Knee Proprioception after Exercise-Induced Muscle Damage

Abstract

The purpose of the present study was to investigate whether exercise-induced quadriceps muscle damage affects knee proprioception such as joint position sense (JPS), force sense and the threshold to detect passive movement (TTDPM). Fourteen young men performed sets of eccentric quadriceps contractions at a target of 60% of the maximal concentric peak torque until exhaustion; the exercise was interrupted whenever the subject could not complete two sets. Muscle soreness, JPS, the TTDPM and force sense were examined before the exercise as well as one, 24, 48, 72 and 96h after exercise. The results were compared using one-way repeated-measure ANOVA. Plasma CK activity, collected at the same times, was analyzed by the Friedman's test to discriminate differences between baseline values and each of the other assessment moments (p<0.05). Relative to the proprioception assessment, JPS at 30 and 70° of knee flexion and force sense were significantly decreased up to 48h, whereas TTDPM decreased significantly at only one hour and 24h after exercise, at 30 and 70° of the knee flexion, respectively. The results allow the conclusion that eccentric exercise leading to muscle damage alters joint proprioception, suggesting that there might be impairment in the intrafusal fibres of spindle muscles and in the tendon organs.

Introduction

Exhaustive and/or unaccustomed exercises, particularly those involving eccentric muscle contractions, are known to induce temporary muscle damage, evidenced by delayed onset muscle soreness and changes in the neuromuscular function, which may persist for several days [8, 14]. Recreational and elite athletes can experience this occurrence often during new demanding phases or different types of training, leading to considerable suffering and handicaps to athletes by temporarily impeding performance and precluding their programmed training [12]. This muscle damage is evidenced by structural and ultrastructural alterations such as irregularities of cross-striated patterns, vacuolization, segmental necrosis, central nuclei, activation of satellite cells and fibroblasts, intra-cellular edema and muscular inflammatory reaction with leukocyte infiltration [1, 11]. These changes in the integrity of the muscle are parallel to indirect signals of muscle injury [18, 21] where reduction in maximal voluntary force [4, 31], increase in muscular stiffness [18, 30] and decrease in range of motion [22, 31] are reported.

After exercise-induced muscle damage, the well-known increase in muscle stiffness and tension might be explained by parallel changes in muscle spindle activity leading to the increased reflexive neural activation of the muscle [25]. However, contrasting with the huge concern of researchers to the extrafusal muscle fibres, the muscle spindle has been neglected on the issue of exercise-induced muscle damage. The hypothetical change in muscle spindle activity induced by eccentric exercise was studied by Saxton et al. [29] and Brockett et al. [3] showing alterations in joint position sense (JPS) using elbow flexors muscles, however, since then this topic has received little attention in the literature. Apart from the commitment of intra and extrafusal muscle fibres, it is possible that tendon organs could also be negatively affected by aggressive exercise, explaining the drop in force-generating capacity through the greater error in force sense experienced by subjects [4, 31]. Despite this suspicion, the reduction of proprioception after exercise-induced
muscle damage has not been sufficiently documented, so as to deeply assess these supposed changes. Therefore, an array of conscious proprioception measures should be considered, such as joint position sense, kinesthesia (active and passive), and force sense.

Assuming that proprioceptive impairments predispose athletes to sports injuries [20], it will be important to understand if such impairment really exists and, if so, to characterize its temporal behaviour following exercise. In this sense, the purpose of our study was to investigate whether exercise-induced muscle damage in the quadriceps muscle affects different components of proprioception such as joint position sense, force sense and the threshold to detect passive movement (kinesthesia).

Material and Methods

Subjects

The study was performed in accordance with the ethical standards [16]. All volunteers completed a medical screening questionnaire and provided written informed consent prior to participation. Fourteen young healthy men were included in the study. Participants did not perform intense or exhaustive exercise nor did they experience delayed onset muscle soreness during the three months preceding the study. They did not report any disturbance of their lower legs and were not permitted to receive any treatment such as heat or ice applications, massage, stretching, analgesics or exercise during the experimental protocol. Mean (± standard deviation) of age, weight, height, body mass index and maximal concentric peak torque were 22.1±2.1 years, 73.9±4.5 Kg, 1.8±0.6 cm, 23.4±1.4 Kg/m², and 219.3±16.7 N.m, respectively.

Induction of muscle damage

After previous familiarization with an isokinetic dynamometer (Biodex System 3pro, Biodex Medical Systems, Inc., Shirley, NY, USA) for the measurement of maximal concentric peak torque, the exercise protocol to induce muscle damage of the quadriceps muscle of the dominant leg consisted of sets of thirty eccentric contractions at a target of 60% of the maximal concentric peak torque. The exercise was interrupted whenever the subject could not complete two sets (defined as a criterion for the incapacity to maintain the contractions at the given intensity and frequency). The subjects were instructed to perform an eccentric quadriceps contraction against the moving lever of the system at 60° per second, using visual feedback from the dynamometer software as a means to maintain the strength intensity. The range of motion was set between 20 and 100° of the knee flexion; resting time between contractions and sets was set at one and thirty seconds, respectively.

Each subject had to warm up on a cycle ergometer (Monarc E-824, Varber, Sweden) at a resistance of 2% of body weight for five minutes. Then the subjects were seated with 100° hip flexion on the dynamometer chair. The standard Biodex knee unit attachment was used to restrain the chest, pelvis, thigh and ankle in accordance with the manufacturer’s instructions (Biodex Pro Manual, Applications/Operations, Biodex Medical Systems, Inc., Shirley, NY). The resistance pad was placed as distally as possible on the tibia while still allowing full dorsiflexion of the ankle. The input axis of the dynamometer was aligned with the axis of the dominant knee.

Dependent variables

Muscle soreness, knee joint position sense (JPS), force sense, the threshold to detect passive movement (TTDPM) and plasmatic creatine kinase (CK) activity were recorded immediately before and 1, 24, 48, 72 and 96h after exercise.

Subjective muscle soreness pain sensation

Muscle soreness pain sensation was measured using a Visual Analogue Scale (VAS), which is commonly used in delayed onset muscle soreness research [17, 19, 24]. The VAS is a 10 cm line with endpoints labelled “no pain” and “worst possible pain” [7]. The subject was instructed to perform a squat (active movement) and rate their perception of soreness, marking the line at the distance corresponding to the intensity of present pain.

Knee joint position sense (JPS)

The protocol for the JPS assessment involved a passive positioning and active repositioning (passive-active test) of the dominant leg. JPS measurements were performed with an isokinetic dynamometer (Biodex Medical Systems, Inc., Shirley, NY, USA). Drouin et al. [10] demonstrated that Biodex System 3 isokinetic dynamometer was a mechanically reliable instrument for the measurement of angular position, isometric torque and slow to moderately high velocities, with high intraclass correlation coefficients (ICC2,3 = 0.99 for each variable). Instructions about the test were given to the subjects prior to their initiation and they were allowed to familiarize themselves with the Biodex System before its use.

The subjects were seated in the dynamometer chair at 100° hip flexion with their eyes closed, wearing headphones and fitted with an air cushion above the leg with pressure at 40 mmHg to minimize cutaneous sensory information [6, 15]. All subjects had the “hold” button in one hand so that they could stop the dynamometer’s lever arm with their thumb when they thought it to be at the target angle. In each trial, the lower leg was passively moved at 10° per second and positioned at an index angle approximating either 30 or 70°; for 5 s the target position was maintained to memorize. The subject actively reproduced the index angle to the best of his ability. The start position was 100° of the knee flexion and the direction of movement was into extension. This procedure was repeated three times for each index angle. The order of the tested angles was randomized. The protocol of the JPS assessment was always completed by the same researcher, who did not give any feedback about the performance obtained during the assessment.

The repositioning absolute error was obtained through the calculation of the difference between the target angle and the mean of the three angles chosen by the subjects.

Force sense

Knee extensor force-matching procedures were conducted at 20% of maximal concentric peak torque (MCPT) obtained in the assessment done prior to exercise. Isometric force sense was utilized to better isolate the role of force [9], and a low load for testing was chosen because the ability to reproduce force is directly related to motor unit recruitment and firing frequency [5]. Subjects were positioned on the Biodex System in the same manner as for the JPS testing and were instructed to obtain the target force using visual feedback from the dynamometer software. Moreover, they were asked to maintain the isometric contraction for 6 s with a 6 s rest during 6 trials. Prior to repeating procedures of the same target force (but without visual feedback), a 1 min rest period was provided. Throughout the test.
subjects were not given feedback about their force-matching performance. Average peak torque to be used for analysis was provided by the dynamometer software without visual feedback to the subject. The difference between the target force and the average peak torque produced in absolute value was calculated and used for analysis.

Threshold to detect passive movement (TTDPM)
The TTDPM in the knee was assessed using the Biodex System. The subjects were evaluated in the same seated condition as described for JPS evaluation; they were also blindfolded and had earphones placed over their ears. Each subject was asked to press the handheld stop button when feeling a sensation of movement or a change in the starting knee position, which was engaged at random in the subsequent 20 s by the tester. Testing at slow angular velocity (0.25 degrees/s) is suggested to maximally stimulate joint receptors and minimally stimulate cutaneous and muscle receptors [28]. Three trials from a starting position of 30 and 70° knee flexion moving into extension were performed and the average number of degrees in these three consecutive trials was collected to determine the TTDPM being the mean values calculated in both joint positions.

Plasmatic creatine kinase (CK) activity
To determine plasmatic CK activity, a sample of 32 μl of capillary blood was collected from the ear lobe into a tube containing heparin (Cat n° 9550532 Reflotron®). The ear lobe was cleaned with 95% ethyl alcohol and, after being dried with cotton, the lobule was pricked with a sterile, single-use lancet device (AccuCheck®, Safe-T-Pro Plus). The blood was immediately piped to a Reflotron Creatine Kinase tab (Cat n° 1126695 Reflotron®) and the tab was inserted into the Reflotron Analyser® (Boehringer, Germany).

Data analysis
All data are reported as mean±standard deviation (SD) or as median with interquartile range (for analysis of CK values). The distribution of all variables was examined using the Shapiro-Wilk test and no significant difference from normal was found except in the plasma CK activity values. A one-way repeated-measure ANOVA was employed to assess changes from baseline in muscle soreness, force sense, joint position sense and kinesthesia within the group using raw data. In the case of evident differences, the Tukey’s Post hoc test for multiple comparisons was applied. Plasma CK activity was analyzed by a Friedman’s test to detect changes between the 6 moments and Wilcoxon’s signed ranks tests to discriminate differences between baseline values and each of the other assessed moments. A paired-samples t test was used to compare JPS and the TTDPM between joint positions at each moment. The level of significance was set at α=0.05. The SPSS version 16.0 was used for all analyses (SPSS Inc., Chicago, Illinois).

Results

All fourteen subjects reached the end of the study. The mean number of eccentric contractions performed to induced muscle damage was 127.8±13.0 and as expected, the markers of muscle injury (● Figs. 1 and 2) demonstrated that the exercise applied induced muscle damage. In fact, muscle soreness increased significantly after the eccentric exercise and was present in all assessments that comprised the experimental protocol. The maximal manifestation of muscle soreness was at 48 h. Plasmatic CK activity also increased significantly (p<0.05) from the baseline values after 24 h. The results for JPS are shown in ○ Fig. 3. The mean absolute error value of JPS assessed before the exercise with the knee at 30° of flexion (2.2±1.5) was similar to the one obtained at 70° (2.9±1.6). In fact, this difference was not statistically significant (p=0.451).

Joint position sense decreased significantly after exercise-induced muscle damage in both joint amplitudes studied up to 48 h. One hour after exercise, the error increased by 3° in the knee at 30°, while with the knee at 70°, the error increased by only 2° (○ Fig. 3). However, this difference was not statistically significant (p>0.05). The force sense significantly decreased one hour after exercise and remained decreased until 48 h (○ Fig. 4). During this period the error of the reproduction force was duplicated, i.e., in the baseline the error was 2.5±4.5 N.m and after exercise-induced muscle damage it was always slightly over 5 N.m.

Changes in the TTDPM (kinesthesia) are shown in ○ Fig. 5. The degrees necessary to perceive passive movement were similar in the 30 (0.7±0.4°) and 70° (0.8±0.3) of knee flexion (p=0.464).
The purpose of the present study was to determine whether eccentric exercise aiming to cause muscle damage could affect the different modalities of proprioception. The significant increase in the values of muscle soreness and plasmatic CK activity during the experimental protocol clearly support the achievement of our goal to induce muscle damage. Our findings demonstrated that exercise-induced muscle damage reduces proprioception immediately after exercise. Indeed, all variables chosen to assess changes in the sensory-motor system showed significant changes after the exercise, and it was necessary to wait 72 h to verify tapering of the changes in proprioception comparable to the pre-exercise condition.

To assess JPS, two target joint positions were chosen, one at 30°, which is the basis for most weight-bearing activities, and another in the middle range, based on the assumption that position matching acuity for movement extent relies on different cues and might depend in part on different mechanisms [27]: the end range of motion is considered to be more sensitive with respect to the sense of the posture of segments [13,23]. In this study, the mean absolute error value verified before doing exercise with the knee at 30° of flexion was slightly lower than that obtained at 70°. However, this difference was not significant, contrary to what was expected. It might be suggested that a 30° knee flexion is not sufficiently near the end of the range of motion to verify more influence of the other receptors, such as Ruffini endings or Golgi-type organs.

The results found in the present study corroborate the results obtained by others [3,29] showing that eccentric exercise-induced muscle damage leads to a reduction in JPS. Indeed, all fourteen subjects who participated in our trial experienced significant changes in knee position sense during the 48 h post-exercise, and the mean absolute error value increased in both joint positions assessed. The relative contribution of joint and muscle mechanoreceptor deficits following exercise-induced muscle damage remains unclear. It is generally established that muscle spindles contribute to the senses of position and motion of body segments [3]. However, it is possible that damage from eccentric exercise is not restricted to the common muscle fibres. The decrease in JPS might theoretically be thought of as secondary to the loss of muscle receptor input with injury in the intrafusal fibres of spindle muscles. According to Brockett et al. [3], although the compliant connections might reduce the amount of intrafusal fibre stretch, both extrafusal and intrafusal fibres are affected by eccentric contractions. If the damaged fibres really died or were ruptured, spindle discharge levels would fall significantly.

With respect to the TTDPM assessments, Boerboom et al. [2] recently demonstrated that the threshold to detect passive movement is an accurate and valid method to measure proprioception in healthy persons. Nevertheless, this approach has never been used to assess neuromuscular dysfunction after exercise-induced muscle damage. Similar to the results found for JPS, the ability to perceive passive movement in the position joint at 30°, did not demonstrate much accuracy. With respect to the effect of eccentric exercise on the threshold to detect passive movement, we found significant changes immediately after doing the exercise in both joint positions. However, at 70° knee flexion this reduction to perceive passive movement remained up to 24 h, while in the 30° condition statistical findings were prevalent only one hour post-exercise. The reasons for the reduc-

The results demonstrated significant changes in kinesthesia one hour after exercise when the knee was set at 30° of flexion and 1.0±0.5° were necessary to perceive motion, while at 70° of knee flexion, joint position kinesthesia remained altered up to 24 h and 1.2±0.6° were necessary to perceive passive motion. The TTDPM was statistically different between joint positions at 24 h.
tion in the ability to perceive passive movement are probably similar to those used to explain the increase of the absolute error in JPS. Since eccentric exercise is harmful to extrafusal fibres, the possibility of this damage being extended to muscle receptors – specifically, the intrafusal fibres of muscle spindles – is plausible.

Apart from muscle spindles, it is reasonable to hypothesize that other mechanoreceptors, such as those presented in joint capsules and ligaments, might also have contributed to the obtained results. As TTDPM was tested at 0.25 degrees/sec, velocity where the stimulation of the joint receptors is maximal [27], the smallest error observed in the position at 30° may be due to the greater participation of receptors in knee ligaments at this joint position. In fact, joint mechanoreceptors do not appear to be sufficiently stimulated through the midranges of motion to contribute substantially to proprioception, especially in relation to the seemingly potent input stemming from muscle receptors; however, near the end range of motion, such as 30° of knee flexion, seemingly potent input stemming from muscle receptors; how-ever, near the end range of motion, such as 30° of knee flexion, the mechanoreceptors present in several structures of the knee joint, namely of cruciate ligaments, become more activated with the stretching of these ligaments, which might contribute to explain the reduced error observed with TTDPM at 30° (for refs see [26]).

To assess the force sense, we chose low loads for testing because the ability to reproduce force is directly related to motor unit recruitment and firing frequency [5] and obviously, if loads increase, the difficulty of the task also increases. The decreased force following eccentric exercise apparently leads to more effort required to maintain the same target force and this sensation probably explains the increase in error in the force sense. When the subjects were asked to maintain isometric force at 20% of maximal concentric peak torque, the error of the target force duplicated in magnitude after exercise, recovering at 72 h. This result was in accordance with those obtained by Brocket et al. [3] when exercise-induced muscle damage in the elbow flexor muscles disturbed force sense over a number of days.

It is still unknown how exercise-induced muscle damage affects the function of the Golgi tendon organs and their ability to perceive the force generated. This study suggests that the receptors responsible for providing the peripheral information necessary to sense force were affected. Indeed, the sensation of force arises from the sense of the tendon generated byafferent feedback from the muscle. However, the importance of the central mechanism in the sense of effort cannot be disregarded.

In conclusion, eccentric quadriceps exercise induces muscle damage and a reduction in knee proprioception. Changes in the afferent information arising from proprioceptors after exercise-induced muscle damage have interfered with all measured variables of proprioception, such as joint position sense, the threshold to detect the passive movement and force sense. Having this in mind, more attention should be paid to the presence of this clinical condition because it is consensual that a reduction in proprioception could increase the risk of sport injury. Future studies should continue to research changes in joint proprioception after exercise-induced muscle damage, particularly with the aim to differentiate whether these exercises interfere in the functioning of muscles spindles and/or in the regulation of spinal cord circuits.

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