Abnormal Volitional Hip Torque Phasing and Hip Impairments in Gait Post Stroke

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Hyngstrom A, Onushko T, Chua M, Schmit BD. Abnormal volitional hip torque phasing and hip impairments in gait post stroke. J Neurophysiol 103: 1557–1568, 2010. First published January 20, 2010; doi:10.1152/jn.00528.2009. The purpose of this study was to quantify how volitional control of hip torque relates to walking function poststroke. Volitional phasing of hip flexion and extension torques was assessed using a load-cell-instrumented servomotor drive system in 11 chronic stroke subjects and 5 age-matched controls. Hips were oscillated from ~40° of hip flexion to 10° of hip extension at a frequency of 0.5 Hz during three movement conditions [hips in phase (IP), 180° out of phase (OP), and unilateral hip movement (UN)] while the knees and ankles were held stationary. The magnitude and phasing of hip, knee, and ankle torques were measured during each movement condition. Surface electromyography was measured throughout the legs. Over ground gait analysis was done for all stroke subjects. During robotic-assisted movement conditions, the paretic limb produced peak hip torques when agonist hip musculature was stretched instead of midway through the movement as seen in the nonparetic and control limbs (P < 0.012). However, mean torque magnitudes of the paretic and nonparetic limbs were not significantly different. Abnormalities of paretic hip torque phasing were more pronounced during bilateral movement conditions and were associated with quadriceps overactivity. The magnitude of flexion torque produced during maximal hip extension was correlated with the Fugl Meyer Score, self-selected walking speed, and maximal hip extension during over ground walking. These results suggest that hyperexcitable stretch reflexes in the paretic limb impair coordinated hip torque phasing and likely interfere with walking function post stroke.

INTRODUCTION

Efficient locomotion depends on the coordination of bilateral hip movements. Control of the hips is complex, and involves not only the execution of supraspinal commands (Nielsen 2003) but also the integration of sensory information by spinal circuitry (Lam and Pearson 2002) for timely organization of multijoint and bilateral limbs movements. For example, data from animal and human studies have demonstrated the critical role that proprioceptive input from hip afferents (e.g., muscle spindles) play in the regulation of walking (Dietz 2002; Grillner and Rossignol 1978; Hiebert et al. 1996; Lam and Pearson 2001, 2002). Synaptic input from the hip flexors signals the transition from the stance phase of walking to swing in spinal and decerebrate cats (Grillner and Rossignol 1978; Lam and Pearson 2001), and passive hip movements can modulate the excitability of ankle and knee motoneurons (Hyngstrom et al. 2008). Likewise, in human spinal cord injury, stretch of the hip flexors is believed to be an important sensory cue for body-weight supported treadmill stepping (Dobkin et al. 1995; Pang and Yang 2000, 2002). In the neurologically intact state, these hip-related sensory effects potentially simplify the volitional control of walking. Following stroke, however, control of the hips during locomotion may be altered due to disruption of supraspinal and spinal circuitry and contribute to impaired generation of joint torques (Chen et al. 2005; Jonkers et al. 2009; Kim and Eng 2003; Nadeau et al. 1999) as well as abnormal multijoint torque coupling throughout the paretic leg.

The neuropathology of impaired hip movements and the contribution to walking dysfunction post stroke is not clear from the current literature. Motor impairments of the paretic leg have been linked to reductions in force generation, presumably by diminished cortical drive (Swayne et al. 2008), but could also due to abnormal processing of movement-related sensory afferents by spinal circuitry (Lewek et al. 2007). Although it is known that velocity-dependent reflex responses are enhanced post stroke (Black et al. 2007), growing evidence shows that abnormal processing of sensory information at the spinal level is far more complex (Sangani et al. 2007, 2009) and involves more than disregulation of classic Ia pathways (Eccles et al. 1957; Nichols 1999). Specifically, stretch-related abnormal reflex coupling of the hip and knee has been observed (Finley et al. 2008; Lewek et al. 2006, 2007) and correlated with “stiff knee gait.” In addition, there are abnormal torque couplings of the hip and knee during isometric tasks with the leg in locomotor related postures (Cruz and Dhaher 2008). The interpretation of the results of the aforementioned studies is limited, however, because they focus on unilateral leg tasks. Because walking involves bilateral movement of the legs, locomotor control strategies are likely to differ from control of unilateral movement.

Clinically, motor impairments following stroke are often assessed and treated unilaterally, but it is plausible that altered sensorimotor interactions between the legs could contribute to reduced walking capability. Using a pedaling paradigm, Ting et al. (1998, 2000) have demonstrated in neurologically intact subjects that movement-related sensory input from the contralateral limb can impact the phasing and magnitude of ipsilateral muscle activity during pedaling. These studies provide evidence of an inhibitory effect of extensor activity of the contralateral leg onto the ipsilateral flexors and demonstrate the functional significance of locomotor-related neural coupling between the antagonist musculature of opposite legs in healthy humans. In chronic spinal cord injury, contralateral leg sensory information appears to entrain ipsilateral activity (Ferris et al. 2004; Kawashima et al. 2005; Lunenburger et al. 2006). However, following stroke, contralateral leg activity has been shown to exacerbate motor impairments during pedaling.
Understanding the nature of alterations in bilateral hip control and the relationship with walking capability is important for post stroke rehabilitation. Over-ground and treadmill walking studies have documented decreased hip range of motion in the impaired leg during gait compared with the nonparetic limb for walking (Chen and Patten 2008; Chen et al. 2005; De Quervain et al. 1996). Despite the limited hip movement, people post stroke are especially reliant on hip control in the paretic limb to the extent that an orthosis is frequently placed at the ankle to limit its negative contribution to gait. Given the relative importance of hip sensory afferents for the regulation of locomotion and the apparent disregulation of stretch-related inputs following injury to the nervous system, it is plausible that abnormal bilateral reflex control of the hips could greatly impact the “normalcy” of gait post stroke. Although current clinical practice acknowledges the existence of stretch-related leg motor impairments, the literature is not clear on the relative contribution of stretch-related impairments (i.e., by quantification of torque or EMG patterns) on walking (Malhotra et al. 2009) despite the rehabilitative implications (Lundstrom et al. 2010).

The purpose of this study was to quantify the coordination (i.e., timing and magnitude) of torque production by the paretic limb during imposed sinusoidal oscillations of the hips during bi- and unilateral movement tasks and relate coordination abnormalities to hip-related impairments during walking. As an example, if stretch-related afferents dominate motor output of the paretic limb during volitional hip movements, then we would expect peak hip torques to occur when corresponding muscles are maximally stretched, resulting in a phase advance of torque as compared with the nonparetic and control legs. Correlations between torque phasing and muscle activity of the paretic limb with clinical measures of leg function and kinematics variables of gait would implicate a role for abnormal stretch responses at the hip in walking dysfunction post stroke.

METHODS

Subjects

Eleven subjects with chronic (>6 mo) post stroke hemiparesis participated in this study (see Table 1). Per reported history, all subjects suffered a single stroke with unilateral sensory and motor effects. Subjects were excluded if they reported any untreated cardiovascular or orthopedic conditions, decubitis ulcerations, osteoporosis, or if cognitive impairments limited their ability to follow instructions or provide informed consent. All subjects could ambulate independently on level surfaces without an orthosis or assistive device except S4, who ambulated with a large base quad cane and articulated orthosis. At the time of the study, none of the stroke subjects were taking oral anti-spasticity medications. S9 had received Botox injections in his paretic ankle musculature (gastrocnemius and soleus) 3 mo prior to the study. Five age-matched controls [mean age = 57.8 ± 10.2 (SD yr)] with no history of neurological injury were recruited for the study and underwent the robot-assisted leg movement portion of the study described in the following section.

The study was conducted in two experimental sessions on separate days ≥1 wk apart. During session one, robotic-assisted tests, the Fugl Meyer test, and the 10-m walk test were performed. Gait testing was done during session two. Subjects reported no change in medical or functional status between testing dates. Informed consent was obtained from all study participants and all experimental procedures were approved by Marquette University’s Institutional Review Board.

### Robot-assisted tests

**TEST APPARATUS AND EXPERIMENTAL SETUP.** Study participants were assisted onto a therapy plinth and asked to lie supine with their legs secured in customized braces (Fig. 1A). Two servomotor systems (Kollmorgen, Northampton, MA) adjacent to the participant’s hips were used to generate leg oscillations about the hip joint. Participants were fitted within the device by aligning the head of the greater trochanter with the axis of rotation of the servomotors. The participants’ legs were supported within the leg braces using a strap securing the thigh, a strap around the heel, and a clamp over the dorsum of the foot to secure it to a plate at the end of the leg brace. The approximate anatomical joint axes of rotation for the knees and ankles, determined by palpation, were aligned with the center axis of torque transducers (S. Himmelstein and Company, Hoffman Estates, IL) integrated within the leg braces. Sagittal plane torques were measured bilaterally for the hip, knee and ankle joints, and right and left hip positions were monitored using optical encoders (US Digital, Vancouver, WA) affixed to the servomotor systems. Surface electromyograms (EMGs) were measured bilaterally using Ag/AgCl electrodes placed over the muscle belly of the following muscles: vastus medialis (VM), rectus femoris (RF), vastus lateralis (VL), medial hamstring (MH), medial gastrocnemius (MG), soleus (Sol), and tibialis anterior (TA). EMG signals were amplified (1,000–10,000 times) and band-pass filtered (10–1,000 Hz: Bortec Medical AMT-16; Calgary, Alberta, Canada). Prior to acquisition, torque and EMG signals were low-pass filtered (500 Hz) and then sampled at 1 kHz using a data-acquisition card (National Instruments, Austin, TX) and PC. Custom-written LabVIEW (National Instruments) programs were used to generate a hip trajectory command signal to the servocon-

### Table 1. Characteristics of stroke subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age, yr</th>
<th>Sex</th>
<th>Hemisphere Affected</th>
<th>Time Since Stroke, mo</th>
<th>Fugl Meyer (Lower Extremity)</th>
<th>Self Selected Walking Speed: m/s</th>
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<tr>
<td>S1</td>
<td>78</td>
<td>F</td>
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<tr>
<td>S2</td>
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<td>M</td>
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<tr>
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<td>58</td>
<td>F</td>
<td>R</td>
<td>220</td>
<td>32</td>
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<td>53</td>
<td>F</td>
<td>L</td>
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<td>18</td>
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<tr>
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<td>F</td>
<td>L</td>
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<td>M</td>
<td>R</td>
<td>77</td>
<td>21</td>
<td>0.77</td>
</tr>
<tr>
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<td>52</td>
<td>F</td>
<td>R</td>
<td>184</td>
<td>31</td>
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<tr>
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<td>L</td>
<td>36</td>
<td>24</td>
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<tr>
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<td>M</td>
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<td>29</td>
<td>0.83</td>
</tr>
<tr>
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<td>F</td>
<td>R</td>
<td>300</td>
<td>28</td>
<td>0.70</td>
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</tbody>
</table>

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controller and acquire all data. EMG data were collected from 6 of the 11 stroke subjects and 5 of 5 noninjured controls.

Experimental protocol

Sinusoidal hip oscillations were imposed to the legs of study participants while they lay supine in the test apparatus. Hip coordination was evaluated using three different sinusoidal hip perturbations: bilateral 180° out of phase (OP), bilateral in phase (IP), and unilateral hip movements with the contralateral leg held stationary at 20° hip flexion (UN). For the unilateral tests, both the paretic and nonparetic legs were tested. The legs were moved through a 50° range, starting from 40° of hip flexion to 10° of hip extension through 10 consecutive cycles at an average velocity of 50°/s (0.50-Hz sinusoidal trajectory). This velocity was chosen as it has previously been shown to elicit a stretch reflex response in chronic spinal cord injured subjects (Onushko and Schmit 2007) and because it is below the reported ranges of maximum hip flexion and extension velocities in normal and spastic gait (Granata et al. 2000). The knee joints were held isometrically in a slightly flexed position of 15–20° and the ankles at 0–5° of plantarflexion for all tests. These ranges approximate leg posture during the late stance and preswing phases of gate (Perry 1992). Specifically, stretch responses have been demonstrated in this posture (Lewek et al. 2007), and this knee range optimizes stretch of hip flexors (Van Dillen et al. 2000). Participants were asked to either assist (“active”) the imposed hip oscillations or to remain relaxed (“passive”). Age-matched control participants were asked to complete the same protocol. Three trials were performed for each movement condition (OP, IP, and UN) for each group (active and passive) in random order using a block design.

At the completion of the experiment, two additional hip perturbations were done to assess the biomechanical properties of the legs and leg braces (Onushko and Schmit 2007; Steldt and Schmit 2004). The gravitational and inertial torque contributions were calculated off-line and subtracted from the filtered torque signals to obtain the torques only due to muscle activation (refer to Analysis). The gravitational torque contribution was approximated by slowly moving the legs through the entire range of motion for each test condition (IP, OP, and UN) for 5 s every 10°. The inertial properties of the leg and leg brace were approximated by rapidly (1.5 Hz) oscillating the leg within a small range (25–10° of hip flexion) to minimize the chances of eliciting reflex responses.

Analysis

The torque measurements recorded during the hip oscillations were corrected for the biomechanical properties of the legs and leg braces to obtain the joint torque associated with muscle activation. Gravitational and inertial torques were estimated for each subject using the method described by Onushko and Schmit (2007). Briefly, gravitational torque was approximated for each segment by first calculating the mean torque from the pause period during the slow incremental hip movements and then fitting a third-order polynomial to the mean torque. The polynomial coefficients were then used to estimate the gravitational torque about each joint during the experimental hip movements (i.e., IP, OP, and UN). To account for the inertial properties of the leg and leg brace, an inertial constant was calculated for each segment (i.e., thigh, shank, and foot) using the torque recorded during the rapid hip oscillations (1st subtracting the gravitational torque). The inertial torque was the product of the inertial constant and the trial acceleration. The estimated gravitational and inertial torques were subtracted from the experimental torque data for each joint. In addition, a mechanical artifact was present within the recorded torque measurement that was not associated with the gravitational or inertial properties of the legs. The mechanical artifact has been attributed to flexion of the brace itself, resulting in energy storage and release that produces an artifact in the recorded torque measurements. To account for the artifact, an ensemble average of torque recordings from neurologically intact participants during the passive hip oscillations (with an absence of EMG) was used to approximate the artifact, which was then subtracted from the experimental torque measurements.

FIG. 1. A and B: test apparatus used to measure hip, knee, and ankle torques during hip oscillations. Leg braces holding the knees and ankles stationary were attached to a servo motor drive system. C and D: schematic illustrating normalization of hip angle to a 360° polar coordinate system. The phase angle represents the relative timing of the generation of peak torque for a given direction. Vector magnitude signifies the repeatability of the timing across cycles.
Surface EMG signals were band-pass filtered (10–350 Hz) to remove baseline drift and possible high-frequency noise and notched filtered (59–61 Hz) to remove line noise using fourth-order Butterworth filters off-line. The root mean square (RMS) of the EMG signals were then calculated using a 100-ms sliding window. RMS EMG was used for all analyses. EMG activity was considered to be present if the signal magnitude exceeded three times the SD of baseline activity recorded during a trial with no movement.

A phase analysis was performed to examine the timing of the peak torque generated during the hip oscillations. The circular statistics methods outlined by Batschelet (1981) were used for this analysis and the details have been previously described (Onushko and Schmit 2007). In brief, torque data were first separated into flexion and extension components and the hip angular data were transformed to 360° of a polar coordinate system, where 0° represented the hip position in full flexion (40°) and 180° represented the hip position in full extension (10°) during one cycle of movement (Fig. 1, bottom). For each movement cycle, the Cartesian coordinates (x, y) of the peak torque were identified, and were converted into a phase angle, θ, and a normalized vector length (unit vector), r. The vectors for each movement cycle (n = 27 across the 3 trials of each condition) were then converted to Cartesian coordinates and the means of the x and y components were calculated. The mean coordinates were then converted back to polar form yielding a mean θ and a mean r. Note that if every cycle had exactly the same phase angle, the resulting r value would be 1. If the θ’s were randomly distributed, the resulting r value would be close to 0. Rayleigh’s test for directedness was performed on the data to determine whether the torque responses demonstrated a significant phasing, based on the r value (α = 0.05). The Watson-Williams Test was done to identify significant differences (α = 0.05) between the mean phase (θ) among the various movement types (OP, IP, and UN). A Bonferroni correction was used for multiple comparisons (α = 0.05/number of comparisons).

To characterize the magnitude of the abnormal phasing of the hip torque, the difference in the torque at maximum hip extension (∆THΕ) between the paretic and nonparetic limb was calculated. The rationale for this measurement was that abnormal control of hip torque could increase hip flexion torque in late stance (i.e., when the hip is extended), thereby limiting hip extension during gait. Torque was determined from each cycle of movement at peak hip extension and averaged across trials for the paretic and nonparetic limbs. The magnitude of the hip torque produced at maximum hip extension (10°) of the nonparetic limb was subtracted from the paretic limb torque at maximum hip extension to obtain ∆THΕ. ∆THΕ was then used for comparison with gait kinematics from the second experimental session.

The peak flexion and extension torque magnitudes for each joint were calculated to compare the relative strength of the paretic and nonparetic limbs during both the active-assist and passive trials. The peak hip, knee, and ankle flexion and extension torque was calculated for each movement cycle. The data were then averaged over three trials for each subject and each movement condition. A repeated-measures ANOVA was done for each torque direction with three trials for each subject and each movement condition. A repeated-measures ANOVA was done for each condition. The average comfortable walking (10-m walk test) speed for the stroke subjects was 0.91 ± 0.19 m/s (minimum = 0.19 m/s, maximum = 1.27 m/s).

**RESULTS**

**Clinical assessment of motor function in stroke subjects**

Stroke subjects (n = 11) had a mean ± SD lower extremity Fugl-Meyer score of 25 ± 5 of a total possible score of 34. The minimum score was 19/34 and the maximum score was 31/34. The average comfortable walking (10-m walk test) speed for the stroke subjects was 0.91 ± 0.39 m/s (minimum = 0.19 m/s, maximum = 1.27 m/s).

**Phasing of hip and knee torque during oscillatory hip movements**

The timing of peak hip and knee torque during imposed cyclical hip movements with and without subject assistance...
was different between the paretic and nonparetic limbs of the 11 chronic stroke subjects and between the paretic limb and the 5 age-matched controls. Example data from one stroke subject and one control subject during an active-assist OP trial are shown in Fig. 2. In general, stroke subjects produced peak hip flexion torque near the maximum hip extension position (−10°) and peak hip extension torque when the hip reached full flexion (−40°) (Fig. 3, top). This finding is consistent with a stretch reflex response of hip musculature. In contrast, the nonparetic and control limb peak hip flexion and extension torques were typically generated approximately midway through the hip movements (see control example, Fig. 3, left bottom). This suggests a volitional motor strategy for producing torque to accelerate the limb to achieve the target position and then a reduction of torque as the limb reaches the target. Only two post stroke subjects exhibited a significant torque response at the hip (defined as 3 times the SD of the mean torque) during the passive trials, and there was no significant torque response in the nonparetic and control limbs during passive trials. Thus group comparisons of phasing were only conducted for the assisted trials (Bonferroni correction: α = 0.05/number of comparisons).

In the group comparisons, the paretic limb peak hip flexion and extension torque was significantly phase advanced as compared with the phasing of the nonparetic and control limb (Watson Williams, P = 0.012, compare dashed to solid lines, Fig. 4). During the OP and IP active trials, peak hip flexion torque occurred at the hip extended position (10° of hip extension), while peak hip extension torque was phased when the hip was maximally flexed (40° of hip flexion). This represents an approximate 90° phase advance relative to the peak torque produced from the nonparetic and control limbs. Although the paretic limb hip torque was phase advanced, peak hip flexion torque during the UN active task was less phase advanced compared with the bilateral hip movement conditions (Watson Williams, P = 0.012, see Fig. 4).

Peak knee torque was produced out of phase with the hip movement in most cases, including the paretic limb (6 of 11 stroke subjects) and all nonparetic and control limbs. In other words, peak knee flexion torque occurred while the hip was extending and vice versa. As seen with the hip torque phasing for the controls and nonparetic limb, peak knee torque was generated during the movement (see Fig. 4, bottom). In the remainder of cases for the paretic limb (5 of 11 stroke sub-

![Fig. 2](http://jn.physiology.org/DownloadedFrom/10.220.32.247/on/September24,2016)

**Fig. 2.** Single subject data from 1 stroke subject’s (S4) paretic (A) and nonparetic (B) leg, and 1 control’s (C) leg during the assisted out-of-phase (OP) movement condition. Flexion and extension torques were measured at the hip, knee, and ankle joints. Electromyography (EMG) was measured from the quadriceps [vastus medialis (VM), rectus femorus (RF), vastus lateralis (VL)] and medial hamstrings (MH).
jects), knee flexion torque was generated during hip flexion and knee extension torque with the hip extending. This pattern of knee torque can be explained as the initiation of a flexor or extensor joint coupling response (i.e., flexor or extensor synergy). The discrepancy in the pattern of knee torque phasing of the paretic limb explains why the vector magnitudes for the group data were small for all movement conditions (Fig. 4, bottom). Phasing of the nonparetic and control limbs was not significantly different for the hip or knee torques across movement conditions (Watson Williams, Bonferroni correction, \( P = 0.012 \)).

**FIG. 4.** Group data showing the phasing of hip (A) and knee (B) torques for the paretic (dashed line), nonparetic (thick line), and controls (thin line) for all assisted movement conditions. The top half of each polar plot (hash marks, counter-clockwise from 0 to 180°) represents the portion of the movement cycle when the hip was extending. Peak extension torque vectors fall within shaded regions. Vector magnitude of 1 signifies high repeatability. The paretic limb flexion and extension torques were significantly phase advanced as compared with the nonparetic and control limbs. Phasing of flexion and extension torque of the paretic limb was not significantly different between bilateral movement conditions. However, during the unilateral condition, hip flexion torque was less phase-advanced as compared with the bilateral movement conditions. B: phasing of knee torques. As a whole, the direction of knee torque was phased opposite of the hip so when the hip was extending (hashed marks) the knee was flexing and vice versa. Paretic, nonparetic, and control were not phased differently, but note the low vector magnitude of the paretic limb indicating inconsistency in the phasing of the torque across subjects. Several of the stroke subjects (5/11) demonstrated knee flexion and extension torque in the same direction as the hip movement.
The peak hip and knee flexion and extension torques were not significantly different between the paretic and nonparetic limbs for any movement condition during the active assist trials (repeated-measures ANOVA, $P > 0.05$, partial $\eta^2 \leq 0.27$, estimated power $\leq 0.42$, Fig. 5). Controls, however, generated significantly greater hip flexion torque as compared with the paretic limb (Fig. 5) and extension torque (Fig. 5) for all movement conditions (ANOVA, $P < 0.05$, partial $\eta^2 \geq 0.2$, power $\geq 0.76$). Additionally, mean flexion and extension torques were greater for the active trials as compared with the passive trials for stroke subjects (repeated-measures ANOVA, $P < 0.05$, partial $\eta^2 \geq 0.59$, power $\geq 0.92$), except for hip extension torque in the paretic limb ($P = 0.081$, partial $\eta^2 \geq 0.27$, power $\geq 0.42$).

During the OP movement condition, the hip torque at the maximum extended hip position was calculated for the active trials. Consistent with the phasing of the hip flexion torque, the paretic limb generated significantly larger flexion torque in the fully extended hip position as compared with the nonparetic limb (paretic: $15.5 \pm 9.0$ Nm; nonparetic: $6.6 \pm 7.2$ Nm, $t$-test, $P < 0.05$). Correlations between $\Delta$THE (see METHODS for calculation) and gait measures are presented in the subsequent sections.

Characteristics of paretic muscle activity during active trials

To examine the extent of modulation in muscle activity, the EMG modulation index and EMG duration index were determined for each cycle of hip flexion and extension during the active trials for all movement conditions. EMG data were rejected in 5 of the 11 stroke subjects due to visually identified contamination from poor grounding. In the paretic limb, VM, VL, RF, and MH EMG modulation indices were larger (i.e., there was a smaller difference between the minimum and maximum RMS values) than in the nonparetic limb (Fig. 6). The EMG modulation index in the paretic limb was significantly greater for RF and VM as compared with the nonparetic limb for both the IP and OP movement conditions [Wilcoxon–
Fugl-Meyer score, self-selected walking pace, and kinematic variables associated with the hip during over ground walking (Fig. 8). Negative correlations between ΔTHE and Fugl-Meyer scores ($P < 0.05$, $r^2 = 0.77$) demonstrated that abnormal hip torque production was associated with a decreased ability to move in and out of lower extremity synergy patterns (i.e., less individuation of movement). A slower self selected walking speed was correlated with a larger ΔTHE ($P < 0.05$, $r^2 = 0.42$, Fig. 8B). Analysis of kinematic variables showed that subjects with larger ΔTHEs also had a greater hip flexion angle at the end of stance ($P < 0.05$, $r^2 = 0.47$, Fig. 8C). During normal gait, heel rise is associated with the continuation of hip extension, and stroke subjects with smaller ΔTHE values demonstrated higher hip extension velocity at heel rise ($P = 0.05$, $r^2 = 0.38$, Fig. 8D).

**DISCUSSION**

In this study, we showed that when stroke subjects attempt isolated hip movements, they are unable to properly time paretic leg hip torque. Peak hip flexion and extension torques were dominated by apparent stretch-related reflex responses as suggested by torques that were phase advanced compared with the torques of the nonparetic and control limbs. A stretch-related response is further supported by measures of EMG over activity in the paretic limb during the robotic-assisted movements. Abnormal phasing of hip torque could not be primarily attributed to weakness of the paretic limb as the peak magnitude of the torque production between the paretic and nonparetic limb was not significantly different for any of the movement conditions. These findings are clinically significant as we demonstrated a relationship between the inability to properly phase hip torque during robot tests with kinematic abnormalities during walking and self-selected walking speed.

**Abnormal stretch-related responses and volitional movements post stroke**

The timing of peak torque, quality of quadriceps EMG activity, and the effect of bilateral movements strongly suggest that abnormal stretch-related responses heavily influence motor output during volitional movement in chronic stroke. We showed that in the paretic limb, peak hip torques were observed when muscles were at their greatest length. That is, peak hip flexion torque occurred at maximum hip extension and peak hip extension torque occurred at maximum hip flexion, corresponding to maximum stretch of the muscles. The differences in the pattern of muscle activity in the paretic quadriceps as compared with the nonparetic limb included decreased modulation and relatively longer periods of activation, which supports the presence of stretch reflex activity during volitional movements and has been shown previously (Kautz and Brown).
to the motor output during the robotic-assisted hip movements in the current study. Five of 11 subjects demonstrated hip flexion coupled with knee flexion—a pattern of movement consistent with the clinically described flexor synergy (Brunnstrom 1966), and all subjects had less than normal Fugl-Meyer scores. However, the phase advance of the hip torque responses in our study, which occurred during lengthening of the hip muscles, strongly favors a stretch response. Because stretch-related responses were larger during active conditions as compared with passive conditions, this suggests that supraspinal input (whether from the cortex or brain stem) following stroke may provide generalized excitation to reflex circuitry or motor pools.

**Mechanisms of abnormal stretch response during volitional movements post stroke**

The pathophysiology of abnormal stretch-related responses during volitional movement in the chronic stroke population could involve increases in intrinsic motoneuron excitability (Nielsen et al. 2007). The increase in torque responses produced by stretched muscles and the observed over activity of the quadriceps in this study, reflected by the prolonged EMG duration and decreased EMG modulation, could be a result of changes in the intrinsic electrical properties of the motoneuron pools. In the presence of monoamines, voltage-sensitive persistent inward Ca^2+ and Na^+ currents (PICs) amplify excitatory synaptic input and lengthen the effect of synaptic input on motoneuronal output via plateau potentials (Heckman et al. 2008; Hounsgaard et al. 1988). Indirect evidence for PIC behavior exists in humans (Collins et al. 2001; Gorassini et al. 1998; Kiehn and Eken 1997), and similar evidence of PICs has been demonstrated in subjects with chronic spinal cord injury (Gorassini et al. 2004). This increased motoneuron PIC behavior in chronic spinal cord injury has been associated with increased stretch reflex responses (Hornby et al. 2006). In this case, the phase advance of the hip torque responses in our study, which occurred during lengthening of the hip muscles, strongly favors a stretch response. Because stretch-related responses were larger during active conditions as compared with passive conditions, this suggests that supraspinal input (whether from the cortex or brain stem) following stroke may provide generalized excitation to reflex circuitry or motor pools.
study, PIC activation could result from excitatory input from stretch afferents or from supraspinal input.

Recently, PIC behavior has been indirectly shown in chronic stroke subjects (McPherson et al. 2008) using excitation of stretch reflex pathways via tendon vibration to initiate plateau behavior, resulting in self-sustained firing of muscles in the paretic arm. The authors hypothesize that following stroke, motor control is more reliant on corticobulbar pathways, which in turn would produce greater monoaminergic drive to the spinal cord and initiate motoneuron PICs. In the intact state, an important modulator of PIC amplitude is synaptic inhibition, specifically reciprocal inhibition (Hyngstrom et al. 2007; Kuo et al. 2003), and this has been reported to be limited in stroke subjects (Crone et al. 2003). Hamstring activity was modulated in a relatively normal fashion in the current study, which is consistent with evidence of more PIC-related activity in extensor motoneurons than flexor motoneurons (Hultborn et al. 2003). Recent work examining the relative states of tonic inhibition and excitation to the cord indicates that extensor motor pools are subject to greater changes in inhibitory conductances (Endo and Kiehn 2008). The cortex, which is compromised by stroke, is an important regulator of reciprocal inhibition (Chen et al. 2006; Pyndt et al. 2003), and the loss of cortical modulation of spinal pathways could account for a decreased ability of the motoneuron to appropriately process synaptic input, thereby increasing the gain and duration of an excitatory input. This is consistent with the decreased modulation and longer lasting quadriiceps EMG observed in this study.

The difference in torque phasing between the bi- and unilateral task also supports the idea of an exaggerated motoneuron response to an excitatory synaptic input. Given the interlimb synaptic connections involved in locomotor-related circuitry in the spinal cord (Kiehn 2006), it is plausible that motoneurons receive more excitatory synaptic input during bilateral movement than during a unilateral movement. This spinal coupling likely underlies activity in the contralateral leg during unilateral stepping in people with complete spinal cord injury (Ferris et al. 2004). In stroke subjects, and to a lesser extent in controls, cyclic activity of one limb induces rhythmic muscle activity in a stationary contralateral limb during pedaling (Kautz et al. 2006). Thus simultaneous hip, knee and ankle movements may further augment the effects of contralateral limb movement. Results from this study are significant as they highlight the relationship between hip control and walking function.

Effect of abnormal stretch related responses on walking function

It is widely debated whether abnormal movement-related reflex responses interfere with or facilitate voluntary movement (Dietz and Sinkjaer 2007; Nielsen et al. 2007; Patten et al. 2004). The role of reflexes in functional movement is relevant, as many medical strategies such botulinum toxin and baclofen as well as physical therapy techniques aim to reduce the strength of “spastic” reflex responses. However, a reduction in spasticity may actually be detrimental in some patients who rely on their spasticity to walk (Berger et al. 1984; Dietz and Sinkjaer 2007; Perry 1993).

The presence of stretch reflex responses during a volitional movement has its most detrimental effect on the timing of torque production if it cannot be modulated by supraspinal or spinal sources of inhibition. For example, Musampa et al. (2007) have proposed that decreased stretch reflex thresholds (SRTs) produce spatially organized spasticity zones. Consequently, SRT and the associated magnitude of stretch response could depend on the position of the limb during active movements. In this scenario, it is possible that subjects may avoid certain positions during movement to prevent an interfering spastic response. Our results support this concept, as subjects who had less hip extension at the end of stance and abnormal extension hip velocity at heel rise also produced significantly more abnormal flexion torque at end range extension during the robot task—where they were forced to move their leg through a specific range.

The translation of our results involving supine robotic-assisted movements to tasks such as walking is supported by studies involving robotic-assisted walking and pedaling. Abnormal phasing of hip flexion torque production (phase advanced as compared with the unimpaired) has recently been demonstrated when stroke subject’s limbs were moved through robotically imposed locomotor patterns (Neckel et al. 2008), where the limb was moved through more hip extension than produced in over-ground walking. Because subjects were at least partially weight bearing and upright, our study extends these findings by implicating stretch afferents as the mechanism of pathology because our robot-imposed movements were measured in supine, a position with relatively less weight bearing load and vestibular inputs. Excavations of impairments (e.g., inappropriate muscle activity) in chronic stroke as a function of “verticality” has been demonstrated in a pedaling paradigm (Brown et al. 1997) albeit stroke subjects demonstrated similar ability to modulate EMG activity as age matched controls. Although the robotic-assisted movements conducted in supine for this study involved different sensory and biomechanical components than over-ground walking, the movement tasks share a goal of coordinating reciprocal hip torque. Based on the findings of the aforementioned studies, it is plausible that our findings in supine would be amplified during walking and our results showed a significant correlation to walking dysfunction. Our findings are clinically relevant because subacute stroke rehabilitation often involves the practice of a mobility task or exercises in a gravity lessened position (i.e., supine) to reduce the negative effects of upright posture on coordination.

Rehabilitation implications

Our data indicate that in chronic stroke, abnormal stretch reflex responses elicited during a volitional task are related to restricted range of motion at the hip and angular velocity at the hip during ambulation. Although this study does not necessarily invalidate past studies implicating ankle (Turns et al. 2007) and knee (Kerrigan et al. 2001) deficits resulting in reduced propulsion of the limb, recent modeling of the lower extremity provides strong evidence that elastic coupling of the hip and knee are a better determinant of limb control than propulsion of the ankle during normal gait (Dean and Kuo 2009). Along with functionally relevant strength training, treatment strategies that aim to reduce the influence of hip muscle overactivity during gait need to be explored. For example, it might be useful to augment reciprocal inhibitory pathways onto hip flexor motoneuron pools during gait training. Future studies are needed

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to address the degree to which overactivity of muscles can be modulated perhaps via augmentation of inhibitory pathways during walking. Given the importance of the hip in locomotion, improving hip-related sensory input may also have downstream effects on limb control. Another option for reducing muscle overactivity is botulinum toxin injections (Ward 2008); however, these injections can be painful, have transient results, and might produce unwanted weakness in the hip flexors. Results from this study demonstrate the need for more dynamic assessment of muscle overactivity to discern the best medical intervention.

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GRANTS

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