecting the release of biochemical markers of muscle damage).
2. The sample was one of convenience and was small (N=10), requiring caution when making extrapolations from the results to larger populations.
3. Variables which could affect enzyme activity such as training level, muscle fiber composition, past injuries, and the degree of hormonal activity, were not controlled.
4. In the study performed by Cantanese (30), sensitivity of CRP was to 1 mg/L. We outsourced the measurement of CRP to a local hospital where CRP was only sensitive to 2.9 mg/L. Clinically, any measurement of CRP of 2.9 mg/L or less is acceptable. However, lower CRP values could have a significant impact on the outcome of the study.

Future Research Recommendations

1. Future research is needed with a larger sample size.
2. Future research examining a different population such as sedentary, untrained, or elderly populations is needed to determine if the NT device would function better with untrained individuals.

3. Future research including other markers of muscle damage and inflammation such as CK, LDH, and Mb is needed to obtain a clearer picture of the mechanism and to determine if there is a relationship between the biochemical markers.
4. Future research is needed that focuses on other types of exercise, besides long distance running, which may also cause muscle damage.

BIBLIOGRAPHY

1. Braun W A, Dutto D J. The effects of a single bout of downhill running and ensuing delayed onset of muscle soreness on running economy performed 48 h later. *European Journal of Applied Physiology*. 2003;90(1-2):29-34.
2. Armstrong, R.B. Mechanisms of exercise-induced delayed onset muscular soreness: A brief review. *Medicine & Science in Sports & Exercise*. 1984;16:529-538.
3. NormaTec (Normal Healing Through Technology). Company History. NormaTec. <http://www.normatecusa.com/about/history.html>. Published 2007. Updated 2013. Accessed April 19, 2013.
4. NormaTec. Science Tab. NormaTec. <http://www.normatecrecovery.com/science.shtml>. Published 2007. Updated 2013. Accessed April 19, 2013.
5. Milias G, Nomikos T, Fragopoulou, Athanasopoulos S, Antonopoulou S. Effects of eccentric exercise-induced muscle injury on blood levels of platelet activating factor (PAF) and other inflammatory markers. *European Journal of Applied Physiology*. 2005;95(5-6):504-513. doi:10.1007/s00421-005-0031-6.
6. Nosaka K, Newton M, Sacco P. Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage. *Scandinavian Journal of Medicine & Science in Sports*. 2002;12(6):337-346.
7. Lee J, Clarkson PM. Plasma creatine kinase activity and glutathione after eccentric exercise. *Medicine & Science in Sports & Exercise*. 2003;35:930-936.
8. Chleboun GS, Howell, JN, Conatser RR, Giesey JJ. Relationship between muscle swelling and stiffness after eccentric exercise. *Medicine & Science in Sports & Exercise*. 1998;30:529-535.
9. Nosaka N, Clarkson PM. Changes in indicators of inflammation after eccentric exercise of the elbow flexors. *Medicine & Science in Sports & Exercise*. 1996;28:953-961.
10. Hosoi T, Kobayashi Y, Loeppky JA, Takeuchi T, Yoshizaki H. Effects of a marathon run on serum lipoproteins, creatine kinase, and lactate dehydrogenase in recreational runners. *Research Quarterly for Exercise and Sport*. 2005;76(4):450-455.

11. Schwane JA, Johnson SR, Vandenaeker CB, Armstrong RB. Delayed-onset muscular soreness and plasma CPK and LDH activities after downhill running. *Medicine & Science in Sports & Exercise*. 1983;15:51-56.
12. Zainuddin Z, Newton M, Sacco P, Nosaka K. Effects of massage on delayed-onset muscle soreness, swelling, and recovery of muscle function. *Journal of Athletic Training*. 2005;40(3):174–180.
13. Moraska A. Therapist education impacts the massage effect on postrace muscle recovery. *Medicine & Science in Sports & Exercise*. 2007;39:34-37.
14. Wiltshire VE, Poitras V, Pak M, Hong T, Rayner J, Tschakovsky ME. Massage impairs postexercise muscle blood flow and “lactic acid” removal. *Medicine & Science in Sports & Exercise*. 2010;42:1062-1071.
15. Millar A, Sims J, Wright H, Oakley B, Show R, McMullen A. The effects of aerobic exercise on DOMS measures. *Medicine & Science in Sports & Exercise*. 2003;35:S318.
16. Mekjavic I, Exner J, Tesch p, Eiken, O. Hyperbaric oxygen therapy does not affect recovery from delayed onset muscle soreness. *Medicine & Science in Sports & Exercise*. 2000;32:558-563.
17. Hinds T, McEwan I, Perkes J, Dawson E, Ball D, George K. Effects of massage on limb and skin blood flow after quadriceps exercise. *Medicine & Science in Sports & Exercise*. 2004;36:1308-1313.
18. Pointon M, Duffield R. Cold water immersion recovery after simulated collision sport exercise. *Medicine & Science in Sports & Exercise*. 2012;44:206-216.
19. Haas C, Butterfield T, Abshire S, Zhao Y, Zhang X, Jarjoura D, Best T. Massage timing affects postexercise muscle recovery and inflammation in a rabbit model. *Medicine & Science in Sports & Exercise*. 2013;45:1105-1112.
20. Mawhinney C, Jones H, Joo CH, Low D, Green D, Gregson W. Influence of cold-water immersion on limb and cutaneous blood flow after exercise. *Medicine & Science in Sports & Exercise*. 2013;45:2277-2285.
21. Wheeler A, Jacobson B. Effect of whole-body vibration on delayed onset muscular soreness, flexibility, and power. *Journal of Strength and Conditioning Research*. 2013;27:2527-2532.
22. Weerapong P, Hume P, Kolt. The mechanisms of massage and effects on performance, muscle recovery and injury prevention. *Sports Medicine*. 2005;35(3):235-56.

23. Konstantinos D, Knaggs A. Duration and amplitude decay of acute arterial leg inflow enhancement with intermittent pneumatic leg compression: An insight into the implicated physiologic mechanisms. *Journal of Vascular Surgery* 2005;42(4):717-725. Doi:10.1016/j.jvs.2005.06.004.
24. Moseley A, Carati C, Piller N. A systematic review of common conservative therapies for arm lymphoedema secondary to breast cancer treatment. *Annals of Oncology*. 2007;18:639-646.
25. Partsch, H. Intermittent pneumatic compression in immobile patients. *International Wound Journal* 2008;5(3):389-397.
26. Avery K, Solomon A, Weber R, Jacobs L. Treatment of congenital lymphoedema with sequential intermittent pneumatic compression therapy. *The Foot*. 2000;10:210-215.
27. Richmand D, O'Donnell T, Zelikovski A. Sequential pneumatic compression for lymphedema. A controlled trial. *Archives of Surgery*. 1985;120:1116-1119.
28. Labropoulos N, Leon L, Bhatti A, et al. Hemodynamic effects of intermittent pneumatic compression in patients with critical limb ischemia. *Journal of Vascular Surgery*. 2005;42:710-716.
29. American College of Sports Medicine and American Heart Association. Recommendations for cardiovascular screening, staffing, and emergency policies at health/fitness facilities. *Medicine & Science in Sports & Exercise*. 1998;30:1009-1018.
30. Cantanese C. *Effect of external counterpulsation (ECP) on delayed onset muscle soreness (DOMS) in long distance runners*. Unpublished Master's Thesis. Cleveland, OH: Cleveland State University; 2007.
31. Cancer Council. Cancer Pain Assessment Scales. Cancer Council. http://www.cancercouncil.com.au/wp-content/uploads/2010/09/overcoming_cancer_pain-16.gif. Updated 2013. Accessed April 19, 2013.
32. Fallon K. The acute phase response and exercise: The ultramarathon as prototype exercise. *Clinical Journal of Sport Medicine*. 2001;11:38-43.
33. Gleeson M, Almey J, Brooks S, Cave R, Lewis A, and Griffiths H. Haematological and acute-phase response associated with delayed-onset muscle

soreness in humans. *European Journal of Applied Physiology and Occupational Physiology*. 1995;71:137-142. doi: 10.1007/BF00854970.

34. Simpson R, Wilson M, Black J, Ross J, Whyte G, Guy K, Florida-James G. Immune alterations, lipid peroxidation, and muscle damage following a hill race. *Canadian Journal of Applied Physiology*. 2005;30:196-211.
35. Clarkson P, Nosaka K, Braun B. Muscle function after exercise-induced muscle damage and rapid adaptation. *Medicine & Science in Sports & Exercise*. 1992;24:512-520.
36. Hubal M, Clarkson P. Counterpoint: Estrogen and sex do not significantly influence post-exercise indexes of muscle damage, inflammation, and repair. *Journal of Applied Physiology*. 2009;109:1012-1014.
37. Dannecker E, Hausenblas H, Kaminski T, Robinson M. Sex differences in delayed onset muscle pain. *Clinical Journal of Pain*. 2005;21:120-126.
38. Rinard J, Clarkson P, Smith L, Grossman M. Response of males and females to high-force eccentric exercise. *Journal of Sport Sciences*. 2000;18:229-236.
39. Sewright K, Hubal M, Kearns A, Holbrook M, Clarkson P. Sex differences in response to maximal eccentric exercise. *Medicine & Science in Sports & Exercise*. 2008;40:242-251.
40. Hubal M, Rubinstein S, Clarkson P. Muscle function in men and women during maximal eccentric exercise. *Journal of Strength and Conditioning Research*. 2008; 22:1332-1338.
41. Clarkson, Priscilla. Too much too soon: The aftermath of overexertion. *Swimming World & Junior Swimmer*. 1996; 37(10):6.
42. Hilbert J, Sforzo G, Swenson T. The effects of massage on delayed onset muscle soreness. *Medicine & Science in Sports & Exercise*. 2001;33:S123.

APPENDICES

APPENDIX A

FUNCTIONAL SCHEMATIC OF NORMATEC RECOVERY DEVICE

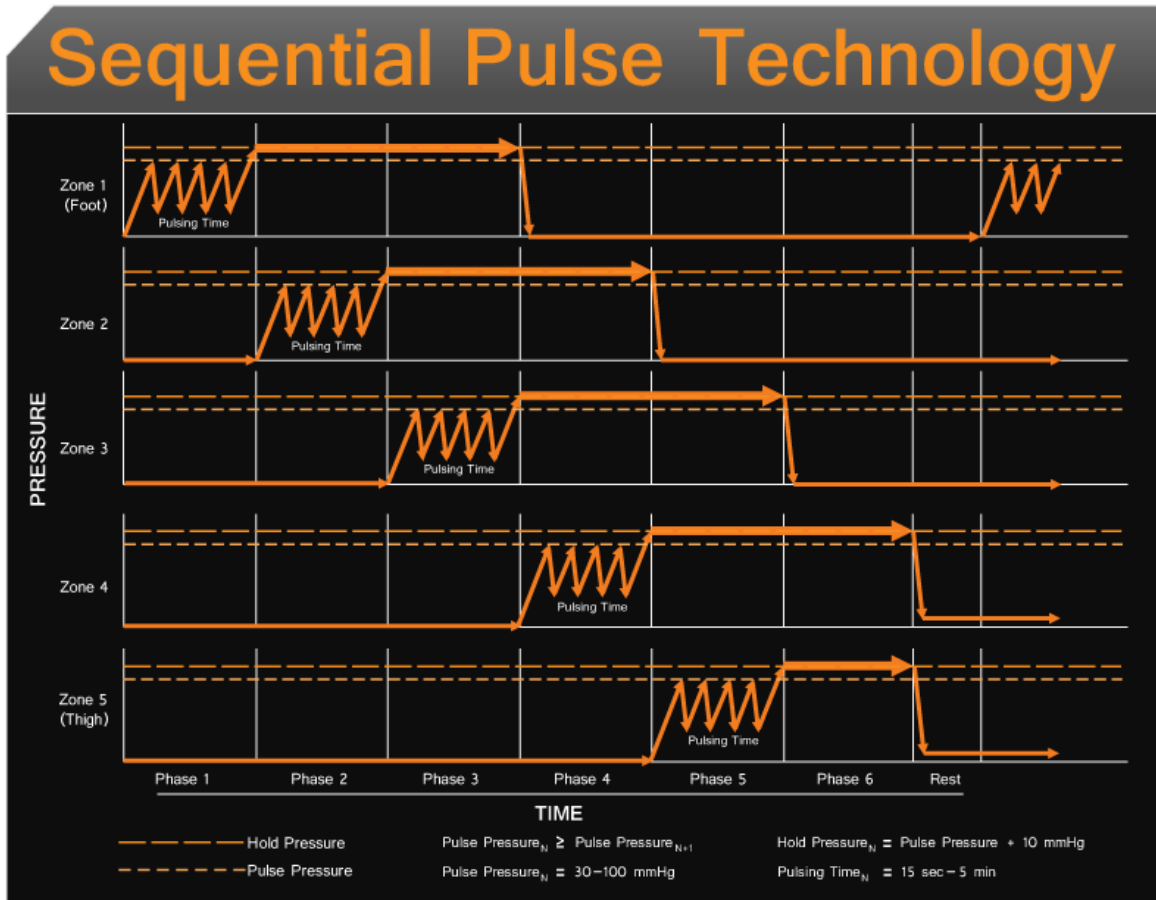


Figure 1

APPENDIX B

AHA/ACSM Health/Fitness Facility Pre-participation Screening Questionnaire

Assess 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**

Symptoms

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

Other health issues

- You have diabetes**
- You have or asthma other lung disease.**
- You have burning or cramping in your lower legs
when walking short distances.**
- You have musculoskeletal problems that limit your physical activity**
- You have concerns about the safety of exercise.**
- You take prescription medication(s)**
- You are pregnant**

If you marked any of these statements in this section, we require a physician's release, attached, in order to continue with the joining process.

Cardiovascular risk factors

- You are a man older than 45 years.**
- You are a woman older than 55 years, you
have had a hysterectomy, or you are postmenopausal.**
- You smoke, or quite within the previous 6 mo.**
- Your blood pressure is greater than 140/90.**
- You don't know your blood pressure.**
- You take blood pressure medication.**
- Your blood cholesterol level is >200 mg/dL.**
- You don't know your cholesterol level.**
- You have a close blood relative who had a heart attack before age 55 (father or brother) or age
65 (mother or sister).**
- You are physically inactive (i.e., you get less than 30 min. of physical activity on at least 3
days per week).**
- You are more than 20 pounds overweight**

If you marked two or more of the statements in this section you are required to obtain a physician's release, attached, in order to join the facility.

None of the above is true.

You should be able to exercise safely without consulting your physician or other healthcare provider in a self-guided program or almost any facility that meets your exercise program needs.

APPENDIX C



Cleveland State University

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College of Education and Human Services
Department of Health and Human Performance

INFORMED CONSENT FOR PARTICIPATION

Effects of Intermittent Pneumatic Compression on Delayed Onset Muscle Soreness in Long Distance Runners

Introduction

You have been asked to participate in a research study to be conducted in the Human Performance Laboratory at Cleveland State University. The purpose of this study is to measure the recovery process from prolonged distance running with the use of NormaTec Recovery system and its effect on reducing delayed onset muscle soreness (DOMS).

Previous studies have documented the delayed onset of muscle soreness is an effect of performing an exercise which places a great deal of strain on the skeletal muscles. The subsequent strain that is placed on the muscles leads to the release of biochemical markers into the blood. These biochemical markers indicate damage has been done to the muscle and can lead to muscle soreness. One of these biochemical markers is lactate dehydrogenase (LDH). Inflammation is the precursor to full recovery of damaged muscle fibers. Inflammation is indicated by another blood marker called C- reactive protein (CRP). C- reactive protein indicates the healing process is underway. NormaTec is a company which has developed a device used by athletes to help them recover faster. It utilizes intermittent pneumatic compression cuffs to inflate, hold, and deflate different areas of the leg aiding in blood flow, profusion, and return back to the heart (similar to massage).

Procedures

I understand that I will be asked to perform a maximal graded exercise test to establish run intensity. I understand a continuous measurement of oxygen consumption will be

recorded throughout the test. Oxygen consumption will be measured using the Cosmed K4 b2 portable oxygen analyzer. I understand that this test involves running at comfortable training pace with the treadmill elevating 3% every three minutes until I reach a maximal grade in which I cannot continue the run. I understand that this test takes approximately 10-15 minutes and is dependent on my conditioning. Values usually range for someone of my ability between 45 and 60 milliliters of oxygen per kilogram of body weight each minute. I know that I can voluntarily stop exercise if I experience any problems.

We ask that you do not do any hard training 48 hours prior to the initial testing. After the initial testing you will report to the Human Performance laboratory where baseline data will be obtained prior to your long run, this involves a venous blood sample, height, and weight. You will then be instructed to complete a 20 mile run at approximately 70 % of your maximal heart rate (moderate intensity). Blood samples will be drawn immediately after the run and each day after until baseline measures are again achieved.

You will complete the same run twice. After one of the runs you will be treated with NormaTec recovery system (60 minute duration) daily until blood markers return to normal. The second run will be the control where only the blood markers will be measured and no NormaTec treatment will be given. During the days after your long run we ask that you refrain from training while recovery is being evaluated.

Risks and Discomforts

I understand that during maximal exercise testing, there exists the possibility of certain changes occurring; these include abnormal blood pressure, fainting, disorders of the heart rhythm, and rare instances of heart attack, stroke or death (1:20,000 exercise tests). I understand the laboratory has emergency procedures in place and every effort will be made to minimize these risks. The laboratory is equipped with an AED and all lab personnel are trained in CPR and First Aid. Emergency procedures including calling EMS (x911) stating to the dispatcher:” We have a medical emergency in the Human Performance Laboratory PE Building- Room B60”. CPR/First aid will be administered until EMS arrives. Emergency procedures are posted throughout the laboratory. I also know that I can voluntarily stop exercise if I experience any problems

I understand other risks associated with this study include muscle soreness resulting from the 20 mile run. Discomfort will also be experienced from giving venous blood samples. The risk associated with the run would be the same experienced from your normal training regimen. Every effort will be made to minimize these risks. A registered nurse will perform all the blood samples drawn and a qualified technician will be responsible for administering the NormaTec treatment.

Benefits

The benefits of the study are significant because it will help further our understanding of therapeutic interventions to help reduce the effects of muscle damage, DOMS and speed of recovery from exercise and athletic endeavors. 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. The NormaTec treatment system can aid in decreasing recovery time so that training can continue sooner.

Confidentiality

To protect your privacy, your name will not be used in any document of the project. The information, however, may be used for a statistical or scientific purpose 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, and if I have any questions about the procedures I can contact Dr. Kenneth Sparks at (216) 687-4831 or Mr. Shane Draper at (440) 313-2909. I also understand that I will be compensated (\$200) for my time.

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.

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 D

Memorandum

Institutional Review Board

To: Kenneth Sparks
HPERD, PE 860

From: Craig M. Zullig
Director
Office of Sponsored Programs & Research

Date: August 26, 2013

Re: Results of IRB Review of your project number: #29876-SPA-HS
Co-Investigator: Shane Draper, Student
Title: Effects of Intermittent Pneumatic Compression on Delayed Onset Muscle Soreness (DOMS) in Long Distance Runners

The IRB has reviewed and approved your application for the above named project, under the category noted below. Approval for use of human subjects in this research is for a one-year period as noted below. If your study extends beyond this approval period, *you must contact this office to initiate an annual review of this research.*

By accepting this decision, you agree to notify the IRB of: (1) any additions to or changes in procedures for your study that modify the subjects' risk in any way; and (2) any events that affect that safety or well-being of subjects. Notify the IRB of any revisions to the protocol, including the addition of researchers, prior to implementation.

Thank you for your efforts to maintain compliance with the federal regulations for the protection of human subjects.

Approval Category:	Approval Date:	August 26, 2013
Expedited (7)	Expiration Date:	August 25, 2014

APPENDIX E

How bad is the pain?

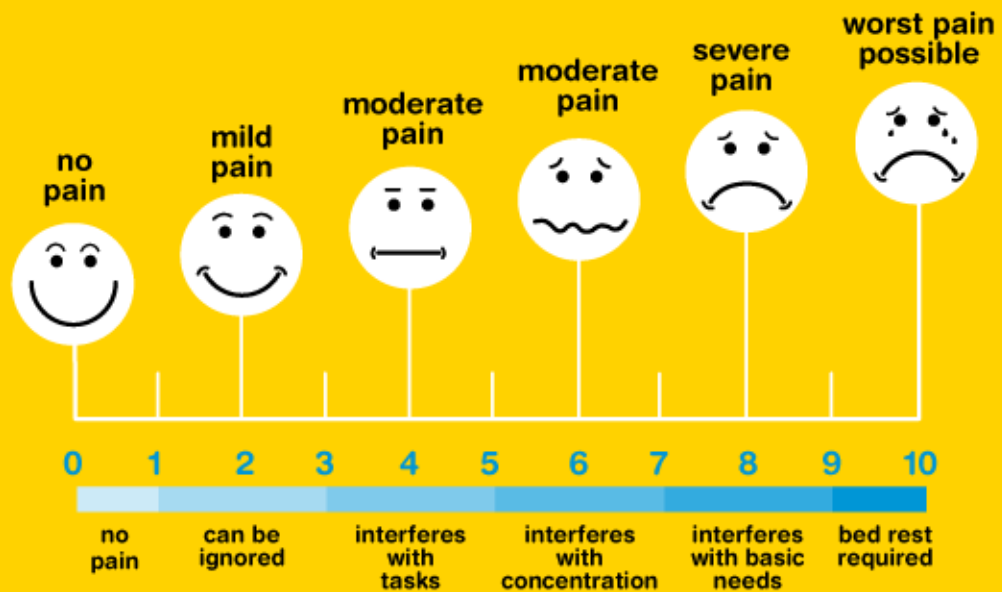
Some people find that rating their pain using a scale can help them describe their pain. There are different kinds of scales:

word scale – this rates the pain from none or mild through to moderate and severe

facial scale – this is the use of facial expressions to show how the pain makes you feel

number scale – this is from 1–10, with the higher the number, the worse the pain

activity tolerance scale – this has statements about how the pain affects your activities



APPENDIX F

NORMATEC TREATMENT SET-UP

