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-contraceptive 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 Staub er 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. Staub er 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; Staub er et al.
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 crossbridges (intrinsic stiffness), and the reflex activation of a muscle following a change in length (reflex stiffness; Carter et al. 1990; Hoff er 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.

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**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 hold, 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