

Performance of plantar flexor muscles with eccentric and isometric contractions in intact rats

MARK E. T. WILLEMS and WILLIAM T. STAUBER

Department of Physiology, West Virginia University, Morgantown, WV 26506

ABSTRACT

WILLEMS, M. E. T., and W. T. STAUBER. Performance of plantar flexor muscles with eccentric and isometric contractions in intact rats. *Med. Sci. Sports Exerc.*, Vol. 32, No. 7, pp. 1293–1299, 2000. **Purpose:** To examine the changes in performance of active plantar flexor muscles of rats by controlled dorsiflexion (i.e., stretching of muscles) at two angular velocities. **Methods:** Repeated stretches (30) at two velocities of ankle rotation [slow stretch ($0.87 \text{ rad}\cdot\text{s}^{-1}$ (i.e., $50^\circ\cdot\text{s}^{-1}$)), fast stretch ($10.47 \text{ rad}\cdot\text{s}^{-1}$ (i.e., $600^\circ\cdot\text{s}^{-1}$))] were superimposed on maximally active muscles from an ankle position of 1.57 rad to 0.70 rad (i.e., from 90° to 40°). Repeated isometric contractions (30) of the same duration (1900 ms) and rest interval (3 min) were performed at 1.13 rad (i.e., 65°). Performance was assessed by measuring the isometric torque at ankle positions of 1.57 and 0.70 rad, work during concentric contractions [range of motion 1.22 rad (i.e., 70°)], and the time to produce 50% of the maximal isometric torque. **Results:** Thirty isometric contractions resulted in a linear reduction in torque (total deficit of 13.8% at 1.57 rad), whereas for slow and fast stretches, half of the total, nonlinear deficit at 1.57 rad (about 30%) was completed after six stretches. Increases in half contraction times were larger for stretches than for isometric contractions. Reductions in isometric torque were greater at an ankle position of 1.57 rad than at 0.70 rad. One hour of rest after the repeated stretches and isometric contractions did not restore muscle performance. **Conclusions:** Isometric contractions of skeletal muscle can create a torque deficit which is much less than that after stretches. Repeated fast and slow stretches resulted in similar torque deficits which did not recover after a rest period of 1 h. **Key Words:** SKELETAL MUSCLE, MUSCLE INJURY, MUSCLE FORCE, VELOCITY, ANKLE, DYNAMOMETER

For humans performing repetitive occupational tasks, the velocity of movements *in vivo* correlated with musculoskeletal disorders (6,19) supporting the hypothesis that velocity may play a role in repetitive strain injury. In humans, movements involving repeated stretches of active skeletal muscles (i.e., lengthening during contraction) result in an immediate reduction in isometric force (2,3,5,22). This reduction in isometric force after repeated stretches of active skeletal muscles might ultimately lead to task-related musculoskeletal disorders.

The reduction in isometric force immediately after a series of repeated stretches of active skeletal muscles could result from the combined effects of cellular injury and fatigue. Structural [sarcomere disruption (7), loss of membrane integrity (9,27)] and functional [E-C coupling failure (13)] evidence for cellular injury have been reported. The role of fiber type (18), initial fiber length, and work input

during the stretch (12) on the reduction in isometric force has been reported. Four studies investigated the effect of velocity of stretch on force deficits using single stretches of muscle fibers (17) or repeated stretches of skeletal muscle (20,24,26). McCully and Faulkner (20) used three stretch velocities (0.2, 0.5, and $1.0 \text{ Lf}\cdot\text{s}^{-1}$) on mouse EDL muscle *in situ*. In this study, a force deficit 3 d after exercise-induced injury was associated with an increase in velocity of stretch because an increase in velocity of stretch correlated with larger peak forces. Warren et al. (26) used three stretch velocities (0.5, 1.0, and $1.5 \text{ Lo}\cdot\text{s}^{-1}$) on soleus muscle *in vitro* of young rats with unphysiological high $[\text{Ca}^{2+}]$ that, as discussed by the authors, influenced the slow decline of muscle function after the “injury” protocol. It cannot be excluded that high calcium levels influenced the force deficits during the “injury” protocol as well. Still, Warren et al. (26) did find some evidence for greater declines in performance at higher lengthening velocities. A weak effect of velocity on changes in muscle function was found by Talbot and Morgan (24) using two stretch velocities ($3.0 \text{ Lo}\cdot\text{s}^{-1}$ and $4.0 \text{ Lo}\cdot\text{s}^{-1}$) on toad sartorius muscle *in vitro*. In the study of single stretches by Lynch and Faulkner (17), five stretch velocities (0.5, 1.0, 2.0, 3.0, and $4.0 \text{ Lf}\cdot\text{s}^{-1}$) were

0195-9131/00/3207-1293/0

MEDICINE & SCIENCE IN SPORTS & EXERCISE®

Copyright © 2000 by the American College of Sports Medicine

Submitted for publication January 1999.

Accepted for publication October 1999.

used on skinned fast twitch muscle fibers from the EDL muscle of adult male Fischer 344 rats. Although fibers exposed to a stretch of $4.0 \text{ Lf}\cdot\text{s}^{-1}$ ($167\% V_{\text{max}}$) had a failure rate of 40% during the stretch compared with a failure rate of only 5% or less for slower velocities, it was concluded that there was no velocity effect (17). Based on these four studies, the evidence for an effect of velocity of stretch on contraction-induced force deficits seems to favor a slightly larger decline in performance with higher velocities of stretch. The effect of velocity of stretch has not been tested on muscle-tendon units in intact animals (11,16). We tested the hypothesis that repeated stretches of skeletal muscle by fast velocity joint-movements would induce a larger decline in muscle performance than slow velocity joint-movements in intact rats. Using the intact animal, stretches occur within the anatomical range of motion.

The purpose of this study was to measure the performance of rat muscles *in situ* during repeated active stretches (velocity-controlled ankle rotations) and repeated isometric contractions. Performance was assessed by measuring the maximum isometric torque at ankle positions of 0.70 and 1.57 rad (i.e., 40° and 90°), positive work during concentric contractions, and time to produce 50% of the maximal isometric torque. Rest periods between isometric contractions and active stretches minimized effects of fatigue from stimulation.

METHODS

Animal care and preparation. Caged-sedentary, female Sprague Dawley rats (3–4 months) were used in this study. Animal care and handling and all experimental procedures were approved by and followed the guidelines of the West Virginia University Animal Care and Use Committee (WVU-ACUC #9511–05) and were conducted in accordance with the principles of the American College of Sports Medicine.

The animals were anesthetized with sodium pentobarbital ($75 \text{ mg}\cdot\text{kg}^{-1}$ i.p.), and supplementary doses were administered as required to maintain an adequate depth of anesthesia. The trachea was cannulated using polyethylene tubing (PE 240, Becton Dickinson, Gaithersburg, MD) to facilitate breathing and the animal was positioned on a heating pad to maintain body temperature.

A midline incision was made in the posterior aspect of the hindlimb and the tibial nerve exposed by blunt dissection through the popliteal fossa. Access to the nerve was facilitated by careful removal of connective tissue and adipose tissue surrounding the nerve. The common peroneal and sural nerves were cut. A bipolar cuff electrode made of silastic tubing (Dow Corning, Midland, MI) was positioned around the tibial nerve. Silastic tubing used had an inner and outer diameter of 1.67 and 2.41 mm, respectively. The position of the cuff was secured by tying nylon sutures around it without compressing the tibial nerve. The distance between the steel wires (Cooner wire AS632, Chatsworth, CO) in the cuff was approximately 1.5 mm. The wires of the cuff were lead out of the popliteal fossa and the skin was

closed using nylon sutures. The free ends of the wire electrodes were connected to the nerve stimulator (Grass SD9 stimulator, Grass Medical Instruments, Quincy, MA).

The animal was placed supine on a X-Y positioning table attached to the dynamometer. Temperature of the left hindlimb was controlled between 30 and 35°C using an infrared lamp and a temperature probe directly attached to the skin. Position of the knee [1.57 rad (i.e., 90°)] was secured in a knee holder. The left foot was compressed on an aluminum plate with the ankle axis, assumed to lie on a line between the medial and lateral malleoli, aligned with the axis of rotation of the motor. The aluminum plate was connected to a custom-built dynamometer (4). The dynamometer involves a permanent magnet servomotor (Type 1000 DC, Model 1410) and an Unidex 1 single axis motion controller (Aerotech Inc., Pittsburgh, PA). The force applied on the aluminum plate at the sole of the foot was translated into a vertical movement of the aluminum plate relative to a $Z\text{-}11.5\text{-kg}^{-1}$ load cell (HBM Inc., Marlboro, MA). The fixture of aluminum plate and load cell will be referred to as force-plate. A schematized drawing of foot placement of the rat on the aluminum plate and knee holder is shown in Figure 1. Force was measured under the sole of the foot of the rat and converted to torque values for the plantar flexors by multiplying force values with the ankle axis of rotation and the border of the proximal component of the thenar pad of the foot (10mm).

During muscle contractions, the foot was kept firmly positioned (compression force: 12–20 N) using two cross-bars. Between tests, the cross-bars were released to avoid swelling of the foot due to compression. Rotational movement of the force-plate and timing of stimulation were controlled by the computer.

Position of the ankle [e.g., 1.57 rad (i.e., 90°)] is defined as the angle between the tibial longitudinal axis and the plantar surface of the foot. Angular position of the force-plate corresponds approximately to the ankle angle (28). Six to eight isometric contractions were performed at a force-plate position of 2.09 rad (i.e., 120°) to establish the stimulus parameters for maximal activation of the plantar flexors by electrical stimulation of the tibial nerve [200 μs pulse

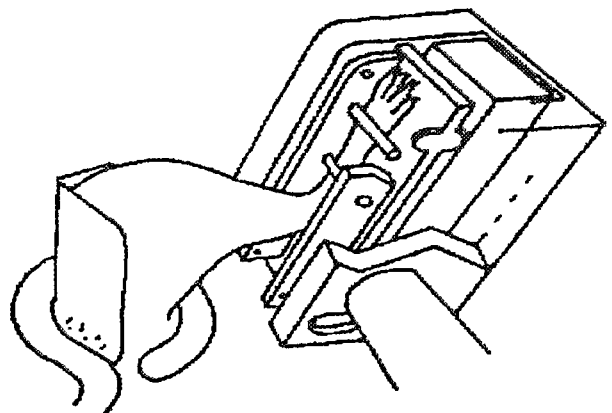


Figure 1—Position of the rat's foot in load cell fixture and knee holder (from ref. 4, reprinted with permission from *Med. Biol. Eng. Comput.* 35:541).

duration, 80 Hz, 5.4 ± 0.3 V (mean \pm SE)]. These stimulation parameters were constant throughout the testing protocols. All dynamic contractions were executed in a range of motion [concentric contractions: range 0.70–2.09 rad (i.e., 40° – 120°); active stretches: range 0.70–1.57 rad; see below for details of contractions] that covered the range of motion that occurs during rat locomotion (10). Once the experiments were ended, the rats were euthanized with a lethal intracardial injection of sodium pentobarbital.

Experimental procedure. The overall experimental protocol for initial testing, the testing with repeated isometric contractions and active stretches, and the early and late recovery testing are presented in Figure 2. Initial and recovery testing consisted of concentric contractions and isometric contractions performed three times each. Concentric contractions provide information on torque production over a specific range of motion and can be used to calculate positive work. For concentric contractions, the force-plate was moved from 2.09 to 0.70 rad with an angular velocity of $0.52 \text{ rad}\cdot\text{s}^{-1}$ (i.e., $30^\circ\cdot\text{s}^{-1}$) with relaxed muscles. After 100 ms pause, the plantar flexors were maximally activated (isometric preload), and 600 ms later the force-plate was returned to 2.09 rad at an angular velocity of $0.52 \text{ rad}\cdot\text{s}^{-1}$, concentric contraction). Total stimulation time was 3400 ms. Each concentric contraction was preceded by a movement of the same magnitude with passive (i.e., noncontracting muscles) and subtracted from the movement with contracting muscles. Each series of three concentric contractions was averaged. These preloaded slow concentric contractions allow analysis of the isometric torque of the plantar flexors at an ankle position of 0.70 rad before the movement. Isometric contractions were performed at a force plate position of 1.57 rad. Stimulation time of isometric contractions was 600 ms. Rest periods were two min for isometric contractions and four min for concentric contractions. Rest periods between contractions were chosen to allow recovery of fatigue from contractions at high stimulation frequency (80 Hz).

Repeated isometric contractions and repeated stretches. The repeated isometric contractions and slow and fast velocity stretches, 30 each, were performed to determine changes in isometric torque, half contraction times and positive work. Six animals each were used for repeated isometric, slow stretch and fast stretch testing, respectively. Stretches were initiated 600 ms after the onset of nerve stimulation from an ankle position of 1.57 rad, isometric preload) by rotational movement of the ankle to an

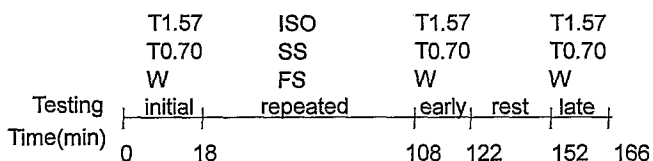


Figure 2—Time sequence of contractions during initial testing, repeated stretches and early and late recovery. W: work during concentric contractions; T0.70 and T1.57: isometric torque at an ankle position of 0.70 and 1.57 rad, ISO, SS, and FS: 30 isometric contractions, 30 slow ($0.87 \text{ rad}\cdot\text{s}^{-1}$) or fast ($10.47 \text{ rad}\cdot\text{s}^{-1}$) stretches (see methods for details of contractions).

ankle position of 0.70 rad at velocities of $0.87 \text{ rad}\cdot\text{s}^{-1}$ (i.e., $50^\circ\cdot\text{s}^{-1}$) and $10.47 \text{ rad}\cdot\text{s}^{-1}$ (i.e., $600^\circ\cdot\text{s}^{-1}$). Based on pilot data of gastrocnemius muscle (1), angular velocities of $0.87 \text{ rad}\cdot\text{s}^{-1}$ and $10.47 \text{ rad}\cdot\text{s}^{-1}$ of rat plantar flexors were estimated to be 5–6% and 60–75% of maximal angular velocity. The repeated active stretches of the plantar flexors during ankle rotations of $0.87 \text{ rad}\cdot\text{s}^{-1}$ and $10.47 \text{ rad}\cdot\text{s}^{-1}$ will be referred to as slow and fast velocity stretches. The passive return of the foot was performed after the end of stimulation. The first slow or fast velocity stretch was preceded by a movement of the same magnitude with noncontracting muscles (passive movement). Isometric contractions were performed at an ankle position of 1.13 rad (i.e., 65°), which was in the midrange of the range of motion during the stretches. Stimulation time was 1900 ms for isometric contractions and stretches with rest periods of 3 min between contractions.

Data collection and analysis. The force-plate position and force output were sampled at 264 Hz. The force-plate position was converted to ankle position by repeated measurements ($N = 3$) of the ankle angle (i.e., angle between a line from the lateral epicondyle of the tibia and the plantar surface of the foot) by the use of goniometry at force-plate positions between 1.05 and 2.09 rad (i.e., 60° and 120°) with steps of 0.35 rad (i.e., 20°). The angular positions of the force-plate were fitted with a one degree polynomial with respect of ankle position ($r^2 = 0.998 \pm 0.001$). The ankle positions at force-plate positions less than 1.05 rad were extrapolated from the curves. Active torque values for dynamic contractions were calculated by subtracting from the total torque during movements with muscle activation the torque during movements with no muscle activation. For isometric torque, the active torque was calculated by subtracting the average torque 100 ms before stimulation from the average total torque between 500 and 600 ms after stimulation (i.e., on the tetanic plateau). For repeated isometric contractions and stretches, third order polynomials were used ($r^2 > 0.99$) to calculate the time to produce 50% of the maximal isometric torque. The time to produce 50% of the maximal isometric torque during each contraction in the series of repeated isometric contractions and stretches were expressed as a percentage of the time in the first contraction in the series. For concentric contractions, positive work was calculated as the area under the curve between force and ankle position in the ankle range of motion between 0.79 and 2.01 rad (i.e., 45° and 115°) omitting the acceleration and deceleration periods at the beginning and end of the movement.

At the end of the experiment the length of the gastrocnemius medialis muscle (GM) (i.e., distance between proximal end of proximal aponeurosis and distal end of the most distal fiber bundle) and length of the plantaris muscle (PL) (i.e., distance between the proximal end of the most proximal fiber bundle and the distal end of the most distal fiber bundle) was measured with the knee in flexion (1.57 rad) and ankle angle at 1.57 rad using a pair of dividers and a ruler with a resolution of 0.5 mm. In order to facilitate

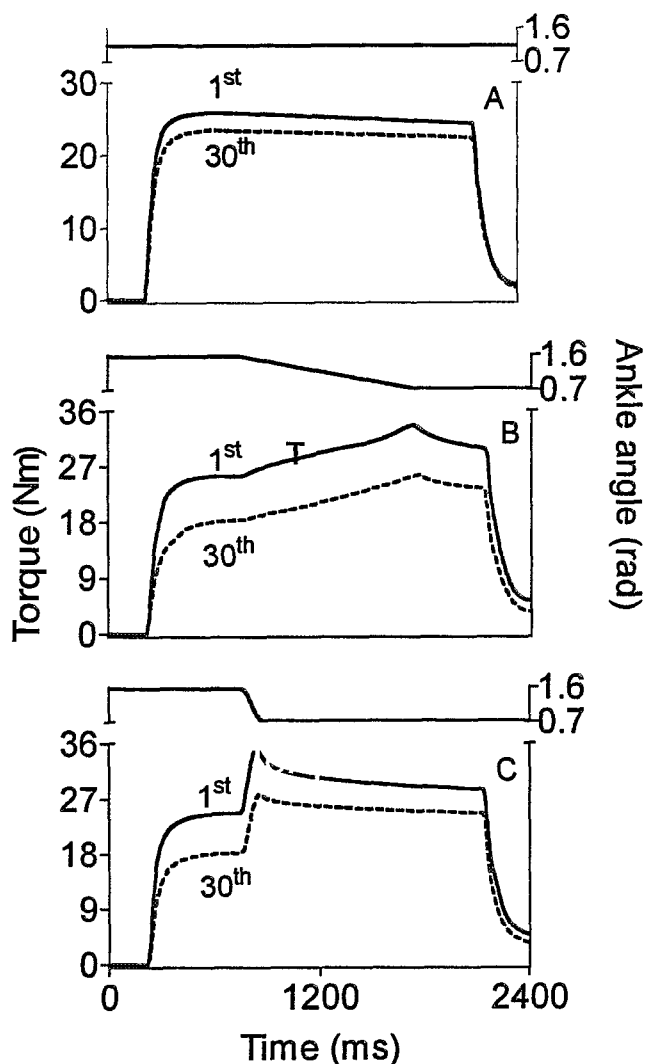


Figure 3—Examples of the torque output of rat plantar flexors working *in situ* during the 1st and 30th contraction in each series of isometric contractions, slow ($0.87 \text{ rad}\cdot\text{s}^{-1}$) and fast ($10.47 \text{ rad}\cdot\text{s}^{-1}$) stretches (A, B, and C, respectively). Top traces in each figure represent ankle position.

length measurements of the PL, GM and part of the gastrocnemius lateralis muscle were removed.

Statistics. One way analysis of variance (ANOVA) was used to test: 1) the effect of repeated isometric contractions and stretches on isometric torque at ankle positions of 0.70 and 1.57 rad and positive work during initial and recovery testing, and 2) to test for length of GM and PL of rats exposed to isometric contractions and slow and fast velocity stretches. Two-way ANOVA was performed to test for the effect of velocity on the isometric preload torque. When a significant *F*-ratio was found, *post hoc* testing was done with a Tukey test to determine where specific differences had occurred. Values were presented as mean \pm SE for $N = 6$ per group. Significance was accepted at $P < 0.05$.

RESULTS

Among the three experimental groups [i.e., isometric contractions (ISO), slow stretch (SS) and fast stretch (FS)],

there were no differences in body weight (ISO: $255.3 \pm 5.6 \text{ g}$; SS: $263.8 \pm 6.5 \text{ g}$; FS: $266.0 \pm 8.2 \text{ g}$), isometric torque at an ankle position of 1.57 rad (ISO: $0.23 \pm 0.01 \text{ Nm}$; SS: $0.23 \pm 0.01 \text{ Nm}$; FS: $0.24 \pm 0.02 \text{ Nm}$) and muscle length of GM (ISO: $31.7 \pm 0.3 \text{ mm}$; SS: $31.5 \pm 0.3 \text{ mm}$; FS: $31.5 \pm 0.4 \text{ mm}$) and PL (ISO: $33.7 \pm 0.5 \text{ mm}$; SS: $33.1 \pm 0.4 \text{ mm}$; FS: $33.0 \pm 0.2 \text{ mm}$) at an ankle position of 1.57 rad. In addition, positive work did not differ during initial testing (data not shown).

Repeated isometric contractions and stretches.

The isometric contractions (Fig. 3A) resulted in nearly constant force levels (i.e., plateau) after the build-up of isometric torque. For slow and fast velocity stretches, ankle rotations were started after the build-up of isometric torque (Fig. 3B and 3C). At both velocities, the torque reached a peak at the end of the rotation (i.e., at the end of the stretch). Peak muscle torques were similar at the end of slow and fast stretches (i.e., at similar joint positions) at comparable contraction numbers during the injury protocol (1st contraction; SS: $0.31 \pm 0.02 \text{ Nm}$, FS: $0.30 \pm 0.02 \text{ Nm}$; 30th contraction; SS: $0.25 \pm 0.02 \text{ Nm}$, FS: $0.24 \pm 0.02 \text{ Nm}$).

Changes in isometric torque during the series of repeated isometric contractions and stretches.

The relationship between the decrease in isometric torque due to repeated isometric contractions was linear ($r^2 > 0.98$) (Fig. 4). The change in isometric torque at an ankle position of 1.13 rad by the series of 30 repeated isometric contractions was 10% and significant (one way ANOVA). In contrast, a larger nonlinear decrease in isometric torque (approximately 30%) was recorded for both slow and fast velocity stretches by the 30th stretch. For both velocities, half of the total decrease in isometric torque after 30 stretches was completed after six stretches. There was no effect of angular velocity on the decrease in isometric torque (two-way ANOVA, $P = 0.09$) (Fig. 4).

Changes in half contraction time during the series of repeated isometric contractions and re-

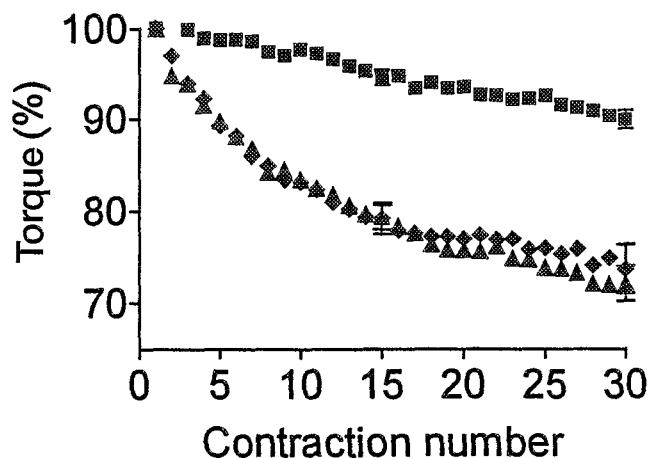


Figure 4—The relationship between contraction number and the isometric torque at an ankle position of 1.57 rad for the stretches and 1.13 rad for the isometric contractions. Torque was normalized by the torque during the first contraction in the series of isometric contractions (■), slow (\blacktriangle) ($0.87 \text{ rad}\cdot\text{s}^{-1}$) and fast (\blacklozenge) ($10.47 \text{ rad}\cdot\text{s}^{-1}$) stretches. For clarity of the figure, only error bars of contraction 15 and 30 are plotted.

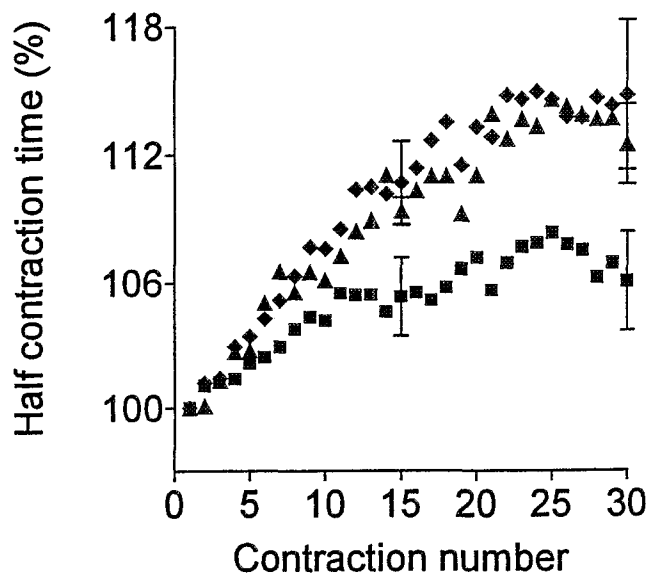


Figure 5—The relationship between contraction number and half contraction time at an ankle position of 1.57 rad for the stretches and 1.13 rad for the isometric contractions. Half contraction times were expressed as a percentage of the half contraction time during the first contraction in the series of isometric contractions (■), slow (▲) (0.87 rad·s⁻¹) and fast (◆) (10.47 rad·s⁻¹) stretches.

peated stretches. During repeated isometric contractions and repeated stretches there was an increase in half contraction time (Fig. 5) that was larger for the stretches. After 1 h of recovery (i.e., rest), the increase in half contraction time due to repeated isometric contractions and repeated stretches was still present.

Isometric torque at two ankle positions and positive work immediately after and 1 h after a series of repeated isometric contractions and stretches. Larger decreases in isometric torque were found at ankle angles of 1.57 rad than at 0.70 rad after repeated stretches but not after repeated isometric contractions (Table 1). At both ankle positions, the decreases in isometric torque due to stretches were larger than the torque deficit created by the isometric contractions. One hour of rest of the muscle after each series of repeated isometric contractions and active stretches (i.e., late recovery) did not alter the torque deficits at each ankle angle (Table 1). The decrease in positive work resulting from the repeated stretches was greater than from repeated isometric contractions (Table 1). There was no effect of velocity on the decrease in positive work. Decrease in positive work did not recover after 1 h of rest (Table 1).

DISCUSSION

Chronic exposure to repeated slow and fast stretches in intact rats (i.e., slow and fast angular velocities) produces muscle injury that can lead to adaptation or pathology (23). In the present study, the effect of slow and fast stretches on muscle injury of plantar flexor muscle working *in situ* was tested in our intact rat model using velocity-controlled ankle rotations. Functional impairments which were present after 1 h of recovery, such as an isometric torque deficit, an increase in half contraction time and decrease in positive work, were taken as indirect evidence of muscle injury. Force deficits induced by 40 preloaded concentric contractions producing fatigue (31% of initial force value) recover within 30 min to 88% of the initial force value (unpublished observations), suggesting that the torque deficits present 1 h after the fast and slow stretches were indicative of muscle injury. Compared with repeated sustained isometric contractions, the active stretches produced larger reductions in isometric torque, larger increases in half contraction time, and larger decreases in positive work. These alterations in muscle performance were taken as indirect evidence of muscle injury and not caused by metabolic fatigue.

The hypothesis that the angular velocity of joint movements with stretches of skeletal muscle working *in situ* is a factor in producing torque deficits was not supported. Likewise, for isolated muscles and single muscle fibers, it has been shown that stretch velocity is not a critical parameter for force deficits due to eccentric contractions (17,24,26). However, the possibility of a larger decline in performance at velocities much above 10.47 rad·s⁻¹ remains.

Most of the torque deficit due to repeated stretching occurred early in the series for both stretch protocols but not for repeated isometric contractions. Several factors likely participate in the exponential character of the decrease in torque from stretching an active muscle. First, each successive stretch in a series of stretches starts with a lower isometric force and attains smaller peak forces during the stretch compared to the previous one. Peak forces during eccentric contractions have been shown to be an important factor in producing subsequent force deficits (26). Second, if some sarcomeres become overstretched by the stretching protocol, other sarcomeres would be at shorter lengths in order to keep the muscle length constant for any specific ankle position. Overstretched sarcomeres would result in functionally shorter muscle lengths and a shift of the length-force relationship of the muscle (e.g., 14). Subsequent

TABLE 1. Early and late recovery after 30 isometric contractions and 30 slow and fast stretches.

Groups	T1.57 (%)		T0.70 (%)		W (%)	
	Early	Late	Early	Late	Early	Late
ISO	13.8 ± 2.0	12.9 ± 1.4	11.7 ± 1.1	12.3 ± 1.2	13.7 ± 1.7	13.6 ± 1.1
SS	31.5 ± 2.7*†	31.2 ± 3.0*†	23.2 ± 2.3†	20.6 ± 3.1†	24.1 ± 2.2†	22.4 ± 2.6†
FS	30.5 ± 3.2*†	27.7 ± 3.5*†	22.9 ± 1.5†	19.4 ± 1.8†	24.0 ± 2.0†	20.9 ± 2.5†

Values are presented as mean ± SE, N = 6 per group. T1.57(%), percent torque deficit at 1.57 rad (i.e. 90°); T0.70(%), percent torque deficit at 0.70 rad (i.e., 40°); W(%), percent decline in positive work; ISO, 30 isometric contractions; SS, 30 slow stretches; FS, 30 fast stretches.

* Significantly different from same group at different ankle position, P < 0.05.

† Significantly different from protocol with repeated isometric contractions, P < 0.05.

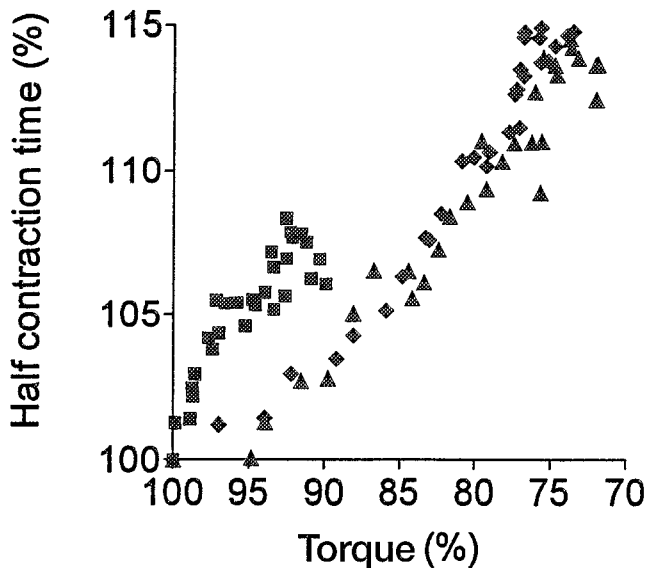


Figure 6—Relationship between isometric force and half contraction time at an ankle position of 1.57 rad for the stretches and 1.13 rad for the isometric contractions. Torque was normalized by the torque during the first contraction in each series. Half contraction times were expressed as a percentage of the half contraction time during the first contraction in each series. ■, ▲, and ◆ indicate isometric contractions, slow ($0.87 \text{ rad}\cdot\text{s}^{-1}$) and fast ($10.47 \text{ rad}\cdot\text{s}^{-1}$) stretches, respectively.

stretches might be initiated or even occur on the ascending limb of the relationship (i.e., at functionally shorter muscle lengths). When single stretches of extensor digitorum longus muscles *in situ* of mice were initiated at relatively short muscle lengths, smaller force deficits were observed (12).

In human skeletal muscle *in vivo* repeated eccentric contractions (stretches) shift the isometric length-force relationship downward (i.e., torque declines) and toward joint positions indicative of functionally longer muscle lengths (14). Due to a shift in length-force relationship, the absolute torque decline at muscle lengths greater than optimum length is smaller than the torque decline at muscle lengths less than optimum length. Our results, torque deficits were larger at relatively short muscle lengths, provide indirect evidence for a shift in the length-force relationship of rat plantar flexor muscles working *in situ*. Shifts in muscle

optimum length (i.e., length-force relationship) after eccentric contractions have also been reported for isolated toad muscle (29), frog muscle (15) and frog single muscle fibers (21). Taken together, these observations support the hypothesis that the cause for the shift in isometric length-force relationship is located within the muscle fibers (i.e., sarcomeres of each fiber).

Failure in the E-C coupling explained 57–75% of the decline in force out to 5 d after contraction-induced injury *in vivo* (13). Excitation-contraction coupling ends with the release of calcium from the sarcoplasmic reticulum. After eccentric contractions, a loss of calcium homeostasis (8) and loss of sarcoplasmic reticulum membrane integrity (25) were observed. The present study provides indirect evidence for altered release of calcium by showing an increase in the time to reach 50% of the maximal isometric torque. The increase in the time to reach 50% of the maximal isometric torque was also observed during sustained isometric contractions (1900 ms), albeit to a lesser extent. Interestingly, similar reductions in torque due to isometric contractions and stretches were associated with smaller changes in half contraction time for the stretches (Fig. 6), suggesting that causal factors for reductions in torque are independent from factors causing changes in half contraction time. Our data do not distinguish which events are causative for deceleration of the rate of torque development. It is concluded that a series of sustained isometric contractions and active stretches reduce the isometric torque output and decelerate the rate of torque development.

In summary, angular velocity within the range tested did not appear to be a critical factor in the decline in performance in skeletal muscle torque working *in situ*. Repeated eccentric contractions decrease the isometric torque and the rate of torque development in rat plantar flexors working *in situ* more than during repeated isometric contractions.

This work was supported by the National Institute of Occupational Safety and Health, Centers for Disease Control (R01-OHAR-02918).

Address for correspondence: William T. Stauber, Ph.D., FACSM, Department of Physiology, West Virginia University, PO Box 9229, Morgantown, WV 26506-9229; E-mail: wstauber@wvu.edu.

REFERENCES

- CAIOZZO, V. J., E. M. A. SAMUEL, A. MCCUE, E. SMITH, R. E. HERRICK, and K. M. BALDWIN. A new model for modulating myosin isoform expression by altered mechanical activity. *J. Appl. Physiol.* 73:1432–1440, 1992.
- CHLEBOUN, G. S., J. N. HOWELL, R. R. CONATSER, and J. J. GIESEY. Relationship between muscle swelling and stiffness after eccentric exercise. *Med. Sci. Sports Exerc.* 30:529–535, 1998.
- CLARKSON, P. M., K. NOSAKA, and B. BRAUN. Muscle function after exercise-induced muscle damage and rapid adaptation. *Med. Sci. Sports Exerc.* 24:512–520, 1992.
- CUTLIP, R. G., W. T. STAUBER, R. H. WILLISON, T. A. MCINTOSH, and K. H. MEANS. Dynamometer for rat plantar flexor muscles *in vivo*. *Med. Biol. Eng. Comput.* 35:540–543, 1997.
- DAVIES, C. T., and M. J. WHITE. Muscle weakness following eccentric work in man. *Pflügers Arch.* 392:168–171, 1981.
- FATHALLAH, F. A., W. S. MARRAS, and M. PARNIANPOUR. The role of complex, simultaneous trunk motions in the risk of occupation-related low back disorders. *Spine* 23:1035–1042, 1998.
- FRIDÉN, J., M. SJÖSTRÖM, and B. EKBLÖM. Myofibrillar damage following intense eccentric exercise in man. *Int. J. Sports Med.* 4:170–176, 1983.
- FRIDÉN, J., and R. L. LIEBER. Ultrastructural evidence for loss of calcium homeostasis in exercised skeletal muscle. *Acta Physiol. Scand.* 158:381–382, 1996.
- FRIDÉN, J., and R. L. LIEBER. Segmental muscle fiber lesions after repetitive eccentric contractions. *Cell Tissue Res.* 293:165–171, 1998.
- GRUNER, J. A., J. ALTMAN, and N. SPIVACK. Effects of arrested cerebellar development on locomotion in the rat. Cinematographic and electromyographic analysis. *Exp. Brain Res.* 40:361–373, 1980.
- HESSSELINK, M. K. C., H. KUIPERS, P. GEURTEN, and H. VAN STRAATEN. Structural muscle damage and muscle strength after incremental number of isometric and forced lengthening contractions. *J. Muscle Res. Cell Motil.* 17:335–341, 1996.
- HUNTER, K. D., and J. A. FAULKNER. Plyometric contraction-induced injury of mouse skeletal muscle: effect of initial length. *J. Appl. Physiol.* 82:278–283, 1997.

13. INGALLS, C. P., G. L. WARREN, J. H. WILLIAMS, C. W. WARD, and R. B. ARMSTRONG. E-C coupling failure in mouse EDL muscle after in vivo eccentric contractions. *J. Appl. Physiol.* 85:58-67, 1998.
14. JONES, C., T. ALLEN, J. TALBOT, D. L. MORGAN, and U. PROSKE. Changes in the mechanical properties of human and amphibian muscle after eccentric exercise. *Eur. J. Appl. Physiol.* 76:21-31, 1997.
15. KATZ, B. The relationship between force and speed in muscular contraction. *J. Physiol. (Lond.)* 96:45-64, 1939.
16. LIEBER, R. L., M. C. SCHMITZ, D. K. MISHRA, and J. FRIDÉN. Contractile and cellular remodeling in rabbit skeletal muscle after cyclic eccentric contractions. *J. Appl. Physiol.* 77:1926-1934, 1994.
17. LYNCH, G. S., and J. A. FAULKNER. Contraction-induced injury to single muscle fibers: velocity of stretch does not influence the force deficit. *Am. J. Physiol.* 275 (Cell Physiol. 44):C1548-C1554, 1998.
18. MACPHERSON, P. C. D., M. A. SCHORK, and J. A. FAULKNER. Contraction-induced injury to single fiber segments from fast and slow muscles of rats by single stretches. *Am. J. Physiol.* 271(Cell Physiol. 40):C1438-C1446, 1996.
19. MARRAS, W. S., and R. W. SCHOENMARKLIN. Wrist motions in industry. *Ergonomics* 36:341-351, 1993.
20. McCULLY, K. K., and J. A. FAULKNER. Characteristics of lengthening contractions associated with injury to skeletal muscle fibers. *J. Appl. Physiol.* 61:293-299, 1986.
21. MORGAN, D. L., D. R. CLAFLIN, and F. J. JULIAN. The effects of repeated active stretches on tension generation and myoplasmic calcium in frog single muscle fibres. *J. Physiol. (Lond.)* 497:665-674, 1996.
22. NOSAKA, K., and P. M. CLARKSON. Changes in indicators of inflammation after eccentric exercise of the elbow flexors. *Med. Sci. Sports Exerc.* 28:953-961, 1996.
23. STAUBER, W. T., K. K. KNACK, G. R. MILLER, and J. G. GRIMMETT. Fibrosis and intercellular collagen connections from four weeks of muscle strains. *Muscle Nerve* 19:423-430, 1996.
24. TALBOT, J. A., and D. L. MORGAN. The effects of stretch parameters on eccentric exercise-induced damage to toad skeletal muscle. *J. Muscle Res. Cell Motil.* 19:237-245, 1998.
25. YASUDA, T., K. SAKAMOTO, K. NOSAKA, M. WADA, and S. KATSUTA. Loss of sarcoplasmic reticulum membrane integrity after eccentric contractions. *Acta Physiol. Scand.* 161:581-582, 1997.
26. WARREN, G. L., D. A. HAYES, D. A. LOWE, and R. B. ARMSTRONG. Mechanical factors in the initiation of eccentric contraction-induced injury in rat soleus muscle. *J. Physiol. (Lond.)* 464:457-475, 1993.
27. WARREN, G. L., D. A. LOWE, D. A. HAYES, M. A. FARMER, and R. B. ARMSTRONG. Redistribution of cell membrane probes following contraction-induced injury of mouse soleus muscle. *Cell Tissue Res.* 282:311-320, 1995.
28. WILLEMS, M. E. T., and W. T. STAUBER. Isometric and concentric performance of electrically stimulated ankle plantar flexor muscles in intact rat. *Exp. Physiol.* 84:379-389, 1999.
29. WOOD, S. A., D. L. MORGAN, and U. PROSKE. Effects of repeated eccentric contractions on structure and mechanical properties of toad sartorius muscle. *Am. J. Physiol.* 265 (Cell Physiol. 34): C792-C800, 1993.

A vertical bar on the left side of the page, consisting of a series of yellow and orange rectangular segments, with a small red diamond at the top.

COPYRIGHT INFORMATION

TITLE: Performance of plantar flexor muscles with eccentric and isometric contractions in intact rats

SOURCE: Medicine and Science in Sports and Exercise 32 no7 JI 2000

WN: 0018301727017

The magazine publisher is the copyright holder of this article and it is reproduced with permission. Further reproduction of this article in violation of the copyright is prohibited..

Copyright 1982-2002 The H.W. Wilson Company. All rights reserved.