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LOW FREQUENCY FATIGUE (LFF) OF QUADRICEPS MUSCLE DURING ECCENTRIC EXERCISE

by

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Healthy men (age 25,4±1,75) (n=12) (weight 74,3±6.2) gave their informed consent to take part in experiments. Experiment was designed to examine changes in muscle force generating capacity after intermittent voluntary eccentric exercise. All voluntary and electrostimulation-induced muscle contractions were registered before work, 2 min (A2), 20 min (A20) and 24 h (A24) after work. Our main finding is that immediately after work there was statistically significant ($p<0.05$) decrease in force at low stimulation frequencies (10 and 20 Hz) as compared to that of 50 Hz. In addition, there is smaller decrease in maximum isometric voluntary force and jump height than in forces evoked by electrostimulation. At 20 min and 24 h after exercise there were no changes in contractile properties, evoked by electrostimulation.

Key words: skeletal muscle, eccentric exercise, low frequency fatigue, jumping, electrostimulation.

Introduction

In humans, low frequency fatigue (LFF) occurs in concentric and isometric exercise (Edwards et al. 1977, Newham et al. 1983, Ratkevicius et al. 1995) but it is mostly observable in eccentric exercise (Newham et al. 1983, Jones et al. 1989). This type of contraction is thought to be metabolically less demanding than concentric contractions (Enoka 1994). Following eccentric contractions muscle can exhibit various forms of damage which results in pain being experienced (Newham et al. 1983, Jones et al. 1989). There is a possibility that in eccentric fatiguing exercise, the end sarcomeres of the fiber overextend and damage those in the middle section of the fiber (Jones 1996, Morgan 1990). In this situation the active sarcomeres would be working at a shorter length than predicted from the overall fibre length and the force-frequency curve would be shifted to the right.

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The primary aim of the present study was to test the hypothesis that when performing eccentric non-intensive intermittent physical exercises there will be a greater increase of LFF in the muscles.

Methods

Subjects. Healthy men (age 25.4±1.75 years, mean ±SD n=12) (weight 74.3±6.2 kg) gave their informed consent to take part in experiment within the study. The subjects were physically active but none took part in any formal physical exercise or sport program

Force measurements. Subjects were placed in the testing apparatus (i.e., experimental chair). They sat upright in the experimental chair with a vertical back support provided. A strap secured the hips and thighs to minimise uncontrolled movements. The right leg was clamped in the force measuring device with the knee semi-flexed. A 6 cm wide plastic cuff, placed around the right leg just proximal to the malleoli, was tightly attached to a linear variable differential transducer. The output of the transducer, proportional to isometric knee extension force, was amplified and digitised at a sampling rate of 1 kHz by a 12-bit analogue-to-digital converter installed in an IBM-compatible AT 286 personal computer. The digitised signal was stored on hard disk for subsequent analysis.

Electrical stimulation. A high voltage stimulator (MG 440, Medicor, Budapest, Hungary) was used. Electrical stimuli to the quadriceps muscle were delivered through lead electrodes (9×18 cm) padded with cotton cloth and soaked in saline solution. One stimulation electrode was placed just above the patella, while the other covered the large portion of the muscle belly in the proximal third part of the thigh. The electrical stimulation was always delivered in trains of square wave pulses of 1-ms duration (voltage 150 V, which induce 65–70 per cent of MVC). The subjects were introduced to electrical stimulation.

Table 1. Control values of indices of men's electrostimulation-induced contractions of quadriceps femoris, MVC and suquatting jump height (mean ±SD)

P10, N	P20, N	P50, N	P10/P50	P20/P50	MVJ, N	H, cm
158,4 58,2	388,2 93,4	539,1 152,1	0,31 0,07	0,73 0,09	782,5 141	36 5,2

The following data were measured: the force of the quadriceps muscle, aroused by electrical stimulation under 10 Hz (P10), 20 Hz (P20) and 50 Hz (P50) frequencies (the duration of each electrical stimulation series was 1 s) and maximal voluntary contraction (MVC) (top of the MVC was reached, held about 2 second and

relaxation). The ratio of P20/P5O and Pl0/P5O forces were calculated for the evaluation of LFF (5). Muscle fatigue index (Fl) was evaluated. The Fl was calculated as follows, Fl = (value after exercies/value before exercise) x 100 per cent.

Vertical jump performance. Each subject performed maximal voluntary jumps using a dynamometric platform of the PD-3A (Russian, Sankt-Peterburg, VISTI) (3 jumping attempts were allowed per person and the best attempt was counted) from semisquatting position with no allowance for preparatory counter-movement. Heights of the jumps (H) were recorded by an earlier technique by applying the following formula (Bosco et al. 1982): $H=l.1226 \times Tf^2$ where: Tf= flight time (s).

The protocol of experiment. Experiment was designed to examine changes in muscle force generating capacity after intermittent voluntary eccentric exercise (eccentric jumps).

Upon arriving at the laboratory, the subject was seated in the experimental chair and after 5 min muscle contractile properties were recorded in the following sequence: P10, P20, P50, MVC (MVC was reached 3 times). Rest interval between registration was 1 min. Then the subjects undertook 5 mm of light exercise as warm-up. The warm-up period was followed by 100 jumps performed by the subjects.

The work of experiment consisted of 100 eccentric muscle exercise of low intensity (the subject performed 100 jumps from a 40-cm platform every 20 s, plastically flexing the knee joints up till 90^0 angle). Immediately after each jump the squatting jump height of the subjects was established. Then the subjects were seated in the experimental chair once again and both voluntary and electrostimulation-induced muscle contraction properties were registered. Following this, the subjects remained seated in the chair for the 20 min during which the muscle contractile properties was tested again. Then the subject performed the standard procedure of warming-up followed by the control squatting jumps. Next day all voluntary and electrostimulation-induced muscle contraction properties were tested once again. In addition, during the next day the subjects subjectively evaluated their muscle pain (during walking) according to 10-point scale.

Statistics. Student's t-test for paired data was used to determine any differences between pre-and post-exercise data within a single experiment. Statistical significance was set at $p<0.05$.

Results

Immediately after exercises there was statistically significant ($p<0.05$) decrease in force (P10, P20 and P50) evoked by muscle electrostimulation. However, there was no change in MVC, but there was decrease in H ($p<0.05$) following exercise (tabl. 2). There was particularly marked decrease in force at low stimulation frequencies (10 Hz and 20 Hz) as compared to that of 50 Hz ($p<0.05$), as was indicated by the significant decreases ($p<0.05$) in P10/P5O and P20/P50.

Table 2. Mean values in per cent of fatigue indices (FI) of electrostimulation-induced muscle contractions, MVC and jump height of men immediately, 20 min and 24 h after exercise (mean ±SD)

P10	P20	P50	P10/P50	P20/P50	MVC	H
Immediately after exercise						
38,1*	56,2*	82,4*	46,2*	68,7*	93,5	94,1*
9,9	9,6	9,5	10,1	12,1	10,4	6,2
20 min after exercise						
44,9*	58,1*	84,1*	53,2*	69,1*	92,7	93,8*
15,6	11,1	12,1	16	10	12	6,2
24 h after exercise						
82,8*	87,6*	95,4*	86,7*	92,4*	97,8	99,2*
22,6	15,8	19,1	14	7,6	9,6	8,9

* significant ($p<0.05$) difference between mean values of P10, P20, P10/P50, P20/P50, MVC, and H to intial level. MVC — maximum voluntary contraction, H — height of jump

At 20 min after the exercise there were no statistically significant changes ($p>0.05$) in muscle contraction forces (Pl0, P20 and P50) as well as in MVC and H when compared to the corresponding values immediattelly after exercise (tabl. 2). Following the exercise the force (Pl0 and P20) values at low stimulation frequencies were still smaller ($p<0.05$) when compared to the corresponding values before exercise (tabl. 2). Also, at 24 h after eccentric exercise the subjects experienced muscle pain (2.7±0.4 points).

Discussion

The main findings of this study are that immediately after exercise there was a statistically significant ($p<0.05$) decrease in force (Pl0, P20 and P50) while there was no change whatsoever in MVC, and there was decrease ($p<0.05$) in H. There were significant ($p<0.05$) decreases in force at low stimulation frequencies (10 Hz and 20 Hz) as compared to that of 50 Hz. This is evident from the significant decreases ($p<0.05$) in Pl0/P5O and P20/P50. At 20 min and 24 h after exercise there were no changes in contractile properties, i.e., no recovery of the muscle contractility took place.

Since physical exercises in the case of both experiments were performed every 20 s we consider there was an adequate period of time for the recovery ATP. In brief test contractions the amount of ATP hydrolysed by actomyosin can covered by the cellular pool of ATP. Studies of the sensitivity of actomyosin ATPase for ATP reveals that even a reduction in ATP would have negligible effect on enzyme activity (Cooke and Bialek 1979). The causes of muscle fatigue therefore could not be at-

tributed to increase in the myoplasm of such metabolites as phosphate and hydrogen ions. Thus, it appears, that the causes of muscle fatigue should be sought after in the mechanics of muscle contraction and extension rather than in muscle metabolism.

It was suggested that LFF may be a consequence of some damage to the structure of muscle fibers and impairment of the electro-mechanical coupling mechanism (Jones et al. 1989, Lännergren et al. 1996), and this type of fatigue is most pronounced following exercise in which the active muscle is stretched (Jones 1996). The mechanism responsible for the damages is not clear. It has been suggested that the series of elements damaged during stretching may be the sarcomeres in the middle of the fiber, elongated by the stronger sarcomeres at the ends of the fiber. Cosequently, the lengthening of contracting muscle (an eccentric contraction) involves the stretching and poping of individual sarcomeres as each reaches its stress limits (Morgan, 1990). The muscle damage is seen predominantly in fast-twitch fibers (Friden et al. 1997), which appear to be selectively recruited for eccentric contractions (Nardone et al. 1989). In this cases is not surprising why LFF was more expressed experimental after F work than after C work.

The mechanism of muscle damage might be largely responsible for evoking both LFF and the decrease in force generated at high frequencies (P50) (tabl. 2). Besides, the origin of subjectively greater pain following 24 hours after the eccentric than after concentric exercise might be also accounted for this fact.

Direct measurements of intracellular Ca^{2+} have now been made in single mammalian fibers (Lännergren et al. 1996) and the results show that for a given stimulation frequency there was a reduced intracellular Ca^{2+} in the fatigued fibers. There was no evidence of altered intracellular buffering of calcium and the relationship between tension and intracellular Ca^{2+} was unchanged, indicating that the cause of LFF in these preparations was reduced release from the sarcoplasmic reticulum rather than decreased Ca^{2+} binding to troponin. In recently has been shown that LFF results from increases in Ca^{2+} during fatigue and these elevations in Ca^{2+} activate some process which leads failure of excitation-contraction coupling and Ca^{2+} release (Chin and Allen 1996). There is a dalay in muscle force development after eccentric exercise, and this dalay is suggested to be due to secondary degradation processes affecting excitation-contraction coupling (Brown et al. 1996). It has been shown, that if fatigue is induced with intermittent tetanic contractions, recovery ir far slower and requires several hours to be complete (Lännergren et al. 1996). During recovery in this case there is often a secondary reduction in the force response to stimulation and extreme cases force may be abolished for 20-30 min. The extreme effect on the force during recovery has been called "post-contractile depression" (PCD, Lännergren et al. 1996) and the underlying mechanisms seem in many respects to resemble LFF in mammalian muscle. In both cases is a reduction of amount of Ca^{2+} released in response to electrical stimulation (Lännergren et al. 1996).

Furthermore, in our experiments on delayed recovery, we have postulated that as a result of muscle fatigue a critical structure in the gap between t-tubules and

sarcoplasmic reticulum becomes demaged or broken. Recently has been shown that in six out of 35 fibres studied, briefly stretching fibres by 20 per cent of their resting length at 6-20 min into recovery resulted almost complete abolition of the force response to electrical stimulation. However, force did recovery its control value, but it is usually took more than 10 h (Bruton et al. 1995).

The fact that in both cases there is more depression of force at low frequencies than at high frequencies (tabl. 2) is explained by the relation between Ca^{2+} and force generation. It has been shown, that this relation is described by an S-shaped curve with a steep rising part followed by plateau (Lännergren et al. 1996). For mammalian fibres 100 Hz stimulation resuhs in a Ca^{2+} which is on the horizontal part of the curve (we speculate that is in our case at 50 Hz), where moderate falls in Ca^{2+} have liffle effect on force. Because at low stimulation frequencies the Ca^{2+} lies on the steep part of the curve, a moderate fall in Ca^{2+} produces a large reduction of force.

It is of interest to note that the force of voluntary contraction (MVC) and jump height decreased to a smaller extent than forces evoked by electrostimulation. In voluntary exercise, the posibility for variability in all these factors is immense. For example, motor unit recruitment, the length and changes in length probably alter from one to another, and may be different in individual muscles and even within single fibers. It is know that submaximal concentric and eccentric contractions may involve the activation of different motor units (Nardone et al. 1989). It is difficult to explain why after eccentric exercise the jump height decreased. It may be connected with changes in mechanics and reflectory mechanisms affecting jumping capacity when in the process of peforming physical exercises the muscle is extended (Gollhofer et al. 1987). Our control jumps however were performed in the squatting position. Thus changes in elastic muscle mechanisms should not have had considerable effect. It is know, that level running at submaximal intensity on a treadmill for 20 min produced a 9% reduction in the amplitude of the soleus H reflex. Downhill running (–10% grade) at the same intensity reduced the H reflex amplitude by 25% (Bulbulian et al. 1992). Hence, in a similar fashion, changes in reflectory muscle mechanisms may have an influence on the height of squat jumps.

In summary, the results indicate that when performing eccentric intermittent exercise of low intensity there is increase of LFF in muscles. In addition, there is smaller decrease in maximum isometric force and jump height than in forces evoked by electro stimulation. At 20 min and 24 h after exercise there were no changes in contractile properties, i.e., no recovery of the muscle contractility took place.

REFERENCES

Bosco C., Viitasalo J.T., Komi P.V., Luhtanen P. 1982. *Combined effect of elastic energy and myoelectric potentiation during stretch-shortening cycle exercise.* Acta Physiol. Scand.,114:557–565.

Brown S.J., Child R.B., Donnelly A.E., Saxton J.M., Day S.H. 1996. *Changes in human skeletal muscle contractile flinction following stimulated eccentric exercise*. Eur. J. Appl. Physiol., 72:515–521.

Bruton J.D., Lännergren J., Westerblad H. 1995. *Mechano-sensitive linkage in excitation-contraction coupling in frog skeletal muscle*. J. Physiol., 484:737–742.

Bulbulian W., Bowles D.K. 1992. *Effect of downhill running on motor pool excitability*. J. Appl. Physiol., 73:968–973.

Chin E.R., Allen D.G. 1996. *The role of elevations in intracellular [Ca^{2+}] in the development of low frequency fatigue in mouse single muscle fibres*. J. Physiol., 491:813–824.

Cooke R., Bialek W. 1979. *Contraction of glycerinated muscle fibres as a function of the of the ATF cocentration*. Biophys. J. 28:241–258.

Edwards R.H.T., Young A., Hosking G.P., Jones D.A. 1977. *Human skeletal muscle function: description of tests and normal values*. Clin. Sc. Mol. Med., 52:283–90.

Enoka R.M. 1994. *Neuromechanical Basis of Kinesiology*. Champaign, W. Human Kinetics.

Friden J., Lieber R.L. 1997. *Muscle damage induced by cyclic eccentric contraction: biomechanical and structural studies*. (In:) S.Salmons (Ed.), *Muscle Damage*, 41–63. Oxford, New York, Tokyo: Oxford University Press.

Gollhofer A., Komi P.V., Miyashita M., Aura O. 1987. *Fatigue during stretch-shortening cycle exercise: changes in mechanical performances of human skeletal muscle*. Int. J. Sports Med., 8:71–78.

Jones D.A., Newham D.J., Torgan C. 1989. *Mechanical influences on long-lasting human muscle fatigue and delayed-onset pam*. J. Physiol., London, 412:415–427.

Jones G.A. 1996. *High- and low-frequency fatigue revisited*. Acta Physiol Scand., 156:265–270.

Lännergren J., Westerblad H., Bruton J.D. 1996. *Slow recovery of single skeletal muscle fibres*. Acta Physiol. Scand., 153:193–202.

Morgan D.L. 1990. *New insights into behaviour of muscle during lengthening*. Biophys. J., 57: 209–221.

Nardone A., Romano C., Schieppati M. 1989. *Selective recruitment of high-threshold human motor units during voluntary isotonic lengthening of active muscle*. J. Physiol, London, 409:451–471.

Newham D.J., Mills K.R., Quigley B.M., Edwards R.H.T. 1983. *Pain and fatigue after concentric and eccentric muscle contractions*. Clin. Sc., 83: 64 (1): 55–62.

Ratkevicius A., Skurvydas A., Lexell J. 1995. *Submaximal-exercise-induced impairment of human muscle to develop and maintain force at low frequencies of electrical stimulation*. Eur. J. Appl. Physiol., 70:294–300.