CHAPTER 5

DISCUSSION

This thesis reported numbers of new findings. First of all, the characteristic study revealed that the symptom of DOMS is similarly to the primary hyperalgesia (i.e., acute tissue injury) as it responded to both mechanical and thermal stimuli. These characteristics have not been reported previously. This also linked to the various outcome measures in this thesis that cover the whole aspect of sensory and motor functions. Thermal stimulus was also included to evaluate DOMS' characteristics as previously mentioned. Furthermore, some sensitive sensorimotor measures (i.e., CPT, PPT, ROM, GS and WES) of the eccentric exercise induced muscle damage of the wrist extensor were also pointed out in this thesis. This is the pioneer report to identify the sensitive and reliable outcome measures in this region. Finally, the potential physical applications including PNF stretching, massage, hot pack and sauna were evaluated for their prophylactic effects in attenuation of DOMS's symptoms in the wrist extensor.

5.1 Reliability of muscle function and sensory perception measurements of the wrist extensors

This study used ICC, CV, and SEMs to assess the reliability of muscle function and sensory perception measures that could be used to assess muscle damage in the wrist extensors. Generally, the ICC of greater than 0.85 is considered as an acceptable reliability (Portney and Watkins, 2000). To determine variation of the values between two test occasions, the percentage of CV was also used. Standard

error of measurements (SEMs) is another indicator of variations and error of measurements. A measure is considered to be reliable if CV is less than 15% and SEM is less than 5% (Atkinson and Nevill, 1998; Portney and Watkins, 2000). ICC of all measures except for JPE were greater than 0.85, only CV of JPE, CP and VIB exceeded 15%, and SEMs were higher than 5% only for JPE and CP. All measurements except JPE are reliable and can be used to investigate effects of a physiotherapy intervention on the wrist extensors. The reliability of JPE-F and JPE-E measures were not within the acceptable ranges. Joint position error is a sensory perception that has the afferent information from peripheral mechanoreceptors located in the skin, muscle, and surrounding joint structures (Voight et al., 1996). The control of this kind of sense is most likely a combination of afferent information, efferent response, and central control (Goodwin et al., 1972), and these factors may make this type of measurement more complicated. The high variation and error of these tests may also cause by the nature of the test in which the subject have to pay attention concisely during the period of test. Therefore, using the JPE should be caution when applying for clinical investigation.

The present study assessed the reliability of muscle function and sensory perception measures that could be utilized in a study to investigate neuromuscular function of the wrist extensors. However, this study may have some limitations such that only healthy young male subjects were included, and the duration of the follow-up was within 1-day interval. It is interesting to investigate how exercise of the wrist extensors affects these measures, and whether these measures are valid to assess the effect of an intervention on muscle damage of the wrist extensors.

5.2 Characteristics of DOM in the wrist extensors

This study investigated the DOMS characteristics in the wrist extensors, followed up within 14 days after the DOMS. The damage of muscle was proved by plasma CK activity. The normal reference range of plasma CK activity with this method is 0-195 IU/L and 57-197 IU/L with a test kit (Lavender and Nosaka, 2006). CK was compared between the pre-exercise values and peak values approximately at the 4th day post-exercise, it showed significant increase from 152.8 ± 13.71 to 301.8 ± 40.46 IU/L with the large inter-subject variability of 65-296 at pre-and 121-759 IU/L at post-exercise. According to Clarkson et al 1992, the plasma CK level in this study is classified as the low responders (Clarkson et al, 1992). High-force eccentric exercise (e.g., maximal contraction of elbow flexors) increased CK, with peak activity (generally 2,000-10,000 IU) occurring about 4 to 6 days post-exercise (Clarkson et al, 1992). Sorichter et al. (1995) suggested that the increased CK activity was due to a messaging out of the CK from EIMD. Therefore, it means that the induction protocol of this study could be able to produce muscle damage in the wrist extensor muscles.

There were significantly increased in pain intensity (VAS) and muscle soreness (modified Likert scales) with a similar pattern at immediately and after the exercise. In this study, the results showed the moderate relationship between VAS and LS ($r = 0.40$, $p < 0.05$) on the peak day (Appendix 6). Pain and muscle soreness were maximal at Day1, similar to the study of Slater and colleagues in 2005. The symptoms of DOMS disappeared within the Day 7. It was hypothesized that pain increased immediately post-exercise due to the eccentric exercise that cause muscle damage. Muscle soreness seems to appear many hours post-exercise of the damage-

inducing exercise and peak 24-48 hours post-exercise (Newham et al., 1983, Clarkson et al., 1992, and Ebbeling and Clarkson, 1989).

The time course and the extent of DOMS in the present study was similar to other previous reports (Armstrong, 1984; Ebbeling and Clarkson, 1989). Clarkson et al. (1986) studied eccentric exercise in forearm extensors using pulley weights and reported small but significant increase, approximately 3 on a 1-10 scale 24 hours after exercise.

This present finding has shown that TPT especially cold pain threshold was the sensitive outcome during the symptoms of DOMS. In the present study, CPT was also more sensitive than HPT at the both sites (lateral epicondyle and ECRB). The cold and warmth receptors locate mainly underneath the skin at discrete separated spots with more cold spots comparing to warmth spots. Moreover, cold signals are transmitted in A delta myelinated nerve fibers and some in type C nerve fibers whereas warmth signals are transmitted only over type C nerve fibers, which suggests that better transmission of cold than warmth sensation (Guyton and Hall, 2006). Frost et al. (1991) have highlighted cold hyperalgesia as an important diagnostic criterion for sympathetically maintained pain. CPT was also sensitive in the lateral epicondyle and the ECRB sites. CPT at the lateral epicondyle site was elevated and peaked at immediately post-exercise, continued to day 1-3, and recovered back to the baseline within 4 days post-exercise. CPT at the muscle site was increased at immediately post-exercise, continued to day 1-5 with the peak on day 2, and recovers back to the baseline within 6 days. CPT is one of the TPT which showed more sensitive in DOMS due to the lower thermal pain threshold in the muscle damage. PPT was sensitive on the both sites of the lateral epicondyle and ECRB, similar to the study of

Slater and colleagues in 2003, and this pattern continued through the period of DOMS. PTT was decreased at immediately post-exercise with the lowest point on the day 2 post-exercise and recovers back to the baseline within 11 days at the lateral epicondyle site and within 13 days at the muscle site. PPT is a mechanical pain threshold which showed more sensitive in DOMS due to the lower threshold in the muscle damage. EIMD generated similar sensori-motor characteristics as tissue injury which response to the process of sensitization of peripheral nociceptive apparatus resulting in lowers the normal mechanical threshold for nociceptors (Graven-Nielson and Mense, 2001).

VIB did not seem to be the sensitive outcomes in this study. Furthermore, there were no significantly different results in VIB at the origin and muscle sites in this study. The vibrating sense has been used to signify the proprioceptive sense via large-fiber mechanoreceptors (Weerakkody et al., 2001). However, the result on this thesis did not show the significant outcome on VIB. Therefore, an involvement of the large mechanoreceptors related to exercise-induced muscle damage of the wrist extensor is remained questioning.

In this study, ROM was assessed in both active and passive of the wrist flexion and extension. AF was decreased at immediately post-exercise, continued to day 1-6 with the lowest point on the day 2 post-exercise and recovers back to the baseline within 7 days post-exercise. AE was decreased and the bottom out at immediately post-exercise, continued to day 1-6, and recovers back to the baseline within 7 days post-exercise. PF was decreased at immediately post-exercise, continued to day 1-14 with the lowest point on the day 1 post-exercise. PE was decreased at immediately post-exercise, continued to day 1-7 with the lowest point on

the day 2 post-exercise, and recovers back to the baseline within 8 days post-exercise. ROM; AF, AE, PF, and PE were significantly decreased during the symptoms of DOMS, espectially PF was significant decrease until the 14th day after exercise. There was moderate relationship between ROM and some measurement outcomes such as AF & GS (D2), AF & WES (D2), AE & PPTO (D1), AE & PPTM (D1), AE & GS (D1), AE & GS (D2), AE & WES (D2), and PF & PPTM (D2) in this study (Appendix 6). It showed that ROM was related to sensory (PPT) and motor functions (muscle strength) during the peak pain period. ROM is a sensitive outcome in DOMS due to the motion course of pain in the muscle damage. The sensation of tender, aching muscles, usually feel during movement (muscle contraction) or being stretched (Smith, 1991). As the confirmation with the negative moderate correlation result of LS & AF and LS & PF on the day 1 and day 2 post-exercise, $r = -0.51, -0.55, -0.60$ and -0.69, respectively which means that the more pain the less in the ROM (Appendix 6).

CRT is a represent of the motor performance; it is the neuromuscular response to the stimuli. In this study, there was not change in CRT during the DOMS period. The nature of this testing requires a combined movement of the whole upper limb (e.g., shoulder, elbow, wrist and hand), so that the unaffected joints may compensate for the selected motion during the CRT test. In addition, CRT was found to be slower on the day of the induction only, this may mainly due to the muscular fatigue. Bisset et al. (2006) demonstrated a different result in that slower response times of upper-limb movement was evidence in the chronic lateral epicondylalgia group. This phenomenon did not happen with EIMD of the wrist extensors in this thesis. Different muscular pathologies (i.e., acute vs chronic) may be one of factors for this

convergence. In this present study, there was trend to reduce of CRT in both sides due to the training effect. CRT of the DOMS side was significantly greater than the normal side. This might be the fact that the DOMS side was "non-dominant" which could take the longer response time than in the normal side.

GS and WES were represented of muscle strengths. There were the sensitive outcomes of the muscle strengths to DOMS especially in pain-free motions and their correlations in this study were in moderate level ($r = 0.49$ and 0.65 on the day 1 and 2 post exercise) (Appendix 6). GS with pain-free motion was decreased at immediately post-exercise, continued to day 1-12 with the lowest point on the day 1 post-exercise and recovers back to the baseline within day 13. GS was not significantly different on the day 8-10 due to the high inter-subjects variation in the results of these days. WES with pain-free motion was decreased and bottom out at immediately post-exercise, continued to day 1-11 post-exercise and recovered back to the baseline within 12 days post-exercise. WES was not significantly different on the day 10 due to the high inter-subjects variation in the results of this day. The reduction in muscle strength at immediately post-exercise might be related to fatigue. Muscle strength decreased and slowly recovery after exercise and throughout the period of DOMS symptoms. The prolonged strength loss after eccentric exercise is considered to be one of the most valid and reliable indirect measures of muscle damage in human (Warren et al., 1999). High-force eccentric exercise can often generate up to 50-60% loss of force-generating capacity when compared with pre-exercise values in elbow flexors (Newham et al., 1987, Nosaka et al., 1991, and Saxton et al., 1995). Prolonged strength loss in the days after eccentric exercise can typically last 1 to 2 weeks (Newham et al., 1987). The reason of the decrease in muscle strength during the

symptom of muscle soreness could be explained with a combination of untrastructure damage, altered neural activation, fatigue, and calcium homeostasis disruption which the importance of each of these factors varies with time after eccentric exercise (Armstrong, 1984; Clarkson et al., 1992; Weber et al., 1994). Interestingly, GSmax was significant greater than the baseline since day 6 and WESmax was significant greater than the baseline since day 10 to the following day. The result was similar to the study of Jamurtas et al. (2005), i.e. they found that exercise peak torque (EPT) of leg was significantly elevated comparing to the pre-exercise level at 72 and 96 hours post-exercise. Reasons for this increase might be the effect of the exercise training and the neuromuscular adaptation results in greater coordination and familiarity with the machine turn to the increase in muscle strength.

This study demonstrated that the induction protocol can induce DOMS. The analyses data showed the sensitive outcomes of DOMS were pain intensity, cold pain sensation, pressure pain threshold, ROM, and muscle strength. For the characteristics of DOMS, muscle soreness symptoms mostly recovered back to the baseline within 7 days excepted for PPTM, ROM-PF, and muscle strength. The results of the reliability section and the investigation of DOMS' characteristics were utilized to the preventative study part.

5.3 The Preventative Studies

From the results the preventative studies, there was no different in the physical characteristics, average of maximal torque and total works between groups while the exercise induction. Therefore, there was no effect of the physical characteristics and

force exertion between the preventative group (PNF, massage, hot pack and sauna) and the control.

5.3.1 A prophylactic effect of proprioceptive neuromuscular facilitation (PNF) stretching on DOMS

This study demonstrated that skin blood flow was not change after application of PNF-stretching as detected by a Laser Doppler. One of the possible reasons is that the PNF technique focuses mainly on the muscle which may not affect much on the skin level. Therefore, Laser Doppler failed to detect a significant change in blood flow of the skin following PNF application in this thesis.

The main findings of this study were the beneficial effects of PNF intervention on DOMS symptoms including CPT, ROM, pain-free grip and wrist extensor strength. However, VAS and LS which were used to detect the pain intensity in this study did not show significant difference between groups. VAS and LS are subjective data collecting that may not sensitive enough to identify the changes between the intervention and the control group. However, they are quite useful measures for reflecting the development of the symptoms of DOMS. The PNF group significantly demonstrated a lesser deficit in cold pain threshold than in the control group. We have demonstrated that using the PNF (hold-relax with agonist contract) technique can help to minimize pain perception and reduce the effect of cold stimuli on CPT at the muscle site. It has been reported that a single bout of resistance exercise is capable of modifying the sensation of experimentally induced analgesia (Koltyn and Arbogast, 1998; Koltyn et al., 2001). Hoeger Bement et al. (2009) also reported that an isometric fatiguing contraction significantly alters the corticomotor pathway during application of a noxious stimulus. These insights help to explain why

PNF intervention has some effect on the thermal receptor adaptation to the stimuli, as shown in the results from the PNF group when compared to the control group. Passive flexion range of the wrist was lesser deficit in the PNF group. Passive extension range of the wrist in PNF group was also fewer deficits. Muscle soreness is more painful and sensitive with stretching (Byrne et al., 2004). Passive range of motion can cause more pain especially in the opposite movement (i.e., flexion direction) of the damaged wrist extensor muscle and more compressed in muscle belly when moved in the same direction (i.e., extension direction) of the damaged muscle. The two directions of experiment lead to muscle guarding during the movement (Jones et al., 1987). Active flexion and extension range of the wrist in the PNF group were lesser deficit than in the control group. Active movement (contraction) in the flexion can stretch the extensor muscle which is the cause of pain during the movement. Active contraction of extension, the soreness muscle can affect the excitation contraction coupling and cause pain during motion (Byrne et al., 2004). In general, DOMS can cause the reduction in ROM by pain and/or stiffness after exercise. Application of PNF has beneficial effects on passive and active movement in this ROM measure.

It seemed that pain-free muscle strength is more sensitive to detect the change of muscle damage than the maximal muscle strength. The pain-free grip strength and wrist extensor strength in the PNF group have fewer deficits than the control group. Muscle strength is one of the best muscle damage indicators, which is normally reduced after exercise with slow recovery (Nosaka and Newton, 2002). The prevention of DOMS by using PNF demonstrated the beneficial effect on muscle

strength of grip and wrist extension in this present study, as showed in a lower deficit of pain-free grip and wrist extensor strength.

Regarding the magnitude of change, the observed attenuation in CPTM (4.84° C), ROM (11° - 16°), grip strength (25 N) and wrist extensor strength (30 N) by the PNF-stretching appears to be clinically important for athletes (e.g., racket sports, fencing) as well as people who required the specific tasks (e.g., artist, graphic designer, musician, and patient).

The result of our study was dissimilar to previous studies. High et al. (1989); Johansson et al. (1999) did not to demonstrate the efficacy of stretching on muscle soreness in quadriceps and hamstrings, respectively. They applied static stretching before the induction exercises in healthy student volunteers, and their results showed no effect of static stretching on DOMS. In this present study, the different stretching technique (PNF-hold relax with agonist contraction) was performed. This technique is a combination of both static and dynamic stretching maneuvers. As a result, some advantageous effects of the PNF were evidenced on DOMS symptoms in terms of sensory perception and muscle function. The application of PNF before exercise was aimed at preparing the localized muscle to prevent DOMS symptoms. PNF technique of hold relax with agonist contraction was used to prepare the wrist extensors with passive and active movement that can improve muscle flexibility via autogenic inhibition and reciprocal inhibition. The benefits of an active warm up may be to minimize muscle stiffness by moving the required muscle groups through their range of motion. As a result, the warm up with PNF stretching may release actin-myosin bonds and thereby reduce the passive stiffness of muscle. This may contribute to an increased rate of force development

and an increase in the efficacy of muscle working during eccentric exercise (Bishop, 2003). Stretching exercises also affect the mechanical properties of the muscle-tendon unit (MTU), i.e. reduce the tension on the muscle-tendon unit that affects the visco-elastic component of tissue leading to an increase in the compliance of muscle and a reduction in muscle stiffness; consequently, less tension will be produced in the muscle during a specified stretch. The resulting improvement of muscular flexibility possibly reduces muscle and connective tissue damage after exercise (Weldon and Hill, 2003; Magnusson and Renstrom, 2006). Apart from the visco-elastic mechanism of the PNF stretching, the neurophysiological mechanism may take part for the effects through neural inhibition of the muscle group being stretched via an inhibitory interneuron. As a result of reducing the activity in the alpha-motor neuron to the antagonist muscle, which then promotes greater relaxation and decreases resistance to stretch (Guyton and Hall, 2006). It is also possible that the descending pain inhibitory systems (e.g., mid brain) may be activated during PNF stimulation (Carrive, 1993). Further neurological studies, such as functional magnetic resonance imaging (fMRI), are warrantee to investigate this notion.

Some limitations should be noted that the placebo condition was not included in the study. Studies in the future should consider the placebo-controlled study design for strengthening the internal validity of the study.

5.3.2 A prophylactic effect of massage on DOMS

All measures (VAS, modified Likert's scale, PPT, cold thermal pain threshold, ROM and strength) showed that muscle soreness developed in the wrist extensors after eccentric exercise. However, a significant effect of massage on pain perception was evident only by passive ROM and active flexion. The passive ROM

in flexion and extension are considered as a sensitive measure to detect a minimal change in pain perception.

Massage prior exercise could result in diminish ROM reduction after the exercise. There was a significant difference in passive flexion range of the wrist between control and massage groups on day 2, the massage group significantly showed a lesser deficit in ROM-PF than the control group. There was significantly lesser deficit in passive extension range of the wrist under the massage group in comparison to the control group on days 1, 2, and 4. Muscle soreness is more painful and sensitive with stretching (Byrne et al., 2004). Passive range of motion can cause more pain especially in the opposite movement (i.e., flexion direction) of the damaged wrist extensor muscle, and more bulged in muscle belly when moved to the same direction (i.e., extension direction) of the damaged muscle (Byrne et al., 2004; Jones et al., 1987). In this present study showed the less reduction of ROM-PF and ROM-PE in the massage group. The reductions in ROM-PF and ROM-PE following exercise were approximately 24.5%, 6.9%, respectively. It has been reported that decreases in ROM after eccentric exercise are associated with muscle soreness, swelling, strength loss and muscle stiffness (Clarkson et al., 1992; Nosaka and Clarkson, 1997). During passive flexion (ROM-PF), the wrist extensors are stretched, and pain and stiffness caused by stretching might have affected the ROM, thus the massage was found to affect ROM-PF. ROM-PE, which was least decreased after exercise, is more associated with flexibility of the wrist flexors that were not affected by the exercise, therefore the treatment on the wrist extensors had only minor effect. Regarding the magnitude of change, the observed attenuation in ROM by the massage

(10.5º - 13.3º) appears to be clinically important for athletes (e.g., racket sports, fencing).

Massage techniques in this study were consisted of muscles compression, effleurage, tapotement, and petrissage. These massage techniques have been used to reduce muscle tension and increase blood flow (Hovind and Nielsen, 1974; Rodenburg et al., 1994; Moraska, 2005; Frey Law et al., 2008). The application of massage on arm muscles was aimed to prepare the muscle before the exercise. The mechanism effect of massage includes an increase in muscle flexibility and reduction in muscle stiffness. It also enhances local microcirculation and lymph flow, and increase muscle compliance (Weerapong et al., 2005). The effects of massage support the result in less severe muscle damage to eccentric exercise. For the physiological effects of massage on blood flow, massage can increase venous blood flow and muscle blood flow (Hovind and Nielsen, 1974), vasodilation in skin tissue produced by massage was caused by the release of vasoregulatory neuropeptides (Morhenn, 2000). In this present study, the result also showed an increasing in tissue blood flow after using the massage. Increase blood flow, improvement CO_2-O_2 exchange rate and nutrients to active structures could be beneficial for preparing and warm up the muscle before exercise (Bishop, 2003). Massage involves the application of mechanical pressure on the muscle tissue in order to decrease tissue bond. Increase muscle-tendon compliance is believed to be achieved by mobilizing and elongating shortened or adhered connective tissue. Improved muscle compliance results in a less stiff muscle-tendon unit (Weerapong et al., 2005). The reduction in muscle spasm could be less susceptible to eccentric exercise-induced muscle damage.

Unfortunately, there is no previous studies related to the prophylactic of massage on the DOMS, thus it is limited evidence which could compared with this study.

5.3.3 A prophylactic effect of hot pack on DOMS

The aim of this study was to investigate prophylactic effect of hot pack treatment on DOMS in the wrist extensors. The results showed that the hot pack treatment increased skin temperature and skin blood flow, and affected the changes in pressure pain threshold, ROM-PF, ROM-PE, ROM-AE, pain-free wrist extensor strength following eccentric exercise, but did not affect other variables (VAS, modified Likert's scale, cold thermal pain threshold, ROM-AF, pain-free grip strength and the maximal strengths). The increases in skin temperature and skin blood flow were expected, since previous studies (Petrofsky et al., 2009; Draper et al., 1998) also reported increases in skin blood flow (e.g. approximately 5-folds) and subcutaneous tissue temperature at 1-cm depth (e.g. approximately 1.1-folds) after hot pack treatment. In the present study, the skin temperature increased approximately 11°C after the hot pack treatment. Draper et al. (1998) reported that calf muscle temperature increased 3.8°C after 15 min hot pack treatment when the subcutaneous tissue temperature increased up to 38°C from 34°C. In the present study, muscle temperature was not measured, because it is an invasive technique that could potentially affect the outcome measures, but it seems reasonable to assume that muscle temperature also increased after the hot pack treatment by at least 3°C as shown in the previous study (Draper et al., 1998).

It should be noted that the previous studies showing prophylactic effect of warm-up on DOMS found approximately 3°C increases in muscle temperature (Gray and Nimmo, 2001; Skurvydas et al., 2008; Evan et al., 2002). For example,

Evans et al. (2002) reported that pulse short-wave diathermy resulting in 3.5°C increase in muscle temperature, attenuated swelling after eccentric exercise of the elbow flexors. Skurvydas et al. (2008) have shown that a 44°C-water immersion that increased muscle temperature by 3°C attenuated the increases in serum creatine kinease activity and muscle soreness of the quadriceps, and decreases in jump height and maximal voluntary isometric contraction strength of the knee extensors following drop jumps. In contrast, Nosaka et al. (2004) reported that pre-exercise microwave treatment for 10 min that increased muscle temperature by 3°C had no effect on muscle soreness as assessed by VAS, range of motion and muscle strength following eccentric exercise of the elbow flexors. It is possible that the therapeutic dose of the heated application and the volume of heated muscle account for the different findings. The optimal treatment duration of a heat modality is 20-30 minutes to produce its potential therapeutic effects (Cameron, 2003). Thus, it may be that the 10-minute duration of microwave application in the study by Nosaka et al. (2004) was not long enough.

It has been documented that increased muscle temperature by warm-up exercise makes muscle and connective tissue less susceptible to eccentric exercise-induced muscle damage possibly by reducing muscle viscosity thereby resulting in smoother muscle contraction (Nosaka and Clarkson, 1997). Safran et al. (1989) stated that warm-up would decrease stiffness of the muscles, increase extensibility of connective tissue within muscle and decrease muscle viscosity. Although it was expected that the hot pack treatment would attenuate changes in all dependent variables, the prophylactic effects of hot pack treatment were limited to PPT and

ROM in the present study. It is also important to note that the effect of hot pack treatment on these variables were not large.

All sensory perception measures (VAS, modified Likert's scale, PPT, cold thermal pain threshold) showed that muscle soreness developed in the wrist extensors after eccentric exercise. However, a significant effect of hot pack treatment on pain perception was evident only by PPT. The PPT is considered as a sensitive measure to detect a minimal change in pain perception, whereas the VAS and modified Likert's scale are categorized as a subjective outcome measure with ordinal scales for monitoring changes in pain (Slater et al., 2005). It may be that the sensitivity of VAS and modified Likert's scale was a reason why the prophylactic effect of hot pack treatment was not found for these measures. The reduction in pressure pain perception in the hot pack condition was observed and the magnitude of reduction in pressure pain threshold by the hot pack in this present study was approximately 22-35%, which is considered to be clinically significant (Slater et al., 2005).

Cold thermal pain threshold was employed to evaluate the primary hyperalgesia, which could be indicated by a reduction in the threshold (Wright et al., 1992; Wright et al., 1994). In the present study, the magnitude of change in cold thermal pain threshold was approximately 20-30%, which was similar to that of PPT. However, the change in cold thermal pain threshold was not significantly different between the conditions, suggesting that the hot pack treatment could not attenuate the primary hyperalgesia. We found that the cold thermal pain threshold had a large variation between trials (i.e., the coefficient of variation is ranged from 12-27%)

comparing with 5-8% for the PPT. This may also be a reason for the non-significant difference between the hot pack and control conditions.

The effect of hot pack treatment was found for passive ROM of both flexion and extension, and active ROM for extension. A difference between active ROM and passive ROM is that the active ROM involves voluntary contraction of agonistic muscles, while no voluntary muscle contraction was involved in the passive ROM measures. The reductions in ROM-AE, ROM-AF, ROM-PF and ROM-PE following exercise were approximately 30%, 20%, 20%, 10%, respectively. It has been reported that decreases in active ROM after eccentric exercise are associated with muscle soreness, swelling, strength loss and muscle stiffness (Nosaka et al., 1997, 2007). The no significant effect of the hot pack treatment on the active flexion (ROM-AF) was probably because the muscles used for active wrist flexion (wrist flexors) were not treated by the hot pack. In contrast, during passive flexion (ROM-PF), the wrist extensors are stretched, and pain and stiffness caused by stretching might have affected the ROM, thus the hot pack treatment was found to affect ROM-PF. ROM-PE, which was least decreased after exercise, is more associated with flexibility of the wrist flexors that were not affected by the exercise, therefore the treatment on the wrist extensors had only minor effect.

The present study found prophylactic effect of hot pack on pain-free wrist extension strength. Immediately after exercise, wrist extensor strength decreased 52%, but returned to the baseline by 1 day post exercise. Slater et al. (2005) reported that wrist extensor strength decreased 34% immediately after the same exercise as that used in the present study (300 eccentric contractions). The magnitude of the strength loss immediately after exercise of the wrist extensors was

similar as that shown in other muscles such as approximately 60% reduction in elbow flexors (Nosaka et al., 2002) and approximately 40% reduction in knee extensors (Prior et al., 2001). It is generally accepted that muscle strength loss indicates the magnitude of muscle damage (Warren, et al., 1999), but the initial strength loss is not solely due to muscle damage, but a combination of fatigue and muscle damage (Miles and Clarkson, 1994). We found the significantly difference of pain-free wrist extensor strength between groups. It appears that the strength loss and fast recovery of wrist extension strength found in the present study indicate that the muscle damage was quite severe. Since no significant differences between the hot pack and control groups were evident for torque and total work during the eccentric exercise, it seems likely that the mechanical stress to the muscle was similar between conditions, and this resulted in the similar changes in strength between the hot pack and control conditions.

Regarding the magnitude of change, the observed attenuation in ROM (11º - 21º) and wrist extensor strength by the hot pack (40.5 N) appears to be clinically important for athletes or anybody who requires the specific tasks.

It should be noted that the present study investigated the effect of a single dose of a passive heating (i.e., hot pack) applied immediately prior to exercise on muscle damage of healthy young male individuals. Nosaka et al. (2007) reported that microwave treatment to increase muscle temperature over $40^{\circ}C$, 16-20 h prior to eccentric exercise attenuated the symptoms of muscle damage. It is possible that a combination of heat treatment at different time points prior to eccentric exercise provides a better prophylactic effect. It is important to note that the findings of this study was investigated only in 28 male healthy volunteers, and are not necessarily

applicable to the other groups of population (e.g. females, elderly individuals, clinical population) and other muscles such as elbow flexors and knee extensors. It should be also noted that the present study did not employ a measurement of muscle temperature, and the sample size might not be large enough to eliminate the type II error for some outcome measures (e.g., cold thermal pain threshold, muscle strength), since the sample size estimation was based on the PPT measures. A further study including a double-blinded experimental design is also warranted for strengthening the internal validity of the muscle damaged study.

5.3.4 A prophylactic effect of sauna on DOMS

The main finding of this study demonstrated some positive effects of sauna intervention on DOMS symptoms including passive ROM and pain-free muscle strength. However, the pain intensity and threshold was not show any different between the sauna and the control groups. The sauna group demonstrated a significantly lesser deficit in passive flexion range of the wrist than the control group on days 1 to 7. In the control group, the passive range of wrist flexion was significantly decreased when comparing the range achieved within the pre-exercise period to the first 5 days of post-exercise, whereas ROM-PF was only significantly decreased on day 2 post-exercise in sauna group. Measurement of the passive extension range of the wrist also demonstrated a lesser deficit in the sauna group on days 1 to 2. The ROM-PE in the sauna group did not significantly reduce from the baseline, while the control group took a few days to recover. Muscle soreness is commonly more painful and sensitive with stretching (Byrne et al., 2004). Passive range of motion can cause more pain especially in the opposite movement (i.e., flexion direction) of the damaged wrist extensor muscle and more compressed in

muscle belly when moved to the same direction (i.e., extension direction) of the damaged muscle. The two directions of movement lead to muscle guarding during function (Clarkson et al., 1992; Jones et al., 1987). This present study indicated less reduction of ROM-PF and ROM-PE in the sauna group. There was no significant difference in ROM-AF and ROM AE between the control group and the sauna group.

It seemed that pain-free muscle strength is more sensitive to detect the change of muscle damage than the maximal muscle strength. The pain-free grip strength demonstrated a lower deficit in the sauna group than the control group immediately post-exercise and on days 1 to 2. In addition, the pain-free wrist extensor strength had fewer deficits in the sauna group than control group immediately post-exercise and on days 1 to 3. Muscle strength is one of the best muscle damage indicators, which is normally reduced after exercise with slow recovery (Clarkson et al., 1992). The prevention of DOMS by using sauna has shown the beneficial effect on muscle strength of grip and wrist extension in this present study, as shown in less deficit of pain-free grip and wrist extensor strengths.

Regarding the magnitude of change, the observed attenuation in ROM (12º - 22º) and the grip and wrist extensor strength by the sauna (27 N and 40.5 N, respectively) appears to be clinically important for the athletes or people who required the specific tasks.

The results of this study were different from previous research in this area. For example, Nosaka et al. (2004) found that an increase of pre-exercise muscle temperature using a microwave heat source had no effect on muscle soreness, range of motion and muscle strength. Symons et al. (2004) also found no prophylactic effects of raising pre-exercise muscle temperature with continuous ultrasound on the muscle

damage symptoms. The localized nature of using microwave and ultrasound as the source of heat on the muscle, and the results achieved by using a sauna to heat the whole body, suggests the latter has more potential for muscle recovery. Another reason for the results may be due to the design employed of assessing muscle damage "between-subjects" as opposed to "within-subjects" as has been mentioned earlier that the within-subjects, arm-to-arm model may interfere to the study's results.

The application of sauna before exercise was used to warm up the muscles in general and thus prepare the target muscles for the prevention of DOMS symptoms. The effects of a sauna are increased body temperature, skin blood flow, and muscle relaxation (Kukkonen-Harjula and Kauppinen, 2006). The effects of warm up have been attributed to temperature-related mechanisms and non-temperature related mechanism. Temperature-related mechanism such as decreased resistance of muscle and joints, increased nerve-conduction, and altered force-velocity relationship, these factors could help the performance during exercise. Additionally, the research results show that non-temperature related mechanisms, for example increased blood flow to muscle, was enhanced as a result of using the sauna. Increased blood flow, CO_2-O_2 exchange rate, and the supply of nutrients to active structures could be beneficial for preparing and warming up the muscle before exercise (Bishop, 2003). The increased circulations also help: (a) to clear inflammatory mediators from the muscle tissue; (b) to improve the fatigue characteristics of skeletal muscle during exercise; and (c) to inhibit pain signals as the analgesic properties of increased blood circulation work to relieve muscle damage (Mayer et al., 2006).

It has been demonstrated that increased tissue temperature leads to more extensibility of collagen fibers in connective tissue. The increase in muscle temperature reduces muscle viscosity thereby resulting in smoother muscle contractions. The increased amount of elongation can occur without rupture, and potentially offers some protection against strain injury in warmed muscles (Mayer et al., 2006). Sauna has some effects on locomotor sytem i.e. muscles relax by reduced activity of the neuromuscular system, the joints become more flexible through altered physical properties of the fibrous tissues in the tendons and joint capsules. The tissues stretch more when warm. Heat enhances the circulation of the joint capsule and reduces the viscosity of synovial fluid (Kauppinen, 1997). It has also been suggested that tissue temperature increased by warm-up exercise would render the environment of muscle and connective tissue, less susceptible to eccentric exercise-induced muscle damage (Shellock and Prentice, 1985; Safran et al., 1988). In this present study the prior tissue hyperthermia showed the obvious effect on muscle functions. A systemic warm-up effect of sauna might have contributed to improve arm muscle motor adaptation. Among other factors, such a warm-up would imply a rise in body temperature and thus, increased speed of chemical reactions, oxygen uptake, and neural drive (Bishop, 2003). Moreover, Nosaka et al. (2007) has shown that passive hyperthermia treatment 1 day prior to eccentric exercise-induced muscle damage has a prophylactic effect. Heat has been mentioned for its prophylactic effect on muscle damage by increasing heat shock protein (HSP). This has the advantage of inducing muscle relaxation, decreasing muscle viscosity, and increasing connective tissue extensibility. In this respect HSP would be the explanation of the reduction of muscle damage after eccentric exercise. It has been proposed that HSP helps to

stabilize actin and intermediate filaments (the cytoskeleton in skeletal muscle) against stress during or after eccentric contractions (Koh, 2002). Further study is warrantee for investigating the HSP to confirm this notion.

5.3.5 Effectiveness of PNF-stretching, massage, hot pack and sauna on DOMS

For clinical application, effects on outcome measures following each preventative method should also be considered and taken into account to balance with the availability of the material. Regarding to the results of comparing interventions among PNF, massage, hot pack and sauna it seemed to demonstrate that the passive warm up by heating prior eccentric exercise provided more beneficial effect than using PNF or massage. For example, there was less deficit in ROM-PF of sauna group than the control group on day 1 post-exercise, and ROM-AF was less deficit in hot pack group than the PNF group on day 8 post-exercise. Moreover, GS pain-free showed the less deficit in sauna group than the PNF and massage group at immediately post-exercise. The explanation for this phenomenon is that the passive warm up by heating the tissues may be able to increase in the extensibility of soft tissue to protect the injury after exercise. Another explanation of protective mechanism of muscle is the creating of heat shock protein after pre-hyperthermia as it was mentioned in the study of Nosaka et al. (2007) and Koh (2002).

Unfortunately, there was no measurement of plasma CK in the preventative studies. Further studies of the EIMD should consider a tracking of CK for everyday to confirm the result of the EIMD precisely.