Supplemental Materials and Methods

The shank and thigh static equilibrium equations can be derived as follows:

$$\vec{F}_k+\vec{F}_{sd}+\vec{F}_{sp}=0;$$

$$\vec{F}_h+T_k\cdot(-\vec{F}_k)+\vec{F}_t=0;$$

$$\vec{M}_k+\vec{M}_{sd}+\vec{M}_{sp}+\vec{r}_{sd}\times\vec{F}_{sd}+\vec{r}_{sp}\times\vec{F}_{sp}=0;$$

$$\vec{M}_h+T_k\cdot(-\vec{M}_k)+\vec{r}_k\times[T_k\cdot(-\vec{F}_k)]+\vec{M}_t+\vec{r}_t\times\vec{F}_t=0;$$

where $\vec{F}_k$, $\vec{F}_h$ are the knee and hip joint reaction forces, respectively. $\vec{M}_k$, $\vec{M}_h$ are the knee and hip torques generated by the subject. $\vec{F}_t$, $\vec{F}_{sd}$, $\vec{F}_{sp}$ are the measured thigh, distal shank, and proximal shank load cell force vectors and $\vec{M}_t$, $\vec{M}_{sd}$, $\vec{M}_{sp}$ are the corresponding load cell torques. The position vectors $\vec{r}_{sd}$, $\vec{r}_{sp}$, and $\vec{r}_t$ are the relative vectors from the center of the distal shank, proximal shank, and thigh load cells to the knee joint center defined in the local shank coordinate system (x_s, y_s, z_s) and hip joint center defined in the thigh coordinate system (x_t, y_t, z_t), respectively. The knee joint center was assumed to be located at the midpoint of the line between the medial and lateral femoral epicondyles,[1] whereas the hip joint center was located using regression relations.[2] The location of the knee joint center with respect to the hip joint center ($\vec{r}_k$) was defined in the thigh coordinate system and estimated using predictive regression equations[3] based on anthropometric measures. Finally, T_k is the transformation matrix between the shank and thigh coordinates, representing the knee flexion/extension angle. Overall, there are 4 vector equations to solve for the 4 unknown vector quantities: $\vec{F}_k$, $\vec{F}_h$, and $\vec{M}_k$, $\vec{M}_h$. The z-components of $\vec{M}_k$, $\vec{M}_h$ describe the flexion/extension torques applied by the subject at hip and knee joints, respectively. The x-components of $\vec{M}_h$ describe the abduction/adduction torque applied by the subject at the hip.

At the end of the experiment, knee flexion and extension maximum voluntary torques were collected to explore how differential knee strength may impact the observed across joint torque coupling.

Data Analysis

The knee and hip torques were divided by the moment of inertia of the mass below each joint about the axis the torque was acting. For each participant, the lower extremity inertias were calculated according to anthropometric regression equations[3] based on weight, height, shank length, foot length, tibial height, and knee, ankle, thigh and calf circumferences. While similar to traditional normalization methods of height and weight, this method takes into account the differences in torque required to move the flexed leg in the frontal versus sagittal plane.

To explore the potential confounding effects of differential knee extension-to-flexion strength ratio across groups, knee-strength data were collected on a subset of stroke and control subjects (n=10). The mean ratio of maximum extension-to-flexion knee torque was calculated for each group. Nonparametric tests were used to investigate statistical differences across groups within posture.

Supplemental Results

Inclusion criteria for individuals with stroke included: (1) clinical symptoms consistent with a single ischemic or hemorrhagic unilateral brain lesion, confirmed by an imaging study, resulting in sensory motor dysfunction no less than 6 months before evaluation; (2) the ability to walk 5-meters without assistance, with a gait speed >0.4 m/s with or without an assistive device; and (3) clinically identifiable circumduction gait pattern. A subject was accepted if at least 2 of the 3 physical therapists who screened him/her concurred that the individual had a circumduction walking strategy. The degree of circumduction was variable, with some subjects making large lateral excursions, whereas others were only slight circumductors. Exclusion criteria for both the stroke and control groups include: (1) severe osteoporosis, cardiorespiratory or metabolic diseases; (2) unhealed decubiti; (3) a history of balance deficits not related to the stroke; (4) persistent infection; and (5) significant cognitive or communication impairment.

Unlike the stroke population, the hip torque biases of the control group were inconsistent. Only 30% of the control group produced resultant torque biases similar to the stroke group, whereas the remaining 70% of subjects exhibited different resultant torque directions. Although the larger number of subjects in the stroke group certainly contributes to the difference in torque bias, the distribution of values about the mean is more likely the cause. To explore this, circular statistics were performed on a random sample (n=11) of stroke subjects, and the torque bias, mean, and median angles were not different from the complete data set (P>0.05).

Using a nonparametric test, there were no statistical differences in the maximum extension-to-flexion knee torque ratios across groups within posture. The mean (SD) in the stroke subgroup was 2.23 (1.69) and 2.64 (2.35) at the toe-off and midswing postures, respectively. The corresponding ratios in the control group were 1.40 (0.59) and 1.39 (0.64) at toe-off and midswing postures, respectively.

Supplemental Discussion

The across group similarity in abduction strength may be attributed to the inclusion criteria for the subject population, which consisted of circumducting stroke subjects. However, this criterion was achieved by visual inspection and may have been influenced by inter-rater variability. Indeed, post hoc gait analysis of the stroke subjects revealed high variability in frontal plane kinematics in the swing phase,[4,5] which was inconsistent with the visual examination. Therefore, it is highly unlikely that the hip strength observed in the stroke group is attributed to the subject inclusion constraints and may reflect an actual maintenance of abduction strength poststroke.

Modeling studies of healthy subjects have shown that the hip torques differ across limb configurations during isometric contractions and are associated with changes in muscle moment arms related to the joint angle and the force-length behavior.[6] Posture effects on torque production have been attributed to "posture dependent weakness" after stroke. For example, Koo et al[7] observed angle-dependent muscle weakness at the hemiparetic elbow joint of stroke survivors during maximal isometric voluntary contractions. Our data indicate that whereas impaired flexion and abduction/flexion torque was observed in both postures (posture independent), reduced hip adduction torque was only observed in the toe-off posture. These findings suggest an uneven distribution of the posture-dependent muscle weakness at the affected hip.

Our data indicated that 3 of the 22 stroke subjects exhibited a different across-joint torque pattern, primarily a knee flexion torque produced during the hip adduction torque generation task. To examine the potential reasons for this imperfect distribution of knee torque among the stroke group,

Table I. Maximum Voluntary Isometric Hip Torque

	Stroke			Control			
Hip Torque Direction	Mean	SD	SE	Mean	SD	SE	*P* Value
	Toeoff (n = 22 for stroke, n = 11 for control)						
Abduction	13.50	7.61	1.62	18.53	13.04	3.93	0.257‡
Abduction/Flexion*	7.12†	5.39	1.15	15.37	6.33	1.91	0.0005
Flexion*	10.23†	6.39	1.34	19.58	9.15	2.76	0.0016
Adduction/Flexion	15.69	7.88	1.68	24.36	14.77	4.45	0.092‡
Adduction*	13.67	9.22	1.97	22.35	12.69	3.83	0.032
Adduction/Extension	14.30	9.96	2.12	17.52	9.21	2.76	0.377
Extension	17.52	8.64	1.84	19.20	13.47	4.06	0.667
Abduction/Extension	14.48	6.21	1.32	16.46	9.12	2.75	0.467
	Midswing (n = 21 for stroke, n = 11 for control)						
Abduction	10.90	6.95	1.52	18.78§	18.53	5.86	0.222‡
Abduction/Flexion*	5.27†	4.54	0.99	14.96†	7.03	2.12	0.00005
Flexion*	5.32†	3.75	0.82	11.54	3.99	1.20	0.00013
Adduction/Flexion	10.21	5.67	1.24	16.96	10.24	3.09	0.063‡
Adduction	14.05	9.52	2.08	19.14§	14.07	4.45	0.244
Adduction/Extension	16.06	8.69	1.90	24.65	15.49	4.67	0.111‡
Extension	17.99	8.52	1.86	16.74	6.90	2.08	0.679
Abduction/Extension	11.79	6.73	1.47	18.54	13.95	4.21	0.154‡

*significant differences ($P<0.05$) between stroke and control for which the *P* values are shown in the last column; †significant differences ($P<0.05$) between that direction and the 180 degrees out of phase direction for the same group (eg, Stroke Abduction and Stroke Adduction). ‡unequal variances; §n=10

a number of characteristics of the 3 stroke subjects were analyzed including medical history, demographics, anthropometry, gender, spasticity, range of motion, and gait parameters. None of these variables explained the different knee torque behavior the 3 stroke subjects exhibited. However, the location and the extent of the infarct and the type of physical therapy each received postinjury may potentially play a role in the observed behaviors.

Peripheral receptors signaling static joint postures may also change the state of the motorneuron pool through the excitability of the corticomotor tract. For example, the flexor carpi radialis H-reflex response to electrical stimulation of the median nerve does not differ significantly across shoulder positions; yet, significant changes in intracortical inhibition were observed with changes in shoulder position.[8] Therefore, shoulder position may influence the recruitment efficiency of the corticospinal volleys to motorneurons of forearm muscles. In the future, a paired-magnetic stimulation paradigm conducted at both lower limb postures used in this study may reveal posture-dependent cortical excitability of the quadriceps and hamstrings muscles.

Table II. Paired *t* Tests on Target Torque: Differences Between Toe-Off and Midswing Postures

Target Direction	*P* value	Greater Torque	*P* value	Greater Torque
	Stroke		Control	
Abduction	0.008*	Toe-off	0.965†	
Abduction/Flexion	0.104		0.996	
Flexion	0.0037*	Toe-off	0.024*	Toe-off
Adduction/Flexion	0.00037*	Toe-off	0.153	
Adduction	0.626†		0.965†	
Adduction/Extension	0.114†		0.039*	Midswing
Extension	0.848†		0.802	
Abduction/Extension	0.039*	Toe-off	0.605	

t test for difference between means; †nonparametric, Wilcoxon signed-rank test for Difference in Medians, with continuity correction. $*P < 0.05$

Supplemental References

1. Blankevoort L, Huiskes R, de Lange A. Helical axes of passive knee joint motions. *J Biomech*. 1990;23:1219–1229.
2. Seidel GK, Marchinda DM, Dijkers M, Soutas-Little RW. Hip joint center location from palpable bony landmarks–a cadaver study. *J Biomech*. 1995; 28:995–998.
3. Zatsiorsky V, Seluyanov V. Estimation of the mass and inertia characteristics of the human body by means of the best predictive regression equations. In: Winter D, Norman R, Wells R, Hayes K, Patla A, eds. *Biomechanics IX-B*. Human Kinetics: Champaign, IL;1985:233–239.
4. Hayes T, Patton J, Dhaher Y. Evidence For Restricted Control Options in The Lower Limbs of Stroke Subjects. Paper presented at: Society for Neuroscience, 2006; Atlanta, GA.
5. Kerrigan DC, Frates EP, Rogan S, Riley PO. Hip hiking and circumduction: quantitative definitions. *Am J Phys Med Rehabil*. 2000;79: 247–252.
6. Arnold AS, Salinas S, Asakawa DJ, Delp SL. Accuracy of muscle moment arms estimated from MRI-based musculoskeletal models of the lower extremity. *Comput Aided Surg*. 2000;5:108–119.
7. Koo TK, Mak AF, Hung L, Dewald JP. Joint position dependence of weakness during maximum isometric voluntary contractions in subjects with hemiparesis. *Arch Phys Med Rehabil*. 2003;84:1380–1386.
8. Ginanneschi F, Dominici F, Biasella A, Gelli F, Rossi A Changes in corticomotor excitability of forearm muscles in relation to static shoulder positions. *Brain Res.* 2006;1073–1074:332–338.