

Technical note

Force output during and following active stretches of rat plantar flexor muscles: effect of velocity of ankle rotation

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Abstract

During the development of force deficits by repeated stretches, velocity-sensitive changes in the extra force produced during and after subsequent stretching has not been studied. In the present study, repeated dorsiflexion of the foot of rats with maximally contracting plantar flexor muscles was performed at two angular velocities [0.87 (slow muscle stretch) and 10.47 rad s⁻¹ (fast muscle stretch)] to examine the active force of the muscles during and following dorsiflexion. Dorsiflexion was performed 30 times with a rest period of 3 min between the stretches to minimize muscle fatigue. The ability of rat plantar flexor muscles to produce additional force during the stretch was not velocity sensitive. In contrast, repeated dorsiflexion with fast muscle stretches, but not with slow muscle stretches, resulted in an increase in the force decay with time following the stretches (i.e. increased stress relaxation), as indicated by a change in the time constant of force decay during stress relaxation. Apparently, the stress-relaxation of rat plantar flexor muscles is sensitive to angular velocity of ankle movements; repeated fast, but not slow dorsiflexion, alters the stress relaxation process of active skeletal muscles exposed to stretches which create a force deficit. The change in time constant of force decay during stress relaxation in response to a series of repeated stretches might provide information on the sarcomere length distribution in skeletal muscles. © 2000 Elsevier Science Ltd. All rights reserved.

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1. Introduction

The production of extra force during and following stretches of activated skeletal muscles has been described for undamaged muscles (Abbott and Aubert, 1952; Cook and McDonagh, 1995; Edman et al., 1978) but not for muscles in which prior stretching produced force deficits indicative of muscle injury (Hesselink et al., 1996). For normal (undamaged) skeletal muscles, there is a relationship between velocity of stretching and the extra force produced during the stretch at low velocities of stretch such that the faster the stretch the greater the extra force produced. At higher velocities of stretching, no such relationship exists as force remains constant with increasing stretching velocity. In addition to velocity, the magnitude of the extra force depends on initial length, length changes and final muscle length (Edman et al., 1978). The

magnitude of the extra force produced by stretching also correlates with the force deficit produced. The force deficit caused by exposure of muscle to unaccustomed stretches is taken as indirect evidence of muscle injury (e.g. McCully and Faulkner, 1986) because there is no correlation between the amount of structural damage (e.g. sarcomere disruption) and the force deficit.

For the extra force recorded after the stretch (i.e. stress-relaxation), velocity of stretching was without effect for undamaged muscles (Edman et al., 1978). Stress-relaxation has not been studied in muscles exposed to repeated stretching or following other types of muscle injury. For injured muscles where structural and functional parameters are changing, the response to subsequent stretching could be quite different than for undamaged muscles. Since repeated stretches with and without muscle damage are common during sports and occupational tasks, knowledge of any altered biomechanics would be useful in predicting functional outcomes.

Using velocity-controlled joint rotations to produce the stretches, the extra force during and following stretches was monitored as a function of repetition

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number to determine the effects of angular velocity of repeated stretches on the capacity for extra force production and stress-relaxation. It was found that stress-relaxation following stretches became sensitive to the angular velocity with repeated fast but not slow stretches.

2. Methods

2.1. Animal care and preparation

Experiments were performed on plantar flexor muscles of female Sprague–Dawley rats (body weight 264.9 ± 5.0 g) and approved by the West Virginia University Animal Care and Use Committee (WVU-ACUC #9511-05). Animal requests for this study complied with Animal Welfare Act P.L. 91-579 and DHHS Guidelines governing the care and use of laboratory animals. Rats were kept anaesthetized with sodium pentobarbital (75 mg kg^{-1} intraperitoneal) and euthanized by an intracardial injection of sodium pentobarbital at the end of the experiments. The dissection procedure for nerve cuff placement, animal positioning, dynamometer and force recording are described elsewhere (Cutlip et al., 1997; Willems and Stauber, 1999).

2.2. Experimental procedure

Angular force-plate position corresponds to ankle angle, defined as the angle between tibia and plantar surface of the foot (Willems and Stauber, 1999). With the force-plate at 2.09 rad, 6–8 isometric contractions were performed to establish maximal muscle activation parameters by electrical stimulation of the tibial nerve ($200 \mu\text{s}$ pulse duration; 80 Hz, 5.4 ± 0.3 V) and used for all contractions.

Active forces of plantar flexor muscles were determined during and following repeated stretches (30) at two velocities of foot dorsiflexion [0.87 rad s^{-1} (slow stretch) and 10.47 rad s^{-1} (fast stretch)]. Six animals were tested for each velocity. Dorsiflexion occurred in a range of motion (0.70 – 1.57 rad) used during rat locomotion (Gruner et al., 1980) and around optimum length of plantar flexors (Willems and Stauber, 1999). Stretches were initiated at an ankle position of 1.57 rad on muscles isometrically contracting for 600 ms by dorsiflexion to 0.70 rad. The return stroke occurred after stimulation. Three minutes before the first active stretch, dorsiflexion of the same magnitude and velocity without muscle activation was performed providing passive force data. Stimulation time for contractions was 1900 ms with rest periods of 3 min between contractions to minimize muscle fatigue. Slow and fast stretches lasted 1000 and 120 ms, respectively. Stress-relaxation was allowed for 300 and 1180 ms for slow and fast stretches and analyzed for the first 300 ms.

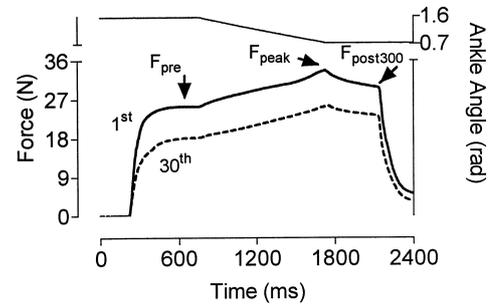


Fig. 1. Typical example of the active force of rat plantar flexor muscles during the first (solid line) and 30th (broken line) contraction with slow ankle rotation (i.e. dorsiflexion at 0.87 rad s^{-1}). F_{pre} , F_{peak} , and F_{post300} represent active force before the stretch, the highest active force during the stretch and the active force 300 ms following the stretch, respectively (arrows).

2.3. Data collection and analysis

Force and force-plate position were sampled at 264 Hz. Active muscle force values were calculated by subtracting the force during dorsiflexion with no activation from the total force during dorsiflexion with muscle activation (peak passive forces were less than 5% of the total peak forces during the first stretch—minor changes in passive force due to subsequent stretches with active muscles were ignored). Only active muscle force values were evaluated in this study. The isometric force preceding a stretch was designated F_{pre} (Fig. 1) and calculated between 500 and 600 ms after stimulation. Slow and fast stretches produced similar force deficits, indicated by the isometric force preceding each stretch [1 h of rest did not alter the force deficit (unpublished observations)]. Force peaked at the end of the stretch (F_{peak} see Fig. 1 for example of slow stretches) and decayed with time after the stretch (i.e. stress-relaxation) (Fig. 1). In the force trace, the kink up to the end of the stretch was observed in 4 out of 6 animals with slow stretches (most extreme shown in Fig. 1) but not in animals with fast stretches. The final stretch force and the force at 300 ms during stress-relaxation were designated F_{peak} and F_{post300} (Fig. 1). The ratio of F_{peak} and F_{pre} represented the force increase during stretch. The ratio of F_{peak} and F_{post300} was considered the relative relaxation of the muscle. Force during stress-relaxation was normalized by $(F(t) - F_{\text{post300}})/(F_{\text{peak}} - F_{\text{post300}})$ where $F(t)$ is the force at t ms during stress-relaxation. The equation:

$$F = A + B \exp[-C(t - t_{\text{peak}})] \quad (1)$$

where F is the force, t the time where force values between F_{peak} and F_{post300} were reached and t_{peak} the time where F_{peak} was reached, was fit to the force decay between F_{peak} and F_{post300} . A , B and C are fitting constants. $0.69/C$ was taken as the time constant (τ) of force decay during stress-relaxation. Exponential fittings yielded r^2 of 0.98 ± 0.02 .

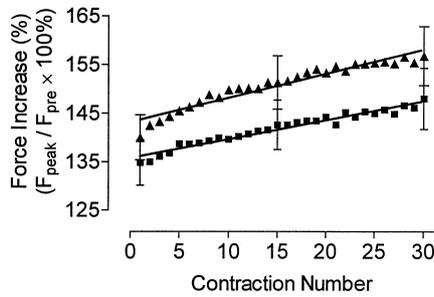


Fig. 2. Relationship between contraction number and the increased force during stretching (i.e. ratio of F_{peak} and F_{pre} multiplied by 100%). The increased force due to the stretches was not velocity sensitive. For clarity, only the SEs of contraction numbers 1, 15, and 30 are plotted. Data are presented as mean \pm SE for plantar flexor muscles of six animals. \blacktriangle = slow stretches, \blacksquare = fast stretches. Lines represent linear fits.

2.4. Statistics

Linear regression analysis was performed to test for velocity on the increase in force during the stretch, relative relaxation, and the time constant of force decay as a function of contraction number (GraphPad Prism version 3.00 for Windows, GraphPad Software, San Diego, CA, USA). GraphPad Prism was used to test slopes between regression lines of data sets with slow and fast stretches. Significance was accepted at $p < 0.05$.

3. Results

The increased force due to the stretches (i.e. $F_{\text{peak}}/F_{\text{pre}}$, see Fig. 1) was not velocity sensitive [Fig. 2, slopes of regression lines were not different ($p = 0.44$)] but the relative relaxation following stretches (i.e. $F_{\text{peak}}/F_{\text{post300}}$) was velocity sensitive [Fig. 3, slopes of regression lines were different ($p = 0.02$)]. Fast stretches resulted in a greater increased relative force during stress-relaxation at 300 ms compared to slow stretches (Fig. 3). All force data for fast stretches showed larger variability than data for slow stretches. Ratios of F_{peak} and F_{post300} (Fig. 3) do not provide information about the non-linear time course of force decay during stress-relaxation.

The time constant (τ) of force decay during stress-relaxation increased with fast but not with slow stretches (Fig. 4) indicating increased relative force production during stress-relaxation following fast stretches.

4. Discussion

Repeated stretches of active skeletal muscles produce force deficits indicative of injury (Hesslink et al., 1996). Injury itself could alter the active force developed during and following subsequent repeated stretches. Velocity

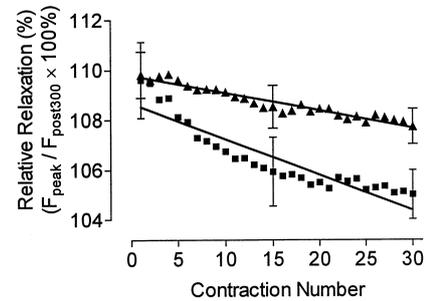


Fig. 3. Relationship between contraction number and relative relaxation (i.e. ratio of F_{peak} and F_{post300} (active force values) multiplied by 100%). The relative relaxation following stretches was larger for fast stretches. Data (\blacktriangle = slow stretches, \blacksquare = fast stretches) are presented as mean \pm SE for plantar flexor muscles of six animals. For clarity, only the SEs of contraction numbers 1, 15, and 30 are plotted. Lines represent linear fits.

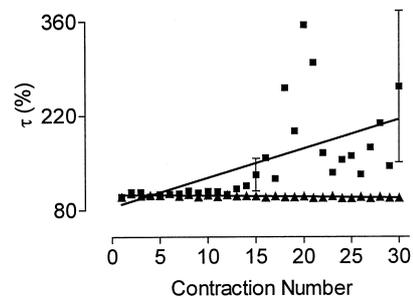


Fig. 4. The time constant (τ) of active force decay plotted as a function of contraction number for slow (\blacktriangle) and fast (\blacksquare) stretches. Fast stretches increased the relative force production during stress-relaxation. The time constants of active force decay were normalized with respect to the time constant in the first contraction in each series of slow and fast movements. For clarity, only the SEs of contraction numbers 15 and 30 are plotted. Data are presented as mean \pm SE for plantar flexor muscles of six animals. Lines represent linear fits.

may play a role. Contractile parameters such as absolute isometric force and the rate of force development have been studied following unaccustomed stretches (Lieber and Fridén, 1993; Warren et al., 1993). We examined in this study the force during and following stretches of maximally activated rat skeletal muscles at slow (0.87 rad s^{-1}) and fast (10.47 rad s^{-1}) velocities of ankle rotation. The force enhancement during stretch was not velocity sensitive. An absence of an influence of the velocity of stretch has also been shown for the force deficit due to unaccustomed stretches of active muscles (Talbot and Morgan, 1998; Warren et al., 1993) and muscle fibres (Lynch and Faulkner, 1998). In contrast, the force decay following the stretch became smaller for fast stretches and was unchanged for slow stretches. We cannot exclude that the changes seen with fast stretches were due to greater effects of uncontrolled variables such as changes in properties of connective tissue and changes in functional muscle length.

In the present study, it was assumed that the heterogeneity of sarcomere lengths was not different in both groups before the stretches. Although non-contractile components (i.e. connective tissue and cytoskeletal proteins) participate, the major change in stress-relaxation following fast stretches most likely results from changes in sarcomere length. Morgan (1990) proposed that during active stretches a non-uniform distribution of sarcomere lengths develops with some overstretched sarcomeres becoming injured. This 'injury' hypothesis was confirmed by observations of sarcomere lesions following stretching (Talbot and Morgan, 1996). Morgan (1994) commented that formation of sarcomere non-uniformity is likely to be influenced by stretch velocity — very slow stretches could result in more uniform sarcomere length distribution, although the velocity of slow stretches was not quantified. Based on pilot data of gastrocnemius muscle (Caiozzo et al., 1992), an angular velocity of 0.87 rad s^{-1} of rat plantar flexors was considered slow (i.e. 5–6% of the maximal angular velocity).

Passive elements, which connect adjacent myofibrils and link together longitudinal elements, act in series with strong myofibrillar segments (Edman and Tsuchiya, 1996). As long as sufficient strong elements were active during slow and fast stretching, little difference would be expected in force during the stretch if the sarcomeres were uniform or non-uniform. However, differences would appear during the active relaxation phases, if non-uniform sarcomeres readjusted their lengths — some becoming shorter and others longer. With uniform sarcomere length distribution, no readjustment would occur as most sarcomeres remain with adequate filament overlap to maintain the enhanced force. If non-uniform sarcomere length distribution exists, readjustment could lead to some sarcomeres being pulled further apart by the sarcomeres with greater filament overlap. As sarcomeres shorten and move closer to optimal filament overlap, increased force would result. Based on this line of reasoning, fast stretches in our study probably produced greater sarcomere length heterogeneity than slow stretches. More sarcomeres would have adequate filament overlap to maintain force.

In summary, repeated fast but not slow stretches of rat plantar flexor muscles by dorsiflexion of the foot produced a smaller relative decline in active relaxation force. The change in time constant of force decay during stress-relaxation of skeletal muscles in response to a series of repeated fast stretches may indicate greater sarcomere length heterogeneity.

Acknowledgements

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