Is eccentric exercise-induced torque decrease contraction type dependent?

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ABSTRACT
A. MICHAUT, M. POUSSON, N. BABAULT, and J. VAN HOECKE. Is eccentric exercise-induced torque decrease contraction type dependent? Med. Sci. Sports Exerc., Vol. 34, No. 6, pp. 1003–1008, 2002. Purpose: This study was designed to determine whether torque decrease following an acute eccentric exercise is contraction type dependent. Methods: Ten active males performed an exercise session consisting of five sets of ten maximal eccentric muscle actions of the elbow flexors. Before and immediately after the exercise, maximal voluntary eccentric (−60°·s⁻¹; Ecc60), isometric (0°·s⁻¹; Iso) and concentric (60°·s⁻¹; Con60 and 240°·s⁻¹; Con240) torque were measured. In order to distinguish central from peripheral factors involved in torque decrement, activation level (twitch interpolation technique), myoelectrical activity (RMS) of biceps brachii, as well as electrically evoked M-wave and peak twitch torque (Pt) were recorded. Results: The eccentric exercise induced a significant torque reduction (P < 0.01), whatever the muscular contraction type [mean (SD): −22.3 (8.1)% for Ecc60; −20.8 (11.2)% for Iso; −18.5 (6.1)% for Con60 and −12.5 (8.9)% for Con240]. Relative torque decrement was however significantly less for Con240 compared with Ecc60, Iso, and Con60 (P < 0.05). Torque decreases were associated with a reduction of both M-wave amplitude (P < 0.01) and Pt (P < 0.001), probably related to an impairment of the excitation-contraction coupling. Concurrently, activation level was reduced (P < 0.01), therefore indicating the occurrence of central fatigue, as also confirmed by RMS decreases for all the conditions (P < 0.05), except Con240. Discussion: An acute eccentric exercise induced a significant voluntary maximal torque reduction during eccentric, isometric, and concentric muscle actions ascribed to both peripheral and central failure of force production capacity. It can be concluded that eccentric exercise-induced torque decrease is not contraction type dependent. Key Words: MUSCLE FATIGUE, MUSCULAR CONTRACTION TYPE, TWITCH CONTRACTILE PROPERTIES, M WAVE

Acute muscular exercise generally induces the development of fatigue that has detrimental effects on performance. Eccentric exercise is well known to induce impairment of force-generating capacity (4,9,14,16,19,22,24), that could persist for several days (4,9,15,16,19). This strength loss is generally ascribed to excitation-contraction coupling impairment and ultrastructural damages, such as wavy Z-band, A-band disruptions, and overstretched sarcomeres (1,5,17). Such damage to the muscle-tendon system could find, at least in part, its origin in the high forces developed during eccentric muscle actions (12,13).

Most of the studies dealing with eccentric muscle actions have primarily focused on strength recovery following an eccentric exercise. Conversely, the mechanisms of early reduction of force are less discussed. Nevertheless, according to some authors (17,21), changes in organization of the sarcomere structure and changes in excitation-contraction (E-C) coupling appear to be the main contributors to the early strength reduction. However, the issue of whether force decrease induced by eccentric muscle actions could also be partly attributed to central fatigue is still unsettled. Indeed, after voluntary eccentric exercise, Pasquet et al. (20) and Saxton and Donnelly (23) did not find any central fatigue, whereas Gibala et al. (9) reported a 6% voluntary activation decrease using the twitch interpolation technique.

On the one hand, if early eccentric exercise-induced maximal torque reduction is mainly caused by a failure of the E-C coupling and of sarcomeres reinterdigitation (1,21), voluntary torque production capacity would be similarly reduced, whatever the muscular contraction type. On the other hand, it has been previously suggested that central fatigue could depend on the contraction type and/or contractile tension developed. For instance, after an isokinetic fatiguing exercise, Newham et al. (18) have reported an activation failure in isometric and slow concentric (20°·s⁻¹) contractions, whereas no activation failure was detected during higher velocity (150°·s⁻¹) concentric contractions. From these results, it could be hypothesized that if the eccentric exercise induced central fatigue, strength reduction could depend on either or both movement velocity and contractile tension developed. According to this hypothesis, torque decrease would be lower during concentric contractions and especially faster concentric contraction velocities. Thus, if the eccentric exercise induced both central and peripheral failure of force production capacity, it could be hypothesized that torque reduction would depend on the contraction type tested. However, eccentric exercise-induced strength loss
and its recovery have been mostly studied in isometric conditions (16,19,22,24). Therefore, the aim of the present study was to further investigate the effects of an acute eccentric exercise on the maximal torque production capacity of the elbow flexor muscles for voluntary eccentric vs isometric vs concentric muscle actions. In other words, this experiment was designed to test the hypothesis that eccentric exercise-induced torque decrease differed among contraction types, i.e., was contraction type dependent.

METHODS
Approach to the Problem and Experimental Design

The effects of 50 maximal voluntary eccentric muscle actions of the elbow flexors on eccentric (-60°·s⁻¹), isometric (0°·s⁻¹) and concentric (60°·s⁻¹) voluntary torque was determined on ten physically active male subjects. The relative contribution of central and peripheral failure of force production capacity to potential torque decrease was evaluated by analyzing the electromyographic (EMG) and mechanical responses obtained under maximal voluntary and electrically evoked conditions. Further, if eccentric exercise-induced torque reduction is both peripheral and central in origin, we hypothesized that exercise-induced torque decrease would differ among contraction types and would therefore be contraction type dependent.

Subjects

Ten healthy male sports science students with no previous history of pathology of the elbow [mean ± SD: 21.0 ± 1.5 yr; 181.8 ± 9.0 cm; 74.9 ± 6.7 kg] took part in this investigation. All subjects were physically active and were regularly trained (two to three times a week) in various ball-games. Written informed consent was obtained from all of the subjects before participation. The experimental procedure was performed in accordance with the Declaration of Helsinki and approved by local human research committee.

Data Collection

The tests were carried out using a Biodex isokinetic dynamometer (System 3: Biodex Shirley Corporation, Shirley, NY). The subjects were seated beside the dynamometer with both the right arm and forearm supported in the horizontal plane to avoid the effects of gravity. The forearm was tightly fixed in a gutter using belts, and the wrist was placed in a neutral position (between supination and pronation). The axis of rotation of the dynamometer was aligned with the epitenon-evi-proxymyondy axis of the arm. Extraneous movement of the upper body was limited using two crossover shoulder harnesses and an abdomen belt. The left arm was along the trunk with the forearm on the abdomen. Subjects performed dynamic muscle actions over a range of motion of 120°, and torque was measured at a constant angle of 60° (0°: full extension), i.e. in the middle of the range of motion. During eccentric muscle actions, the arm was brought back passively by the motor of the dynamometer from 0° to 120° and inversely (from 120° to 0°) during concentric contractions. Whatever the contraction type, the passive displacement of the arm to the starting position was made at the same angular velocity as the considered testing velocity (i.e. 60°·s⁻¹ for Ecc60 and Con60, and 240°·s⁻¹ for Con240).

The EMG recordings were obtained from two pairs of silver-chloride surface electrodes positioned parallel to the longitudinal axis of the muscle fibers on the middle portion of the muscle belly of the biceps brachii and lateral head of the triceps brachii muscles of the right arm. Low impedance at the skin-electrode interface was obtained (Z < 1.5 kΩ) by light abrasion of the skin. The inter-electrodes distance was 2 cm (center to center). The reference electrode was fixed to the left wrist. The position of the electrode was marked with indelible ink to ensure consistent electrode placement throughout the experiment. The EMG was amplified with a bandwidth frequency ranging from 10 Hz to 2 kHz (Common Mode Rejection Ratio = 90 dB; Z input = 100 MΩ; gain = 1000). Myoelectric activity amplitudes were calculated as Root Mean Square (RMS) values.

Single supramaximal electrical stimulations were delivered using a high-voltage stimulator (Digitimer Stimulator DS7, Digitimer Ltd., Hertfordshire, England) to determine elbow flexor twitch contractile properties and to estimate the voluntary activation level, using the twitch interpolation technique. The cathode was on the musculo-cutaneous nerve, midway between epicondylus medialis and processus coracoideus. This position was marked with indelible ink on the skin to ensure identical electrode placement throughout the experiment. The anode was a self-adhesive stimulation electrode (5 x 5 cm) located on the acromion. The stimulation parameters were: total output 400 V (max); fixed pulse width 1 ms; current range 100–200 mA. Stimulation intensity was increased until no further increase in elbow flexor twitch torque was observed. Supramaximal stimuli were then delivered at a 60° elbow angle (i) at rest to evoke a control twitch, (ii) during a maximal voluntary isometric contraction of the right elbow flexors to estimate the level of voluntary activation according to the twitch interpolation technique, and (iii) on relaxed muscles, two seconds after a maximal voluntary isometric contraction, to determine the postactivation potentiation. Torque, angular position, and EMG signals were digitized on-line (sampling frequency 1000 Hz) using a digital computer and stored on hard disk for further analysis.

Experiment Design

All subjects performed one session of familiarization with the measurement apparatus the week before the experiment. Each subject reported then to the laboratory for the test session. They carried out a standardized warm-up composed of ten submaximal concentric contractions and five submaximal isometric elbow flexions in the testing position (i.e. 60° elbow flexion). After the warm-up, subjects were asked to perform three maximal voluntary muscle actions of the elbow flexors at three different angular velocities, ran-
domly presented: $-60^\circ \cdot \text{s}^{-1}$ under eccentric conditions (Ecc60), $60^\circ \cdot \text{s}^{-1}$ and $240^\circ \cdot \text{s}^{-1}$ under concentric conditions (respectively Con60 and Con240). Subsequently, ten single twitch traces (1-s intervals) were applied on the relaxed muscles, and a set of three maximal voluntary isometric contractions (Iso), held for 2 s, was superimposed with single twitches. Two seconds after the end of each of the maximal voluntary isometric contractions, a single twitch was delivered on the relaxed muscles. A 1-min rest period was permitted between each set of three muscular actions in order to minimize the effects of fatigue and recuperation, before and after the exercise session, respectively. The same experimental procedure (lasting 15 min) was carried out before and immediately after the exercise session described below.

**The Exercise Session**

All subjects performed an exercise session consisting of 5 sets of 10 maximal eccentric muscle actions of the right elbow flexors using the isokinetic dynamometer described above. The angular velocity was set at $60^\circ \cdot \text{s}^{-1}$, over a $120^\circ$ range of motion. Thus each eccentric muscle action lasted two seconds, separated by a 2-s rest period. A 1.5-min rest period was allowed between each set.

**Data Analysis**

Maximal voluntary isometric and isokinetic torque, as well as EMG signals were averaged for the three trials. During dynamic muscle actions, RMS amplitude was calculated over a range of $20^\circ$ (10° on either side of the $60^\circ$ angle at which torque was measured). During isometric contractions, the RMS was calculated over a 0.5-s period after the torque had reached a plateau. RMS amplitudes of the biceps brachii were expressed as a fraction of the RMS value obtained during the maximal isometric contraction performed before the exercise. RMS amplitudes of the triceps brachii were expressed as a fraction of its RMS value obtained during a maximal isometric contraction when acting as an agonistic muscle.

The following parameters were recorded and averaged from ten mechanical twitch traces: peak twitch torque ($Pt$, N-m), and both twitch maximal rate of torque rise ($+dPt/dt$, N-m s$^{-1}$) and maximal relaxation rate ($-dPt/dt$, N-m s$^{-1}$). The postactivation potentiation (PAP, % of the control twitch) was determined on three trials by calculating the ratio between the amplitude of a twitch delivered 2 s after the end of a maximal isometric contraction and that obtained under control conditions. The peak-to-peak amplitude expressed in volts (V) as well as the duration (ms) of the biceps brachii M-wave associated with the control twitch were also recorded and averaged. Pt values were also expressed as a fraction of the M-wave amplitude (Pt-to-M-wave ratio). Finally, the level of voluntary activation was estimated for each test session according to the formula:

$$\text{Voluntary activation (%) } = \left[ 1 - \frac{\text{(Superimposed twitch torque/ Mean control twitch torque)}}{\text{100}} \right] \times 100.$$
The RMS values of biceps brachii are expressed in percentage of RMS obtained during ISO performed before the exercise. RMS values of triceps brachii are expressed in percentage of RMS obtained during a maximal voluntary elbow extension performed before the exercise. Values are means (SD). * Significantly different from value measured before the exercise (P < 0.05).

**DISCUSSION**

The main finding of the present study was that an eccentric exercise session induced a significant torque decrease whatever the contraction type considered. These torque decreases were associated with lower EMG (except for Con240), lower activation level, and modifications of twitch contractile properties.

Torque decrements, observed in isometric, concentric, and eccentric action modes (each of the contraction types considered) could partly have arisen from a peripheral failure of force production capacity. Such a phenomenon has been put in prominent position by the analysis of both EMG (M-wave) and mechanical (twitch) responses to electrical stimulation of the motor nerve. Indeed, the amplitude of the M-wave significantly decreased, and this event could indicate a failure in neuromuscular transmission and sarcoplemmal excitation (8), probably resulting from an alternation of Na+-K+-pumping (10). As a consequence, the twitch torque amplitude was reduced after the present exercise session. Nevertheless, Pt reduction would not result exclusively from the impairment of neuromuscular transmission. Indeed, the Pt-to-M-wave ratio was significantly reduced after the exercise session, indicating either or both an excitation-contraction (E-C) coupling failure and contractile process impairment. According to Ingalls et al. (11), the E-C coupling failure accounts for approximately 75% of the immediate force decrease after eccentric muscle actions performed in mouse extensor digitorum longus. These authors suggested that the E-C coupling defect was located at the t-tubule-sarcoplasmic reticulum (SR)-Ca2+ release channel interface. The exact causes of such failure are still unknown, but Ingalls et al. (11) hypothesized that it could result from ions and metabolic products accumulation reducing SR Ca2+ release, eccentric exercise-induced physiological disruptions and/or Ca2+ activated proteolytic degradation. Other authors have also hypothesized that changes in Ca2+ release could be induced by an eccentric muscle action (1). Since it is usually believed that, at maximum activation, the Ca2+ concentration is larger than the one required for the maximum activation of the contractile proteins (3), changes in Ca2+ release following eccentric muscle actions could partly explain the greater reduction in Pt compared with voluntary torque. Nevertheless, part of Pt reduction, as well as +dPt/dt and -dPt/dt decrease, could also be ascribed to other mechanisms, such as a reduction of the myofibrillar protein sensitivity for Ca2+ (resulting from acidosis and/or elevated Pi concentration) and/or of acto-myosin cross-bridges function (e.g., a reduction of tension developed by the cross-bridges and/or a reduced number of attached interactions).

**TABLE 1.** Normalized root mean square (RMS) amplitude values of the agonist biceps brachii and antagonist triceps brachii muscles at the different angular velocities, before and after the exercise session.

<table>
<thead>
<tr>
<th>Angular velocity</th>
<th>Biceps brachii Before</th>
<th>Biceps brachii After</th>
<th>Triceps brachii Before</th>
<th>Triceps brachii After</th>
</tr>
</thead>
<tbody>
<tr>
<td>-60°·s⁻¹</td>
<td>0.94 (0.25)</td>
<td>0.76 (0.26)*</td>
<td>0.27 (0.11)</td>
<td>0.29 (0.13)</td>
</tr>
<tr>
<td>0°·s⁻¹</td>
<td>1.00 (0.00)</td>
<td>0.83 (0.17)*</td>
<td>0.31 (0.10)</td>
<td>0.32 (0.09)</td>
</tr>
<tr>
<td>60°·s⁻¹</td>
<td>1.18 (0.28)</td>
<td>0.90 (0.23)*</td>
<td>0.32 (0.15)</td>
<td>0.33 (0.17)</td>
</tr>
<tr>
<td>240°·s⁻¹</td>
<td>1.02 (0.28)</td>
<td>0.87 (0.21)</td>
<td>0.37 (0.16)</td>
<td>0.39 (0.21)</td>
</tr>
</tbody>
</table>

RMS values of biceps brachii are expressed in percentage of RMS obtained during ISO performed before the exercise. RMS values of triceps brachii are expressed in percentage of RMS obtained during a maximal voluntary elbow extension performed before the exercise. Values are means (SD). * Significantly different from value measured before the exercise (P < 0.05).
cross-bridges), as well as changes in organization of the sarcomere structure (e.g. failure in thick and thin filament reinterdigitation). However, the hypothesis of a reduced Ca$^{2+}$ sensitivity could be partly rejected, because PAP, which mainly depends on contractile protein Ca$^{2+}$ sensitivity (25), was not modified by the present exercise. Moreover, the contractile process failure, and especially $+\Delta P_{d}/\Delta t$ reduction, could also be due to an increased compliance. Since an eccentric exercise did not induce any significant modification of the series elastic component intrinsic properties (16), it could be assumed that Pt reduction could partly be attributable to either or both a reduced number of attached cross-bridges (7) and of tension developed by each cross-bridge. Thus, even if the exact mechanism underlying peripheral failure of force production capacity assessed in the present study cannot be ascertained, it could mainly be attributed to an impairment of the E-C coupling, a failure of cross-bridges function, and also to eccentric exercise-induced ultrastructural fiber disruption (1,21,27).

As suggested in the introduction, the occurrence of peripheral failure of force production capacity could be partly responsible for torque decrease, whatever the contraction type considered. Nevertheless, when considering electromyographic activity as well as activation level results, it is likely that our exercise session also induced central fatigue. Indeed, voluntary activation level estimated during isometric contractions was significantly reduced by about 12%. This result is in accordance with Gibala et al. (9) who reported approximately 6% decrease of the activation level following eight sets of eight submaximal eccentric elbow flexions. Then, part of the isometric torque decrease could be ascribed to neural drive failure, as also indicated by the concomitant agonist EMG reduction (i.e. about 17%), as previously reported in humans (6,26). If one assumes that activation level and EMG activity are similarly depressed by fatigue whatever the contraction type (i.e. dynamic and isometric), one could conclude that central fatigue also occurred during Ecc60 and Con60. What remained unclear was whether central fatigue was induced by the eccentric exercise or was an adaptive response to force production capacity failure. Indeed, Morgan and Allen (17) have suggested that the way the brain drives muscle activity was changed by eccentric exercise-induced pain and weakness. According to this hypothesis, central fatigue should be considered as a consequence of eccentric exercise-induced contractile failure of force production capacity. Nevertheless, no significant reduction of biceps brachii myoelectrical activity was observed for Con240. This last result could suggest that central changes were minor or absent during muscular contractions performed at the angular velocity of 240° s$^{-1}$. This hypothesis could be partly reinforced by the observation of Newham et al. (18). Following an isokinetic fatiguing exercise, these authors have effectively shown an activation failure in isometric and slow (20° s$^{-1}$) concentric contractions, whereas no activation failure was detected during higher velocity (150° s$^{-1}$) concentric contractions. Thus, the eccentric exercise-induced fatigue would be related to different neural patterns according to the movement velocity and/or the contractile tension developed (i.e. torque value). First, because of the higher movement velocity of Con240, the absence of RMS modification for Con240 could be attributed to the duration of the contraction, which was four times shorter than for Ecc60 and Con60. Second, the lack of activation failure for Con240 could be ascribed to a force-related mechanism, resulting in either a gradual decrease in the amount of activation failure as velocity increases, or alternatively a critical force above which none occurred (18,28). Finally, one would expect that the central fatigue measured for Ecc60, Iso, and Con60 after the exercise could partly arise from a reinforcement of the neural inhibiting mechanism, previously described during eccentric (2,28) and slow concentric (28) muscle actions, when the volunteer is fresh, that act as a protective mechanism by limiting overuse and damage. This may not occur during fast concentric contractions. These hypotheses could therefore account for the lower relative agonist EMG activity and torque decrements measured for Con240.

In conclusion, the present eccentric exercise-induced torque decrease was not contraction type dependent since voluntary torque was significantly reduced, whatever the muscular contraction type considered. This torque decrement could mainly be attributed to peripheral failure of force production capacity probably related to an impairment of the E-C coupling. However, torque reduction was much more pronounced during isometric and slow eccentric and concentric muscle actions compared with faster concentric contractions. This could be attributed, at least in part, to neural pattern changes, with the occurrence of central fatigue during Ecc60, Iso and Con60. Although the present study did not examine myoelectrical activity of the brachial- and brachioradialis involved in elbow flexion, it should be carefully examined in future studies. Thus, further investigations are necessary to target the mechanisms involved in the lack of significant modifications during fast concentric muscle actions following an acute eccentric exercise.

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