Reduced strength after passive stretch of the human plantarflexors

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Reduced strength after passive stretch of the human plantarflexors. J Appl Physiol 89: 1179–1188, 2000.—The purpose of this study was to assess strength performance after an acute bout of maximally tolerable passive stretch (PSmax) in human subjects. Ten young adults (6 men and 4 women) underwent 30 min of cyclical PSmax (13 stretches of 135 s each over 33 min) and a similar control period (Con) of no stretch of the ankle plantarflexors. Measures of isometric strength (maximal voluntary contraction), with twitch interpolation and electromyography, and twitch characteristics were assessed before (Pre), immediately after (Post), and at 5, 15, 30, 45, and 60 min after PSmax or Con. Compared with Pre, maximal voluntary contraction was decreased at Post (28%) and at 5 (21%), 15 (13%), 30 (12%), 45 (10%), and 60 (9%) min after PSmax (P < 0.05). Motor unit activation and electromyogram were significantly depressed after PSmax but had recovered by 15 min. An additional testing trial confirmed that the torque-joint angle relation may have been temporarily altered, but at Post only. These data indicate that prolonged stretching of a single muscle decreases voluntary strength for up to 1 h after the stretch as a result of impaired activation and contractile force in the early phase of deficit and by impaired contractile force throughout the entire period of deficit.

isometric; motor unit activation; stiffness; viscoelastic

PASSEIVE STRETCH HAS BEEN USED in a number of research contexts to study mechanical factors of force production (13), stress-relaxation characteristics of muscle (26, 30, 37, 40), neuromuscular reflex patterns (17, 21), factors contributing to muscle damage (2, 25), mechanisms of hypertrophy (38), and stretching parameters relating to the development of flexibility (26, 39). Despite this breadth of information, there is very little research on factors that may influence measured force in twitch or maximum contractions as affected by passive stretch.

Many factors may influence muscle force generation directly after passive stretch. Stretches maintained at the same joint angle for >45 s result in reduced passive tension (i.e., muscle stiffness) (28, 30, 40), and repeated intense stretching increases muscle length (27, 37). Reduced muscle stiffness can affect evoked muscle twitch amplitude and shape because of greater time needed to “take up slack” in compliant in-series elements (7), and increased muscle length may alter the fine balance of muscle properties and joint kinematics that combine to produce force at a given joint angle (24). Altered force-length characteristics may influence neural activation patterns because of altered proprioceptive feedback and coordination, although changes in activation would be of greater importance in dynamic or submaximal movements than isometric maximal contractions (6, 18).

Armstrong et al. (2) observed a 61% decrease in twitch force after a 2-h stretch of rat soleus. Compromised maximum tetanic force after passive stretch has been demonstrated in animal muscle experiments (25) and human experiments in which maximal strength performance was tested (23). Greater “slack” after stretching can explain the reduction in twitch force but is unlikely to explain the reduced peak contraction force, because all slack should be taken up during maximum contractions. Therefore, factors other than in-series compliance may affect force generation after passive stretch.

The following study evaluated the contractile response of human plantarflexor muscles to prolonged passive stretch to identify possible sites of altered contractile capability after stretch. The duration of stretch performed in this experiment is more similar to prolonged stretch procedures employed in animal experimental models and, therefore, may have limited application to sport stretching performed in conjunction with athletic performance. However, the finding of reduced contractile ability after stretch provides valuable insight into important influences on the generation of maximum voluntary force in human skeletal muscle.

METHODS

Subjects

Eight men [22.3 ± 0.8 (SE) yr] and four women (20.3 ± 0.1 yr), who were recreationally active and had no history of injury or abnormality affecting the ankle joint, completed a total of three trials in two experiments. Informed, written consent was obtained from each subject before participation.
in the experiment. The study was approved by McMaster University's Human Ethics Committee. Two men completed all trials of the experiment but were removed from analysis because electromyogram (EMG) above the criterion threshold was detected during the passive stretch protocols. Therefore, data were collected on 12 subjects and analyzed for 10 subjects (6 men and 4 women).

**Experimental Design**

To determine possible factors that could contribute to contractile deficits after maximally tolerable passive stretch (PS$_{\text{max}}$), two experiments were completed. In experiment 1, the time course for alterations in contractile response after PS$_{\text{max}}$ was examined and compared with a no-stretch control condition. Measures included evoked twitch, isometric maximum voluntary contractions (MVC), and passive stiffness at a number of time points within 1 h after the intervention. At least 3 wk after experiment 1, experiment 2 was performed to assess contractile performance after the same stretch protocol at three joint angles, as opposed to the single testing angle assessed in experiment 1. Testing at three angles in experiment 2 was designed to control for contractile performance alterations due to any changes in the muscle force-length relation after stretch.

**Apparatus**

A leg-holder device described by Sale et al. (35) was used for all procedures. Subjects were positioned so that the knee and hip angles were at 90°. By having the knee joint set at 90°, the gastrocnemius, which crosses the knee joint and therefore was at a more shortened length, bears less of the load imposed in the stretch and contributes less force during active plantarflexion than the soleus (35). Ankle movement is limited to 48° of dorsiflexion (D) or plantarflexion (P) from the midposition of a 90° ankle angle to the tibia (or 0°D). Subjects did not wear shoes and were firmly secured with Velcro straps and anterior tibial and femur compression supports during the protocol and for each test. The axis shaft of the apparatus was aligned with the axis of rotation of the ankle through the medial malleolus. Strain gauges at the axis shaft translated pressure to the metal foot plates into a torque signal. The torque signal was amplified (Honeywell Accudata 143 bridge amplifier) at 2 kHz, converted to a digital signal, and fed into a 12-bit analog-to-digital converter (Datqa Electronics, Akron, OH) and then into an IBM computer for on-line analysis. CODAS data acquisition software (Datqa Electronics) was used to process the data.

**Testing Protocol**

After a brief familiarization and practice period and after 2 days of rest from strenuous activity with the lower legs, subjects underwent a PS$_{\text{max}}$ or a neutral ankle angle control (Con) in random order. The general experimental procedure for experiment 1 was as follows: preexercise measures (Pre), 10-min rest period, the PS$_{\text{max}}$ or control (Con) protocol, and contractile measures immediately after (Post) and at 5, 10, 15, 30, 45, and 60 min after testing. All force testing in experiment 1 was completed at 10°D, the optimal joint angle for plantarflexion force production (35). Force testing in experiment 2 was completed at joint angles above and below this optimal joint angle (total of 3 joint angles). Resting twitches were omitted at the 5-min time point because of the confounding effects of postactivation potentiation (41). At >3 days after the first trial, subjects returned to the laboratory to perform the remaining protocol (Con or PS$_{\text{max}}$) with the same leg that was tested in the previous trial.

The testing procedure for Con was performed identically to that for PS$_{\text{max}}$, except no stretch of the ankle plantarflexors occurred but, rather, the ankle was kept in a resting joint angle position selected by the subject (~10°P). During the Con trial the subject was secured into the apparatus with the same tension on the supports and Velcro straps and for the same total duration as during PS$_{\text{max}}$.

**Experiment 2** involved testing at three joint angles (0°D, 10°D, and 20°D). Twitches at the three joint angles were assessed at Post and 15, 30, 45, and 60 min after PS$_{\text{max}}$. MVCs at three joint angles were assessed only at 30 and 60 min to keep the total number of MVCs approximately the same between trials. Rest periods of 20 s were allowed between successive twitches, and 100-s rest periods were allowed between successive MVCs. Joint angle testing order was randomized between subjects.

PS$_{\text{max}}$

Without prior warm-up or stretching, the subject’s leg was secured in the device and pretested. After a 10-min rest interval, the plantarflexors were passively stretched by the experimenter to the maximum possible dorsiflexed position achievable without pain. The joint angle was then locked into place, and at intervals of 2 min 15 s the ankle joint was released to a neutral joint angle for 5 s and then manually passively stretched over 5–10 s at ~2/3 to a new maximal joint angle as limited by the tolerance of the subject. Torque was zeroed between each stretch to eliminate the effects of drift from the torque transducer. A total of 13 maximal stretches were imposed in 33 min (i.e., 30 min of time under stretch). Subjects were given visual feedback of torque and EMG activity during the stretch protocol. Maximum joint angle achieved with each stretch during the protocol was visually read by the experimenter from the apparatus (angle ±0.25°). Posttesting began directly after cessation of PS$_{\text{max}}$ and for up to 1 h.

**Force Measurements**

Isometric MVC was held for 3–5 s; an interpolated stimulus (a maximal twitch superimposed on the maximal contraction) was delivered after ~2 s, when a plateau in the torque trace was clearly visible to the tester. The inflection in torque above the plateau was identified as the interpolated twitch. Because of the number of MVCs performed in the experimental protocols, only single MVCs were performed for each time point, except in the Pre measurements for experiment 1, where the best of two MVCs was taken. The methods of Belanger and McComas (4) were used to calculate motor unit activation from the interpolated twitch torque values: [(resting twitch – interpolated twitch)/resting twitch] × 100%. An additional calculation was used to determine the distribution of force loss between activation and force-generating components from the methods of Duchateau (10). This extrapolates maximum voluntary force to a theoretical maximum value at 100% activation. The force loss due to activation is calculated from the motor unit activation calculation above; the remainder of the force loss is partitioned to the force-generating component by subtraction.

Twitch contractions were evoked by percutaneous electrical stimulation. The stimulating electrodes were lead plates wrapped in gauze and impregnated with conducting gel. The cathode (2 × 3 cm) was positioned in the popliteal fossa overlying the posterior tibial nerve. The anode (1.5 × 1.5 cm) was positioned at the motor point for the soleus, along the medial line directly below the belly of the medial and lateral gastrocnemius muscles. Skin over the stimulation sites was...
abraded and cleaned with isopropyl alcohol pads. Stimuli were delivered from a high-voltage Grass S88 stimulator through a Grass SIU5 stimulus isolation unit with single rectangular voltage pulses of 150 μs. The intensity (voltage) was adjusted to elicit a maximal twitch peak torque for an individual subject trial. Maximal twitch response was achieved when further increases in stimulating voltage did not result in increased twitch torque. A single pulse of parameters identical to that eliciting the single twitch was employed for the interpolated twitch. Stimulating voltage remained constant during a single testing session for all twitch and interpolated twitch measures. When an MVC and a twitch were measured in the same time point, the twitch always preceded the MVC. Maximal twitch responses were analyzed on a computer software program especially designed in our laboratory to evaluate the contractile parameters for peak twitch torque ($P_t$), peak MVC torque, and interpolated twitch torque for the MVCs.

**EMG**

EMG recordings were made with 10-mm-diameter Ag-AgCl (Meditrac 60) surface electrodes. Electrodes were placed over the soleus (~15 cm proximal from the lateral malleolus and 1.5 cm lateral from the medial line and at the lateral insertion into the Achilles tendon; interelectrode distance was ~12 cm). One ground electrode was positioned on the lateral side of the subject. The skin was shaved, abraded with high-grit sandpaper, and cleaned with alcohol. Electrode positioning was not altered during a single testing trial. The EMG signal was passed through an alternating-current amplifier (Honeywell Accudata 135A). The gain was calibrated to optimize signal amplitude for analog-to-digital conversion. Because the sampling frequency was 6 kHz total for the system, sampling was divided by 2 for twitch recordings (3.0 kHz/channel for twitch torque and EMG) and divided by 3 for MVC recordings (2.0 kHz/channel for MVC torque, EMG, and interpolated twitch torque). EMG and torque were only sampled at 50 Hz during PS$_{max}$ to provide a visual signal of passive torque and EMG activity to the subject and experimenter during PS$_{max}$. WinDaq 200 software (Dataq Electronics) was used to acquire and analyze EMG records. Raw EMG signals were full-wave rectified, and the resulting signal was integrated over the duration of the contractions. Integrated EMG in two 0.5-s windows, before and after the interpolated stimulus in each MVC, was divided by time (1 s) to achieve average integrated EMG (AEMG, mV). Four PS$_{max}$ trials were selected at random and sampled at 1.0 kHz/channel to estimate AEMG at time intervals during PS$_{max}$.

**Muscle Stiffness (Passive Tension)**

Passive tension was measured as the passive torque at ankle angles of 0°D, 10°D, and 20°D after torque was zeroed at 10°P. Therefore, the passive torque measure is the increment in torque from 10°P, where it is observed that passive torque is negligible for the plantarflexors (33). Passive torque remained constant during a single testing session for all ankle angles of 0°D, 10°D, and 20°D after torque was zeroed at 10°P. Therefore, the passive torque measure is the increment from 10°P, where it is observed that passive torque is negligible for the plantarflexors (33). Passive torque was measured in successive order from 10°P, 0°D, 10°D, to 20°D. Passive torque was measured before and after contractile measures at each time point and before and after PS$_{max}$.

**Data Analysis**

Statistical analysis was performed on Statistica for Windows R.4.5 software (Statsoft, Tulsa, OK, 1993). Values are means ± SE. Multifactor ANOVA with repeated measures was used to analyze performance measures. Two-factor ANOVAs were completed to compare differences between measures in Con and PS$_{max}$ conditions for experiment 1. One-factor ANOVAs compared changes with the Pre values for experiment 2. Post hoc analysis of mean values was performed using Tukey’s honestly significant difference method. $P$ ≤ 0.05 was accepted for statistical significance.

**RESULTS**

The results are presented separately for experiments 1 and 2. To avoid duplication, only pertinent results from experiment 2 are presented, that is, those unique from experiment 1.

**Experiment 1: PS$_{max}$**

The PS$_{max}$ protocol simulated an intense stretch of the ankle plantarflexors. To the subjects, the sensation was similar to that produced by standing on one leg with the ball of the foot on a chair and allowing the extended heel to drop and passively stretch the calf muscles. The experimental setup allowed measurement of ankle joint angle, passive torque, and soleus EMG activity with each stretch (Fig. 1).

**Angular displacement.** PS$_{max}$ caused an increase in maximum joint angle from 31.3 ± 1.5 to 37.8 ± 1.7°D. A significant increase in joint angle was achieved between the first and second stretch, which was 31% of the total increase in joint angle achieved during the course of 13 repeated stretches ($P < 0.05$). Over one-half of the joint angle increase (57%) attained in 13 stretches was achieved by the fourth stretch (Fig. 2A). One woman reached the end range of the apparatus at 48°D by the seventh stretch.

**Passive torque.** Passive torque traces during PS$_{max}$ indicated stress relaxation. Passive torque decayed rapidly after initiation of a single stretch and then decayed gradually ~30 s after stretch onset (Fig. 1B). Average peak passive torque at initiation of a stretch interval increased from 38.2 ± 2.3 N·m in the first to 41.2 ± 2.0 N·m on the third stretch (relative increase 7.8%, $P < 0.05$) and then did not change for any stretches thereafter, indicating a “set point” for stretch tolerance within the subjects (Fig. 2B).

**PS$_{max}$ EMG.** EMG recorded during the stretch protocol indicated that two subjects were not totally “passive” during PS$_{max}$ and were therefore disqualified from the analysis. The EMG for the subject presented in Fig. 3 and the other subject removed from the analysis indicated activity approaching 10–12% of MVC AEMG. The disqualified subjects reported no “intent” to voluntarily contract, so the activity was likely reflex in origin. All other subjects’ AEMG was at or below the lower detectable limit of the collection equipment (~1–3% of MVC AEMG, similar in amplitude to the noise of the recording system; Fig. 1C).

**Experiment 1: Force Measurements**

**Isometric MVC.** PS$_{max}$ caused a 28% decrease ($P < 0.05$) in MVC (Fig. 4A). MVC had recovered to 80% of the Pre value at 5 min and to 87% at 15 min. MVC was still below (9%) the Pre value at 60 min after the
stretch \((P < 0.05)\). MVC did not change significantly in the Con condition. Motor unit activation calculated from the interpolated twitch was significantly decreased at Post (16\%) and 5 min after \(\text{PS}_{\text{max}}\) (13\%; Fig. 4B). MVC AEMG was similar in Con (0.610 ± 0.096 mV) and \(\text{PS}_{\text{max}}\) (0.581 ± 0.083 mV) at Pre, although AEMG in \(\text{PS}_{\text{max}}\) was reduced by 15.1\% at Post (not significant), recovered by 15 min to Pre values, and was elevated over Pre at 45 min and 60 min \((P < 0.05)\). Because of an increase in Con AEMG observed after \(\text{PS}_{\text{max}}\), the reduced AEMG at Post and 5 min in \(\text{PS}_{\text{max}}\) was significantly different from Con, following the pattern of reduced motor unit activation immediately after \(\text{PS}_{\text{max}}\). Nonsignificant changes in the MVC-to-AEMG ratio over time (results not presented) also indicated that reduced force at Post and 5 min was due primarily to reduced activation.

The decrease in MVC after \(\text{PS}_{\text{max}}\) was partly the result of decreased motor unit activation, but \(\text{PS}_{\text{max}}\) also decreased muscle force-generating capacity. Estimates of the relative contributions to the MVC deficit, of reduced motor unit activation and reduced muscle force-generating capacity after \(\text{PS}_{\text{max}}\), were made using the method described by Duchateau (11). These estimates are shown in Fig. 5. Of the 44 N⋅m force deficit immediately after \(\text{PS}_{\text{max}}\), most of the force loss was caused by reduced motor unit activation (~25 N⋅m) and less by reduced muscle force-generating capacity (~19 N⋅m). Reduced motor unit activation played a minor role in the force deficit at 15, 30, 45, and 60 min after \(\text{PS}_{\text{max}}\).

\[\begin{align*}
\text{Pt.} & \quad \text{Pt} \quad \text{significantly decreased immediately after PS}_{\text{max}} (18\%) \quad \text{and also in the Con condition (9\%; Fig. 6). The decrease after PS}_{\text{max}} \quad \text{was greater (condition} \times \text{time interaction, } P < 0.05). \quad \text{In the PS}_{\text{max}} \quad \text{condition, Pt} \quad \text{recovered to 92\% of the Pre value at 15 min and then decreased to 87\% of Pre values for the remaining time points. Con Pt} \quad \text{recovered to 96\% of its Pre value at 15 min but remained depressed by ~9\% at 30, 45, and 60 min. Although attempts were made to keep the legs}
\end{align*}\]
covered and warm during the protocol, muscle cooling may have occurred. Reduced muscle temperature (9) may explain the depressed twitches in testing after the Con or PS$_{\text{max}}$ conditions. The 15-min twitch may have exhibited some lingering effects of potentiation from the MVC completed at 5 min.

**Muscle stiffness (passive torque).** Mean passive torque was calculated as the average torque at a time point for three joint angle measurements before and after an MVC. Therefore, each data point contains 6 measurements for each subject, for a total of 60 data points. Mean passive torque was reduced by 27% directly after the PS$_{\text{max}}$ protocol ($P < 0.05$; Fig. 7). Muscle stiffness recovered quickly to 14% below Pre at 15 min ($P < 0.05$) but did not completely recover within 1 h, inasmuch as mean passive torque was still depressed below Pre by 8% at 45 min (not significant, $P = 0.078$) and 60 min (not significant, $P = 0.058$). Mean passive torque was significantly correlated to average P$_t$ for Con and PS$_{\text{max}}$ twitch measures over the six testing time points ($n = 12$ sample points, $r = 0.62, P < 0.05$), indicating a relation between muscle stiffness and force generated in a twitch.

**Experiment 2: Relative Joint Angle Testing**

There were no significant differences in the maximum joint angles achieved, total increase in maximum joint angle over 13 stretches, or peak passive torque at initiation or at the end of a stretch interval between the PS$_{\text{max}}$ protocols in experiments 1 and 2 ($P > 0.4$).

**Isometric MVC.** MVC was tested at three joint angles in experiment 2 and only at three time points. MVC testing at Post and 15 min was eliminated to avoid confounding effects of reduced motor unit activation and to limit the total number of MVCs performed by each subject. The 45-min time point was also excluded to limit the total number of MVCs. There was no difference in results between experiments 1 and 2 when the MVCs tested at 10°D were compared at 30 and 60 min, indicating a consistent decrease in force in the two experiments (i.e., main effect for time conserved, $r > 0.93, P < 0.05$).

Peak MVC torque occurred at 10°D at Pre (Fig. 8), although there was no significant difference between MVC torque at 10°D and 20°D. The shape of the torque curve was not different at the 30-min time point after "lengthening" of PS$_{\text{max}}$ inasmuch as there was no interaction of joint angle tested with time ($P = 0.2$). The shape of the torque curve was not different when calculated relative to an individual's 100% MVC. This would indicate that the intensive stretching performed in this protocol was not sufficient to maintain a "lengthened" state for 30 min after PS$_{\text{max}}$ or was not sufficient to cause a significant shift in the contractile element force-length relation at a time when MVC was still depressed. The mean MVC torque of three joint angles was below Pre by 7% and 6% at 30 and 60 min, respectively ($P < 0.05$).

Interpolated twitch and calculated motor unit activation indicated a main effect for testing joint angle, although no effect for time ($P = 0.73$) or interaction ($P = 0.13$) was evident. Motor unit activation was lower at the 20°D testing angle than at 0°D or 10°D by 6% and 4%, respectively ($P < 0.05$). MVC AEMG exhibited main effects for joint angle and time ($P < 0.05$), although there were no interactions of joint angle $\times$ time, meaning that PS$_{\text{max}}$ did not cause length-specific changes in activation. In contrast to the lower motor unit activation, mean AEMG of tests at 20°D (0.721 ± 0.66) were greater than those at 10°D (0.656 ± 0.62, $P < 0.05$) and 0°D (0.613 ± 0.073, $P < 0.005$). Consistent with the findings in experiment 1, AEMG was significantly elevated over Pre values (0.600 ± 0.077) at 30 min (0.671 ± 0.070) and at 60 min (0.719 ± 0.078, $P < 0.05$), although the values for 30 and 60 min were not different. Rather than an increase in motor unit activation, the increase in AEMG after PS$_{\text{max}}$ was mostly the result of increased amplitude of muscle fiber action potentials, as indicated by the increase of the whole muscle compound action potential (M wave, data not presented). AEMG expressed relative to MVC...
torque (AEMG-to-MVC ratio) showed no significant interactions (data not presented).

After PSmax, the normal Pt main effect for joint angle of $10^\circ D > 20^\circ D > 0^\circ D (P < 0.05)$ was temporarily altered (Fig. 9). At Post the Pt was 1.6% greater at $20^\circ D$ than at $10^\circ D$ (not significant), whereas at Pre the Pt was 6.9% greater at $10^\circ D$ than at $20^\circ D (P < 0.05$). This “shift” in the force curve to a more optimal testing angle of $20^\circ D$ at Post had disappeared by 15 min.

**DISCUSSION**

PSmax of the human plantarflexor muscles for 30 min resulted in a 25% loss in maximum voluntary force. The loss in force was partly due to reduced activation and partly due to compromised muscle force-generating capacity. By 15 min of recovery, when full activation of the plantarflexors had been restored, muscle force-generating capacity in an MVC was still reduced so that it remained 12% to 8% below Pre values up to 1 h after the stretch. Any significant reduction in maximal force-generating capacity after PSmax is a relevant finding. The results of the present study corroborate a recent report that stretching compromises maximum voluntary force in the muscles participating in the preactivity stretching routine (23).

Depression of plantarflexor MVC directly after PSmax was associated with a significant increase in interpolated twitch torque, an indication of reduced muscle activation (4). Although the relation of interpolated twitch to activation may be nonlinear and exponential with declining extra torque (10), the indication is that activation decreased after PSmax. Using a formula described by Duchateau (11) to account for force decrements as neural or muscular in origin, we estimated that 60% of the 25% reduction in MVC directly after PSmax was neurally mediated and 40% of the reduction originated in the muscle. By 30 min, reduced activation accounted for only ~1% of the 10% decrement in MVC. It is important to note that the “Duchateau” formula relates activation to an assumed 100% activation, which is an extrapolated maximum muscle force larger than that achieved by voluntary effort. This assumption has been challenged (10). Nevertheless, application of the Duchateau formula indicates that the immediate PSmax-induced force decrement is neural and contractile in origin.

Several neuromuscular feedback responses could contribute to activation failure after PSmax; namely, the Golgi tendon reflex, mechanoreceptor (type III afferent) and nociceptor pain feedback (type IV afferent), and/or fatigue responses. The Golgi tendon reflex is an autogenic inhibition that occurs when the Golgi tendon organs, located at myotendon junctions, detect high force combined with muscle lengthening. The Golgi tendon organs’ feedback inhibits agonist activation to lower force production and reduce potentially injurious strain on the muscle. However, an extremely intense stretch is necessary to activate Golgi tendon organs (20); moreover, Golgi tendon organ discharge rarely persists during maintained muscle stretch, and the
inhibitory effects are momentary (1). The facts that peak passive torques in PS max averaged only ~28% of MVC (range 21–45% of MVC) and the drop in activation occurred at time points after cessation of the stretch discount Golgi tendon organ feedback as the likely mechanism for reduced voluntary activation in this experiment.

Mechanoreceptor (type III afferent) and nociceptor pain feedback (type IV afferent) may reduce central drive (5, 31). Any perceptions of discomfort or pain were not present during the poststretch MVCs, so the sensation of stretch and discomfort as a cause of temporary activation failure would have to be short lived. Some subjects commented that their muscle “just didn’t want to contract” in the Post and 5-min MVCs, despite maximal voluntary effort. Reduced reflex assistance (H reflex) or “spindle support” as a result of stretch could also serve to reduce muscle contraction; however, this effect is also more prevalent during stretch and recovers immediately (17).

Enoka and Stuart (14) describe situations of fatigue that can cause central activation failure in addition to local metabolic effects to reduce force. The initial activation failure and the force decrements that persisted in this experiment might then be thought to result from fatigue. The normal response to muscle elonga-

Fig. 4. A: effect of PS max on maximal voluntary strength (MVC). *Significantly different from Pre, P < 0.05. #Significantly different from PS max and control, P < 0.05. Values are means + SE. B: motor unit activation (calculated from interpolated twitch values) after 30 min of PS max of the ankle dorsiflexors or neutral angle control. *Significantly different from Pre, P < 0.05. #Significantly different from control, P < 0.05. Values are means + SE. C: isometric MVC AEMG in PS max and control. The drop in AEMG immediately (Post) and 5 min after PS max is due to the drop in motor unit activation in B. #Significantly different from Pre, P < 0.05. *Significantly different between PS max and control, P < 0.05. Values are means + SE.

Fig. 5. Estimated contributions of reduced motor unit activation and reduced muscle force-generating capacity to the MVC deficit after passive stretch. MVC deficits were significantly different from Pre values at all time points after PS max (P < 0.05).

Fig. 6. Peak twitch torque (P) after PS max or neutral angle control. All time points after PS max were significantly decreased from Pre (P < 0.05). Control values were also significantly decreased from Pre (P < 0.05), except for the 15-min time point (P = 0.45). *Significant decrease below Pre. #Significant decrease of PS max below control, P < 0.05. Values are means + SE.
tion is the stretch reflex, which is a feedback loop from muscle spindles that cause agonist contraction resisting the stretch. Silent EMG activity during passive stretching indicates a lack of stretch reflex response. We confirmed that the slow stretching procedure performed in this experiment was truly passive with no EMG activity, as has been achieved by other researchers using stretch protocols in human subjects (8, 27, 29, 30, 32). Our two subjects that exhibited EMG activity during PS\textsuperscript{max} were not included in the analysis. Therefore, fatigue could not play a major role in the responses to PS\textsuperscript{max}.

Irrespective of the mechanism responsible for temporary loss in activation, the fact that a reduced MVC persisted after recovery of activation indicates that reduced muscle force-generating capacity was caused by additional factors after PS\textsuperscript{max}. Factors that may affect force-generating abilities after stretch may be due to changes in the length-tension relationship and/or plastic deformation of connective tissue, which could affect force generation during a maximal contraction.

PS\textsuperscript{max} produced a marked decrease in muscle passive torque (force) measured at the same absolute joint angles after PS\textsuperscript{max}, confirming previous observations (39, 40). The reduction in passive torque is a result of the muscle lengthening during the stretch, so that, when returned to the same absolute joint angle after the stretch, the muscle is effectively at a shorter muscle length at the same absolute test angle than before the stretch. This would place the muscle in a different point in the passive torque curve and would exhibit as lowered passive torque after stretch. Slow passive stretch of the human plantarflexors has been observed to directly lengthen the muscle belly and not the tendon (19). This effect was confirmed for our experiment by B-mode ultrasound, which measures muscle fascicle lengths (22). With use of a similar PS\textsuperscript{max} protocol in a single subject, PS\textsuperscript{max} was observed to facilitate muscle fascicle elongation of 8, 8, and 2 mm for the soleus, lateral gastrocnemius, and medial gastrocnemius muscles, respectively.

A possible explanation of the reduced force in the present study was that the lengthened muscle fascicles would be in a less optimal portion of the length-tension curve when returned to the same absolute testing angle (as in experiment 1) after PS\textsuperscript{max} but may not be compromised at a joint angle relative to the increase in muscle length (as in experiment 2). We observed that PS\textsuperscript{max} altered the twitch torque-joint angle relation, such that the greatest torque occurred at greater ankle dorsiflexion directly after PS\textsuperscript{max}. The alteration was short lived; the Pre torque-joint angle relation was restored by 15 min, even though passive torque was still significantly decreased. The MVC torque-joint an-
gle relation measured at 30 min was not significantly influenced ($P = 0.20$) by the temporarily reduced passive torque ($P = 0.07$); however, even subtle differences in fiber pennation angles (caused by the changes in muscle stiffness in this experiment) have been observed to affect muscle specific tension (22).

Elongation of the muscle-tendon unit in this experiment was facilitated by stress relaxation and tissue “creep.” Stress relaxation is viscoelastic and purely mechanical in nature and is indicated by a decay in passive torque over time for a given stretched muscle length (37). Viscous or hydraulic “pistonlike” elements and elastic “springlike” elements within the muscle are taken up early in a single stretch (40) or in a stretch routine (37). Creep results from maintained tissue strain, which causes a reorientation of the supporting connective and soft tissue-supporting structures of the muscle (i.e., plastic deformation) to more-ordered (i.e., parallel) arrays (34), which allows muscle lengthening over time. Fifty-seven percent of the joint angle increase (i.e., muscle elongation) occurred in the first 4 stretches of this 13-stretch protocol, or 71% of lengthening occurred in the first 4 of 10 stretches. This implies that stress relaxation occurred early in $P_{\text{max}}$ and that creep may have allowed angular displacement in the later stages of $P_{\text{max}}$. The rapid and prolonged phases of stiffness recovery observed in this experiment may be related to the muscle viscoelastic recoil and recovery from plastic deformation, respectively. This would explain why $P_{\text{t}}$ significantly correlated with mean passive torque and a significant shift in the twitch-torque curve was observed directly after testing but was not observed in twitch or MVC testing after 15 min.

The presence of plastic deformation may also explain the reduced force that persisted after $P_{\text{max}}$ in this experiment. A relation between muscle stiffness and contractile performance is indicated in a study by Garfin et al. (16), which evaluated the effects of fascia and compartment pressure on force production in contractions of dog hindlimb. Garfin et al. found that using a surgical fascial release to apply a small slit in the epimysium resulted in a 15% reduction in force production loss with passive stretch protocols in animals (2, 25). Creatine kinase enzyme activity is used as a marker of exercise-induced muscle damage and increased by 250% after acute stretching in chickens (3) but only by 62% after 17 min of passive hamstring/low back stretching in humans (36). It is unlikely that contractile element damage contributed to the force decrement in the present experiment, because maximal force production is restored to 100% at 24 h after $P_{\text{max}}$ (15). Five to 14 days are required for recovery of force decrements due to eccentrically induced myofiber injury to normal values (12).

This experiment simulated an intense maximal stretch far beyond what an athlete may attempt before activity or as part of a flexibility training program. The intensity and duration of stretching required to produce lasting stiffness changes in muscle are unknown (27), although the upper conceivable limit of stretching performed here was not sufficient to produce significant muscle stiffness changes lasting 1 h. By its viscoelastic nature, muscle has a strong tendency to return to its resting or genetically and biomechanically determined length. It may be questionable to oppose this tendency with the use of intense stretching to enhance performance, when performance can be compromised by altering the fine dynamic balance of neural, architectural, and electrophysiological factors that exists in muscle to create force. Further testing with a stretching protocol more similar to that regularly performed in the athletic context should be evaluated under the controlled conditions of this study.

In summary, an intense prolonged stretch of the ankle plantarflexors reduces maximum voluntary force for up to 1 h after the stretch. Decreased maximum voluntary force directly after stretch was partly due to reduced activation and partly due to reduced muscle force-generating capacity. Voluntary activation is quickly recovered, as is any shift in the muscle torque-joint angle relation encouraged by the lengthening stretch. Complete recovery of force-generating capacity is more prolonged, similar to the recovery in muscle stiffness. Stiffness recovery may represent the mechanical recoil from the stretching activity. Elements contributing to muscle stiffness may “stabilize” the muscle to generate force, and any alteration of those elements may compromise force production.

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