CHAPTER 6
DISCUSSION

The purpose of the study was to determine whether a specific Physiotherapy modality would be an effective intervention in decreasing or minimizing the negative impacts on performance and perceived soreness that are associated with Delayed Onset Muscle Soreness.

The Physiotherapy interventions used in the present study were Cryotherapy, Ultrasound, and Exercises. The specific exercise used in the present study was, mild full ROM elbow flexion and extension exercises, with only the gravitational pull on the hand and arm providing resistance. The hypothesis was that there would be differences in both perceived pain and Influence on biochemical markers means between the groups, experimental and control. Investigations using exercise as an intervention for DOMS did not find any reduction in muscle soreness and performance deficits (Hasson et al., 1989; Donnelly et al., 1992; Isabell et al., 1992; Weber et al., 1994).

In the present study, three different Physiotherapy modalities were compared to facilitate the recovery of DOMS, and the influence of three different physiotherapy interventions on biochemical markers were investigated.

This study had the following steps in the procedure: the pre-outcome measurements (baseline), the induction of delayed onset muscle soreness, randomization of the participants into one of the four groups group I-Cryotherapy, group II-Phonophoresis, group III-Exercises and group IV-Control, the post -test measurements of creatine kinase, lactate dehydrogenase and maximum isometric voluntary contractions after 24
hours, 48 hours, 72 hours and 96 hours, and the visual analog scale ratings for pain taken at 24 hours from baseline and at 96 hours. For the three Intervention groups, significant differences were found between post-test and baseline measures for each dependent variable. The performance of the control group was negatively affected by the soreness inducing exercise session with the presumptive diagnosis of DOMS. It appears that the soreness inducing exercise session used in the present study was sufficient to induce DOMS and affect muscular performance negatively. Also the data suggests that the exercise intervention and phonophoresis was successful in minimizing the pain and muscle functional deficits associated with DOMS. But the parameters of the biochemical markers in the data suggest that the exercise intervention (Group 3) in the experimental group was comparatively better than the other three groups in limiting the creatine kinase and lactate dehydrogenase enzymatic activity by 96 hours post exercise.

6.1 CREATINE KINASE COMPARISONS

In the Present study, before induction of Delayed Onset Muscle Soreness (DOMS), the baseline parameters of CK were within the same range in all the four groups. However after induction of DOMS, CK were started to rise in all the four groups, and immediately after intervention, there is variation of recovery with respect of CK in all the groups. In the present study, it is noted that CK values did not differ significantly in the four groups at baseline and at 48 hours. But where as significant differences were observed at 24 hours, after 72 hours and after 96 hours (Table 5.1). We found that electrotherapy modalities Cryotherapy and Phonophoresis do not have much influence on reducing the CK activity where as exercise has better influence on reducing CK activity. This might indicate that exercise protocol used in this study
reduces some of the pathological alterations following unaccustomed and strenuous eccentric exercise. Thus it is interesting that increases in plasma CK activity were significantly smaller for the Exercise group than the control and other two physiotherapy interventions.

The probable reason for the blunted CK response in the exercise group could be explained either by smaller CK efflux from the damaged muscle or increased clearance of CK from the circulation. It may be that exercise enhances the transport of CK from the damaged muscle to the circulation via the lymph fluid leading to accelerated CK clearance from the blood by increasing blood and lymph flow (Smith et al., 1994). It can also be assumed that exercise assists in flushing neutrophils and macrophages from the injured area, thus avoiding fiber necrosis and CK efflux. However, no concrete evidence to support these speculations is available till now.

Commonly accepted mechanisms of CK release are damage to muscle tissue and changes in myocyte membrane permeability. The pattern of CK response after eccentric exercise in the present study is likely be due to additional CK release from damaged muscle tissue (Byrnes et al., 1985). With regard to membrane permeability, there are various theories of ion-distribution change, enzyme deficiency, and ATP depletion (Wrogemann and Pena, 1976).

During exercise, the muscle repeatedly contracts and uses energy substrates. When the exercise intensity is within the normal range of metabolism, the muscle tissue is exercised without marked changes in membrane permeability. However, when the exercise intensity exceeds this permissible range, the membrane permeability temporarily changes, resulting in CK release from the active muscle. The boundary of this permissible range is its break point. In untrained individuals, relatively greater
muscle tension was required than in well trained individuals to complete the same exercise. In addition, mobilization of free fatty acids, which acts as an energy substrate during exercise, tended to be lower in the untrained individuals than in the well trained individuals. This suggests that the main active muscles of untrained individuals might not be supplied with enough energy substrate to endure repeated tension during intense exercise. Therefore, the relative exercise intensity for the muscle seemed to rise according to developing muscle fatigue with continued exercise. It was estimated that metabolic enhancement of the glycolytic pathway induces the production of lactic acid. Thus untrained individuals like recreational athletes seem to have exercised beyond this break point, resulting in an increase in serum CK activity (Totsuka et al., 2002).

An increased concentration of CK is assumed to indicate sarcolemma disruption (Hortobagyi et al., 1989), but because CK is a very large molecule (80,000 Da) CK should not be able to enter the blood directly due to the small pore radius in the capillaries 4-7 nm, but is thought to be released into the blood stream via the lymphatics (Vander, 1991). This explains the delayed CK concentration peak following eccentric exercise. However, Paaske and Sejrsen in 1989 found a larger pore radius in the capillaries being 145-160 Å rather than 40-70 Å, which would allow CK to enter the blood stream directly from the interstitial space. Since it could be argued that the CK release is a one-time event (during and right after the eccentric exercise), stretching might help in removing CK from the interstitial space close to the damaged muscle fibers. Anderson. (2005) conducted a review study and it was concluded that the results do not support the role of pre-exercise or post exercise stretching as an intervention addressing post exercise muscle soreness. Another systematic review analysis performed by Herbert. (2007) also concluded, the evidence
derived from laboratory-based studies of stretching indicated that muscle stretching does not reduce delayed-onset muscle soreness in young healthy adults.

Exercise programs that include eccentric muscle contractions can result in significant serum CK elevations. One study followed 203 participants to evaluate the magnitude of CK elevation and the effect on renal function produced by exercise (Rogers, 1985). After performing 50 maximal eccentric elbow flexor contractions, 55% of participants had CK elevations >2000 IU/L at 4 days after exercise; 25% had CK elevations >10,000 IU/L; 13% had levels >20,000 IU/L. Another study found significant increases in CK (approximate mean of 15,000 IU/L) after repetitive eccentric elbow flexor contractions in college-age males (Margaritis, 1999).

Eccentric weight lifting and similar activities, like downhill running, may result in an increase in serum CK levels of 10 to 20 times normal, whereas other nonweight-bearing exercises and exercise involving no or minimal eccentric contractions, such as swimming and cycling, cause only nominal increases in serum CK (McPherson and Pincus, 2007). One study found that mean total CK elevations 24 hours after a marathon were 3322 IU/L (22.3 times baseline) for men and 946 IU/L (8.6 times baseline) for women (Craig, 2006).

Similarly in the present study, after performing 4 sets of total 40 eccentric elbow flexor contractions, Participants in the groups cryotherapy, phonophoresis and in control group had CK elevations > 900 IU/L, at 72 hours after exercise; and had CK elevations > 1000 IU/L, at 96 hours after exercise. Where as in the exercise group the participants had CK elevations < 500 IU/L only. Thus increase in serum CK activity were significantly less in exercise group when compared to other two experimental groups and when compared with the control group (Fig 5.2).
Large inter-subject variation in CK in response to exercise has previously been reported as a limitation in its use in establishing the extent of muscle damage (Sorichter and Puschendorf, 1999). It is therefore interesting to note that in the current study, although there is considerable variation between subjects in the exercise group and when compared to other three groups, the exercise group showed very little inter-subject variation, indicating that exercise protocol used in this study provided a protective effect to the participants in that group.

In the present study Subjective Creatine kinase (CK) enzymatic values remained elevated for at least 4 days after exercise induced muscle damage in group 1 (cryotherapy), group 2 (phonophoresis) and in group 4 (control), but in group 3 (exercises), the values remained elevated for 2 days only, than the values started to decrease at 72 hours and the values were reaching near to the baseline values in day 4 (Figure 5.1)

6.2 LACTATE DEHYDROGENASE COMPARISONS

With this biochemical marker, in this present study, Cryotherapy and Phonophoresis do not have much influence on reducing the Lactate dehydrogenase (LDH) activity, whereas the exercise have better influences on reducing the LDH enzymatic activity.

It has been widely acknowledged that blood lactate is removed more quickly during active recovery because blood flow remains elevated through the active muscle, which in turn is believed to enhance lactate removal from the muscle cell (Nichols, et al., 2006). This has been researched extensively and has been given the green light as the most prominent way to enhance recovery from excessive lactic acid levels. This
may be the probable reason to support the present study, because the data in the present study shows that, Lactate dehydrogenase (LDH) enzymatic values remained elevated for at least 4 days after exercise induced muscle damage in group 1 (cryotherapy), group 2 (phonophoresis) and in group 4 (control), but in group 3 (exercises), the values remained elevated for 2 days only, than the values started to decrease at 72 hours and the values were reaching near to the baseline values in day 4 (Figure 5.3). The data shows that in the present study, after performing 4 sets of total 40 eccentric elbow flexor contractions, Participants in the groups cryotherapy, phonophoresis and in control group had LDH elevations > 700 IU/L, at 72 hours after exercise induced muscle damage; and had LDH elevations > 700U/L, even at 96 hours after inducing DOMS. The LDH levels > 400 IU/L, at 72 hours and it was 378 IU/L at the end of 96 hours. (Table 5.4)

The rate, at which the glycolytic / lactate system burns to provide energy in the form of ATP, is critical to the development and maintenance of high power outputs or speed. However, a problem can arise if the product of glycolysis (pyruvic acid) is not being removed and funneled into the aerobic system for further metabolism, as fast as it is being produced by glycolysis. When there is high concentration of pyruvic acid, it has the potential to stop the glycolysis along with the energy it generates. To avoid this dilemma, an enzyme called lactate dehydrogenase steps in and converts some of the pyruvic acid to lactic acid (removing pyruvic acid and half of the free H+ ions produced during glycolysis) and hence ‘buys some time’ to allow glycolysis to continue to reform the ATP molecule.

Scientific evidence has shown that approximately 70% of the lactic acid formed during any intensity of exercise is converted back to pyruvic acid and is used as a
substrate by the heart and skeletal muscle for energy. After 30-60 minutes following intense athletic events, there remains lactic acid concentration in the blood due to the body’s circulatory system (Dodd et al., 1984). This means that the lactic acid level in the blood as well as muscles, come to physiological resting level after a period of 60 minutes continued rest. The body’s physiological functions convert the excess lactate that results were converted back into glucose in the liver. This glucose can then be used in the process of resynthesizing glycogen that was reduced while exercising. To completely remove the lactic acid that was formed during the exercises, it requires approximately 20 minutes to half an hour.

Due to this fact, we cannot say that lactic acid (which comes to resting levels) is responsible for the sore and paining muscles that occur after grueling exercises. Muscle soreness that occurs 24-72 hours after exercise is most likely to be delayed onset muscle soreness which is not affected by lactic acid levels. In addition to it, good levels of lactic acid are also sources of energy and are required to produce energy in the normal process.

So, from the data analysis, it is known that muscle soreness was not affected by lactic acid and therefore, lactate dehydrogenase enzymatic activity is not much influenced by delayed onset muscle soreness, and there seems to be an active recovery for the enzyme to come back near to the baseline/normal range, and the exercise protocol given in this study accelerates even more the enzymatic activity to reduce down to the normal range when compared to other three groups.

Hence therefore in the present study Lactate dehydrogenase (LDH) enzymatic values remained elevated for at least 2 days (till 48 hours) after exercise induced muscle damage in all the four groups, and by 72 hours and after 96 hours the LDH values
does not get elevated and it remained to be in the same level in the group I (Cryotherapy), group II(Phonophoresis) and in group IV (Control), but in group III (Exercise) alone the LDH values started getting reduced by 72 hours and after 96 hours the values were reached near to the baseline/normal range (Figure 5.3 & Figure 5.4).

6.3 COMPARISONS BETWEEN DOMS, CK, LDH

Numerous studies have examined the effects of eccentric exercise using the elbow flexors on DOMS, CK, and LDH. Though the indices of muscle damage are the same, variations have been shown to exist between eccentric exercise performed on the arms and legs. Arm exercises have been shown to elicit greater DOMS, CK, and LDH activity than the legs. Overall, muscle damage is significantly greater in the arms, resulting in greater recovery time. It is suggested that changes and trends in CK, LDH, and DOMS are situational, based on the exercise protocol administered (Paschalis et al., 2005). It is noteworthy to recognize that studies reporting indices of muscle damage of the elbow flexors may not accurately represent the trends in blood markers and DOMS following eccentric exercise of the lower extremities.

The greatest elevation among the dependent variables CK AND LDH in the present study was observed after 48 hours in all the groups after inducing DOMS and the values remain elevated at 72 hours and even till 96 hours in Group I, Group II & Group IV. In Group III alone, the CK and LDH values elevated to peak level at 48 hours and started to decline after 72 hours and at 96 hours the values continued to reduce even more (Figure 5.2 & Figure 5.4).
Previous literature review often mentioned peak levels of CK, LDH, CRP, and DOMS that differed from the present study, though they did not account for all the days post-exercise to recovery, therefore not providing the most ideal means of comparison (Clarkson et al., 2006; Simpson et al., 2005; MacIntyre et al., 2001; Milias et al., 2005; Paschalis et al., 2007; Jamurtas et al., 2005).

6.4 MAXIMUM ISOMETRIC VOLUNTARY CONTRACTIONS (MIVC) COMPARISONS

In the present study maximum isometric voluntary contraction (MIVC) of elbow flexors were recorded by modified hand held dynamometer, and it has been found that there is an immediate reduction in maximum voluntary contraction for all the participants included in all the four groups at 24 hours after exercise induced muscle damage, and the reduction in maximum voluntary contraction was there even after 96 hours in control group and to the cryotherapy group. Whereas the other two experimental groups group II (Phonophoresis) and group III (Exercise) showed much improvement in recovering the maximum voluntary contraction force by 72 hours after inducing delayed onset muscle soreness (Figure 5.5).

The eccentric loading protocol designed in this study, produced a reduction of 40% MIVC of the elbow flexors in group IV (Control) and in group I (Cryotherapy) after 96 hours, but after same time line of 96 hours, in group II (phonophoresis) the eccentric exercises produced a reduction of 17 % MIVC and in group III (Exercises) the maximum voluntary force reduction was even less of 12% only. The percentages of reduction were noted from the mean scores (Figure 5.6).

This can be explained by the quick and prolonged reduction in the excitation-contraction factor. But it does not concur with the animal studies. Eccentric exercise
disrupts the T-tubule network immediately after downhill running in rats (Takekura et al., 2001). The increase in intracellular $\text{Ca}^{2+}$ concentration during tetanic stimulation is decreased immediately after eccentric damage (Balnave and Allen, 1995). Furthermore, the maximal $\text{Ca}^{2+}$ activated force is reduced less than the force produced by tetanic stimulation, particularly when the eccentric exercise protocol is severe (Warren et al., 1994). That is, contraction can occur when $\text{Ca}^{2+}$ is provided, but the link between excitation of the sarcolemma and release of $\text{Ca}^{2+}$ is disrupted.

The variation in the relation between length and tension of the elbow flexors is also a reason for the changes in the contractile force after exercise. There is an acute shift in the length of the muscle fibre immediately after eccentric exercise, towards longer lengths for the peak of this relation for a twitch contraction (Saxton and Donnelly, 1996; Jones et al., 1997). This is in fact felt in the focal lengthening and impairment in the occasional sarcomeres (Proske and Morgan, 2001), in order to increase the muscle length beyond the original optimum to produce the same myofilament overlap in undamaged sarcomeres. Some studies in animals (Lynn and Morgan, 1994) and humans (Brockett et al., 2001) suggest that incorporation of new sarcomeres in series produces a sustained shift to longer lengths in the length–tension relationship. Presumably changes in both excitation–contraction coupling and the length–tension relation underlie the reduction of the maximum voluntary force after eccentric damage in the present study. It is difficult to find out the individual contribution of the two effects as the forces were assessed at 90 degrees of elbow flexion which is close to the angle used for maximum voluntary force. However, a previous study suggests that for voluntary forces the shift in the length–tension relation after eccentric damage of human elbow flexors may explain only a modest part of the observed changes (Philippou et al., 2004).
Eccentric exercise lengthens muscle during contraction and damages its muscle fibers. A prolonged reduction in force occurs in animal (Faulkner et al., 1993; Friden, 2002) and human studies (Newham et al., 1983, 1987; Howell et al., 1993). Exercises that are eccentric can also lead to soreness and softness of the muscles. This muscle pain is the reason for activity in group III and IV muscle afferents (O’Connor and Cook, 1999), although a contribution from muscle spindle afferents has been postulated (Weerakkody et al., 2003). The reduction in maximal force is thought to be secondary to sarcomere ‘popping’ and disorganization (Morgan, 1990), as well as damage to components of the excitation–contraction coupling process (Warren et al., 1993; Balnave et al., 1997). Under some circumstances sarcolemmal function is affected (Yeung et al., 2003). These peripheral mechanisms have been reviewed (Proske and Morgan, 2001; Warren et al., 2001; Clarkson and Hubal, 2002; Lieber et al., 2002).

Exercise intervention included in this study has proved to show only mild reduction (12%) in maximum voluntary force of elbow flexors when compared to other two Intervention groups and to the control group. One of the theory supporting this finding is that perimysium plays an important role in preventing overstretching of muscle fiber bundles. Purslow. (1989) suggested that perimysium is arranged in different directions at different sarcomere lengths, and that perimysium collagen networks can be rearranged in some circumstances such as when muscle action performed concentrically and more rearrangement takes place when the muscle is immobilized. When muscle was moved in a lengthened position, the angle of the collagen fibers was arranged at a less acute angle than when the muscle was in shortened position (Williams and Goldspink, 1984). Any changes in the arrangement of perimysium can impact the compliance of muscle. Therefore the exercise intervention in the present study causes an acute adaptation of the parallel elastic components by rearranging
connective tissue of muscle and thereby helping to reduce the degree of muscle damage. Thus the mechanisms for reducing the severity of the muscle damage might be muscle fibers would be more flexible and more tolerant to mechanical overload and, as a result, would show minimal symptoms of muscle damage.

It has been suggested that ultrasound interacts with one or more components of inflammation, and earlier resolution of inflammation, accelerated fibrinolysis (Harpaz et al., 1993) stimulation of macrophage derived fibroblast mitogenic factors, heightened fibroblast recruitment, accelerated angiogenesis (Young and Dyson, 1990) increased matrix synthesis, more dense collagen fibrils (Friedar, 1988) and increased tissue tensile strength (Dyson and Luke, 1986) have all been demonstrated in vitro. Such findings form the basis of the rationale for the use of ultrasound to promote and accelerate tissue healing and repair.

Ciccone et al. 1991 reported that phonophoresis with trolamine salicylate was more effective than ultrasound alone in decreasing the effects of DOMS. In a review of phonophoresis, Byl in 1992 reported that ultrasound transmitting topical agents are required in order to get maximum treatment effectiveness. The method of Phonophoresis involves sending in low molecular weight drugs through ultrasound energy and cell permeability. Ultrasound may induce thermal and non-thermal physical effects in tissues. There is a chance of non-thermal effects occurring when the method is used for thermal effects. Reported thermal effects of ultrasound upon tissue include increased blood flow, reduction in muscle spasm, increased extensibility of collagen fibers, and a pro-inflammatory response. It is estimated that thermal effects occur with elevation of tissue temperature to 40 - 45 degrees C for at least 5 minutes (Prentice, 1994). The non-thermal properties of ultrasound include
cavitation and acoustic micro streaming. An event called cavitation occurs through ultrasound energy that form gas bubbles in the tissue fluids (Wells, 1977). Due to this, there is an enhanced flow in the surrounding liquid. Stable (regular) cavitation is considered to be beneficial to injured tissue. Due to one directional movement of fluids along the cell membranes, mechanical pressure modifies in the ultrasonic field and acoustic micro streaming is formed. This can change the structure of cell membranes, their functioning and also the permeability which has been considered to trigger cell repair (Dyson and Suckling, 1978).

A heavy decline in the strength and power during DOMS has been reported widely. These reductions are most notable in eccentric muscle actions, although concentric and isometric strength losses have also been reported (Paddon-Jones and Quigley, 1997; Donnelly and Maughan, 1990; Clarkson and Ebbeling, 1989; Francis and Hoobler, 1988; Hasson et al., 1993; Brown et al., 1997; Eston et al., 1996; Yates et al., 1990). Peak torque deficits are most pronounced 24-48 hours following DOMS-inducing exercise and are more profound and persistent during eccentric testing (Smith, 1992). The duration of strength reduction is also greater following eccentric activity and may require up to 8-10 days to return to normal baseline levels (Ebbeling and Clarkson, 1989). It takes 4 days to recover isometric and concentric strength.

Other researchers have reported a delayed return of eccentric peak torque of the elbow flexors following isokinetic eccentric exercise, with eccentric peak torque remaining at a value 15% less than initial peak torque until 7 days post-DOMS inducement (Yates and Armbruster, 1990). Similar trends have also been observed with respect to the lower limb (Eston et al., 1996) measured isokinetic, eccentric and concentric knee extension peak torque values of the dominant leg at 0.52 and 2.83
radians/sec 2, 4 and 7 days following DOMS-inducing isokinetic exercise. The results showed an immediate post-exercise loss in peak torque for both modes of exercise at slow and fast velocities up to day 4, and a return to normal levels between days 4–7. Significant reductions in concentric and eccentric peak torque in the lower limb 48 hours following DOMS-inducing exercise have also been reported in other studies (Hasson et al., 1993). In spite of these findings, the time taken to recover strength is not clear. Many a times, researches have not collected data at the right intervals over the 2 day period following DOMS inducement. Because of this, the time taken for peak torque to come back to normal level is not absolute. This may have important implications for the athlete as an alteration in the strength ratio of agonist to antagonist muscle groups may contribute to an increased risk of injury (Orchard et al., 1997).

With some forms of exercise, impaired voluntary activation or an inadequate drive to muscle fibers occurs, and such a mechanism could contribute to the prolonged reduction in voluntary force after eccentric exercise (Gandevia, 2001). After eccentric muscle damage, voluntary activation (and hence muscle force) may be reduced as a result of muscle pain and tenderness. Saxton and Donnelly. (1996) used a type of twitch interpolation with nerve stimulation during isometric contraction of the elbow flexors in the days after eccentric exercise. They found inconsistent changes in the force added by tetanic stimulation. Using motor cortical stimulation to evoke force increments, Loscher and Nordlund. (2002) found some impairment of voluntary activation immediately after eccentric exercise, but this recovered within 5 min. Twitch interpolation using nerve stimulation after eccentric exercise of the elbow flexors also revealed an immediate reduction in voluntary activation (Michaut et al.,
2002). In the latter two studies it was not possible to separate an acute effect of muscle fatigue from a longer-term effect related to muscle damage.

A compliant muscle tendon unit might help to reduce the severity of muscle damage by increasing the optimal length of muscle fibers from the length-tension curve. When muscle is lengthened beyond the optimal length, the sarcomeres become more unstable (Morgan and Allen, 1999). Each sarcomere is stretched until it is unable to support the tension. Within a muscle at a given length, shorter sarcomeres are stretched more and become disrupted after repeated lengthening (Brockett et al., 2001). The area of disrupted sarcomeres spreads throughout muscle fibers and leads to muscle damage. This hypothesis is supported by the finding that eccentric exercise at a longer muscle length (stretch position) caused more muscle damage than the shorter length. Newham et al. (1988) and Nosaka et al. (2001) compared two different starting positions with the same range of motion (short and long muscle range 50-130° and 100-180°, respectively). The participants reported more soreness and demonstrated more strength and range of motion loss, and more swelling in the long muscle length condition than in the short muscle length condition. The images from magnetic resonance and ultrasound also showed greater damage in the long muscle length condition than the short muscle length condition. Therefore, if an intervention helps to increase optimal length of muscle, it may help to reduce muscle damage from eccentric exercise. Similarly in the present study the exercise intervention was believed to reduce muscle damage by increasing optimal length of the muscle.

Many modalities have been advocated for enhancing the recovery of pain and range of motion in the treatment of Delayed Onset Muscle Soreness (DOMS), however there is lack of concrete information regarding the efficacy of those modalities.
Evidence with regard to the efficacy of ultrasound (US) on DOMS was conflicting. Some study (Hasson et al., 1990) showed that US helps relieve DOMS while the other have observed contradictory findings of no effects (Craig et al., 1999) and also adverse effects (Ciccone, 1991). The present study was designed to evaluate the possible contribution of pulsed ultrasound for recovery of, pain and muscle power following DOMS.

The data analysis showed that at baseline and at 24 hours the MIVC measures between the four groups were found to be not significant statistically (p>0.05), but after 48 hours it was having significant difference between the groups (p<0.001) (Table 5.7). Thus the MIVC Measures were found to be reduced even until 96 hours after inducing delayed onset muscle soreness in group I (Cryotherapy) and in group IV (Control), but where as in group II (Phonophoresis) and group III (Exercise) the MIVC measures were found to be reduced only till 48 hours and by 72 hours the MIVC measures started to get increased and after 96 hours the values were found to reach near to the baseline values (Figure 5.6)

6.5 PAIN COMPARISONS

Muscle soreness was evaluated using a visual analog scale consisting of a 10-cm continuous line representing “no pain” at one end (0 cm) and “very, very painful” at the other (10 cm). Subjects were asked to indicate the soreness level on the line when an investigator extended the elbow joint.

In this study, the pain scores at 24 hours between the groups were not found to have significant difference (p>0.05) but at 96 hours the pain scores showed statistically significant difference (p<0.001). In the present study, the pain scores were much
reduced and near to normal in group II (Phonophoresis) and in group III (Exercise), there was 88% of pain relief noted in group II, 72% pain relief in group III, where as in group I (Cryotherapy) and in group IV (Control) pain relief was 43% and 44% only noted after 96 hours (Table 5.11).

The onset of muscle pain in all the four groups occurred within the first 24 hours following the eccentric exercise protocol, which is consistent with other studies, investigated the onset of DOMS resulting from exercise-induced muscle damage. (Nosaka, 1995). The possible responsible mechanisms for pain relief are neurological (pain gate theory), physiological (biochemical substances), and mechanical (realignment of muscle fibers). Phonophoresis may reduce the pain by activating the neural-gating mechanism in the spinal cord. Micro massaging effect received from ultrasound therapy produces tactile information which might stimulate large fast nerve fibers and then block the smaller, slower nerve fibers that detect pain. This effect presumably results from local lateral inhibition in the spinal cord (Guyton and Hall, 2000) and explains why touching the painful area is an effective strategy for relieving pain. However, there are no objective data to support this idea. Ultrasound can also increase biochemical substances such as serotonin (Leivadi et al., 1999), which is a neurotransmitter that plays a role in reducing pain (Guyton and Hall, 2000).

Physiotherapists usually use treatment modalities to break the vicious cycle that causes muscle spasm, and consequently, muscle pain. Muscle spasm causes muscle pain directly by stimulating mechanosensitive pain receptors or indirectly by compressing the blood vessels resulting in ischemia (Guyton and Hall, 2000). Exercise might help to rearrange muscle fibers and increase microcirculation. The realignment of fibers helps to reduce muscle spasm that stimulates pain receptors and
helps to reduce the pressure on blood vessels. The increase in blood microcirculation helps to increase nutrition to the damaged area. However, there is no scientific evidence to support these ideas because exercise is unlikely to increase muscle blood flow and there is no published study on the effects of exercise on the realignment of fibers.

Exercise is one of the most effective strategies for alleviating DOMS (Armstrong, 1984). However, pain relief is also temporary and rapidly resumes again following exercise cessation (Smith, 1992). It has been proposed that the temporary alleviation of pain during exercise may be due to the breakup of adhesions in the sore muscles, an increased removal of noxious waste products via an increased blood flow or an increased endorphin release during activity (Hough, 1902). The latter result is an analgesic effect that minimizes the sensation of DOMS. Elevated afferent input from large, low threshold sensory units (groups Ia, Ib and II fibers) may also interfere with the pain sensation carried by group III and IV fibers, thus reducing pain (Carlsson, 1982). Studies that have investigated the therapeutic effects of exercise on the development of DOMS have shown mixed findings. Upper arm ergometry performed for 8 (Weber et al., 1994)–10 minutes (Gulick et al., 1996) immediately following DOMS inducing eccentric muscle activity of the elbow and wrist extensors revealed no statistically significant differences in muscle soreness at 24, 48 and/or 72 hours post-exercise when compared with a control group. The performance of 25 sub maximal eccentric contractions 1 day following a heavy eccentric DOMS-inducing exercise regimen for the forearm flexor and extensor muscles also showed no effect on muscle soreness (Donnelly, 1992). Hasson et al. (1989), reported a significant decrease in DOMS at 48 hours following high velocity concentric isokinetic exercise (6-20 maximum voluntary contraction of the knee flexors and extensors at 5.23
rad/sec) performed 24 hours following stepping exercise. The contrast in research findings was attributed to differences in exercise protocols, including type of exercise performed, timing of exercise and degree of effort (sub maximal vs. maximal) (Gulick et al., 1996).

In the present study, exercise protocol as an intervention was designed in such a way that, the individuals in the exercise group performed mild full ROM elbow flexion and extension exercises, with only the gravitational pull on the hand and arm providing resistance. The repetitions were performed continuously during a 20-second period and then rested for 40 seconds. This exercise/rest interval was continued for a total treatment time of 15 minutes. This exercise protocol showed the differences of change in pain measure from 24-96 hours were found to be statistically significant in between the four groups (p<0.001) and pain scores at 24 hours between the groups were not found to have significant difference (p>0.05) but at 96 hours the pain scores were showing statistically significant difference (p<0.001) (Table 5.11). There was a pain relief experienced in all the three experimental groups and in the control group, however Phonophoresis and exercise intervention were found to relieve pain in much better way when compared to cryotherapy intervention and with the control group (Figure 5.7).

This may have important implications for athletes who train on a daily basis or who are preparing for an event that will comprise some eccentric activity. In particular, attention must be paid to the scheduling of training programmes in order to minimize the amount of muscle soreness experienced at or near competition times. Future research should be directed towards the identification of pain relief strategies during exercise, and whether compensatory mechanisms are adopted to help alleviate pain.
Although this may not be relevant to the recreational athletes who will most likely rest for 2–3 days following strenuous exercise, the elite athlete, who must train daily or twice daily, may be predisposed to further injury if biomechanical adaptations are adopted to help relieve intense muscle soreness. Therefore we strongly recommend that exercises can be advised for the early recovery of DOMS in recreational athletes and for also to the elite athletes to facilitate their early participation.

Our results reject the null hypothesis.

Summary:
In the present study, our focus was to find out the effect of various Physiotherapy modalities in remunerating DOMS in recreational athletes. This is the first Randomized control trial in India to find out the effect of these three modalities in single research design. Since DOMS is the major limiting factor and it hinders the performance of athletes, as sports physiotherapist it is important for us to treat this complication and rehabilitating the athletes to return back to the event as early as possible. Therefore we need to understand the biochemical changes, clinical findings and as well as the muscle performance following DOMS. In the present study based on the result analysis, we found that there is a significant improvement and reduction in biochemical parameters following Physiotherapy interventions especially exercise group found to be more recovered as compared to other groups.

Based on the result analysis of Maximum isometric voluntary contraction and pain scale (VAS), Phonophoresis and exercise is found to be effective when compared to cryotherapy, but as compared to control group, all the three modalities is found to be effective. Therefore we reject the null hypothesis and recommend phonophoresis and exercise can be used as a non invasive approach for the management of DOMS in recreational athletes.