

Force deficits by stretches of activated muscles with constant or increasing velocity

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ABSTRACT

WILLEMS, M. E. T., and W. T. STAUBER. Force deficits by stretches of activated muscles with constant or increasing velocity. *Med. Sci. Sports Exerc.*, Vol. 34, No. 4, pp. 667–672, 2002. **Purpose:** Force deficits produced by constant (CV) versus increasing velocity (IV) stretches of rat plantar flexor muscles at low and high levels of nerve activation were studied. **Methods:** Twenty repeated stretches were imposed on isometric contractions by ankle rotation from 90° to 40° at 300°·s⁻¹ and at 3000°·s⁻² during 80-Hz (CV80 and IV80) and 20-Hz stimulation (CV20 and IV20). Rest periods between contractions were 3 min. Isometric and peak stretch forces during the stretch protocols and force-frequency relationships before and 1 h after the stretch protocols were measured. **Results:** Peak stretch forces were similar for IV80-CV80 and for IV20-CV20 rats but were lower for IV20-CV20 than for IV80-CV80 rats throughout the stretch protocol. At the end of the stretch protocol, isometric force deficits were similar for IV80 (49.9 ± 2.1%) and CV80 (54.5 ± 2.5%) and for IV20 (16.4 ± 2.8%) and CV20 (15.8 ± 1.9%) but lower for IV20-CV20 rats. In contrast, for all groups, deficits in peak stretch force were similar at the end of the stretch protocol (IV80: 35.0 ± 1.8%, CV80: 32.3 ± 2.2%, IV20: 26.8 ± 3.6%, CV20: 28.0 ± 2.0%). After 1 h, isometric force deficits were similar for either IV80-CV80 or IV20-CV20 at 5, 10, 20, 40, 60, and 80 Hz stimulation but were lower for IV20-CV20. **Conclusions:** Variation in velocity of ankle rotation with similar peak stretch forces did not influence the amount of stretch-induced force deficits. High peak stretch forces produced greater isometric force deficits than low peak stretch forces, but the relative loss in peak stretch force was not force dependent. Different mechanisms may account for isometric force deficits and peak stretch force deficits caused by repeated stretches of activated skeletal muscles. **Key Words:** SKELETAL MUSCLE, INJURY, ECCENTRIC CONTRACTION, JOINT ROTATION, ACCELERATION, KINEMATICS

Skeletal muscles show functional (22) and structural signs (1) of injury after unaccustomed stretches during activity (i.e., strain injury). Strain-injured muscles lose the ability to produce isometric force at both low and high stimulation frequencies (5,8), commonly referred to as an isometric force deficit, requiring days to recover (11).

The effect of several mechanical factors that are associated with producing force deficits such as peak stretch force, initial muscle length, length changes, and velocity have been studied in human and animal models. During stretches of activated skeletal muscles, peak stretch force has been shown to be the most important determinant for the isometric force deficits after stretches (5,14,19). Peak stretch force was varied by fatigue (isometric contractions), stretch velocity, stimulation frequency, and amount of lengthening in isolated EDL of mice (14), by stimulation frequency and amount of lengthening in isolated soleus of rats (19), and in humans by using percutaneous stimulation (5). The amount of force deficits was a function of peak stretch force. Repeated eccentric contractions of human knee extensor muscles resulted in larger losses in maximum voluntary force when tested at long compared with short muscle lengths (6). In animals, the amount of work during stretching and initial length of the muscle before stretching proved to be factors

that influence the magnitude of the loss in isometric force (10). Several studies used a range of constant velocities to test velocity as a mechanical factor for producing force deficits in single muscles (19) and single muscle fibers (13). Large stretch velocities in isolated rat soleus muscle resulted in greater declines in isometric force (19). We showed in intact rats that the isometric force deficits that developed with slow and fast velocity of ankle rotations were similar (22). Whether functional changes by stretches with constant acceleration are different than stretches with constant velocity has not been tested.

In the present study, we examined the functional changes that result from increasing-velocity (i.e., constant acceleration) and constant-velocity stretches with submaximal and near maximal activation of skeletal muscles. The functional changes were assessed by the development of isometric and peak stretch force deficits during the series of stretches and by the isometric force deficits as a function of stimulation frequency 1 h after the series of stretches.

MATERIALS AND METHODS

Animal care and preparation. Female Sprague-Dawley rats (4–5 months) were used in this study. Experimental procedures were approved by and followed the guidelines of the West Virginia University Animal Care and Use Committee (WVU-ACUC no. 9809-02) and were conducted in accordance with the principles of the American College of Sports Medicine. The use of rats for this study complied with Animal Welfare Act P.L. 91-579 and guidelines from

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the Department of Health and Human Services governing the care and use of laboratory animals. Rats were anesthetized with sodium pentobarbital (initial dose $75 \text{ mg}\cdot\text{kg}^{-1}$ i.p.), and supplementary doses were administered to suppress the hindlimb withdrawal reflex when squeezing the foot. The susceptibility of plantar flexor muscles to reductions in isometric and peak stretch force during repeated stretches with high and low levels of nerve activation (stimulation frequency 80 Hz and 20 Hz, respectively) during ankle rotations with constant velocity and increasing velocity was studied.

Details on the dissection procedure for placement of the nerve cuff around the tibial nerve, positioning of the rats, and dynamometer and force recording have been described elsewhere (7,21). Briefly, the rat's foot was positioned on an aluminum plate that was connected to a dynamometer. The knee was held at 90° . The dynamometer consists of a DC permanent magnet servomotor (Model 1410C) and an Uni-dex 100 single axis motion controller (Aerotech Inc, Pittsburgh, PA). A load cell was positioned below the aluminum plate. Contraction of the plantar flexor muscles was induced by electrical stimulation of the nerve, and the reaction force was recorded under the sole of the foot (21–23). Rotational movement of the aluminum plate and timing of stimulation were computer controlled. Isometric contractions of 600 ms with rest periods of 2 min were used to determine the voltage for maximal force production with a stimulation frequency of 80 Hz. Voltage and pulse duration ($200 \mu\text{s}$) were kept constant for each muscle preparation. Skin temperature of the left hindlimb was maintained around 32° by using an infrared lamp and a surface temperature probe.

Experimental procedure. Stretches of activated plantar flexor muscles were performed during ankle rotations controlled for increasing velocity (acceleration $3000^\circ\cdot\text{s}^{-2}$) at a stimulation frequency of 20 Hz [i.e., group IV20, body weight: $279 \pm 7 \text{ g}$ (mean \pm SE)] and 80 Hz (i.e., group IV80, body weight: $273 \pm 7 \text{ g}$) and controlled for constant velocity ($300^\circ\cdot\text{s}^{-1}$) at a stimulation frequency of 20 Hz (i.e., group CV20, body weight: $269 \pm 2 \text{ g}$) and 80 Hz (i.e., group CV80, body weight: $283 \pm 8 \text{ g}$). Six animals were used in each group. In each group, 20 stretches with activated muscles were initiated 400 ms after the onset of stimulation from an ankle position of 90° by rotational movement to 40° ; the stretching time was the same for all groups (Fig. 1A). The return movement from 40° to 90° with an angular velocity of $90^\circ\cdot\text{s}^{-1}$ but without muscle activation was performed 1.0 s after end of each stretch. Rest periods between the stretches were 3 min to minimize fatigue. For the stretches controlled for increasing velocity, there was nonlinear change of ankle position throughout the range of motion (Fig. 1A) with a linear increase in angular velocity from 90° to 50° and a linear decrease in angular velocity from 50° to 40° providing constant acceleration for 80% of the range of motion (Fig. 1B). For the constant velocity stretches, there was a linear change of ankle position throughout most part of the range of motion (Fig. 1A) with a nonlinear change in velocity from 90° to 86° (i.e., acceleration) and from 48° to 40° (i.e., deceleration) providing constant velocity for 76%

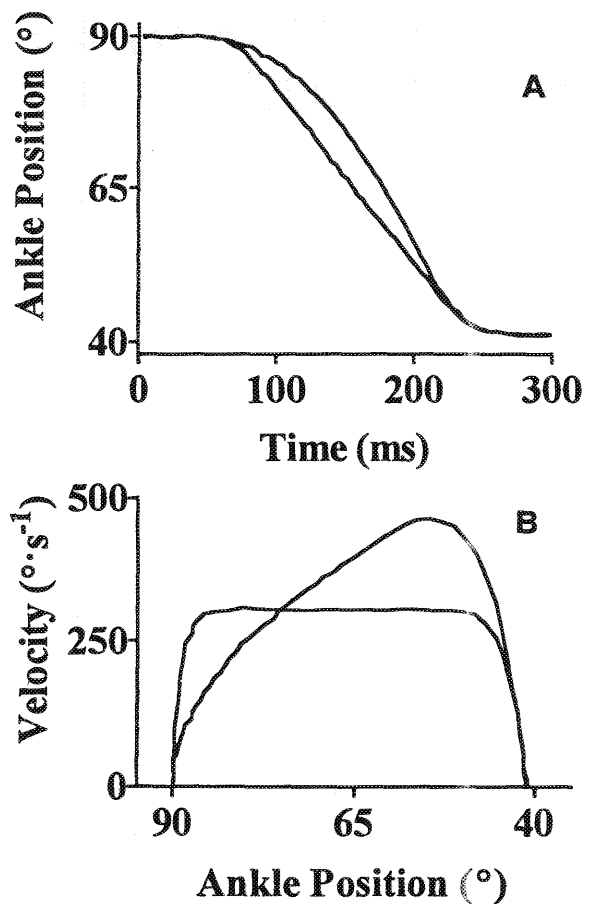


FIGURE 1—Relationships between ankle position and time (A) and angular velocity and ankle position (B) showing the performance of the dynamometer.

of the range of motion (Fig. 1B). Relative changes in isometric and peak stretch force have been taken as indirect evidence of stretch-induced muscle injury because initial force levels could be different but produce the same relative deficits.

Force-frequency measurements. Force-frequency measurements were performed at an ankle position of 90° before the stretch protocol and after 1 h of rest after the stretch protocol using isometric contractions. Plantar flexor muscles were stimulated at 5 Hz (1500 ms), 10 Hz (1500 ms), 20 Hz (1500 ms), 40 Hz (900 ms), 60 Hz (600 ms), and 80 Hz (600 ms). A stimulation frequency of 80 Hz provided over 90% of maximal force values that were obtained with a stimulation frequency of 120 Hz (unpublished observations). Stimulation frequencies between 5 and 80 Hz cover the range from low to near maximal force production of rat plantar flexor muscles. In our setup, plantar flexor muscles recovered almost completely within 30 min from a fatigue test consisting of 40 contractions without stretches that resulted in force losses of about 70% (23). Furthermore, isometric force deficits after 1 h of rest after the repeated stretches of activated muscles have been taken as indirect evidence of stretch-induced muscle injury (2). At the end of the experiments, intracardial injections of sodium pentobarbital were used to euthanize the rats.

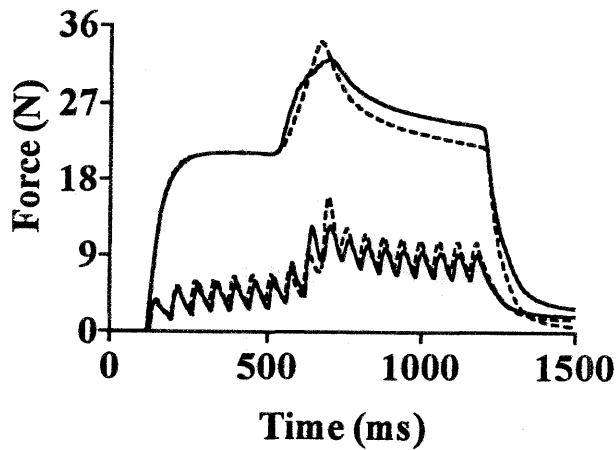


FIGURE 2—Examples of the force of rat plantar flexor muscle for the first stretch imposed on isometric contractions. IV80, rats exposed to 20 stretches with increasing velocity (i.e., acceleration of $3000^{\circ}\text{s}^{-2}$) and a stimulation frequency of 80 Hz (top traces broken line); IV20, rats exposed to 20 stretches with increasing velocity and a stimulation frequency of 20 Hz (bottom traces broken line); CV80, rats exposed to 20 stretches with a constant velocity (i.e., 300°s^{-1}) and a stimulation frequency of 80 Hz (top traces solid line); CV20, rats exposed to 20 stretches with a constant velocity and a stimulation frequency of 20 Hz (bottom traces solid line).

Statistics. For all groups, one-way analysis of variance (ANOVA) was used to test: 1) peak stretch forces of the 1st stretch, 2) peak stretch forces of the 20th stretch, 3) isometric force deficits before the 20th stretch, 4) peak stretch force deficits of the 20th stretch, and 5) isometric force deficits at each stimulation frequency after 1 h of rest after the stretches. Two-way ANOVA with repeated measures on contraction number was performed to test 1) peak stretch force, 2) isometric force deficits, and 3) peak stretch force deficits. Two-way ANOVA with repeated measures on stimulation frequency was performed to test the isometric force deficits 1 h after the stretches. When a significant *F*-ratio was found, *post hoc* testing was done with a Bonferroni test to determine where specific differences had occurred. Values were presented as mean \pm SE. Significance was accepted at $P < 0.05$.

RESULTS

Peak stretch forces during the repeated stretches. Typical examples of force traces for the 1st stretch in each of the four groups are illustrated in Figure 2. Stretches with a stimulation frequency of 20 Hz resulted in lower peak stretch forces than stretches with a stimulation frequency of 80 Hz (Fig. 2). For constant-velocity and increasing-velocity stretches with a stimulation frequency of 20 Hz, peak stretch forces were similar at comparable contraction numbers during the stretch protocol (CV20: 1st stretch: 13.6 ± 0.8 N, 20th stretch: 9.7 ± 0.9 N) (IV20: 1st stretch: 16.5 ± 1.8 N, 20th stretch: 12.1 ± 1.5 N) but lower throughout the stretch protocol than stretches with a stimulation frequency of 80 Hz (Fig. 3). For stretches with constant velocity and increasing velocity with a stimulation frequency of 80 Hz, peak stretch forces were similar at

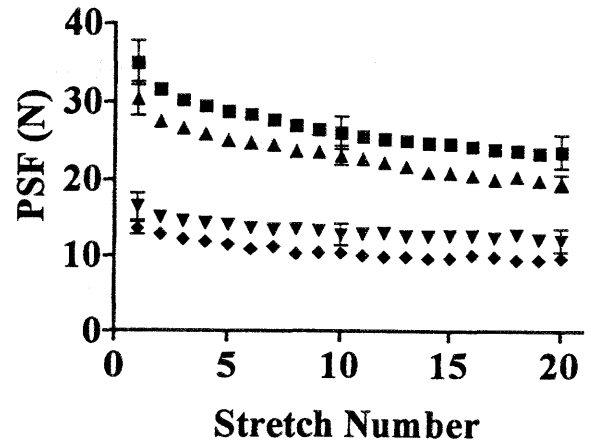


FIGURE 3—The relationship between stretch number and peak stretch force (PSF). \blacktriangle , IV80: rats exposed to 20 stretches with increasing velocity (i.e., acceleration of $3000^{\circ}\text{s}^{-2}$) and a stimulation frequency of 80 Hz; \blacksquare , CV80: rats exposed to 20 stretches with a constant velocity (i.e., 300°s^{-1}) and a stimulation frequency of 80 Hz; \blacklozenge , IV20: rats exposed to 20 stretches with increasing velocity and a stimulation frequency of 20 Hz; \blacktriangledown , CV20: rats exposed to 20 stretches with a constant velocity and a stimulation frequency of 20 Hz. For clarity of the figure, only error bars of contractions 1, 10, and 20 are plotted.

comparable contraction numbers during the stretch protocol (1st stretch; CV80: 34.9 ± 2.8 N, IV80: 30.4 ± 2.1 N; 20th stretch; CV80: 23.7 ± 2.1 N, IV80: 19.6 ± 1.0 N) (Fig. 3).

Force deficits that developed during the repeated stretches. During the stretch protocol, isometric force decreased similarly for stretches with constant velocity and increasing velocity with a stimulation frequency of 80 Hz (two-way ANOVA) (Fig. 4A). Deficits in isometric force before the last stretch (20th) were $54.5 \pm 2.5\%$ (CV80) and $49.9 \pm 2.1\%$ (IV80) ($P = 0.2$). Throughout the stretch protocol, smaller deficits in isometric force resulted for stretches with constant velocity and increasing velocity with a stimulation frequency of 20 Hz; deficits in isometric force before the last stretch (20th) were $15.8 \pm 1.9\%$ for CV20 and $16.4 \pm 2.8\%$ for IV20, respectively (Fig. 4A). For all groups, the deficits in relative peak stretch force that developed during the stretch protocol were similar (Fig. 4B). Deficits in peak stretch force before the last stretch were $32.3 \pm 2.2\%$ (CV80), $35.0 \pm 1.8\%$ (IV80), $28.0 \pm 2.0\%$ (CV20), and $26.8 \pm 3.6\%$ (IV20) ($P = 0.1$).

Force deficits 1 h after the repeated stretches. Typical examples of the force-frequency relationship with isometric contractions of rat plantar flexor muscles before and 1 h after the stretches with activation are shown in Fig. 5A. One hour after the stretches with activation, the force-frequency relationship was shifted downward, resulting in isometric force deficits at all stimulation frequencies. For the groups tested with constant velocity and increasing velocity stretches with a stimulation frequency of 80 Hz, isometric force deficits 1 h after the stretches were not different at any of the stimulation frequencies. For the groups tested with constant velocity and increasing velocity stretches with a stimulation frequency of 20 Hz, the isometric force deficits were not different at any of the stimulation frequencies but were substantially smaller at frequencies of

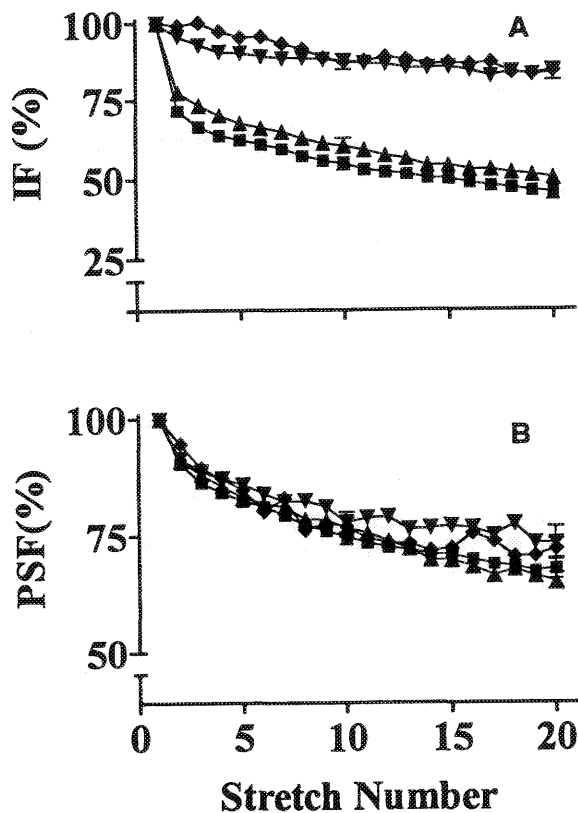


FIGURE 4—The relationship between stretch number and the isometric force (IF) at an ankle position of 90° (A) and peak stretch force (B). Force was normalized for the isometric force or peak stretch force during the first contraction in the series of stretches (\blacktriangle , IV80: rats exposed to 20 stretches with increasing velocity (i.e., acceleration of 3000°s^{-2}) and a stimulation frequency of 80 Hz; \blacksquare , CV80: rats exposed to 20 stretches with a constant velocity (i.e., 300°s^{-1}) and a stimulation frequency of 80 Hz; \blacklozenge , IV20: rats exposed to 20 stretches with increasing velocity and a stimulation frequency of 20 Hz; \blacktriangledown , CV20: rats exposed to 20 stretches with a constant velocity and a stimulation frequency of 20 Hz). For clarity of the figure only, error bars of contractions 10 and 20 are plotted. Line connecting data points for illustrative purposes only.

5–80 Hz than for muscles originally stretched while stimulated at 80 Hz (Fig. 5B).

DISCUSSION

In intact muscle-tendon joint systems, the kinematic parameters that have been studied for effects on stretch-induced force deficits include peak stretch force (5), angular velocity (22) and range of motion (6). Peak stretch force has been shown to be an important determinant of stretch-induced force deficits (5,14,19). Stretches with activation result in higher forces than forces during isometric contractions, but it remains uncertain which skeletal muscle structures are exposed to these high “damaging” forces because of the complexity of the pathway of force transmission from contractile proteins to tendon (9). In previous work, velocity of ankle rotations (velocities tested were 50°s^{-1} and 600°s^{-1} that had similar peak stretch forces) was not related to stretch-induced force deficits of activated plantar flexor muscles (22). Interestingly, wrist movements with

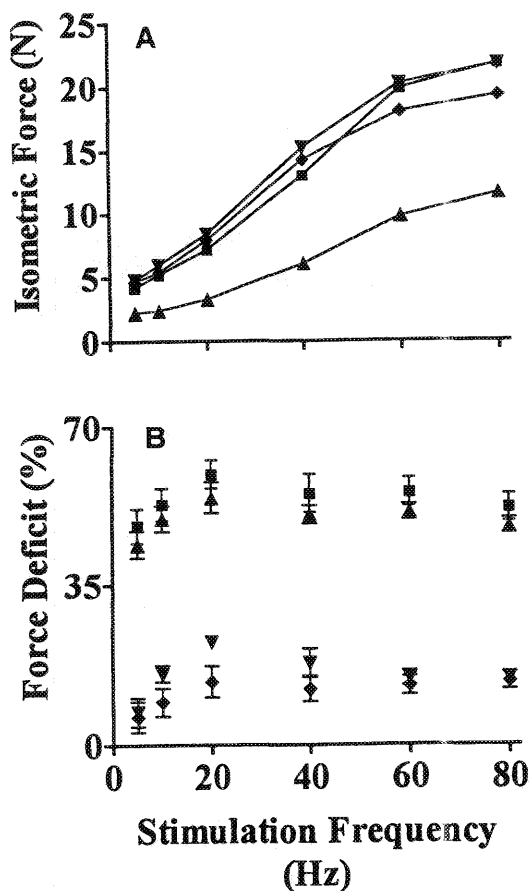


FIGURE 5—Typical examples of the relationship between stimulation frequency and isometric force (A) before and 1 h after the series of stretches and the isometric force deficits (B) 1 h after the series of stretches (symbols in A; \blacksquare (before stretches) and \blacktriangle (1 h after the stretches) of a rat exposed to 20 stretches with increasing velocity (i.e., acceleration of 3000°s^{-2}) and a stimulation frequency of 80 Hz, \blacktriangledown (before stretches) and \blacklozenge (one h after the stretches) of a rat exposed to 20 stretches with increasing velocity and a stimulation frequency of 20 Hz) (symbols in B; \blacktriangle , IV80: rats exposed to 20 stretches with increasing velocity (i.e., acceleration of 3000°s^{-2}) and a stimulation frequency of 80 Hz; \blacksquare , CV80: rats exposed to 20 stretches with a constant velocity (i.e., 300°s^{-1}) and a stimulation frequency of 80 Hz; \blacklozenge , IV20: rats exposed to 20 stretches with increasing velocity and a stimulation frequency of 20 Hz; \blacktriangledown , CV20: rats exposed to 20 stretches with a constant velocity and a stimulation frequency of 20 Hz).

high-acceleration components (i.e., increases in velocity) have increased risk for cumulative trauma disorder (18). During such movements, skeletal muscles must produce high breaking forces during stretches to decelerate moving body parts. Therefore, stretches with increasing velocity could be more damaging than stretches with constant velocity stretches if stretches with increasing velocity were accompanied by larger peak stretch forces. In our study, increasing velocity during ankle rotations, which resulted in a maximal angular velocity of 450°s^{-1} , produced similar deficits for isometric and peak stretch force compared with ankle rotations with controlled constant velocity at 300°s^{-1} . The similarity in stretch-induced force deficits in each group with high and low levels of nerve activation was probably due to identical peak stretch forces in these two groups. Thus, for plantar flexor muscle-tendon complex in

intact rats, stretch-induced force deficits were not dependent on parameters of ankle rotation but on peak stretch forces that occurred in the same range of motion with the same duration.

In the present study, contractions with high stimulation frequency (e.g., 80 Hz) could have induced muscle fatigue. However, several reasons indicate that the force deficits produced by stretching of activated muscles were not caused by fatigue but by stretch. First, isometric contractions with 3-min rest intervals resulted in an isometric force loss of 0.5% per contraction (22). Second, isometric force deficits induced by 40 preloaded concentric contractions producing fatigue (31% of initial force value) were found to recover within 30 min to 88% of the initial force value (23). Third, in the present study, there was no major recovery of the isometric force deficits after 1 h of rest after the stretches (e.g., $7.2\% \pm 1.4\%$ for stretches with 80-Hz stimulation). It is concluded that for ankle rotations with stretches of activated rat plantar flexor muscles, acceleration of $3000^{\circ}\cdot\text{s}^{-2}$ is not an additional risk factor in producing stretch-induced force deficits.

For activated rat plantar flexor muscles, muscle force during stretches is a causal factor for isometric force deficits. Substantially smaller isometric force deficits resulted from stretches of activated plantar flexor muscle with low levels of nerve activation compared with high levels of nerve activation. This observation confirms work on EDL *in situ* of mice (14), soleus muscle *in situ* of rats (19), and knee extensor muscle *in vivo* in humans (5). Low forces during stretches of activated skeletal muscles probably produce less damage to sarcomeres [e.g., desmin (12)] and sarcolemma (15). However, smaller isometric force deficits with low levels of nerve activation during stretches might also be explained by another factor other than lower peak stretch forces (14,19). For rat gastrocnemius muscle, the force-length relationship of submaximally activated muscle (e.g., 15 Hz) shifts to longer muscle lengths compared with maximal activation (17). Such a shift is probably accounted for by length-dependent calcium sensitivity of the myofilaments (3). Because of this shift during low levels of nerve activation, the plantar flexor muscles would be stretched with lower force and at shorter muscle lengths than stretches with high levels of nerve activation. Stretches initiated at a relatively short muscle length resulted in smaller stretch-induced force deficits (10). Low frequencies of activation during stretches of activated skeletal muscles produced smaller force deficits due to reduced stretch force and prob-

able shifts in muscle length. In the present study, probable shifts in muscle length at low frequencies of activation were not controlled.

To our surprise, deficits in relative peak stretch force were not dependent on the muscle force (i.e., 20- and 80-Hz stimulation) during stretches. Traditionally, the functional susceptibility for stretch-induced muscle injury has been evaluated based on isometric force deficits. Stretch-induced isometric force deficits result from 1) excitation-contraction uncoupling, probably caused by a functional change of the voltage sensor of the t-tubules (20); and 2) alteration of force producing and/or transmitting structures (16). However, the difference between the relative losses in isometric and peak stretch force, which was most obvious for stretches with low force (i.e., 20-Hz stimulation), must be due to different mechanisms that are responsible for the losses in isometric and peak stretch force or that additional mechanisms contribute to the loss in peak stretch force. In normal muscles, cross-bridge formation between myosin heads and actin molecules results in the production of muscle force. Additional force is produced during stretches than during isometric contractions. This additional force is thought to be caused by the ability of attached cross-bridges to produce larger forces when stretched than attached cross-bridges during an isometric contraction. Interestingly, stiffness measurements on injured soleus muscle of rabbits have revealed that cross-bridges attach but do not enter the force-generating state (4). For injured skeletal muscles by stretches with high and low levels of nerve activation, it is possible that 1) some cross-bridges lose their ability to attach during the stretch and 2) some attached cross-bridges reduced their ability to produce larger forces during stretch.

In summary, stretches of activated skeletal muscles during joint rotations with increasing velocity did not appear to be an additional risk factor for the development of isometric and peak stretch force deficits, whereas muscle force was critical for the development of the deficits in isometric force but not the deficits in peak stretch force.

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