Neural and Muscular Determinants of Dorsiflexor Weakness in Chronic Stroke Survivors

Cliff S. Klein, Geoffrey A. Power, Dina Brooks, and Charles L. Rice

Few examined the contribution of neural and muscular deficits to weakness in the same stroke subject. We determined maximal voluntary contraction (MVC) and 50 Hz torques, activation (twitch interpolation), electromyographic (EMG) amplitude and antagonist coactivation, and muscle volume using magnetic resonance imaging (MRI) of the dorsiflexors bilaterally in 7 chronic stroke subjects (40–67 y). Recordings of MVC and 50 Hz torque were also done in 7 control subjects (24–69 y) without stroke. The MVC torque was smaller in the contralesional than ipsilesional limb (29.8 ± 21.3 Nm vs. 42.5 ± 12.0 Nm, p = .04), and was associated with deficits in activation ($r^2 = .77$) and EMG amplitude ($r^2 = .71$). Antagonist coactivation percentage was not significantly different between limbs. Muscle volume, 50 Hz torque, and specific torque (50Hz torque/muscle volume) were also not different between sides. The concept that atrophy is commonplace after stroke is not supported by the results. Our findings indicate that dorsiflexor weakness in mobile stroke survivors is not explained by atrophy or reduced torque generating capacity suggesting an important role for central factors.

Keywords: muscle function, special needs populations, strength, isometric

Chronic stroke survivors (>1 y poststroke) have varying degrees of weakness and impaired motor control that compromises daily function (Arene & Hidler, 2009). During walking the dorsiflexors help to stabilize the ankle during stance and optimize foot-ground clearance during swing (Dubo et al., 1976). These dorsiflexion functions may be impaired by weakness and thereby contribute to the slow gait and falls following stroke (Knutsson & Richards, 1979). Weakness, defined as the inability to generate normal levels of torque during a maximal voluntary contraction (MVC), predominates in the contralesional (paretic) limb after hemiparetic
stroke (Adams, Gandevia, & Skuse, 1990; Frontera, Grimby, & Larsson, 1997; Horstman et al., 2008; Klein, Brooks, Richardson, McIlroy, & Bayley, 2010; Knorr, Ivanova, Doherty, Campbell, & Garland, 2011; Landau & Sahrmann, 2002; Levin & Hui-Chan, 1994). Whether the loss of strength arises solely from neural impairments or also reflects altered intrinsic muscle properties is uncertain (Frontera, et al., 1997; Jakobsson, Edstrom, Grimby, & Thornell, 1991; Landau & Sahrmann, 2002; Odajima, Ishiai, Okiyama, Furukawa, & Tsukagoshi, 1987; Ramsay, Barrance, Buchanan, & Higgison, 2011; Scelsi, Lotta, Lommi, Poggi, & Marchetti, 1984; Slager, Hsu, & Jordan, 1985).

Impaired muscle activation likely contributes to poststroke weakness (Frontera, et al., 1997; Horstman, et al., 2008; Klein, et al., 2010; Knorr, et al., 2011; Newham & Hsiao, 2001). For instance, during a dorsiflexor MVC, the surface electromyographic (EMG) amplitude or motor unit discharge rates of the tibialis anterior (TA) are often reduced in the contralesional limb (Frontera, et al., 1997; Knorr, et al., 2011). In addition, activity of the antagonist muscles during a MVC may reduce MVC torque. Antagonist coactivation in stroke survivors has been shown to be excessive during an MVC in some cases (Klein, et al., 2010; Levin & Hui-Chan, 1994; Yusevich, 1968), although other reports show that coactivation is similar between the contralesional and ipsilesional limb (Clark, Condliffe, & Patten, 2006; Lum, Patten, Kothari, & Yap, 2004; Newham & Hsiao, 2001; Wagner, Dromerick, Sahrmann, & Lang, 2007).

The contribution of poststroke changes in muscle properties to dorsiflexor weakness is uncertain (Frontera, et al., 1997; Jakobsson, et al., 1991; Landau & Sahrmann, 2002; Odajima, et al., 1987; Slager, et al., 1985). The study by Landau and Sahrmann (2002) compared the side-to-side differences in dorsiflexor MVC force and maximal force generating capacity evoked by 50 Hz stimulation over the TA. In the 14 subjects with chronic stroke, the mean MVC force was significantly less in the contralesional than ipsilesional limb, but mean 50 Hz forces were not different. However, contralesional 50 Hz force was 20–70% smaller than the ipsilesional value in four persons, and was unexpectedly larger by 30–150% in five others, but the cause of these differences was not evaluated (Landau & Sahrmann, 2002). A reduction in 50 Hz force may result from muscle atrophy or reduced specific tension (i.e., torque per unit area or volume), or both, whereas an increase could indicate opposite responses. Poststroke dorsiflexor (Odajima, et al., 1987; Ramsay, et al., 2011) or TA muscle fiber (Frontera, et al., 1997; Jakobsson, et al., 1991; Scelsi, et al., 1984; Slager, et al., 1985) morphology has been determined, but the findings are not always in agreement. One study reported smaller dorsiflexor cross-sectional area (CSA) in the contralesional than ipsilesional limb based on computer tomography images (Odajima, et al., 1987), but another found no differences in TA volume determined by MRI (Ramsay, et al., 2011). At the fiber level, Scelsi and coworkers (1984) reported progressively smaller type II fiber diameter in the contralesional TA with increased time (1–17 months) since stroke, but Jakobsson et al., (1991) found that fiber CSA was not different between chronic stroke subjects and healthy controls.

Although central neural activation, muscle size, and maximal muscle force generating capacity are primary determinants of voluntary strength, all three determinants have not been assessed concurrently in the same stroke subjects; and thus the purpose of this study. More specifically, we addressed whether weakness
arises solely from neural impairments or also results from atrophy and reduced maximal force generating capacity. In addition to this comprehensive assessment of the determinants of weakness, the findings should provide some insight into the conflicting results noted above pertaining to dorsiflexor (TA) size and force generating capacity. We hypothesized that most of the contralesional dorsiflexor weakness would reflect reduced activation, but that intrinsic changes in muscle properties also would contribute in some cases.

**Methods**

**Participants**

Five men and two women (56.9 ± 9.0 y, mean ± SD) who had a stroke at least 2 years before the study were recruited (Table 1). These individuals participated in a previous study of plantar flexion weakness (Klein, et al., 2010), and showed significant limb differences in strength, activation, and muscle volume. Therefore the same subjects were tested in the current study providing a sufficient sample size for these comparisons. Motor impairment of the leg and foot (Gowland et al., 1993) ranged from mild to moderate (Table 1). Three subjects (no. 4–6) walked with a more pronounced reduction in knee flexion than the others. Two subjects (no. 4 and 7) used a single point cane when walking, and two others (no. 3 and 6) wore an ankle orthotic. All participants could walk without assistance, but their gait speed varied threefold, and the average speed (0.83 ± 0.33 m/s) was about 30% slower than healthy adults (Klein, et al., 2010). Four subjects (subjects 1, 3, 5, and 6) engaged in 20–30 min of aerobic exercise about two times per week at a fitness club over the previous year. We also determined bilateral MVC and evoked torque in 7 men with no history of neurological impairment (38 ± 16 y, range, 24–69; 175.2 ± 14.7 cm; 82 ± 13 kg). Informed written and verbal consent were obtained in all subjects and the study was approved by the local research ethics committees of our institution.

**Table 1  Characteristics of the Stroke Subjects**

<table>
<thead>
<tr>
<th>Subject no.</th>
<th>Sex</th>
<th>Age (y)</th>
<th>Ht (cm)</th>
<th>Wt (kg)</th>
<th>CMSA Leg/foot</th>
<th>Speed (m/s)</th>
<th>TSO (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>67</td>
<td>185.4</td>
<td>104.5</td>
<td>5/5</td>
<td>1.35</td>
<td>36</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>63</td>
<td>180.3</td>
<td>81.8</td>
<td>4/4</td>
<td>1.0</td>
<td>43</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>61</td>
<td>162.6</td>
<td>61.4</td>
<td>4/3</td>
<td>0.63</td>
<td>37</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>56</td>
<td>177.8</td>
<td>68.2</td>
<td>4/3</td>
<td>0.49</td>
<td>27</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>51</td>
<td>185.3</td>
<td>95.4</td>
<td>3/3</td>
<td>0.58</td>
<td>24</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>40</td>
<td>167.6</td>
<td>68.2</td>
<td>3/3</td>
<td>0.82</td>
<td>51</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>60</td>
<td>178.0</td>
<td>61.4</td>
<td>4/2</td>
<td>0.91</td>
<td>48</td>
</tr>
</tbody>
</table>

Abbreviations: M, male; F, female; Ht, height; Wt, weight; CMSA, Chedoke-McMaster Stroke Assessment Score; Speed, walking speed; TSO, time since onset of stroke
Experimental Set-Up

Only one leg was assessed during each of the two visits to the laboratory to minimize general fatigue of the participants. The testing order of the contralesional and ipsilesional limb, and right-left side of the controls, was randomized. Subjects were seated with their leg positioned in a custom torque measuring device (Klein, et al., 2010). The knee and hip joints were flexed to 90°, and a clamp stabilized the knee from above. The foot rested on a plate and was secured with straps across the instep and proximal to the great toe. The ankle was plantar flexed 30° from the neutral (0°) position. At this angle, the peronei are shortened and are therefore less effective in counteracting the evoked torque of the dorsiflexors. In addition, 30° is near optimal for MVC torque (Marsh, Sale, McComas, & Quinlan, 1981) and optimal for 50 Hz torque (van Schaik, Hicks, & McCartney, 1994).

The compound muscle action potential (M-wave) and MVC EMG were recorded with disposable silver-silver chloride disc electrodes positioned in a bipolar configuration (Bortec Biomedical Ltd., Calgary, Alberta). The recording electrodes were placed over the distal third of lateral gastrocnemius (LG) medial gastrocnemius (MG), and midline of the soleus (2 cm below the distal end of the MG) using a 2 cm center-to-center spacing. For the TA, the distal electrode was placed at the border of the muscle belly and tendon, and the other electrode was placed 4–5 cm proximally.

The EMG was amplified (×500) and filtered between 30 Hz and 1 kHz (Astro-Medical, Model P511, West Warwick, RI, USA). Torque was sensed by a transducer beneath the ankle joint (LCDA-500, Omega Engineering Inc, Stamford, CT, USA), and amplified (×400) and filtered (DC-100 Hz). The EMG and torque signals were digitally converted by a 12-bit converter (1401 Plus, Cambridge Electronic Design, Cambridge, UK) at sampling rates of 2000 Hz and 500 Hz, respectively, and analyzed with Spike 2 software (Spike 2, version 7.03, Cambridge Electronic Design).

To find the optimal site for stimulation of the common fibular nerve, a bar electrode (1 cm diameter discs, 3 cm spacing; Medtronic, Skovlunde, Denmark) was placed immediately inferior to the fibular head and manually adjusted until M-waves were evoked during submaximal stimuli. For all subsequent recordings, 3 cm diameter round carbon rubber electrodes (Empi, St. Paul, Minnesota, USA), coated in conductive gel, were positioned at the optimal site and secured with tape. These electrodes minimized subject discomfort during 50 Hz stimulation.

Protocol. Single pulses (50 μs) of progressively greater current intensity (5 mA increments) were applied by a constant current stimulator (Model DS7AH, Digitimer) every 5 s until the TA M-wave peak-to-peak amplitude did not increase further (i.e., Mmax). Subjects generated a few practice voluntary contractions of the dorsiflexors at ~50% effort. Three to four MVCs, each lasting about 4–5 s followed by a two min rest, were recorded. Strong encouragement was provided during the MVC and a display of the on-going torque trace was provided. Before and after each MVC, single supramaximal (120% of Mmax current) pulses were applied so that percentage activation could be determined (Belanger & McComas, 1981). The stimuli were triggered manually by the investigator about 2–4 s after the beginning of the MVC, when there was a plateau in the torque trace, and also about 1 s after the MVC finished.
Three minutes after recording the MVCs, two practice 50 Hz contractions (each 0.5 s) were evoked at progressively greater submaximal current intensities. A 50 Hz train (0.5 s) was then evoked using the supramaximal current. Supramaximal stimulation of the dorsiflexors at 50 Hz is well tolerated and the evoked response reflects the maximal torque that can be generated by the dorsiflexor muscles (independent of subject volition) (Marsh, et al., 1981). Inclusion of this measure together with the assessment of muscle volume allowed us to determine whether changes in muscle properties contribute to the loss of MVC strength. Specifically, smaller contralesional than ipsilesional 50 Hz torque would indicate a loss in torque generating capacity, either because of atrophy or reduced specific tension (force per unit area) of the muscle fibers, or both. Specific torque (50 Hz torque/muscle volume) was calculated in the current study as an indirect measure of specific tension. After recording the 50 Hz torque, subjects completed three to four plantar flexor MVCs, each lasting 4–5 s.

**MRI.** The participants were placed in a supine position in a 3T whole body scanner with the shank in a quad head coil (Signa Excite HDx, General Electric Company). The knee was extended, the leg supported with padding, and the ankle was stabilized in ~20° of plantar flexion. Images were acquired from each leg separately using serial T1-weighted spin-echo sequences. Initially, scout images of the leg were acquired in the coronal and sagittal planes. After the scout images, about 40–50 axial slices was aligned perpendicular to the tibial shaft in the coronal and sagittal planes between the knee and ankle joints. The axial images were acquired in an interleaved manner using the following parameters; echo time 18 ms, repetition time 850 ms, matrix 256 × 256, field of view 200 mm, slice thickness 10 mm, interslice gap 0 mm, number of acquisitions 2. Our results in the stroke subjects were compared with the ample age-matched control data reported for this muscle group from our laboratory (McNeil, Vandervoort, & Rice, 2007) and others (Kent-Braun & Ng, 1999).

**Data Analysis**

**MVC.** The peak MVC torque was determined for each trial. The TA root-mean-square EMG over a 500 ms period was determined just before the interpolated twitch, and was normalized to the Mmax (i.e., RMS EMG/peak-to-peak amplitude of the M-wave). The interpolated torque was equal to the torque at the onset of the stimulus minus the evoked peak torque. Activation percentage was equal to the following: \[1 - (T_s/T_r)] \times 100\%,\] where \(T_s\) was the interpolated torque evoked by the stimulus during the MVC, and \(T_r\) was the torque of twitch after the MVC.

**Antagonist Coactivation Percentage.** The root-mean-square EMG of the soleus, LG, and MG over 500 ms of the dorsiflexor MVC was expressed as a percentage of the muscle’s maximum EMG recorded during the plantar flexor MVC.

**Dorsiflexor Volume.** The MR images were converted to TIFF files from their original DICOM format. All images were analyzed by one investigator using available software (ImageJ, ver 1.38, National Institutes of Health, Bethesda, MD). The border of the dorsiflexor muscle group that included the tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius,
was manually traced for all axial slices from muscle origin to insertion and the corresponding CSAs determined. Intramuscular fat in each image was subtracted from the muscle CSA with a user-defined threshold. Dorsiflexor volume was calculated as the sum of fat-free CSA area times slice thickness for all slices of the muscle group. Specific torque was calculated as maximal 50 Hz torque divided by dorsiflexor volume.

**Statistics.** Paired and unpaired *t* tests were used to compare side-to-side differences within a group and between groups, respectively, for all measures except voluntary activation percentage, for which the Mann-Whitney *U* test was used. The relationship between measures was determined with Pearson’s product-moment correlation coefficients. Data are presented as means ± SD, and differences were considered significant when *p* < .05.

**Results**

**MVC Torque**

Representative MVC recordings in two stroke subjects are shown in Figure 1. In one, peak MVC torque was similar in both legs, although contralesional torque declined slightly toward the end of the contraction (Figure 1A, uppermost two

![Figure 1](image_url) — Ipsilesional and contralesional dorsiflexor MVC torque (uppermost two traces) and raw EMG of the TA, soleus, LG and MG in two stroke subjects (contralesional EMG is the lower trace of each pair). (A) In this subject (no. 2), peak MVC torque is similar between sides (B) In this subject (no. 4), MVC torque was much less in the contralesional than the ipsilesional limb. Note the interpolated twitch and sparse TA EMG in the contralesional limb.
traces). In addition, there was no obvious interpolated twitch in either leg. In the
other subject, peak contralesional MVC torque was 30% of the ipsilesional side
(Figure 1B). Reduced contralesional activation was apparent, shown by the inter-
polated twitch and sparse TA EMG. In six of the seven subjects, the MVC torque
was less in the contralesional than ipsilesional limb, and the mean values were
different ($p = .04$, Figure 2, Table 2).

### Table 2 Dorsiflexor Properties in Stroke and Control Subjects

<table>
<thead>
<tr>
<th>Measure</th>
<th>Contralesional</th>
<th>Ipsilesional</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>TA Mmax (mV)</td>
<td>7.8 ± 1.2</td>
<td>8.0 ± 2.8</td>
<td>7.4 ± 2.6</td>
</tr>
<tr>
<td>MVC (Nm)</td>
<td>29.8 ± 21.3*</td>
<td>42.5 ± 12.0</td>
<td>49.7 ± 7.1</td>
</tr>
<tr>
<td>MVC EMG</td>
<td>0.043 ± 0.028*</td>
<td>0.118 ± 0.045</td>
<td>0.09 ± 0.01</td>
</tr>
<tr>
<td>MVC activation (%)</td>
<td>78.1 ± 37.8</td>
<td>98.7 ± 1.0</td>
<td>98.2 ± 1.5</td>
</tr>
<tr>
<td>Soleus coactivation (%)</td>
<td>35.4 ± 37.6</td>
<td>16.3 ± 3.9</td>
<td>19.1 ± 13.2</td>
</tr>
<tr>
<td>Gastrocnemii coactivation (%)</td>
<td>25.8 ± 26.6</td>
<td>10.1 ± 8.1</td>
<td>18.0 ± 6.1</td>
</tr>
<tr>
<td>50 Hz torque (Nm)</td>
<td>32.3 ± 12.6</td>
<td>30.4 ± 10.5</td>
<td>35.7 ± 6.6</td>
</tr>
<tr>
<td>Dorsiflexor volume (cm³)</td>
<td>260 ± 69</td>
<td>250 ± 82</td>
<td>_</td>
</tr>
<tr>
<td>Dorsiflexor CSA (cm²)</td>
<td>13.2 ± 3.0</td>
<td>12.9 ± 4.1</td>
<td>_</td>
</tr>
<tr>
<td>50Hz/volume (Nm/cm³)</td>
<td>0.12 ± 0.03</td>
<td>0.12 ± 0.02</td>
<td>_</td>
</tr>
</tbody>
</table>

Note the MVC EMG values were normalized to the TA Mmax. *Significantly different compared with the ipsilesional limb and control limb.
MVC EMG Amplitude and Activation Percentage.

The TA Mmax was not significantly different between the contralesional and ipsilesional limb ($p = .70$, Table 2). The MVC root-mean-square EMG, normalized to the Mmax, was less in the contralesional than ipsilesional limb in all subjects, and the mean values were different ($p = .01$, Figure 2, Table 2). Activation percentage via twitch interpolation was less in the contralesional than ipsilesional limb, but the mean values were not different ($p = .2$, Table 2). Those with greater weakness tended to have larger activation deficits. Hence, relative contralesional MVC (% ipsilesional side) was associated with relative EMG ($r^2 = .71$, $p = .01$) and activation percentage ($r^2 = .77$, $p = .01$). Because subject 7 could not generate any voluntary dorsiflexor torque, a value of zero was assigned for each of his contralesional MVC torque, EMG amplitude, and activation scores and these zero values were included in the means presented in Table 2. The reported statistically significant differences between the contralesional and ipsilesional means for MVC torque, EMG amplitude, and percent activation were unchanged when data were reanalyzed with subject 7 excluded ($N = 6$); $34.8 \pm 18.3$ vs. $43.3 \pm 13$ vs. Nm ($p < .05$), $0.050 \pm 0.022$ vs. $0.120 \pm 0.046$ ($p < .05$), and $91.1 \pm 17.2$ vs. $98.9 \pm 1.0$ ($p = .30$), respectively.

Antagonist Coactivation Percentage

The mean antagonist EMG amplitude (mV) was less in the contralesional than the ipsilesional leg in the soleus ($0.015 \pm 0.005$ vs. $0.27 \pm 0.004$, $p = .03$) and gastrocnemii ($0.013 \pm 0.012$ vs. $0.023 \pm 0.016$, $p = .005$). The soleus coactivation percentages ranged from 10.6% to 110.7% and 11.4–21.7% in the contralesional and ipsilesional limb, respectively. The corresponding values for the gastrocnemii were 6.8–70.3% and 4.3–26.4%. The mean soleus and gastrocnemii coactivation percentages were larger in the contralesional than ipsilesional limb, but the differences were not significant ($p = .2$, Table 2). The larger contralesional means were mainly due to the values recorded in subject 6 (110% and 70% in the soleus and gastrocnemii, respectively). The augmented coactivation percentage in this subject reflected a larger absolute EMG coactivation (mV) in the gastrocnemii, but not the soleus, combined with a dramatically smaller absolute MVC EMG of both muscles; 12% and 5% of the ipsilesional side, respectively. Similar to the findings of others (Clark, et al., 2006; Lum, et al., 2004; Newham & Hsiao, 2001; Wagner, et al., 2007), our results reveal little evidence of excessive coactivation in the contralesional limb.

50 Hz Torque, Muscle Volume, and Specific Torque

The 50 Hz torque recordings of two subjects are shown in Figure 3 and demonstrate that the evoked torques were similar between sides. In all subjects except one (no. 4), contralesional 50Hz torque was equal to or larger than the ipsilesional value, and the means were not different ($p = .3$, Table 2). The lack of a difference in mean 50 Hz torque generating capacity between limbs supports the findings of a previous study (Landau & Sahrmann, 2002), but, unlike their large side-to-side 50 Hz differences in individual subjects (up to 150%), differences in the present subjects were $\leq 25\%$ (see discussion). The total dorsiflexor volume (cm$^3$), fat volume (cm$^3$), and percentage of total volume that was fat, was not significantly different.
between the contralesional and ipsilesional limbs; 264.8 ± 69.0, 4.6 ± 2.3, 1.9 ± 1.1% vs 255.5 ± 81.2, 5.6 ± 2.5, 2.5 ± 1.7% (p = .4, p = .2, p = .1, respectively). Contralesional fat-free volume (and CSA) was similar to or larger than ipsilesional volume across subjects (Figure 2), and the means were not different (p = .4, Table 2). The differences in the 50 Hz torque/volume ratio between limbs were not different (p = .9, Table 2).

Comparisons to Control Subjects

The MVC torque (p = .04) and MVC EMG (p = .003) were significantly less in the contralesional limb compared with the right limb of the 7 controls, but TA Mmax, activation, coactivation, and 50 Hz torque did not differ significantly (Table 2). There were no significant differences between ipsilesional and control means for any parameter (Table 2). Right-left differences in mean TA Mmax, control MVC torque, MVC EMG, and 50 Hz torque were modest (<16%) and not significant (p > .05).

Discussion

In this study, we assessed both central and peripheral determinants of weakness in chronic stroke subjects. Our findings indicate that most of the weakness reflects a reduction in central neural drive to the motoneuron pool, with little contribution

Figure 3 — Evoked dorsiflexor torque and EMG during supramaximal 50 Hz stimulation in subject 2 (A) and subject 7 (B). For each subject, the torque and EMG are shown for the contralesional limb (dotted line and lower EMG trace) and ipsilesional limb (continuous line and upper EMG trace).
from antagonist muscle activity. The idea among some clinicians that atrophy is commonplace after stroke is not supported by the present results.

Because muscles receive some of their innervation from the ipsilateral motor cortex, the ipsilesional side may show impairments in activation and weakness following stroke (Adams, et al., 1990; Newham & Hsiao, 2001). However, in the current study, MVC and 50 Hz torques did not differ between the ipsilesional and control limbs (Table 2). Although the controls were younger on average than the stroke subjects, previous work showed little age-related differences in dorsiflexor properties between 30 and 70 y of age. Hence, mean ipsilesional MVC, 50 Hz torque, and dorsiflexor CSA of the five men with stroke (47 Nm, 34 Nm, and 14.2 cm²) are similar to the means reported for older (65 y) healthy men (44 Nm, 30 Nm and 14.3 cm², 65y) (Kent-Braun & Ng, 1999; McNeil, et al., 2007; van Schaik, et al., 1994). The preserved ipsilesional dorsiflexor motor function may reflect overuse of this limb to compensate for contralesional impairments (Olney, Griffin, Monga, & McBride, 1991).

Contralesional weakness was evident in six of the seven subjects and was associated with deficits in EMG amplitude and activation percentage. The recorded weakness, particularly evident in subjects 4, 6, and 7, may be partially attributed to a diminished capacity to recruit and fully activate the dorsiflexor motoneuron pool. Indeed, one study of stroke subjects found that the motor unit firing rates of the contralesional TA did not rise above 15 Hz during an MVC (Frontera, et al., 1997). This level of motor unit firing is less than half of the normal rate and would be expected to generate smaller peak forces. Others have reported contralesional dorsiflexor weakness (Adams, et al., 1990; Eng, Lomaglio, & Macintyre, 2009; Landau & Sahrmann, 2002) and reduced EMG amplitude (Frontera, et al., 1997; Jakobsson, et al., 1991; Knorr, et al., 2011), but the contribution of changes in whole muscle morphology was not evaluated.

Our results indicate that reduced central neural drive is the primary determinant of contralesional dorsiflexor weakness in these subjects because of a lack of atrophy or any contractile impairment combined with their deficits in activation and EMG amplitude. However, the percent contribution of reduced neural drive to weakness may be underestimated when based on twitch interpolation data and overestimated when based on the EMG amplitude results (Figure 2) (Klein, et al., 2010). Although twitch interpolation is considered to be one of the better methods to assess voluntary activation, it is not without limitations. The sensitivity of the method, or the ability to detect small deficits in dorsiflexor activation, is reduced at higher (> 80% MVC) torques, possibly because the fibular (peroneal) muscles can act as antagonists when activated during electrical stimulation (Belanger & McComas, 1981). In healthy adults, the relationship between relative (%MVC) EMG amplitude and dorsiflexor torque is curvilinear (Garland, Garner, & McComas, 1988). Hence, at a contraction strength equal to 50% MVC torque, the corresponding EMG is 30% of the MVC EMG. This difference between relative EMG and relative torque may widen after stroke because of a reduction in maximal motor unit firing rate in the contralesional limb (Frontera, et al., 1997), and could explain the relatively larger deficits in contralesional EMG than MVC torque (Figure 2).

Dorsiflexor atrophy was not evident in the present subjects and supports the concept of little to no loss in dorsiflexor mass after stroke. Our results confirm previous studies of chronic stroke subjects that found no evidence of TA atrophy
based on the determination of muscle volume derived from magnetic resonance images (Ramsay, et al., 2011) and fiber area of biopsies (Jakobsson, et al., 1991). In one study, dorsiflexor CSA, derived from computer tomography images, was about 12% smaller in the contralesional than the ipsilesional limb (Odajima, et al., 1987). Another reported evidence of contralesional TA fiber atrophy in some stroke subjects, but they may have been less mobile than the present stroke subjects since they were to undergo corrective foot surgery (split anterior tibial tendon transfer) to improve ambulation (Slager, et al., 1985).

The lack of atrophy and contractile impairment in the present subjects is evident despite deficits in EMG and activation (i.e., no. 4 and 7). Subject 7 was unable to generate any EMG or torque during the MVC, and his TA activity during gait was also found to be negligible. Immediately after the torque recordings of the contralesional and ipsilesional limbs, we had subject 7 walk back and forth a distance of 5 m in the laboratory at his preferred speed while recording the associated EMG activity using the same electrodes (data not shown). During this 5 m walk, his contralesional mean TA EMG amplitude at heel strike was found to be about 5% of the ipsilesional TA MVC EMG amplitude; much less than typically recorded in healthy controls (Jakobsson, et al., 1991). Our whole muscle results support and extend the findings of a previous study in which there was no significant contralesional TA fiber atrophy, even though the TA EMG amplitude during walking was less than 10% of adult controls (Jakobsson, et al., 1991). Together, these observations suggest that recruitment of a large portion of the dorsiflexors is not a necessary precondition for maintenance of muscle mass and force generating capacity following stroke. Rather, factors independent of muscle activation, including passive mechanical tension or neurotrophic support may play important roles (McComas, Sica, Upton, & Aguilera, 1973; Yucesoy & Huijing, 2007).

In subject 6, hypertrophy was apparent in the contralesional dorsiflexors (+28%) and the peronei (+42%) but with atrophy of the gastrocnemii (-50%) (Klein, et al., 2010), indicating that responses may be muscle-dependent in some cases (Odajima, et al., 1987; Ploutz-Snyder, Clark, Logan & Turk 2006; Prado-Medeiros et al., 2012). Scattered fiber hypertrophy has been noted in the TA and other muscles after stroke (Jakobsson, et al., 1991; Slager, et al., 1985). Interesting, in some individuals with anterior cruciate ligament injury, TA volume was also unexpectedly larger in the injured limb compared with the noninjured limb (Binder-Macleod & Buchanan, 2006). Like these types of injuries, peculiar changes in gait mechanics following stroke (Perry, Waters, & Perrin, 1978) may lead to stretch-induced hypertrophy of some muscles and disuse atrophy of others. In subject 6, the equinovarus foot position during gait may have resulted in stretch-induced hypertrophy of the TA and peronei.

We explored intrinsic contractile function by comparing side-to-side 50 Hz torque differences using maximal stimulation of the common fibular nerve and which could be related to muscle quantity. In one previous study of chronic stroke, contralesional 50Hz torque, tested by using pad stimulation over only the TA, was found to differ widely (~20–150% of the ipsilesional value in one-half of the subjects), which was not easy to explain (Landau & Sahrmann, 2002). Compared with their data (Landau & Sahrmann, 2002) we found that the side-to-side 50 Hz torque differences were more modest in the stroke subjects (≤ 25%) and controls (≤ 9%). The design to isolate the current field to the TA in the prior study (Landau...
& Sahrmann, 2002) may not have accounted for variable activation spread to other local dorsiflexors and plantar flexors (peronei). Supramaximal nerve stimulation, as applied in the current study, is likely a more precise method for assessing limb differences in torque generating capacity because all dorsiflexor and peronei muscles are maximally activated.

**Study Limitations**

With a limited sample size and inclusion of only persons with chronic neurological impairments who are mobile and physically active, these findings are not necessarily representative of the wider stroke population. Dorsiflexor atrophy and weakness would be expected to be greater in those who are less mobile and inactive (Eng, et al., 2009; Odajima, et al., 1987). In addition, weakness and antagonist coactivation may be more pronounced when measured during dynamic contractions as opposed to isometric conditions employed here (Lum, et al., 2004).

**Conclusions**

Despite weakness and reduced EMG amplitude, muscle volume, 50 Hz torque, and specific torque were well preserved in the stroke subjects. Our results suggest that much of the weakness can be attributed to a loss of central neural drive to the agonist motoneuron pool. The preservation of contralesional dorsiflexor muscle properties may be explained in part by their relatively preserved mobility and regular physical activity.

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**References**


