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ORIGINAL ARTICLE

Andrew B. Leger · Theodore E. Milner

Passive and active wrist joint stiffness following eccentric exercise

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Abstract The purpose of this study was to investigate the effects of exercise-induced muscle injury on passive and active wrist joint stiffness. Ten male subjects were repeatedly tested over a period of 11 days, once prior to, and four times following a bout of eccentric exercise with the wrist extensor muscles. Static wrist stiffness was measured by applying a 3° ramp and hold displacement of the manipulandum, which stretched the wrist extensor muscles. Wrist extension maximum voluntary contraction (MVC) declined by 24.5% from pre-exercise to 24 h after the exercise bout ($P < 0.001$). There was a reduced passive range of motion (ROM) from 82.8° pre-exercise to 70.2° on day 1 ($P < 0.01$), but no change in the passive joint stiffness at the neutral joint position, suggesting mechanical changes in the non-contractile tissues, or swelling that only resisted movement at the extremes of the ROM. Active joint stiffness at 50% pre-exercise MVC declined from 0.299 Nm deg^{-1} pre-exercise to 0.254 Nm deg^{-1} on day 1 ($P < 0.025$). Active joint stiffness at 10% pre-exercise MVC did not change on any of the days of testing compared to pre-exercise. These findings may indicate that large muscle fibers were more affected by the injury than small muscle fibers.

Key words Stiffness · Muscle injury · Wrist extensor muscles · Eccentric exercise

Introduction

Mild injury occurs frequently during eccentric exercise and is associated with muscle weakness, soreness, and the sensation of stiffness (Clarkson et al. 1992; Ebbeling and Clarkson 1989; Stauber 1989). Although commonly reported by subjects following exercise-induced muscle injury (Fridén et al. 1983; Jones et al. 1987; Kuipers 1994), stiffness has not been studied extensively. The sensation of stiffness has been described as a reluctance to stretch the affected muscle, and has been most commonly evaluated by measuring the resting position of the joint post-exercise (Clarkson et al. 1992; Howell et al. 1985; Jones et al. 1987). As an example, work by Stauber et al. (1990) has shown that the elbow angle of the relaxed arm in standing subjects becomes more acute following eccentric exercise of the elbow flexors. Immediately after exercise, this angle begins to decrease and continues to decrease until the 3rd day. The resting angle then gradually increases over the next week (Clarkson et al. 1992).

Various theories have been proposed to account for the increased stiffness and soreness. Initially, it was believed that muscle spasm was involved (DeVries 1966). Electromyography (EMG) recordings from the injured muscles during passive extension have since shown that this is not the case (Bobbert et al. 1986; Jones et al. 1987). Clarkson et al. (1992) and others (Ebbeling and Clarkson 1989) proposed that an influx or accumulation of calcium could activate specific enzymes and cause excessive contractures in the damaged fibers. Howell et al. (1985) felt that the restriction of motion and apparent decrease in resting length of the muscles was due to edematous changes in the perimuscular connective tissue. Stauber et al. (1990) concurred and proposed that the swollen tissue pushing against the fascia could shorten the muscles passively. However, the relationship between the time courses of swelling and of changes in resting arm angle has not supported this theory (Clarkson et al. 1992).

Quantification of muscle stiffness following exercise-induced injury has predominantly involved the evaluation of resting joint angle or amount of force required to move a joint through its full range (Clarkson 1992; Howell et al. 1985; Jones et al. 1987; Stauber et al.

A. B. Leger (✉)
School of Rehabilitation Therapy, Queen's University,
Kingston, Ontario K7L 3N6, Canada
e-mail: AL7@post.queensu.ca
Fax: +1-613-5336776

T. E. Milner
School of Kinesiology, Simon Fraser University,
Burnaby, British Columbia V5A 1S6, Canada

1990). Howell et al. (1993) quantified the stiffness of relaxed muscle (passive stiffness) by determining the relationship between static torque and elbow angle from the middle to the end of the range of motion. This procedure revealed an increase in passive stiffness that was quantified as an average value over the range of motion tested. Since the effects have tended to be greatest at the extremes of the range of motion (ROM), it is not clear whether the stiffness of relaxed muscle also increases significantly in the mid-range of motion where the joint is most frequently positioned during normal activities.

Mechanical stiffness, particularly the component associated with active muscle force, is recognized as being important in the normal control of posture and movement in humans (Bennett et al. 1992; DeSerres and Milner 1991; Hogan 1984; Kearney and Hunter 1990; Milner and Cloutier 1993; Milner et al. 1995). The sensation of increased muscle stiffness has been associated with exercise-induced muscle injury (Fridén et al. 1983; Jones et al. 1987; Kuipers 1994). However, it is unclear whether this sensation of stiffness is in any way related to mechanical stiffness. The mechanical stiffness of a joint is defined as the ratio of the change in joint torque to change in joint angle (Kearney and Hunter 1990). It is composed of three components that have different origins and can vary independently of one another. They are the elastic properties of non-contractile tissue (passive stiffness), the elastic properties of attached cross-bridges (intrinsic stiffness), and the reflex activation of a muscle following a change in length (reflex stiffness; Carter et al. 1990; Hoffer and Andreasson 1981; Nichols and Houk 1976; Sinkjaer et al. 1988; Toft et al. 1991).

Warren et al. (1993), in an attempt to separate the contribution of contractile elements and activation to the force deficit following eccentric exercise, calculated the total stiffness of the rat soleus muscle after it had been stimulated to tetanus. They showed that there was a similar pattern of decline in tension and muscle stiffness. This would suggest that the force deficit is due to fewer attached cross-bridges. However, this finding has not been corroborated under conditions where motor units are recruited in their normal order or at force levels below maximum voluntary contraction (MVC), by measuring the joint stiffness of a voluntarily activated muscle after the induction of exercise-induced muscle injury. From histological evidence, it appears that type II muscle fibers are more susceptible to injury than type I fibers during eccentric exercise (Fridén et al. 1983; McCully and Faulkner 1986; Stauber et al. 1990). If type I muscle fibers are much less affected by eccentric exercise than type II muscle fibers, it is also likely that muscle stiffness at low levels of muscle force will not change as a result of eccentric exercise. However, muscle stiffness may change at levels of muscle force requiring large type II muscle fibers.

The purpose of this study was to investigate the changes in mechanical parameters following exercise-induced muscle injury. These include passive joint stiffness at a mid-range neutral position and active joint stiffness at low and high levels of muscle force.

Methods

Subjects

Ten normal male subjects participated in this study [mean (SD) age 27.3 (4.7) years]. Eight of the subjects were right-handed, the other two were left-handed. None of the subjects reported any previous history of neuromuscular disorders. Each gave their written informed consent to participate prior to the experiment. None had previously participated in any studies involving eccentric exercise of the muscles of the forearm. Subjects were asked not to participate in any weight-training activities, specifically for the upper extremities, for the duration of the study. The experiment was approved by the University Research Ethics Review Committee.

General design

Subjects were tested on five separate occasions, previous to exercise-induced muscle injury (pre-injury), 24 h (day 1), 48 h (day 2), 96 h (day 4), and 240 h (day 10) after muscle injury. The manipulandum was designed to test one side only. All testing was performed on the left hand. The left hand was chosen because it was more likely to be the non-dominant hand. Those for whom the left hand was dominant were not eliminated as they acted as there own control and all comparisons were made to themselves.

Apparatus

A torque motor (PMI U16M4), which was coupled to the manipulandum, was used to generate loads that were computer controlled. The torque motor was used to produce offset torque during an initial holding, period and a servo-controlled ramp was used to control angular displacement. The maximum torque that could be produced by the torque motor was 5 Nm. In tasks that required a torque greater than 5 Nm, a stiff spring (6.55 N cm^{-1}) was attached to the manipulandum. When the spring was stretched it produced a large torque that opposed wrist extension.

Position and velocity were measured using a potentiometer and a tachometer, respectively, which were attached to the motor shaft. The torque was measured by a linear strain gauge that was mounted on a cylinder, coupling the motor shaft to the manipulandum. The EMG was recorded using active, bipolar, stainless steel, surface electrodes (Liberty Mutual MYO 111), with electrode contacts 3 mm in diameter and 13 mm apart. A diagrammatic representation of the apparatus used is shown in Fig. 1.

Recording

EMG activity was recorded from the six forearm muscles that would contribute to the torque produced at the wrist: the extensor carpi ulnaris (ECU), extensor carpi radialis longus (ECRL), extensor digitorum communis (EDC), flexor carpi ulnaris (FCU), flexor carpi radialis (FCR), and flexor digitorum superficialis (FDS). Optimal placements of the electrodes on the 1st day of testing (pre-injury) were determined by observing the EMG activity during brisk test movements. These included: ulnar deviation and wrist extension for the ECU, ulnar deviation and wrist flexion for the FCU, finger flexion for the FDS, finger extension for the EDC, wrist flexion (fingers relaxed) for the FCR, and wrist extension (fingers relaxed) for the ECRL. Each electrode was positioned to maximize the signal obtained during the appropriate movement, and to minimize the signal obtained during others. The position of the electrode on the skin was marked with indelible ink to allow reproducible positioning of electrodes on days subsequent to the

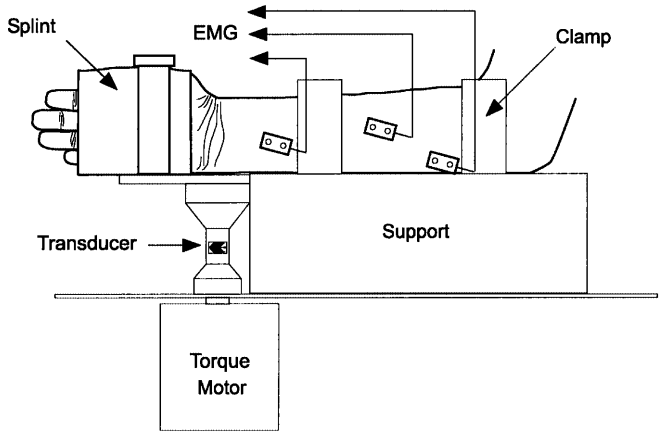

Fig. 1 Experimental setup for the left wrist. The hand and fingers were splinted and secured to the manipulandum such that the axis of rotation of the wrist joint was directly over the shaft of the torque motor. A torque transducer measured motor shaft torque. Angular position was measured with a potentiometer that was positioned in line with the shaft of the torque motor shaft. (*EMG* electromyogram)

induced injury. Each day the recorded signal was tested by the above procedure. Active bipolar electrodes with high-input impedance and a small interelectrode distance were used, which minimized the effects of electrode/tissue interface impedance, crosstalk from other muscles, and pick-up from distant sources (de la Barrera and Milner 1994). Signals were bandpass filtered from 45 to 550 Hz, amplified and digitized at 2 kHz, and stored on disk for later analysis. The position, velocity and torque of the wrist were amplified and digitized at 2 kHz, and then stored on disk.

Exercise protocol

The exercise protocol for the wrist extensors was performed with free-weights on an apparatus designed in the laboratory. The subject's forearm was supported on an inclined plane. Free weights were attached via a pulley system to the hand of the subject such that only the wrist extensors were used to support the weight. Subjects initially completed a regime of concentric-eccentric wrist extension exercises with weights of 4.5, 3.4 and 2.3 kg. Repetitions were performed with each weight until subjects were unable to complete a cycle. This regime was repeated three times with 5 min of rest between each set. Following this series of exercises, the subject performed a set of eccentric exercises only. The subject lowered free-weights of 5.7, 4.5, 3.4 and then 2.3 kg. One weight was used until the subject was unable to lower it in a controlled continuous manner. At this point, the weight was exchanged for the next smaller weight. Following each eccentric contraction, the weight was returned to the starting position by the experimenter. The subject was encouraged to move the wrist through the entire ROM, and to perform as well as possible. This protocol was designed to exhaustively exercise the wrist extensor muscles such that exercise-induced muscle injury occurred. The free-weights were chosen such that all subjects could perform the protocol in a progressively taxing manner, and so that the eccentric component of the protocol was emphasized.

Testing procedures

Following thorough cleaning with alcohol and mild abrasion of the skin, EMG electrodes were placed over the designated muscles. Each subject was seated comfortably in a chair with the left forearm secured and supported at comfortable height and elbow angle. The forearm and wrist were orientated midway between pronation and supination. The hand and fingers were splinted and the splint was rigidly fixed to a manipulandum. The manipulandum allowed movement of the wrist in the flexion-extension direction only. The hand was positioned such that the axis of rotation of the wrist joint was directly over the shaft of the motor (Fig. 1).

Most subjects were familiar with the experiments performed on the manipulandum. Those subjects who were not familiar with a particular task were given the opportunity to practice prior to data collection. To avoid fatigue during the testing session subjects were given at least 30 s rest between each trial, and up to 5 min between different tasks.

The following experiments were performed:

1. Range of motion. The mid-range neutral position of the wrist was determined pre-injury and was used in subsequent experiments on each day of testing. The subject's wrist was passively moved into flexion and then extension on the manipulandum. The joint angle, measured from the neutral position, at which the resistance to movement was 0.5 Nm, was determined for both directions. The total ROM was the sum of the two angles. Three trials were performed.
2. MVC – extension. The manipulandum was locked at the neutral position of the wrist and the subject performed a sustained (up to 3 s) maximal isometric contraction. There was a warm-up period prior to data collection, which involved a series of submaximal contractions. Data were collected from three trials performed at maximum effort.
3. Passive joint stiffness. The manipulandum was set at the neutral position of the wrist and the subject was instructed to relax completely. Once relaxed, the experimenter triggered a small ramp displacement of 3° in 30 ms in the flexor direction. Any trials in which there was evidence of EMG activity or torque prior to the displacement were not accepted. Five acceptable trials were saved.
4. Active joint stiffness. The manipulandum was set at the neutral position of the wrist and the target window on the computer screen was set to one of two torque levels, 10% of pre-injury wrist extension MVC ± 0.5%, or 50% of pre-injury wrist extension MVC ± 2.5%. The subject was instructed to contract the wrist extensors and move a cursor representing torque into the target window on the computer screen. Once the subject had held the cursor in the target window for a random period of time ranging from 1 to 2 s, the experimenter triggered a ramp displacement, as above. The subject was instructed not to respond to the displacement. Trials in which there was evidence of voluntary intervention were not accepted. Five acceptable trials per torque level were saved.

Analysis

To determine the total ROM, each of three trials was averaged. MVC was determined as the highest average torque developed over an interval of 125 ms. To quantify joint stiffness, the five recorded trials were averaged. From the averaged data, mean values of position and torque were calculated for the interval 0–125 ms before the onset of displacement, and for the interval 100–200 ms after the onset of displacement. These intervals were chosen based on the observation that during these periods the torque and position were constant. The joint stiffness was calculated as the ratio of the change in mean torque divided by the change in mean position.

The EMG for each of the three wrist extensor muscles was rectified and averaged for the five chosen trials in each task. The mean rectified EMG (MEMG) was calculated for the interval 0–125 ms before the stretch, as a measure of background activity. Three consecutive intervals beginning 20 ms following the onset of displacement, at short latency (20–60 ms), intermediate latency (60–100 ms) and long latency (100–150 ms) were used for analysis of reflex responses. These intervals were chosen to represent the time of occurrence and duration of the myotatic, late myotatic, and post-myotatic reflex responses (DeSerres and Milner 1991; Jaeger et al. 1982; Milner et al. 1995). The change in MEMG following the displacement was computed for each interval by subtracting the

MEMG prior to displacement (background) from the MEMG over the reflex interval. This is expressed as a percentage change by dividing by the background value. The background MEMG was also determined for the wrist flexors.

Statistics

To test for significant differences over time, individual parameters were analyzed using a repeated-measures analysis of variance design. The level of statistical significance for post-hoc comparisons was set at $P < 0.05$.

Results

The exercise protocol in the present study involved repeated eccentric contractions by the wrist extensors with submaximal loads, performed until the subject was performing maximally and was unable to move in a controlled continuous manner. The exercise bout varied between subjects, involving approximately 150 repetitions and lasting 25–30 min.

Wrist extension isometric MVC changed significantly during the days of the study ($F_{(4,36)} = 13.47$, $P < 0.0001$). It declined by 24.5% from 12.5 Nm pre-exercise to 9.4 Nm on day 1 ($P < 0.001$). It remained significantly lower on day 2 compared to pre-exercise values ($P < 0.005$). Figure 2A shows the recovery of wrist extension MVC back to pre-exercise values by day 10.

Passive wrist ROM dropped significantly after the exercise bout ($F_{(4,36)} = 7.73$, $P < 0.001$). It declined from 82.8° pre-exercise to 70.2° on day 1 ($P < 0.005$). The passive ROM remained significantly lower on day 2 at 71° ($P < 0.01$), but then continually increased from day 4 to day 10, back to pre-exercise values (Fig. 2B).

Passive wrist joint stiffness measured at a single neutral joint position did not change as a result of eccentric exercise-induced muscle injury (Fig. 2C), nor was there any significant change in the MEMG background activity of the wrist extensor muscles during the passive joint stiffness experiment (Fig. 3).

Active wrist joint stiffness at 10% of pre-exercise wrist extension MVC did not differ significantly on any day of testing compared to pre-exercise ($n = 6$) (Fig. 2C). It is presumably at this torque level that mostly small motor units would have been active. Although there was no change in joint stiffness, the

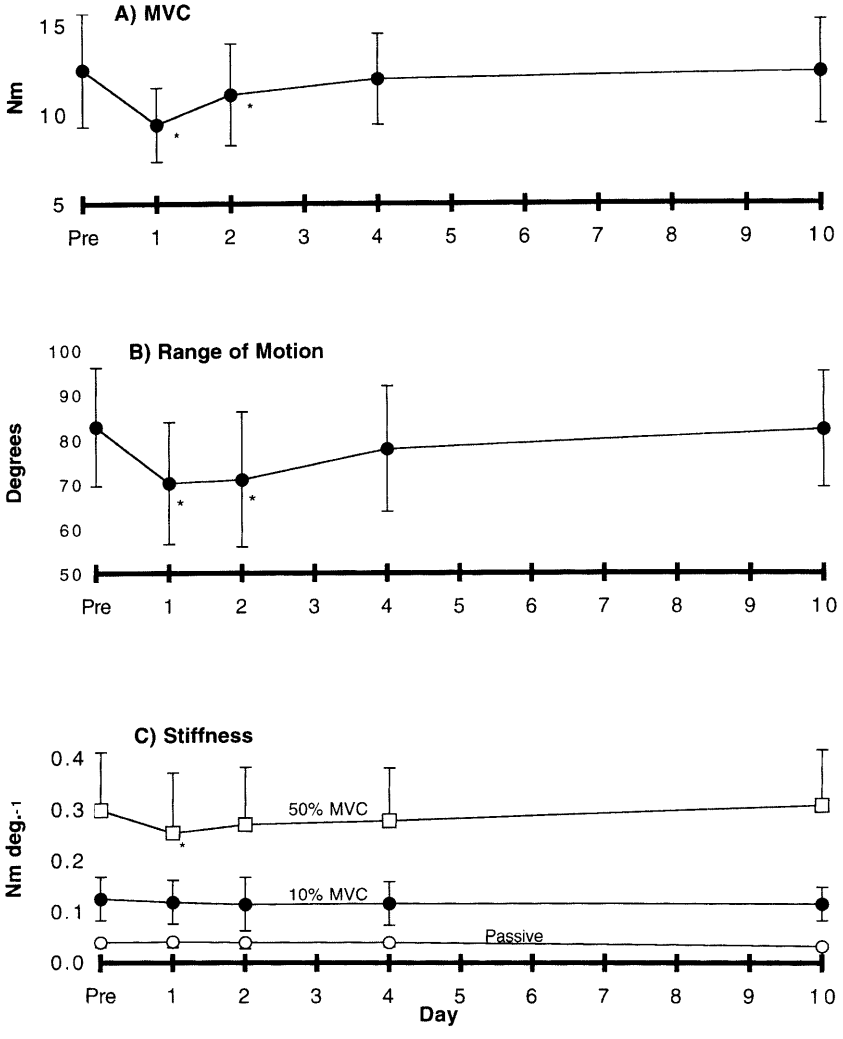

Fig. 2 **A** Maximal wrist extension torque (maximal voluntary contraction, *MVC*). **B** Passive wrist range of motion (*ROM*), **C** wrist joint stiffness. Values are means ± SD. *Significantly different than pre-exercise

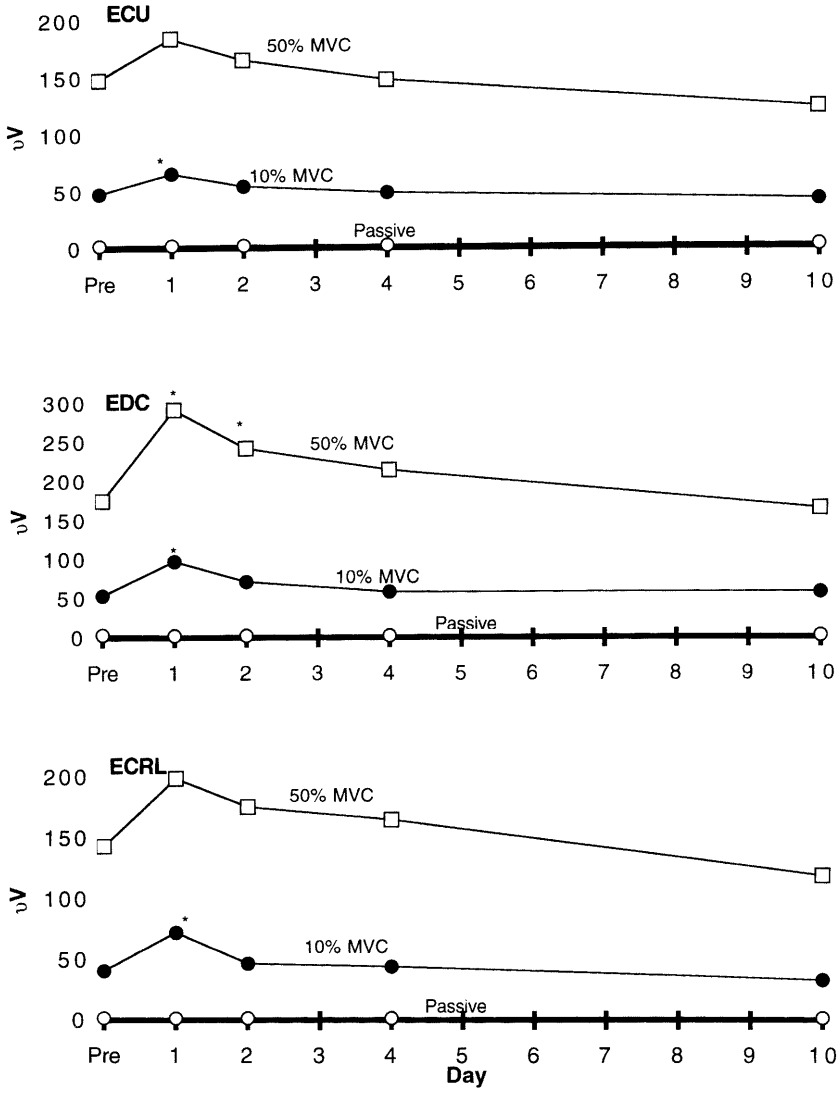

Fig. 3 Background mean integrated EMG (MEMG) of the wrist extensors muscles. *Significantly different than pre-exercise. (*ECU* extensor carpi ulnaris, *EDC* extensor digitorum communis, *ECRL* extensor carpi radialis longus)

background MEMG of the ECRL muscle was significantly greater on day 1 compared to pre-exercise ($P < 0.005$), however it was found that the short-latency reflex response in this muscle was significantly lower on day 1 compared to pre-exercise ($P < 0.0005$). The background MEMG of the EDC and ECU muscles was also significantly greater on day 1 compared to pre-exercise ($P < 0.0001$ and $P < 0.025$, respectively; Fig. 3). However, the reflex responses for the EDC or ECU muscle were not different following injury (Fig. 4A). All three wrist extensor muscles showed a similar pattern of change and recovery during the 10 days of testing.

Active wrist stiffness at 50% pre-exercise wrist extension MVC declined following the exercise protocol ($F_{(4,36)} = 2.56$, $P < 0.05$). It declined by 15% from a pre-exercise value of 0.299 Nm deg^{-1}, to 0.254 Nm deg^{-1} on day 1 ($n = 7$, $P < 0.025$). Figure 2C shows that by day 2 through day 10 the values of active wrist joint stiffness at 50% pre-exercise MVC had returned to pre-exercise values. Accompanying the decline in active joint stiffness, the background MEMG of the EDC muscle was significantly greater on day 1 and day 2 compared to pre-exercise ($P < 0.005$ and $P < 0.01$, respectively). The background MEMG of the ECRL and ECU muscles was also greater on day 1. However, the difference was not significant. The pattern of increase in background MEMG and recovery during the period of testing was similar for all three wrist extensors (Fig. 3). The background MEMG of all three wrist flexor muscles was also greater on day 1 compared to pre-exercise, indicating more co-contraction of the antagonist muscles ($P < 0.05$, respectively) (Fig. 4B). The short-latency reflex response of the EDC muscle was significantly lower on day 1 compared to pre-exercise ($P < 0.025$). It was also lower in the other two wrist extensors, but the difference was not significant (Fig. 4A).

Discussion

The purpose of this study was to investigate the changes in mechanical parameters that occur following exercise-

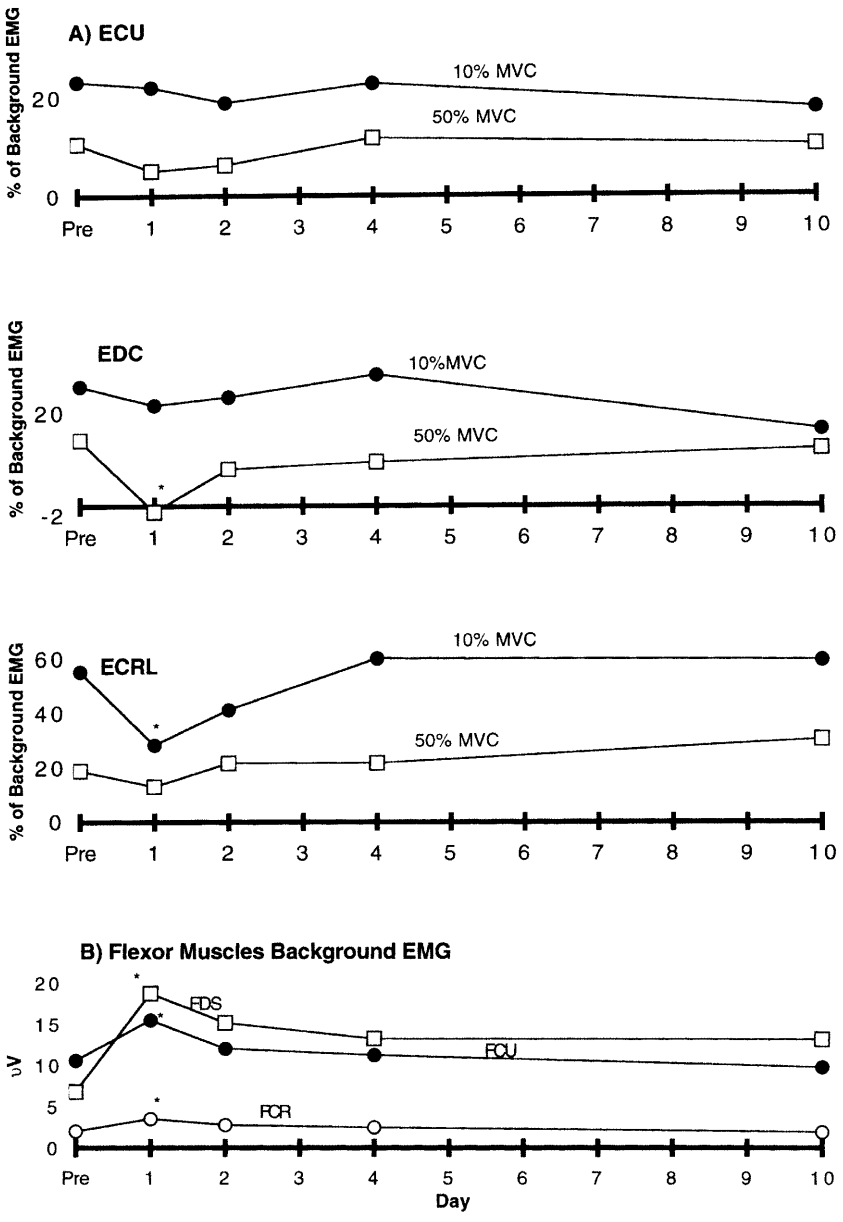

Fig. 4 **A** Short-latency reflex response of the wrist extensor muscles. **B** Background MEMG of the wrist flexor muscles at 50% of pre-exercise wrist extension MVC. *Significantly different than pre-exercise

induced muscle injury. These included passive joint stiffness at a mid-range neutral position, and active joint stiffness at low and high levels of muscle force. Evidence of injury to these muscles included a reduced range of passive wrist movement, and reduced maximal isometric wrist extension torque. These symptoms are similar to those that have been reported in other studies of exercise-induced muscle injury where damage to muscle fibers has been confirmed by histological and ultrastructural analysis (Newham et al. 1987; Stauber et al. 1990). The time course of recovery for parameters evaluated in the present study was also similar to that reported after injury to other muscle groups (Crenshaw et al. 1994; Howell et al. 1993; Jones et al. 1986; MacIntyre 1994; Stauber et al. 1990). Therefore, there is compelling evidence that injury to muscle fibers results from the exercise protocol used in the present study.

Clarkson et al. (1992) reported findings from 109 subjects in a review of the changes that occur in the elbow flexor muscles following an exercise regime of 2 sets of 35 maximal eccentric contractions. Their subjects showed a dramatic loss of isometric strength of up to 50% 1 day after the exercise bout, which was gradually restored such that by 10 days after exercise only a small deficit remained. The exercise protocol used in the present study was at least as strenuous as that used by Clarkson et al. Although the loss of isometric strength in the wrist extensors in the present study was only 24.5% 1 day after the exercise bout, it recovered in a similar manner, such that by day 10 no deficit remained.

The passive range of wrist motion was reduced by 12.6%. This decline is analogous to the change in relaxed joint angle reported by Clarkson et al. (1992) and others (Howell et al. 1985; Jones et al. 1987; Stauber

et al. 1990). These measures illustrate the effects of injury at the extremes of the ROM. Overall, these findings are consistent with the expected effects of an exercise-induced muscle injury to the wrist extensors, resulting from damage to muscle tissue.

The reduced ROM could indicate increased resistance to stretch of connective tissue, as has been suggested in other studies in which no increase in muscle electrical activity during passive joint movements have been reported (Bobbert et al. 1986; Jones et al. 1987). Stauber et al. (1990) and Howell et al. (1985) have suggested that there is a decrease in the resting length of muscle, caused by swollen tissue pushing against the fascia and causing the muscle to shorten passively. Bobbert et al. (1986) have reported swelling and increased circumference of the lower leg after eccentric exercise of the gastrocnemius muscle, with a time course similar to the decreased ROM reported in the present study. Although swelling was not measured, there is reason to believe from these other studies that it occurred and contributed to reduced ROM in either direction, due to increased internal pressure.

Quantification of passive joint stiffness by means of resistance to a quick stretch at the neutral position of the wrist showed no change after muscle injury. This finding would suggest that there was no change in the mechanical properties of the non-contractile tissue of the muscle in the mid-range position. Although increased stiffness and difficulty moving a joint have been reported to occur following exercise-induced muscle injury (Howell et al. 1985; Jones et al. 1987; Stauber et al. 1990), the findings of the present study indicate that there is increased resistance to movement only at the ends of joint ROM. Since the mechanical properties of non-contractile tissue appear to be unaffected by eccentric exercise, this resistance is more likely to be due to swelling within the tissue.

No previous studies have quantified active wrist joint stiffness following muscle injury. Wrist joint stiffness at 10% of pre-injury MVC in the present study did not change after the muscle injury. However, the joint stiffness at 50% of pre-injury MVC was lower 1 day after the exercise bout. Following injury, there was an increase in the extensor muscle background MEMG at 50% of MVC, which was most evident on day 1. This elevated extensor muscle activity was accompanied by greater activity in the wrist flexor muscles on day 1. Such an increase in co-contraction would normally result in greater joint stiffness. However, this was not the case.

The wrist extensor and flexor muscles are composed of approximately 50% slow oxidative and 50% fast glycolytic muscle fiber types (Johnson et al. 1973). It is expected that by 50% of MVC the large fast-twitch motor units would have begun to be recruited. The decline in wrist joint stiffness that occurred despite the increased muscle activity supports the hypothesis that there were changes in the mechanical properties of large motor units, which are required for large torques, and that also affected the muscle stiffness. Had there been less co-contraction of the wrist flexor muscles, the joint stiffness would probably have decreased following injury.

Howell et al. (1993) quantified elbow joint stiffness over a range from intermediate to end-range joint positions, and reported a significant increase following eccentric exercise. They suggested that increased stiffness near the end-range of motion was due to swelling, and that the increased stiffness in the mid-range positions was due to muscle cell injury. The present study does not rule out the possibility of swelling causing increased joint stiffness at the end of ROM. It does, however, show that if swelling was present it was not sufficient to cause measurable changes in stiffness in the mid-range joint positions, and thus does not affect the ability to move at these joint positions.

The lack of change in active joint stiffness for low torque suggests that the muscle injury did not affect the mechanics of the small motor units required for low torques. Contrary to the findings for low torque, the active joint stiffness for high torque was significantly lower 24 h after exercise-induced muscle injury. The decrease in stiffness for high torque may have been due, in part, to lower reflex stiffness, since the reflex EMG was reduced. The finding that only the short-latency reflex responses were affected suggests that the changes associated with the decline in reflex excitation occurred at the spinal cord level. A decline in the short-latency reflex response has been reported to occur after fatiguing contractions (Balestra et al. 1992; Duchateau and Hainaut 1993). It was suggested that this decline was mediated by group III and IV receptors, which are sensitive to the metabolic and mechanical changes associated with muscle fatigue. It is possible that these receptors are activated by injury to the large muscle fibers during eccentric exercise, leading to presynaptic inhibition of the stretch reflex.

It should also be noted that the intrinsic stiffness also appeared to be lower for a number of subjects, since the torque was lower both during and following displacement of the wrist. This would suggest that muscle fibers have a lower stiffness to force ratio following injury.

The presumed greater proportion of injury to the large motor units is in agreement with other studies that have provided morphological evidence of preferential damage of the large type II fibers (Fridén and Leiber 1992; Fridén et al. 1983; Jones et al. 1986). Nardone and Shieppati (1988) and others (Dick and Cavanagh 1987; Moritani et al. 1988) have suggested that this preferential involvement is due to different patterns of use during eccentric exercise. However, this does not preclude the possibility of greater intrinsic susceptibility to damage of the type II fibers. The results of this study do not address this issue, but they are consistent with the occurrence of a mechanical deficit in the large fibers following eccentric exercise.

In summary, the purpose of this study was to examine changes in mechanical wrist joint stiffness following exercise-induced muscle injury. It was shown that under passive conditions there were no changes in joint stiffness at mid-range joint positions. This study is the first

to quantify joint stiffness at a single mid-range joint position, where the joint is most frequently positioned during normal activities. The findings suggest that there were no changes in the mechanical properties of the non-contractile tissue at the mid-range of joint motion, and that the commonly reported sensation of stiffness would have no effect on the ability to function or move in neutral joint positions. The decline in passive joint ROM in the wrist suggests that the resistance to movement found at the ends of ROM might have been due to swelling. However, this was not specifically measured.

Quantification of active joint stiffness revealed no changes in joint stiffness while the wrist extensors were producing low torque. This suggests that the mechanical properties of the small slow-twitch motor units, which would have been active during these tests, were unaffected by the eccentric exercise. On the other hand, there were significant changes in joint stiffness while producing high torque. It is at these levels of torque that the large fast-twitch motor units would be assumed to be active. The decline in active joint stiffness at high torque thus suggests that the mechanical properties of the large motor units had changed as a result of eccentric exercise. These findings support the previously reported specificity of injury to the large muscle fibers.

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