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Changes in hardness of the human elbow flexor muscles after eccentric exercise

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Abstract The purpose of this study was to investigate changes in muscle hardness after eccentric exercise of the elbow flexors muscles that produce muscle shortening and swelling. To assess muscle hardness, a pressure method was used in which the force required to deform the tissue (skin, subcutaneous tissue, muscle) was recorded. Eleven healthy male students performed 24 maximal eccentric actions of the elbow flexor muscles with their non-dominant arms. Muscle hardness, maximal isometric force (MIF), muscle soreness, plasma creatine kinase (CK) activity, relaxed elbow joint angle (RANG), upper-arm circumference (CIR) and B-mode ultrasound transverse images were measured before, immediately after, and 1–5 days after exercise. A long-lasting decrease in MIF, muscle swelling shown by increases in CIR and muscle thickness, large increases in plasma CK activity, and development of muscle soreness indicated that damage occurred to the elbow flexor muscles. The RANG had decreased by approximately 20° at 1–3 days after exercise and showed a gradual recovery thereafter. The CIR increased gradually after exercise and peaked on day 5 post-exercise, the mean amount of increase in CIR being 18 mm. Muscle hardness measured at the relaxed elbow position did not change until 3 days after exercise, but increased significantly ($P < 0.01$) on days 4 and 5 post-exercise. On the other hand, muscle hardness measured when forcibly extending the shortened elbow joint increased significantly ($P < 0.01$) with time and peaked at 3 days after exercise. Muscle hardness assessed by the pressure method seems to reflect changes in muscle stiffness and swelling.

Key words Muscle stiffness · Muscle damage · Muscle shortening · Swelling · Pressure method

Introduction

Unaccustomed eccentric exercise damages muscle and connective tissue, and has been shown to cause inflammatory responses subsequently (Armstrong et al. 1991; Smith 1991). Development of muscle soreness, long-lasting decrease in force-generating ability, increases in muscle proteins in the blood, decrement of range of motion, and muscle swelling have all been considered outcomes of exercise-induced muscle damage (Ebbering and Clarkson 1990; Clarkson et al. 1992). Following eccentric exercise of the elbow flexor muscles, relaxed elbow joint angle has been found to decrease significantly immediately after exercise, and an additional decrease was observed for 3 days after exercise (Clarkson et al. 1992). The mechanism underlying the decrease in relaxed elbow joint angle has not been fully elucidated, but a shortening of the connective tissue due to swelling and/or a spontaneous contraction of muscle fibres (contracture) have been postulated (Clarkson et al. 1992). It has been demonstrated that the decrease in relaxed elbow joint angle is also related to increased muscle stiffness (Jones et al. 1987; Howell et al. 1993; Chleboun et al. 1998). Jones et al. (1987) measured the force required to extend the flexed elbow joint after eccentric exercise of the elbow flexor muscles and showed that muscle stiffness increased after exercise. Howell et al. (1993), and more recently Chleboun et al. (1998), have also reported increases in muscle stiffness after eccentric exercise of the elbow flexor muscles. They used a device that extended the elbow joint and recorded

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the torque required to hold the forearm at successive angles.

Physiotherapists and/or massage therapists often examine the condition of muscle by palpation. In our experience for several days after exercise, palpation of a *stiff* muscle after eccentric exercise has suggested to us that the muscle and surrounding tissues have become *harder* than in the pre-exercise condition. It has been reported that muscle becomes harder in a pathological condition such as spasm, cramps, myopathy, and oedema (Fischer 1987). Muscle stiffness is defined as the instantaneous dependence of tension on length, and has been described by the slope of the tangent to the length-tension curve (Woledge et al. 1985). Therefore, the increased tension of a *stiff* muscle may produce greater resistance when pressure is applied to the fibre transcutaneously, just like pressing a tightened string. A method of assessing muscle hardness in a similar way to palpation should be established, which could then be applied to delineate what muscle hardness reflects. However, changes in muscle *hardness* after eccentric exercise have not been systematically evaluated.

Several non-invasive methods of measuring human muscle hardness have been proposed, such as a quick-release method (Pousson et al. 1990), an impedance method using resonance frequency (Wilson et al. 1994), and a pressure method (Fischer 1987; Horikawa et al. 1993; Komiya et al. 1996). To assess muscle hardness, Horikawa et al. (1993) elucidated the relationship between the application of pressure to human muscles and the amount of distortion of the muscle. Sakai et al. (1995) applied this pressure method to evaluate the hardness of the pericranial muscles in relation to tension-type headaches. This method has an advantage over other methods, because it resolves hardness values into subcutaneous and muscle areas using a two-layer spring model.

Therefore, the purpose of this study was to apply this pressure method to evaluate changes in muscle hardness following eccentric exercise of the elbow flexor muscles. It was hypothesized that muscle hardness would increase as muscle stiffness increases after eccentric exercise. It might also be possible that muscle hardness would be affected by muscle swelling after eccentric exercise, because accumulation of water caused by oedema would increase internal pressure.

Methods

Subjects

Eleven male students (aged 18–23 years) were used as subjects. Their mean age, height, and body mass were 20.1 (SD 1.5) years, 1.71 (SD 0.04) m, and 60.4 (SD 3.5) kg, respectively. They had not been involved in any resistance-training programme before this study. All the subjects signed an informed consent form consistent with the Yokohama City University policy for the protection of human subjects.

Exercise

The subjects performed 24 maximal eccentric actions of the elbow flexor muscles with their non-dominant arms. The other arm did not perform any exercise and served as an internal control. In each eccentric action, the subjects were asked to generate maximal isometric force at the starting position (1.57 rad, 90°) for 1 s. The forearm was then forcibly extended from an elbow flexed (1.57 rad) to an elbow extended (3.14 rad, 180°) position in 5 s on a modified arm-curl machine, and this action was repeated every 15 s as has been described by Nosaka and Clarkson (1996). During the eccentric action, the force of the elbow flexor muscles transduced at the wrist was measured by a load transducer (9E01-L43, NEC Sanei, Japan) installed in a specially designed wrist attachment and monitored and recorded using a digital indicator (F360A, Unipulse Corp., Japan). The peak force of each eccentric action was displayed on the digital indicator to motivate the subjects to generate maximal force.

Indicators of muscle damage

The following indicators were used to evaluate the degree of muscle damage: maximal isometric force, relaxed elbow joint angle, muscle soreness, upper-arm circumference, B-mode ultrasound images, and plasma creatine kinase (CK) activity. Maximal voluntary isometric force of the elbow flexor muscles at an elbow joint angle of 90° was measured for 3 s using a dynamometer. Relaxed elbow joint angle was measured using a goniometer in two ways: when the subject was standing with his arm relaxed at his side – the method which has been used by Nosaka et al. (1991) and by Nosaka and Clarkson (1996) – and when the subject was lying on a bed and with his upper arm relaxed on the bed. In the latter measurement, the forearm was supported with a towel to maintain the elbow joint angle in a completely relaxed position when the elbow joint angle decreased. This was necessary for the accurate measurement of muscle hardness, by stabilizing the elbow flexor muscles and the forearm position. The perception of muscle soreness was assessed using a visual analogue scale in which a 50 mm line (0 = no pain, 50 = very sore) was used. This evaluation has been used successfully in previous studies (Nosaka and Clarkson 1994, 1996, 1997). Upper-arm circumference was measured using a tape measure at four marked sites (40, 60, 80, and 100 mm above the elbow joint) on the upper arm when the subject let his arm hang down at his side. All measurement sites for the elbow angles and upper-arm circumference were marked with a semi-permanent marker, and the same investigator took the measurements throughout the experiment. An ultrasonography machine (SSD-500, Aloka Co. Ltd., Japan) was used to obtain transverse and longitudinal B-mode ultrasound images from the elbow flexor muscles, and changes in muscle thickness of the biceps brachii and the brachialis muscles, as well as the echointensity of the muscles, were evaluated.

Approximately 5 ml of blood was collected from an antecubital vein in heparin-coated tubes (Termo Co. Ltd., Japan) and centrifuged for 10 min to obtain plasma. The plasma samples were frozen and stored at −20° C until analysis. Plasma CK activity was measured in duplicate using an ultraviolet test kit (Dinabot Co. Ltd., Japan). All muscle damage indicators, except muscle soreness and plasma CK activity were taken before and immediately after and once a day for 5 days after exercise. Muscle soreness and plasma CK activity were not measured immediately after exercise.

Muscle hardness

The muscle hardness was measured by a pressure method based on the study by Horikawa et al. (1993). The device used to assess muscle hardness is shown in Fig. 1. The sensor deformed the tissue at a constant rate and simultaneously detected the responding force from the tissue. A linear motor vibrator system (DPM-270: Dia Medical Systems Co. Ltd., Japan) which had an electrical feedback servomechanism was used to deform the belly of the biceps brachii

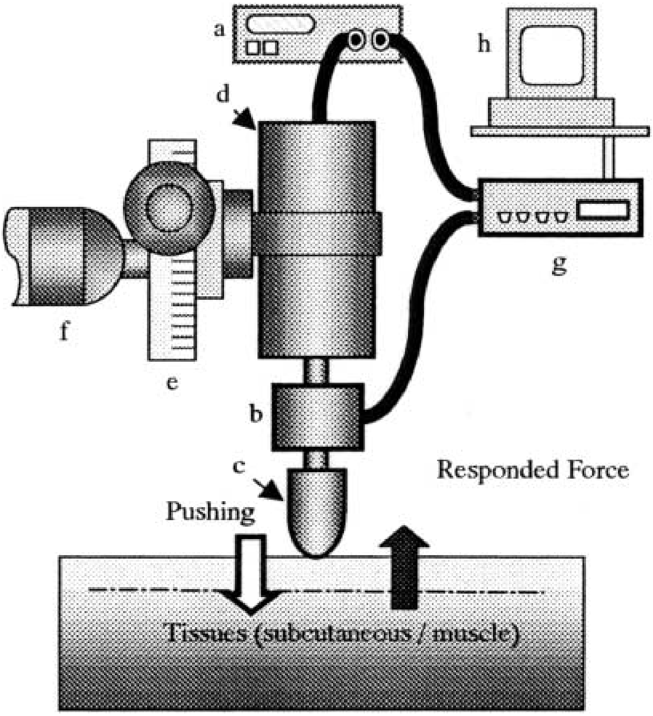


Fig. 1 Diagram of the system for measuring muscle hardness. Mechanical stimulator unit (*a*) controlled the pressure speed (2.5 mm · s^{-1}) and distance (8 mm) of the axle of the vibrator, which was connected to the strain gauge force transducer (*b*) and the plastic displacement probe (*c*). The vibrator (*d*) was coupled to the manipulation device (*e*) and ball and socket head (*f*) for the depth and angle adjustment for the target measurement point of the tissue. Displacement and the force response were recorded using the data recorder (*g*) and computed by the A–D converter and data acquisition system (*h*)

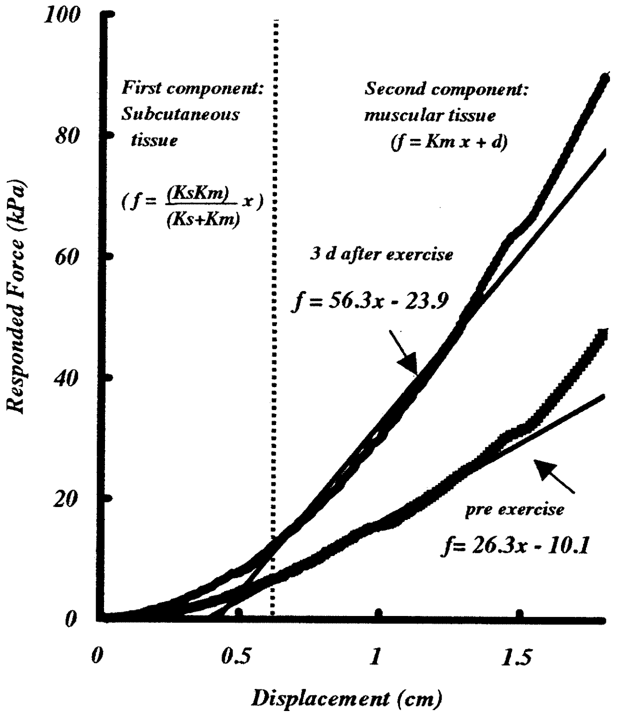


Fig. 2 Relationship between the displacement of the tissue and the force response. A typical shift of the displacement (x)-force (f) curve from pre-exercise to 3 days after exercise is shown in the figure. The muscle hardness value (E) was calculated from the equation: $E = Id(1 - \mu^2)K_m$ (where I is the influence coefficient (0.85), d is the diameter of the displacement probe, and μ is Poisson's ratio (0.5). According to a two-layer spring model (Horikawa et. al. 1993), K_s and K_m represent, respectively the individual hardnesses of subcutaneous tissue and of muscle. The K_m of pre-exercise and 3 days after exercise were 26.3 and 56.3, respectively, from the regression line of each curve. Therefore, the E value of pre-exercise was 13.4 kPa, and 3 days after exercise it was 28.7 kPa

muscle. The vibrator included a variable inductance displacement-frequency converter to detect the linear motor displacement of the axle. The axle of the vibrator moved linearly in the centre of a cylindrical magnetic coil. A strain gauge force transducer and a plastic probe (diameter = 8 mm) were screwed to the top of it. The vibrator could be adjusted to the appropriate angle against the muscle belly by means of a ball socket head. The vibrator system also included a generator of input pulse that controlled the axle's pressure movement. In accordance with the study by Komiya et al. (1996), the vibrator speed was set at 2.5 mm · s^{-1} with an amplitude of 8 mm as a safety stroke within the linear range of the axle (full range = 10 mm at 10 Hz). Therefore, for measurements over an 8 mm depth of tissue, the investigator displaced the vibrator in advance using a manipulation device that had a scale marked in centimetres (Fig. 1). The displacement and the responding force were recorded simultaneously using a data recorder (PC208 A: Sony Magnescale, Inc., Japan). These data were digitized using a 200 Hz sampling frequency using an A-D-converter and data acquisition system (MP100 A: Biopac Systems, Inc., USA). According to the method of Horikawa et al. (1993), muscle hardness can be evaluated using the straight part of the relationship between displacement and the force response (Fig. 2). It was calculated from the following equation:

$$E = Id(1 - \mu^2)(y \cdot x^{-1}) \tag{1}$$

where E is muscle hardness, I is the influence coefficient (0.85), d is the diameter of the probe, μ is Poisson's ratio (0.5), x indicates amount of displacement, and y indicates force recorded. In this equation, $y \cdot x^{-1}$ is equivalent to K_m (see legend to Fig. 2), the hardness of muscle tissue alone. The muscle hardness was measured at 80 mm above the elbow joint on the upper arm when the subjects lay on a bed on their back. Measurements of muscle hardness were made in two different arm positions: a relaxed position and a passively extended position. In the relaxed position, the elbow flexor muscles were quite relaxed because the forearm was supported with a towel to eliminate the tension generated by the forearm. In the passively extended position, the investigator extended the subject's elbow forcibly to full extension by holding the subject's wrist and shoulder. The muscle hardness was measured not only from the exercised arm but also from the control arm before, immediately after, and once a day for 5 days after exercise at approximately 24 h intervals.

Relationship between muscle hardness and elbow joint angle

The general relationship between muscle hardness and elbow joint angle in the absence of muscle damage was also established by using the control (non-exercised) arm. The muscle hardness was measured at seven different elbow joint angles between 120 and 180° (10° intervals).

Statistical analysis

A repeated-measures ANOVA was used to assess the changes in all muscle damage indicators and muscle stiffness. Statistical significance was set at $P < 0.05$.

Results

Maximal isometric force changed significantly ($P < 0.01$) with time. Maximal isometric force dropped to approximately 40% of the pre-exercise level immediately after exercise and had recovered to 60% of the pre-exercise level by 5 days after exercise (Table 1).

Relaxed elbow joint angle in both the standing and lying positions decreased significantly ($P < 0.01$) after exercise, but the former showed a larger decrease than the latter. Relaxed elbow joint angle had decreased approximately 20° from its pre-exercise level by 2 days after exercise, and did not start to recover until 4 days after exercise (Fig. 3).

Muscle soreness developed 1 day after exercise and was maintained for 3 days after exercise. Peak soreness scores upon palpation and extension were 36.3 (SEM 4.6) mm and 40.7 (SEM 2.8) mm, respectively (Table 1.).

Upper-arm circumference increased significantly ($P < 0.01$) after exercise at all the sites. For example, upper-arm circumference measured at the middle (80 mm above the elbow joint) portion had increased from 248 (SEM 3.3) mm (pre-exercise) to 266 (SEM 2.9) mm by 5 days after exercise (Table 1).

The ultrasound images showed enlargement of muscle thickness after exercise as identified by increases in the distance between the skin and the edge of the humerus. The subcutaneous thickness did not change during the 5 days after exercise. Maximal enlargement of muscle thickness was observed at 5 days after exercise (Table 1).

Plasma CK activity increased significantly ($P < 0.01$) and peaked at 4 days after exercise [13,729 (SEM 3,617) IU $\cdot$ l^{-1};Table 1].

Changes in muscle hardness following exercise are displayed in Fig. 4. The muscle hardness of the elbow flexor muscles at the relaxed elbow position, when the muscle was relaxed, did not change until 3 days after exercise, but showed a significant increase from the value of day 3 [15.0 (SEM 1.4) kPa] to days 4 [19.1 (SEM 1.7) kPa] and 5 [20.6 (SEM 4.1) kPa]. However, muscle hardness in the extended position increased significantly ($P < 0.01$) even immediately after exercise and peaked on day 3 after exercise [27.4 (SEM 4.0) kPa]. The muscle hardness increased to approximately twice as much as the pre-exercise value 3 days after exercise. The control arm did not show any changes in muscle hardness with time.

Figure 5 shows the relationships between muscle hardness and elbow joint angle without generating force using the control arm. The muscle hardness increased with increasing elbow joint angle, and the larger the angle, the greater the hardness.

Discussion

The eccentric exercise of this study produced long-lasting decreases in maximal isometric force and relaxed

Table 1 Maximal isometric force, muscle soreness (palpation and extension), upper-arm circumference, muscle thickness and plasma creatine kinase (*CK*) activity immediately before (*Pre*), after (*Post*), and for 5 days (1–5) after exercise ($n = 11$)

Indicator	Pre		Post		1		2		3		4		5	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
Maximal isometric force (N)	177.0	5.2	70.0	4.2	79.0	5.0	83.4	4.6	90.0	5.9	99.0	7.2	103.7	7.7
Muscle soreness (palpation) (mm)	0	0			24.5	3.9	36.3	4.6	28.4	4.3	11.9	3.2	2.5	1.0
Muscle soreness (extension) (mm)	0	0			33.8	3.4	40.7	2.8	40.5	2.4	26.0	3.4	12.0	2.9
Upper-arm circumference (mm)	248.0	3.3	258.0	3.1	257.0	2.9	261.0	3.1	263.0	3.3	264.0	2.9	266.0	2.9
Muscle thickness (mm)	19.6	0.6	24.9	0.7	24.7	0.6	25.1	0.7	25.3	0.9	25.3	0.9	25.3	0.9
Plasma CK activity (IU $\cdot$ l^{-1})	100.0	13.0			910	302	5469	2171	9867	2357	13729	3617	9501	2418

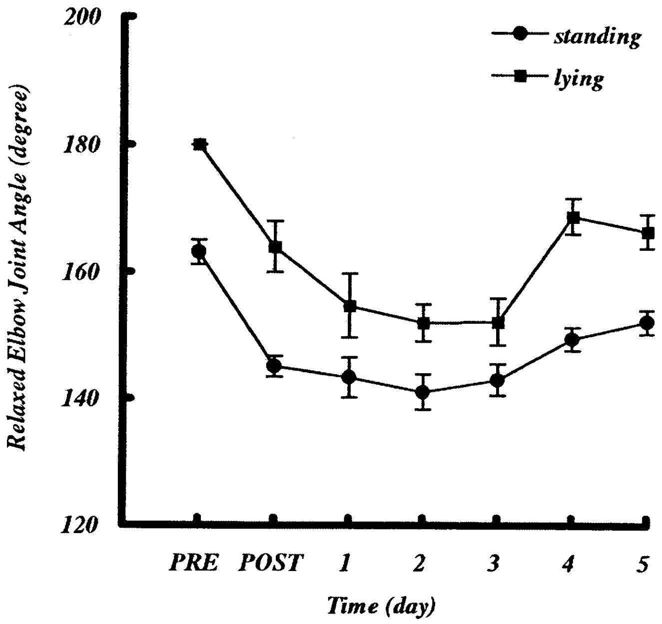


Fig. 3 Changes in relaxed elbow angle before (*PRE*) and immediately after (*POST*) and for 5 days after exercise. Relaxed elbow joint angle was measured with subjects in the standing and lying positions. Mean and SEM values are shown

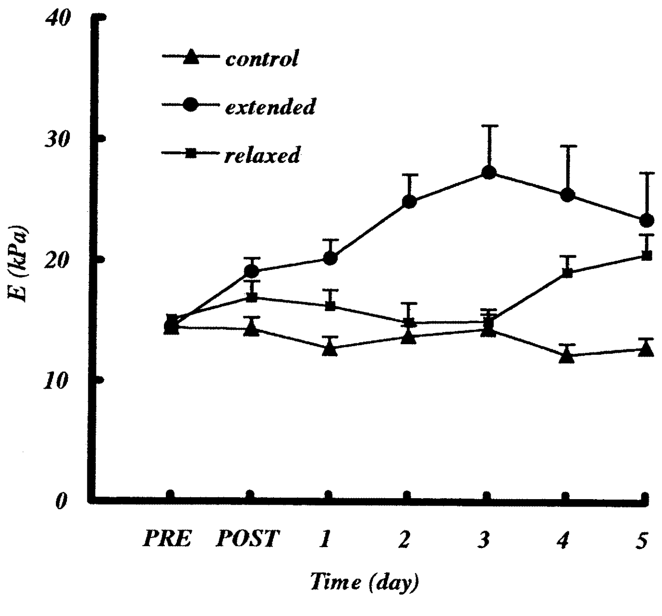


Fig. 4 Changes in muscle hardness (*E*) measured at a relaxed arm angle position (*relaxed*) and at an elbow extended position (*extended*) before (*PRE*) and immediately after (*POST*) and for 5 days after exercise. Changes in muscle hardness of the control arm (*control*) are also plotted. Mean and SEM values are shown

elbow joint angle (Fig. 3), large increases in upper-arm circumference and plasma CK activity, and severe muscle soreness (Table 1). It has been documented that the prolonged decrements in maximal isometric force and the large increases in plasma CK activity reflect structural damage to muscle fibres (McCully and Faulkner 1985; Jones et al. 1986; Friden et al. 1989), and that muscle soreness and muscle swelling indicate acute inflammatory responses (Smith 1991; Clarkson et al. 1992). Changes in relaxed elbow joint angle and upper-arm circumference in the present study were similar to or larger than those that have been reported in the previous studies that investigated changes in muscle stiffness of the elbow flexor muscles after eccentric exercise (Jones et al. 1987; Howell et al. 1993; Chleboun et al. 1998). Therefore, it appeared that the eccentric exercise in the present study induced muscle damage sufficient to study the effect of eccentric exercise on muscle hardness and its relation to muscle stiffness.

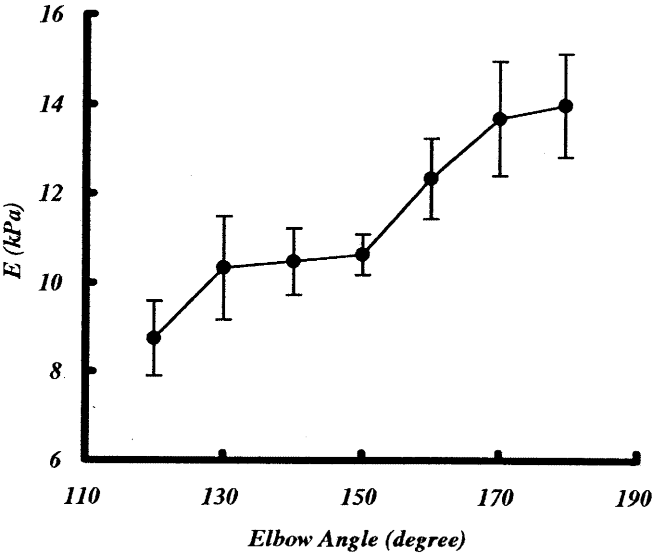


Fig. 5 Relationship between elbow angle of the control arm and muscle hardness (*E*). The elbow joint angle was changed between 120 and 180° in 10° intervals and muscles were kept relaxed during the measurement. Mean and SEM values are shown

As shown in Fig. 5, hardness of the control muscle was larger at the elbow-extended position than at the elbow-flexed position. Komiya et al. (1996) have also reported that muscle hardness decreased with flexing of the elbow joint. Since the relaxed elbow joint angle decreased for 5 days after exercise (Fig. 3), muscle hardness would have decreased after eccentric exercise, if the muscle fibres were slack at the flexed elbow positions. However, no changes in muscle hardness at the relaxed elbow position were observed up to 3 days after exercise (Fig. 4). This would suggest that muscle fibres were not slack but taut, probably due to increased muscle fibre stiffness.

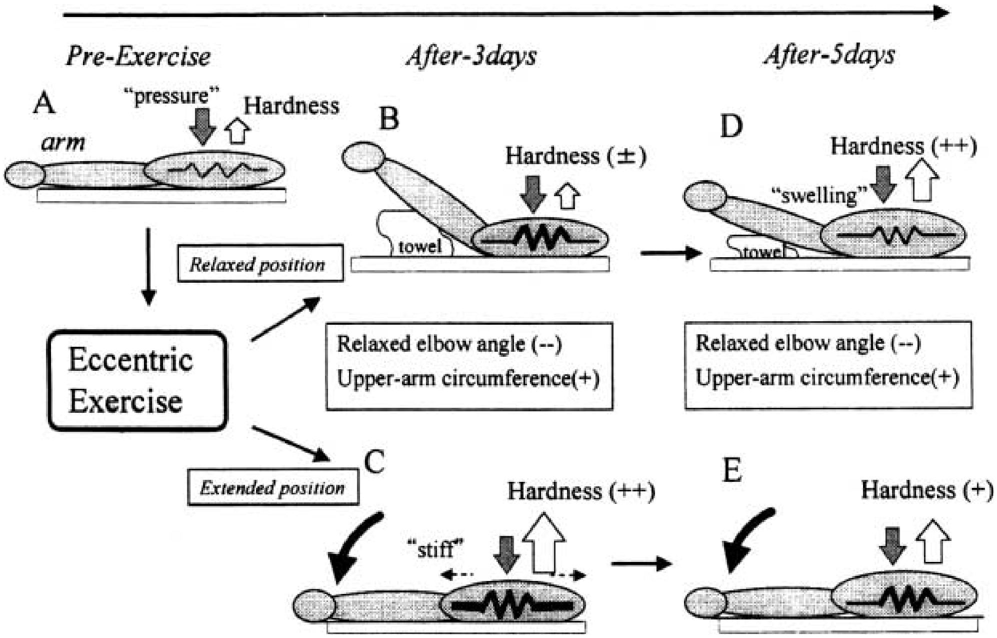
The muscle hardness at the extended position increased immediately after exercise and peaked on day 3 post-exercise by approximately 100% of the pre-exercise value, then gradually decreased on days 4 and 5 (Fig. 4). This change was similar to that of muscle stiffness that has been shown by previous studies (Jones et al. 1987; Howell et al. 1993; Chleboun et al. 1998). Jones et al. (1987) and Howell et al. (1993) have reported that muscle stiffness increased with decreases in relaxed elbow-joint angle after exercise, and the time course of changes in relaxed elbow joint angle matched that in muscle stiffness. The electromyogram activity from the biceps brachii muscle causing a decrease in relaxed elbow joint angle was found to be silent after eccentric exercise (Howell et al. 1985; Jones et al. 1987). It has been assumed that this abnormal flexion is caused by a shortening of non-contractile elements and oedema within the connective tissue network (Jones et al. 1987; Howell et al. 1993), or by a "contracture" or "rigor" state of the damaged fibres caused probably by increased Ca^{2+} in the cytosol (Nosaka et al. 1991; Howell et al. 1993). It seems likely that the increases in muscle hardness were caused by increased tension of muscle fibres or connective tissues. In this sense, muscle hardness measured at the extended elbow position seems to be the same as muscle stiffness.

The muscle hardness at the relaxed elbow position increased on day 4 after exercise (Fig. 5), when the relaxed elbow joint angle started to recover (Fig. 3). This delayed increase in hardness seemed to be associated with the increases in upper-arm circumference and muscle thickness (Table 1). It has been postulated that fluid accumulation is localized in the endomysium of muscle fibres or in the intracellular space of muscle fibres for the first 5 days after exercise, then moves to the subcutaneous area (Nosaka and Clarkson 1996). The fluid accumulation is likely to increase intramuscular pressure. Weigner and Watts (1986) have reported that muscle stiffness is proportional to upper-arm volume even in the resting condition. In the present study, there is no evidence that intramuscular pressure was increased after exercise; however, increases in tissue volume (Table 1) seemed likely to be the cause of the delayed increase in muscle hardness.

Figure 6 illustrates a possible relationship between muscle hardness and muscle stiffness after eccentric exercise. At 3 days after exercise, muscle hardness in the extended elbow position would be greatest, and muscle stiffness also would reach its maximum (Fig. 6C). At this stage, the damaged muscle fibres may be like a harder spring compared to the pre-exercise level. On the other hand, muscle hardness in the relaxed elbow position would not have changed from the pre-exercise level at 3 days after exercise, because the relaxed elbow joint angle decreases maximally (Fig. 6B). In the normal muscle, muscle hardness would decrease with flexing of the elbow joint (Fig. 5). This would make the muscle fibres taut to produce force that would compensate for the decrement of muscle hardness. At 5 days after exercise, muscle hardness at the relaxed position would become greatest because of the maximal swelling (Fig. 6D), but muscle hardness at the extended position would become smaller because of decreased stiffness (Fig. 6E).

In conclusion, the pressure method was able to assess changes in muscle hardness after eccentric exercise. The difference between the present study and previous studies that have assessed muscle stiffness was that muscle hardness at relaxed elbow positions did not change up to 3 days after exercise and then increased from 4 days after exercise. Muscle hardness measured by the pressure method seemed to reflect changes in muscle

Fig. 6 Hypothetical explanation of the results. **A** pre-exercise condition. **B** Three days post-exercise in the relaxed position. The relaxed elbow joint angle decreased maximally and the muscle hardness measurement was made whilst supporting the forearm by a towel to maintain the angle. **C** Three days post-exercise in the extended position. Muscle hardness was measured when the forearm was forcibly extended to stretch maximally. **D** Five days post-exercise in the relaxed position. The relaxed elbow joint angle had started to recover and the muscle fibres were less stiff than that at 3 days post exercise, but muscle swelled maximally. **E** Five days post-exercise at the extended position. Muscle hardness had become smaller than in the **C** stage in spite of maximal swelling, because of less stiffness



stiffness as reported in previous studies and changes in tissue volume that were induced by muscle swelling as well. Therefore, the increased muscle hardness after eccentric exercise that we have felt when palpating the muscle reflects increases in muscle stiffness and swelling.

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