ABSTRACT: Impaired torque production is a major physical impairment following stroke, and has been studied extensively in isometric conditions. However, functional use of a limb requires torque production during movement, and the effects of velocity on maximal torque production may be abnormally enhanced in the paretic limb. The purpose of this study was to quantify the effects of movement velocity on maximal torque production during isokinetic, concentric flexion and extension of the elbow in poststroke subjects. Three speeds were tested (30, 75, 120 deg/s) over a 100-deg range of motion. To control for strength variations between subjects and limbs, isokinetic torques were normalized by peak isometric torque. As flexion velocity increased, paretic limb torque decreased at a greater rate than in the unaffected limb. During extension, paretic limb torque was much lower than torque in the unaffected limb at all speeds. In both flexion and extension, the disparity between limbs in the constant-velocity torque–angle curves became more pronounced as velocity increased. Torque decreased 44% ± 7% in flexion and 63% ± 9% in extension as velocity increased from 30 to 120 deg/s, whereas the corresponding decreases in the unaffected limb were only 9% ± 5% in flexion and 16% ± 4% in extension. No electromyographic (EMG) abnormalities were observed during flexion. During extension, EMG data provided evidence for abnormally increased antagonist coactivation in brachioradialis and markedly reduced activation in triceps as potential contributors to the decreased extension torques. The finding that movement velocity produces large deficits in maximal torque might explain why functional use of the paretic limb is often impaired even though isometric strength appears adequate.

EFFECTS OF VELOCITY ON MAXIMAL TORQUE PRODUCTION IN POSTSTROKE HEMIPARESIS

PETER S. LUM, PhD, CAROLYNN PATTEN, PhD, DHARA KOTHARI, MS, and RUTH YAP, MS

Rehabilitation Research and Development Center, VA Palo Alto Health Care System, 3801 Miranda Avenue, Palo Alto, California 94304-1200, USA

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Among the most prominent impairments contributing to physical disability following stroke is impaired torque production, defined as decreased joint torques in the paretic limb compared to the unaffected homologous limb during maximal-effort tasks. Several studies have emphasized the contribution of impaired torque production to compromised motor function following stroke. Impaired torque production is most commonly quantified by measuring peak torque during maximal isometric contractions. However, other aspects of impaired isometric torque production may contribute significantly to loss of function of the limb. For example, limb function might be affected by slowness in the rate of torque generation, task-dependent weakness, or abnormalities in maximal torque–angle curves. Another study reported that strength imbalances between flexors and extensors results in abnormal torque production when attempting to stabilize the joint by co-contraction. All of these studies have measured isometric torque, and few studies have examined the effects of velocity on torque production. Torque production during dynamic contractions is critical to limb function.

The force–velocity relationship of muscle demonstrates that torque produced during concentric contractions is reduced relative to peak isometric torque. At low velocities, the reduction is small; however, in the range of velocities encountered in functional activities, the reduction can be more

Abbreviations: CNS, central nervous system; EMG, electromyogram; RM-ANOVA, repeated-measures analysis of variance; RMS, root-mean-square

Key words: elbow; hemiparesis; isokinetic; stroke; weakness

Correspondence to: P. S. Lum, Biomedical Engineering, Virginia Commonwealth University, MCV Campus, 1112 East Clay Street, Room 220, PO Box 980694, Richmond, VA 23296-0694; e-mail: plum@vcu.edu

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meaningful. Torque production during movements can be further reduced following stroke due to the negative effects of reduced agonist activation, antagonist muscle co-activation, increased stretch reflex excitability, and changes in the mechanical properties of muscles. Enhanced velocity effects might also result from structural changes in sarcomeres that occur after brain injury. Spastic muscle cells develop passive tension at significantly shorter lengths, and have nearly double the stiffness of normal muscle cells. These results point to dramatic remodeling of structures within muscle cells after brain injury that may influence their mechanical properties, including the force-velocity relationship.

Aberrant muscle activation patterns after stroke can be explained by a physiologically based model developed by Levin and colleagues, who established a link between abnormal co-activation during active movement and the inability of the central nervous system (CNS) to regulate stretch reflex thresholds during passively imposed movement. Stretch reflex thresholds were within the physiological joint range in most stroke subjects, but beyond the physiological range in normal limbs (normal subjects could relax the stretched muscles during passive movement at all speeds). They defined a joint range that is bordered by the stretch reflex thresholds of the flexors and extensors. The CNS can modulate stretch reflex thresholds within this range. In stroke subjects, co-activation and movement deficits were apparent if the movement started from an angle outside of this joint range, or beyond the range of CNS modulation. In contrast, if movement was performed within this joint range, reciprocal patterns of muscle activation were observed.

The purpose of this study was to better understand the effects of movement velocity on impaired torque production in persons with poststroke hemiparesis. The primary hypothesis was that movement velocity would have a greater effect on torque production in the paretic than unaffected limb following stroke. To test this, we measured torque production during maximal effort, constant velocity, concentric flexion and extension of the elbow. We examined the effects of increasing velocity on torque production and compared torque production in the paretic and unaffected limbs to determine whether the deficit varied as a function of velocity. We also examined the torque-angle profile at constant velocity to determine whether these profiles were altered following stroke. A secondary hypothesis was that abnormal muscle activation patterns in the paretic limb would be consistent with the torque abnormal-

**METHODS**

**Subjects.** Fourteen adults with poststroke hemiparesis of greater than 6 months’ duration were recruited from the local community. Criteria for participation included clinical presentation of hemiparesis resulting from stroke, absence of pain or contracture in the major upper-limb joints, no more than minor impairment of upper-limb sensation or proprioception, ability to comprehend and follow three-step commands as evidenced on the Cognistat examination, and demonstration of at least 70 deg of active elbow flexion with gravity eliminated. All subjects provided informed consent and all aspects of this study were approved by our local institutional review board. Once informed consent was obtained, a physical therapist assessed the subject with the upper-limb portion of the Fugl-Meyer test of motor function. Eleven men and three women were tested. Five subjects had a right hemiparesis and nine a left hemiparesis. The paretic limb was the dominant limb before the stroke in six cases. The average age was 70.6 ± 11.5 years, and the duration of hemiparesis ranged from 7 to 19 months, with an average duration of 12.8 ± 4.2 months. Fugl-Meyer test scores ranged from 24 to 61 (out of 66 points), with a mean of 43.1 ± 11.8 points.

**Procedures.** A commercially available dynamometer (Biodex System 3.0 Pro, Shirley, NY) was used to isolate and measure transverse-plane elbow flexion and extension. The standard elbow attachment was modified with a prefabricated wrist splint and straps to accommodate persons with impaired grasp (Fig. 1). Subjects were seated in the Biodex chair with the back angled at 85 deg, the trunk stabilized using waist and trunk straps, and the feet supported using the leg rest. A hand-held goniometer was used to position the shoulder in 5 deg of forward flexion and 70–75 deg of abduction. The arm was stabilized with an adjustable support that balanced the weight of the limb and eliminated excess motion at the shoulder. The dynamometer axis of rotation was aligned...
with the medial epicondyle of the humerus. The range-of-motion limits were defined by alternately extending the elbow to the physiological end-range and then flexing until a range of 100 deg was achieved. Subjects who could not achieve this 100-deg range because of decreased extension range-of-motion were excluded.

Surface EMG was recorded from biceps, brachioradialis, and triceps (long head) using active, preamplified electrodes (MA-311 double-differential surface EMG electrodes, Motion Lab Systems, Baton Rouge, LA). The biceps electrodes were placed over the bulk of the muscle at mid-arm and reflected activity in both heads of the biceps. Electrodes were positioned relative to landmarks as described by Perotto and Delagi.41 The EMG was bandpass filtered between 30 and 500 Hz, and amplified by a factor of 5000. Custom-written software was used to acquire data and present the test stimuli. Position, torque, and EMG data were sampled at 1 kHz and written to disk for offline analysis.

Once the range-of-motion was defined, the sequence of trials was as follows: to measure the passive stiffness at the elbow, the subject was instructed to relax while the dynamometer moved the limb through its full range at 10 deg/s for two continuous cycles of flexion and extension. The elbow was then positioned at 90 deg, the dynamometer placed in isometric mode, and two maximal voluntary effort contractions were performed in both flexion and extension. A computer video display presented the subject’s torque production and provided visual feedback of their performance. During isometric trials, subjects were instructed to produce and hold their maximum voluntary effort for at least 3 s. Following each maximal isometric effort, subjects were allowed to rest for 60 s. Isometric maximal contractions were followed by two discrete trials of isokinetic concentric flexions at each of three target speeds: 30, 75, and 120 deg/s. Speeds were presented in order from slowest to fastest. A parallel sequence of isokinetic elbow extensions was performed. Thirty seconds of rest was provided following each isokinetic contraction. Verbal encouragement was provided to facilitate maximal effort during the isometric and isokinetic trials. Subjects performed the sequence first with the unaffected limb. The entire procedure was repeated in the paretic limb on a separate day.

Data Analysis. To determine the peak torque produced during maximal voluntary isometric contractions, the raw torque data were treated using a 500-ms moving window average. Following this averaging, the peak value achieved in either trial was defined as peak isometric torque. Similarly, the peak root-mean-square (RMS) EMG level (500-ms window) achieved during these isometric trials was defined as peak RMS EMG.

We examined torque production during the isokinetic trials over the middle 45 degrees of the movement. Anatomically, this corresponded to a range of 27.5–72.5 deg of flexion. Because the range of the movements was 100°, there was ample time for subjects to accelerate and reach the criterion speed of the dynamometer before the middle 45-deg interval was reached. This middle 45-deg range was divided into three 15-deg intervals corresponding to early, middle, and late phases of the contraction. The average torque produced during each phase was calculated for each trial. At each target speed, the trial with the larger torque values was retained for statistical analysis. This best trial provided a metric of peak isokinetic torque production as a function of speed and contraction phase. Some subjects were unable to produce the higher targeted velocities throughout the entire trajectory. When velocity decreased below the targeted velocity, the dynamometer provided minimal resistance to movement, and thus minimal torque was produced. In such cases the measured torque was zero, which indicated the subject could produce no torque at the target velocity during the corresponding phase of contraction.

To assess the possibility of increased passive joint stiffness in the paretic limb, the procedure described above was performed for passive movements and yielded average passive torques for the early, middle, and late contraction phases. To control for the effects of strength differences between subjects and limbs, isokinetic torques were normalized by the
peak isometric torque. The EMG data were handled similarly. The RMS EMG level during a particular phase and velocity was normalized by the peak RMS EMG level during the maximal effort isometric trials. The linear relationship between force and EMG is preserved in paretic muscles after stroke.46

Data were analyzed using two-factor, repeated-measures analysis of variance (RM-ANOVA). To test for the effects of velocity, a RM-ANOVA was performed on isokinetic torque and RMS EMG produced in the middle contraction phase, with limb and speed entered as within-subject factors. Increased co-activation in the paretic limb would be indicated by a significant limb effect in the RM-ANOVA, and increased mean EMG activity in the antagonist muscles of the paretic limb compared to the unaffected limb. To test for the effects of phase, RM-ANOVA was performed on isokinetic torque at each speed, with phase and limb entered as within-subject factors. A Bonferroni correction was applied to control for type I errors. To test for differences in passive stiffness between limbs, passive torque was analyzed with a RM-ANOVA with phase and limb entered as within-subject factors.

Correlation analysis was performed to determine the possible relationship between torque deficits and clinical assessments of functional ability of the limb (Fugl-Meyer test score). Torque deficits were quantified by (1) isokinetic torque normalized by peak isometric torque and (2) the decrease in isokinetic torque from the early to late phase of contraction normalized by peak isometric torque. We also performed correlation analysis to determine whether the isokinetic torque deficits were related to isometric torque deficits (peak isometric torque in the paretic limb normalized by peak isometric torque in the unaffected limb).

RESULTS

Representative torque, angle, and EMG data during a trial at 120 deg/s are presented in Figure 2. By considering only the middle 45 deg of the movement, transients and accelerations associated with the start and end of the movement were avoided. In the paretic limb, the flexion torque peaked very quickly and then slowly declined over the remainder of the movement. In the unaffected limb, the flexion torque increased through the early and middle phases and plateaued in the late contraction phase. During extension, torque in both limbs decreased in magnitude as the contraction progressed. Notice that the EMG activity in agonist muscles had peaked before the start of the early phase, indicating that the muscles had reached their maximum contraction levels before the middle 45-deg interval was reached.

In the 45-deg interval of interest, the passive stiffness of the two limbs was not significantly different. In both flexion and extension, levels of passive torque were no different between limbs (P > 0.1), and did not vary as a function of contraction phase (P > 0.05). There were also no phase * limb interactions (P > 0.05).

When normalized by peak isometric torque, paretic limb isokinetic torque was significantly decreased relative to the unaffected limb, especially at the higher velocities. Figure 3 summarizes the torque levels during the middle contraction phase averaged across all subjects. In flexion, torque in both limbs decreased with increasing velocity [F(2,26) = 16.5, P < 0.001], but the decline in torque as velocity increased was greater in the paretic than unaffected limb [limb * velocity interaction, F(2,26) = 6.2, P = 0.006]. In extension, torque was much lower in the paretic limb [F(1,13) = 43.3, P < 0.001]. Extension torque in both limbs decreased with increasing velocity [F(2,26) = 25.6, P < 0.001], and the rate of decline did not differ between limbs (P = 0.6).

As the contraction progressed from the early to late phases, the torque deficit in the paretic limb increased (Fig. 4). In flexion, the shape of the torque–angle curve at constant velocity differed between limbs (limb * phase interactions) [30 deg/s, F(2,26) = 8.8, P = 0.027; 75 deg/s, F(2,26) = 11.5, P = 0.009; 120 deg/s, F(2,26) = 20.8, P < 0.001]. This interaction was further investigated with RM-ANOVA of data from each limb separately. This additional analysis revealed that as the flexion contraction progressed through the phases, paretic limb torque decreased [F(2,26) = 5.4, P = 0.036], whereas unaffected limb torque increased [F(2,26) = 29.5, P < 0.001]. Examination of effect sizes indicated that this disparity between limbs increased as velocity increased. The percentage of the variance attributed to the limb * phase interaction increased from 40% to 62% as velocity increased from 30 to 120 deg/s. In extension, torque in both limbs decreased as the contraction progressed through the phases [30 deg/s, F(2,26) = 96.1, P < 0.001; 75 deg/s, F(2,26) = 78.1, P < 0.001; 120 deg/s, F(2,26) = 81.8, P < 0.001]. Although the rate of decline did not differ between limbs at 30 and 75 deg/s (P > 0.05), the rate of decline at 120 deg/s was faster in the paretic limb [limb * phase interaction, F(2,26) = 8.8, P = 0.018]. The percentage of the variance attributed to the limb * phase interaction
FIGURE 2. Representative torque, angle, and EMG data during elbow flexion (left column) and extension (right column) at 120 deg/s in a single subject. The dotted vertical lines mark the early, middle, and late phases of the contraction.
increased from 7% to 40% as velocity increased from 30 to 120 deg/s. This indicates that as velocity increased, the disparity in extension torque–angle curves increased.

Examination of the EMG data provided evidence of both impaired agonist activation and antagonist co-activation (Fig. 5). RMS EMG levels observed during isokinetic contractions were normalized against peak levels during maximal isometric contractions. Compared to the unaffected limb, decreased levels of normalized RMS EMG in agonists would be evidence for activation impairment, whereas increased levels in antagonists would be an indication of increased co-activation. During flexion, there were no significant differences between limbs in any of the muscle EMG levels. During extension, increased levels of antagonist co-activation were observed in brachioradialis; RMS EMG was higher in the paretic than unaffected limb \( [F(1,13) = 9.0, P = 0.010] \). There was also evidence of impaired activation of triceps during extension; RMS EMG was reduced in the paretic limb compared to the unaffected limb \( [F(1,13) = 6.2, P = 0.027] \). The effects of contraction phase on RMS EMG levels did not differ between limbs in either flexion or extension \((P > 0.05)\).

Subjects who had more severe functional impairments also had larger torque deficits in the paretic limb. Torque deficits were largest in the late phase of contraction at 120 deg/s. Therefore, we calculated the Pearson correlation coefficient between torque during this phase and the functional level of the limb. In the paretic limb, significant correlations were found between Fugl-Meyer test score and isoki-

![Figure 3](image1.png)

**Figure 3.** Torque as a function of velocity. Isokinetic torques during the middle phase of contraction were normalized to peak isometric torque and averaged across all subjects. Error bars are standard error of the mean.

![Figure 4](image2.png)

**Figure 4.** Torque as a function of contraction phase. Isokinetic torques at 120 deg/s were normalized to peak isometric torque and averaged across all subjects. Error bars are standard error of the mean.
netic torque in both flexion and extension (flexion: \( r = 0.59, P = 0.027 \); extension: \( r = 0.68, P = 0.007 \)). Subjects with more severe functional impairments also had larger decreases in torque as the contraction progressed through the phases. We calculated the decrease in torque from the early to the late phase of contraction at 120 deg/s and normalized this torque change by peak isometric torque. In the paretic limb, this normalized torque change was significantly correlated with Fugl-Meyer test score in flexion \( (r = -0.69, P = 0.006) \), but not extension \( (r = -0.05, P = 0.87) \). No significant correlations were present in the unaffected limb.

We also performed correlation analysis to determine whether isokinetic torque deficits were related to isometric torque deficits. Isokinetic torque during

**FIGURE 5.** RMS EMG values during the middle contraction phase normalized to levels obtained during maximal voluntary isometric contraction, and averaged across all subjects. In extension, brachioradialis EMG levels were higher in the paretic limb compared to the unaffected limb \( [F(1,13) = 9.0, P = 0.010] \), and triceps EMG was reduced in the paretic limb compared to the unaffected limb \( [F(1,13) = 6.2, P = 0.027] \).
the late phase at 120 deg/s was normalized by peak isometric torque. The isometric torque deficit was quantified by the paretic limb peak isometric torque normalized by unaffected limb peak isometric torque. In flexion, the isokinetic torque deficit was larger in subjects with larger isometric torque deficits \((r = 0.58, P = 0.03)\). In extension, there was no correlation \((r = 0.31, P = 0.28)\).

**DISCUSSION**

Our primary hypothesis was that movement velocity would have a greater effect on torque production in the paretic limb than in the unaffected limb following stroke. Our results demonstrate that this was indeed the case. As flexion velocity increased, paretic limb torque decreased at a greater rate than in the unaffected limb. During extension, paretic limb torque was much lower than unaffected limb torque at all phases and speeds. In both flexion and extension, the disparity between limbs in the constant-velocity torque–angle curves became more pronounced as velocity increased. A secondary hypothesis was that abnormal muscle activation patterns in the paretic limb would be consistent with the torque abnormalities. We found this to be the case for extension, where EMG data provided evidence for abnormally increased antagonist co-activation in brachioradialis and markedly reduced activation in triceps. However, we observed no EMG abnormalities during flexion. We also hypothesized that the torque deficits would be largest in subjects with more severe functional impairments. This was found to be true for both flexion and extension when the deficit was in terms of torque produced in the late phase at 120 deg/s. As the contraction progressed, declines in flexion torque were larger in more functionally impaired subjects, but this was not the case in extension.

**Potential Mechanisms.** The most striking result of the present study was the large deficit in elbow extension torque during dynamic contractions. Even after normalization for isometric strength differences between limbs, isokinetic extension torque was markedly reduced compared to the unaffected limb. EMG data provided evidence of decreased triceps activation in the paretic limb that was accompanied by increased co-activation of brachioradialis. These aberrant muscle activation patterns could have contributed to the large deficits in torque production. Due to the alteration in spinal reflex pathways known to occur in poststroke hemiparesis, the sensitivity to stretch velocity may be markedly increased in elbow flexors. This increased sensitivity to stretch may enhance activation of flexors during extension movements, and also may inhibit triceps through the reciprocal Ia inhibitory pathway. An alternative explanation for the disrupted muscle activation patterns is that hemiparetic subjects are unable to modulate and inhibit the stretch reflex response in flexors through descending pathways. In neurologically normal subjects, stretch reflex pathways are modulated during voluntary movements and vary as a function of elbow position. Descending modulation results in lower tonic and reflex activation in antagonists during lengthening contractions (e.g., stretch) as compared to isometric and shortening contractions. Following stroke, there is evidence that this descending modulation of reflex pathways is impaired. Such inability to inhibit properly the stretch reflex pathways of flexors during extension movements may have contributed to the increased co-activation of the antagonist flexor muscle (brachioradialis), and reciprocal inhibition of triceps, leading to the observed EMG patterns.

During flexion, increasing velocity led to a greater decline in torque in the paretic than unaffected limb. However, there were no significant between-limb differences in EMG levels. One possible mechanism that could explain this observation is selective decrease and atrophy of fast-twitch, type II muscle fibers, resulting in a higher percentage of type I fibers in the elbow flexors of the paretic limb. A related but alternative explanation is that decreased descending drive to the motoneuron pool of the paretic elbow flexors resulted in the inability to recruit high-threshold, fast-twitch motor units. This decreased descending drive would result in lower isometric torque levels, as well as reduced ability to generate torque at high velocities since the type II, fast-twitch motor units would not be recruited. Atrophy or inability to recruit type II muscle fibers is consistent with our finding that subjects with larger isometric torque deficits also had larger deficits in torque production at the higher velocities.

Our EMG results during extension can be explained by the physiologically based model suggested by Levin and colleagues. In their model, stroke subjects have a much smaller joint range over which the CNS can modulate stretch reflex thresholds. Movements outside of this range result in abnormal co-activation. To produce extension movements, the CNS shifts the stretch reflex thresholds of both agonist and antagonist muscles to beyond the physiological limit of joint extension. This produces the activation of agonist muscles and inhibition of
increased. Thus, a given velocity will produce a length change under the concavity of the force–velocity curve is increased as velocity increased. However, abnormally increased antagonist co-activation was not observed in biceps. Another aspect to this model is that the current activation level in agonists is proportional to the angular separation between the current joint angle and the reflex threshold. Thus, inability to shift the reflex threshold of triceps as far into joint extension as is done normally can explain the decreased levels of triceps activation that we observed in paretic limbs. Levin and colleagues also reported that the coefficient (μ) that characterizes the velocity sensitivity of the stretch reflex is increased after stroke. This is consistent with our observation that the torque deficit increased with increasing velocity.

We observed enhanced velocity effects in paretic limbs that could be partly due to alterations in the structure of muscle cells after brain injury. Lieber and colleagues reported that when the wrist joint is fully flexed, sarcomeres in the spastic flexor carpi ulnaris were significantly longer than normal (3.48 vs. 2.41 μm). It is generally assumed that sarcomere length scales the force–velocity relationship of muscle in proportion to the amount of overlap between actin and myosin filaments, but does not change the shape of the relationship. However, there is some evidence that at long sarcomere lengths, the concavity of the force–velocity curve is increased.27 Thus, a given velocity will produce a greater reduction in force when sarcomeres are at longer rather than shorter lengths (assuming normalization for length–tension effects). However, some caution should be exercised in applying these results to the population we tested. Their data represent severely spastic muscles in patients with joint contractures that were serious enough to warrant surgical interventions. Although some degree of spasticity was present in our subjects, none had contractures. Nevertheless, some degree of the structural remodeling observed in severely spastic muscles might have been present in some of our subjects.

Limitations of This Study. One of the limitations of this study relate to the interpretation of the phase analysis. As the flexion contraction progressed and agonist muscles reached shorter lengths, paretic limb torque decreased, whereas unaffected limb torque increased modestly (Fig. 4). This disparity could be explained by a previous study in hemiparetic subjects that reported greater deficits in elbow isometric strength when agonist muscles were at shorter lengths. However, in the paretic limbs of subjects who had no contractures, data from that study indicate that peak isometric flexion torque increased and reached a plateau as the test angle of the elbow was increased over the range of movement we analyzed (27.5–72.5 deg; see Table 2 in Ada et al.3). Only after approximately 80 deg of elbow flexion does the torque decrease. Thus, the striking downward trend in paretic limb flexion torque as the movement progressed through the phases (Figs. 2 and 4) is unlikely to have been due to the isometric torque–angle deficits reported by Ada and colleagues. In future studies, this could be verified by measuring both isometric and isokinetic torque–angle curves in the same subjects.

The disparity between limbs in torque–angle curves increased as velocity increased. This is additional evidence that this disparity was not due entirely to isometric torque–angle relations. Length-dependent deficits might be due to impaired motor unit rate coding at shorter muscle lengths. Neurologically normal subjects produce markedly increased motor unit firing rates at short muscle lengths, and hemiparetic persons may not be able to achieve these increased firing rates due to decreased descending drive. Subjects also may not be able to maintain motor unit firing rates for long periods of time in paretic muscles. If abnormally low motor unit firing rates were indeed a factor in our study, our data suggest that the effects on torque production are greater during movement than in isometric conditions.

Clinical Implications. Several studies of isometric elbow strength have reported that extension strength is more preserved than flexion after stroke. This observation appears to contradict clinical dogma that flexion strength is more preserved in hemiparesis. Data from the present study might explain this apparent contradiction. Consistent with the findings in the literature, we observed that peak isometric flexion torque in the paretic limb was 51% ± 5% of levels in the unaffected limb, whereas extension torque was 64% ± 7% of levels in the unaffected limb. However, during the late contraction phase at 120 deg/s, isokinetic torque was only 33% ± 6% of the unaffected limb in flexion and 14% ± 7% in extension. Thus, under dynamic conditions, extension was more impaired than flexion; under isometric conditions, flexion was more impaired than extension. The clinical dogma that elbow extensors are
more weakened than elbow flexors after stroke result from observations during performance of functional tasks under dynamic conditions, whereas studies that have reported greater impairment in flexion than extension are based on observations made under isometric conditions.

The velocity-dependent effects observed in the present study may have a large impact on functional ability of the limb. Our correlation analysis illustrated that the velocity-dependent torque impairment was larger in subjects with more severe functional limitations. Furthermore, the velocities we tested are not excessive and correspond with the range of speeds needed to perform routine activities of daily living. The magnitude of the effects are emphasized by examining the percentage decrease in torque as velocity was increased from 30 to 120 deg/s. In the late contraction phase, unaffected limb torque decreased 9% ± 5% in flexion and 16% ± 4% in extension. The corresponding decreases in the paretic limb were 44% ± 7% in flexion and 63% ± 9% in extension. Thus, a 90 deg/s increase in velocity had a profound effect on the paretic limb. Compared to the unaffected limb, decreases in paretic limb isokinetic torque were approximately five times larger in flexion and four times larger in extension.

Recent studies have emphasized the potential functional benefits of strengthening weakened muscles after stroke.118,40,45,49 Our results strongly suggest that velocity-dependent deficits in isokinetic torque are considerable and may contribute significantly to loss of function in the paretic limb. Isokinetic resistance training is one approach that isolates a targeted joint, introduces movement velocity in resistance training paradigms, and may prove effective for addressing the velocity-dependent deficits we observed. Our results also indicate that the effects of interventions should be evaluated not only in terms of active torque production, but also in terms of the effects on co-activation of antagonist muscles and velocity-dependent activation impairment in agonist muscles.

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REFERENCES


