Neurorehabil Neural Repair. Author manuscript; available in PMC 2011 September 27.

Published in final edited form as:
Neurorehabil Neural Repair. 2010 January ; 24(1): 78–87. doi:10.1177/1545968309345269.

Greater activation of secondary motor areas is related to less arm use after stroke

Kristen J Kokotilo, MSc[1,2], **Janice J Eng, PhD**[1,2,3], **Lara A Boyd, PhD**[1,3], and **Martin J McKeown, MD**[1,4,5]

[1]Graduate Program in Neuroscience, University of British Columbia, Vancouver, Canada

[2]Rehabilitation Research Lab, GF Strong Rehab Centre, Vancouver, Canada

[3]Department of Physical Therapy, Faculty of Medicine, University of British Columbia, Vancouver, Canada

[4]Division of Neurology/Faculty of Medicine, University of British Columbia

[5]Pacific Parkinson's Research Centre, Brain Research Centre, University of British Columbia Vancouver, BC, Canada

Abstract

Background—Past studies have identified reorganization of brain activity in relation to motor outcome through standardized laboratory measures, which are quantifiable surrogates for arm use in real-life. In contrast, accelerometers can provide a real-life estimate of arm and hand usage.

Methods—Ten persons with chronic, subcortical stroke and ten healthy controls of similar age performed a squeeze motor task at 40% maximum voluntary contraction during fMRI. Use of the upper extremity was quantified over 3 consecutive days using wrist accelerometers. Correlations were performed between arm use and peak percent signal change (PSC) during grasp force production in six regions of interest (ROIs): bilateral primary motor cortex (M1), supplementary motor area (SMA) and premotor cortex (PM).

Results—Results demonstrate that in healthy controls, PSC across all ROIs did not show a relationship between arm use and brain activation during force production. In contrast, after stroke, contralesional PM and M1 showed a significant ($P \leq 0.05$) correlation between increasing activation and decreasing paretic arm use, while ipsilesional PM showed a significant correlation ($P \leq 0.05$) between increasing activation and decreasing non-paretic arm use.

Conclusions—The results of this pilot study demonstrate a negative relationship between brain activation and actual arm use after stroke. Larger studies using accelerometers that can detect amount and types of movement may offer further insight into brain reorganization and rehabilitation interventions.

Keywords

fMRI; imaging; rehabilitation; hand; cerebrovascular disease; accelerometer

Corresponding Author: Janice J Eng; Address: Department of Physical Therapy, University of British Columbia, 212-2177 Wesbrook Mall, Vancouver, BC, V6T 1Z3; Phone: 604-714-4105, Janice.Eng@ubc.ca.

INTRODUCTION

Functional reorganization of the central nervous system is thought to be one of the fundamental mechanisms involved in recovery after neurological injury, such as stroke. Past animal studies have established that structural plasticity can occur in the damaged cortex and connected brain areas, and that functional recovery is associated with this plasticity 1–3. Similarly, neuroimaging studies in humans have demonstrated that reorganization of brain activation relates to functional outcome after stroke 4–6. More specifically, a systematic review of 26 studies on force production in stroke 7 showed that persons with stroke are more likely to activate motor areas when using their paretic limb, such as the ipsilesional M1, PM, SMA, parietal cortex and cerebellum. This activation is higher when function is lower as indexed by the Barthel activities of daily living (ADL) index, Action Research Arm Test (ARAT) and grip strength 4. In addition, altered activation of the undamaged hemisphere has been described where increased activation of the contralesional cortical motor areas occur during paretic hand tasks compared to healthy controls, and this activation is greatest in the more impaired patients 4, 8, 9. However, conflicting results have been reported in the literature for changes in contralesional activation post-stroke7 and these variations (increased versus no change in contralesional activation) may arise from the varying type and chronicity of stroke, in addition to the task, imaging technique and analysis. Lastly, brain activation may also be increased when using the non-paretic arm in the ipsilesional hemisphere 10 as well as contralesional hemisphere 11, although cortical connectivity (using dynamic causal modeling) has been found to be similar to controls 12. Neuroimaging studies have also examined brain reorganization in relation to rehabilitative interventions and outcome measures of paretic arm use. After implementation of constraint-induced movement therapy (CIMT), persons with chronic stroke can exhibit cortical reorganization which is accompanied by increased use of the affected arm as measured by the motor activity log (MAL) 13,14 (semi-structured interview which assesses the individual’s perception of how much and how well they use their paretic arm during ADL).

Although these past studies have provided valuable information on the relationship between recovery and reorganization of brain function after stroke, the functional outcome measures evaluated in a laboratory setting or the tasks used during brain imaging protocols are a surrogate for actual arm activity in real life. Yet, measuring the level of arm use in real life is important, as one might expect that if the paretic arm is utilized less over time, activation patterns of the brain should reflect lower levels of arm use. The use of accelerometers is a relatively novel method of monitoring arm and hand usage in daily activities which may assess actual upper limb activity outside the laboratory. Contrary to specific outcome measures of hand function, accelerometers can provide a real life estimate of the quantity and intensity of arm and hand usage.

We hypothesized that a relationship would exist between paretic arm use as measured by accelerometry and brain activity during functional magnetic resonance imaging (fMRI) when performing an activation task with the paretic hand for both the ipsilesional and contralesional hemispheres. We selected a grasping task performed at 40% maximal voluntary contraction (MVC) because this is representative of forces used in many ADL 15.

We expected that individuals who use their paretic arm less would exhibit greater activation of secondary motor areas not affected directly by the brain lesion (e.g., SMA and PM of the ipsilesional side or motor areas of the contralesional side). We also hypothesized that non-paretic arm use might be associated with greater brain activity. We hypothesized that no relationship would exist in the control participants between arm use and brain activation.

METHODS

Participants

Participants with hemiparesis following a subcortical stroke were recruited from our local hospital database. Inclusion criteria specified that participants were greater than 6 months post-stroke, had some degree of impairment (<66 on Fugl-Meyer)16, were free from musculoskeletal and other neurological conditions, were able to use a squeeze grip and able to follow instructions in English. Healthy individuals of similar age with no known neurological or serious musculoskeletal conditions were recruited from local community centres. All participants were required to be right hand dominant (prior to stroke for stroke participants) according to the Edinburgh Handedness Inventory 17. Approval was obtained from the local university and hospital ethics committees and all participants provided an informed consent. For stroke participants, motor recovery of the paretic upper extremity was assessed with the upper extremity component of the Fugl-Meyer Motor Impairment Scale 16,18.

Accelerometer Protocol

Activity level of the upper extremities of all participants was quantified using accelerometers (Actical™, Mini Mitter Co, Bend, OR). The accelerometers used in the present work were small (28×27×10 mm), light (17g), waterproof and had a frequency range of 0.3–3 Hz, sensitive to 0.05–2.0 g-force and sampled at 32 Hz. Data was stored as activity counts every 15 seconds and the accelerometer detected motion in all 3 planes.

Two wireless watch-like accelerometers were worn, one on each wrist of participants during all waking hours for 3 consecutive days. The mean total activity counts per day over the 3 consecutive days for both the paretic and non-paretic arms of stroke participants and left arm of control participants were used as the measure of upper extremity activity. Past literature has established the test-retest reliability of accelerometers for measurement of upper limb activity in stroke patients (r>0.86) and a period of 3 consecutive days has been demonstrated to be a valid index of arm activity during normal day-to-day activities (r=0.74 with the Motor Activity Log)19, 20.

fMRI Protocol

A custom-built, MR-compatible rubber squeeze-bulb connected to a pressure transducer was used to index motor related brain activity during fMRI. Participants lay supine and were positioned with their elbow flexed at 90° and forearm in a resting position on their stomach with their hand pronated, gripping the squeeze-bulb. The MVC of each subject was measured prior to fMRI and all subsequent movements were scaled to MVC. Before the scanning session, all participants were trained on the motor task to ensure that they were

familiar with the task requirements. EMG measurements were taken during this practice session to ensure mirror movements did not occur during fMRI task performance. During fMRI scanning, participants viewed a computer screen via a back projection-mirror system. Each trial began with the appearance of a vertical bar on the display to cue movement and to illustrate the force to be exerted. Using the squeeze bulb, participants squeezed until the pressure level matched the target level and then released when the target disappeared, allowing the pressure level to return to baseline; visual feedback was given when participants overshot or undershot the target bar. Three target force levels (10%, 40% and 70% of MVC) were performed; the force generated varied across trials in a pseudo-random order. The three forces were utilized as part of another study investigating the modulation of force post-stroke, however, the 40% value was selected a priori for analysis for this study as a large majority of everyday tasks occur in this force range 15. An event related design was used, where a total of 24 trials, each 4s in length occurred in each run, and each trial was followed by a jittered inter-trial interval lasting between 10s – 16s 21. To allow the hemodynamic response to return to baseline, blank trials constituted 60% of all repetition times (TRs). The experiment was conducted in a total of 4 runs, each approximately 6.8 minutes long; the hand required to squeeze the bulb (paretic or non-paretic for stroke participants, dominant or non-dominant for healthy controls) was alternated between each run and the non-paretic hand (for stroke participants) or dominant (for healthy controls) was tested first. Although both hands were tested for healthy control participants, only the non-dominant arm was used for analysis because this arm corresponded to the paretic arm in the majority (7/10) of stroke participants.

fMRI Data acquisition

A Philips Gyroscan Intera 3.0 T scanner (Philips, Best, the Netherlands), equipped with a head-coil, was used to acquire both T1-weighted anatomical images (170 axial slices) and T2*-weighted echo-planar (EPI) images (matrix size = 128 × 128, pixel size = 1.9 × 1.9 mm, TR=2000 ms, TE = 3.7) with blood oxygenation level-dependent (BOLD) contrast. Each functional run lasted 6.8 minutes. Thirty-six axial slices of 3 mm thickness were collected in each volume, with a gap thickness of 1mm. A total of 206 volumes were acquired continuously during each run.

Behavioral data analysis

Custom Matlab software (Mathworks) and the Psychtoolbox 22 were used to design and present visual stimuli (target force), and to collect behavioral data (pressure) from the response devices.

fMRI data analysis

The functional MRI data were pre-processed for each subject using AFNI software package23. fMRI data were 2D and 3D motion corrected and the skull was stripped from the structural image. Anyone having an average across runs of maximum displacement of 4 mm or over was excluded 24. Functional data from all runs was concatenated and the skull stripped structural image was aligned to the concatenated functional data. A Deconvolution Analysis of functional data was then performed in AFNI where the impulse-response functions were estimated for each of six conditions based on the input stimulus functions

and the observed fMRI time series data. The impulse-response functions were then convolved with the stimulus functions to yield the estimated response. Baseline coefficients for each run and regression coefficients for the first 6 TRs of each condition were calculated. Percent signal change (PSC) was calculated over 6 TR intervals by averaging the baseline constants from each run to create a single baseline constant and then dividing this constant by the regression coefficients for each of the first 6 TRs for each condition. The use of signal change of intensity as a measure of activation has been found to be consistently more reliable than voxel counting in healthy participants 25 and more reliable than both voxel counting, and voxel by voxel analysis in participants with stroke 26.

All images were smoothed with a Gaussian filter with a root mean square deviation of 4 mm. Six specific regions of interest (ROIs) (3 bilaterally) were manually drawn on each aligned structural scan using Amira software. The following ROIs were drawn separately in each hemisphere, guided by a neurological atlas 27 primary motor cortex (M1), supplementary motor cortex (SMA), premotor cortex (PM). We chose M1, SMA and PM, as these three regions all activate during numerous motor tasks, including during individual finger movements and when opening and closing the whole hand 28 and thus, are most likely to be active during daily motor tasks

Structural landmarks were used to trace the ROIs in the cortex in individual participants 29. The area of M1 was defined as extending from the anterior bank of the central sulcus to the anterior edge of the precentral gyrus 26, 29, 30. PM was defined as the area between M1 and the sulcus nearest the coronal plane through the anterior commissure, bounded inferiorly by the inferior edge of the frontal lobe 29. The entire PM was drawn for our analysis, as we did not have an a priori hypothesis regarding differences in activation between dorsal and ventral regions. SMA was defined as the medial region of the hemispheres superior to the dorsal bank of cingulate sulcus along the same anterior-posterior extent as PM 29–31. Within each of these ROIs, the maximum value of PSC across the 6 TRs was used as peak PSC for each condition.

Statistical Analysis

The distribution of the accelerometer data was assessed and deemed to be not significantly different from a Gaussian (Shapiro-Wilks test for normality). Paired t-tests were used to determine differences between amount of arm use in: the paretic vs. non-paretic arms of stroke participants, independent t-tests determined differences between amount of arm use of the paretic arm of stroke participants vs. non-dominant arm of healthy controls and the non-paretic arm of stroke participants vs. the non-dominant arm of healthy controls. In addition, paired t-tests were used to determine differences between PSC at 40% MVC for paretic versus non-paretic respective brain structures of the stroke group while independent t-tests were used between control versus stroke respective brain structures.

We performed a correlational analysis to determine whether a relationship existed between task-related changes in activation of motor areas of the brain and activity of the upper extremity as assessed using wrist accelerometers. For all correlational analyses, we selected the force of 40% MVC to use, as this condition represented a force indicative of functional daily tasks 15. In stroke participants, and for each ROI, we calculated Spearman correlations

between activity of the paretic arm and peak PSC as well as between activity of the non-paretic arm and peak PSC. For healthy controls, and for each ROI, correlations were calculated between activity of the left (non-dominant arm) and peak PSC.

RESULTS

Clinical Data

A total of 17 control participants and 14 participants with stroke responded to our recruitment advertisements, met initial eligibility and were scheduled for further evaluation. However, of the controls, three were removed due to non-compliance with the study (e.g, refused to wear the accelerometers, changed their mind about participating), and one was revealed to have Restless Legs Syndrome. In addition, three control participants were removed after analysis of the fMRI and forces because of excessive head motion (n=1), artifacts from dental implants (n=1), and inappropriate forces being applied during the task (n=1). Of the stroke participants, two were removed due to non-compliance with the study. An additional two stroke participants were removed after analysis because of excessive head motion (n=1) and identification of cortical, rather than sub-cortical lesion (n=1). Characteristics of all stroke participants are listed in Table 1 (Age: mean 62.3 years; std dev 8.9; range 48–80) and they had a similar age to the controls (Age: mean 63.8 years; std dev 8.5; range 50–78).

Seven stroke participants had experienced left hemiparesis and three right hemiparesis. The site of stroke was determined from the T1-weighted structural MRI. All patients had subcortical stroke encompassing basal ganglia structures or subcortical white matter (Figure 1). No cortical or cerebellar strokes were included. Nine of the participants with stroke were mild to moderate stroke (FM range 43–64, mean 57), and the tenth subject was severely impaired (FM 15).

Accelerometer Data

The mean (SD) activity kilocounts per day for both the stroke and control participants appear in Table 2. The activity counts for the paretic hand were significantly ($P<0.05$) lower compared to the non-paretic hand of stroke participants and both hands of healthy controls. The paretic hand activity counts were less than half the values of control participant values. No significant differences were found between activity counts for the non-paretic hand of stroke participants and either hand of healthy controls.

Imaging Data

Brain activation during Relative Force Production—Table 3 displays brain activation, as peak PSC, at 40% MVC force production with the non-dominant hand of control participants compared to the paretic and non-paretic hands of stroke participants. No significant differences were found for peak PSC during force production between the non-dominant hand of healthy control participants and either hand of stroke participants (p-value of 0.2 to 0.9). A trend was found for less activation of the ipsilesional M1 during the non-paretic arm task compared to the same structure during the paretic arm task (p=.070) or control value (0.079).

Brain activation during Relative Force Production versus arm use—In healthy controls, no significant correlations were found between task-related peak PSC of motor regions of either hemisphere and total activity counts of the non-dominant upper limb (Table 4). For the paretic upper limb of stroke participants, no correlation was found between task-related peak PSC in the ipsilesional M1 and total activity counts. However, the correlation analysis did identify several contralesional motor regions in which there was a correlation between brain activation and paretic arm activity (Table 4). Significant negative correlations were found in contralesional M1 ($R = -.721$, $p = 0.019$) and PM ($R = -.648$, $p = 0.043$) and a trend for contralesional SMA ($R=-.624$, $p=0.054$) (Figure 2). Thus, increased activation occurred in motor regions of the hemisphere not affected by the lesion in participants with less paretic arm use. For the non-paretic upper limb of stroke participants, a significant correlation was also found between task-related PSC in ipsilesional PM and total activity counts ($R = -.685$, $p = 0.029$) and SMA ($R = -.661$, $p = .038$) (Figure 3).

DISCUSSION

Despite varying levels of arm activity among individuals in the control group, no relationships were found between increased brain activation and decreasing arm activity in any of our ROIs (right or left hemisphere), with all p-values greater than 0.10. This may indicate that there is a minimal threshold of arm use, relative to motor capacity, that is required to influence brain activation. In healthy participants, day-to-day arm use most likely does not approach their potential activity capacity, and thus a relationship between arm use during daily activities and brain activation during force production would not be expected. In contrast, in stroke participants, movement of the paretic and non-paretic arms may approach their potential limit of activity, resulting in accelerometer data that is an actual measure of motor capacity. This data is then more likely to correlate with brain activation in secondary motor areas. In fact, the increased activation in secondary motor areas (other than ipsilesional M1) that we noted in our stroke participants appears to be indicative of a reorganization process of novel areas of recruitment occurring in the brain after stroke that is influenced by the amount of daily arm usage.

Our analyses of brain activation in relation to paretic arm use as measured by accelerometers demonstrated a lack of a relationship between PSC in ipsilesional M1 during relative force production. The basis for this finding could be a result of the lesion locations for our subset of participants. All participants had subcortical lesions, with the majority (n=6) affecting white matter tracts that may descend from M1. If the corticospinal tract descending from M1 is damaged, then this region will be less useful in producing force output, and therefore less likely to be activated. In contrast, contralesional areas are not damaged and thus may be relied on during performance of a motor task 32. Alternatively, Calautti et al.9 reported low or non-significant correlations between ipsilesional M1 activation and finger tapping and suggested that abnormal inhibitory drive from contralesional M1 onto the ipsilesional M1 could impair recovery and confound this relationship.

Past literature has demonstrated increased activation occurs in several motor areas with increasing severity of stroke or decreasing function after stroke during performance of a motor task with the paretic hand. Specifically, chronic stroke participants having greater

corticospinal tract damage showed increased activation in motor areas including bilateral M1, bilateral PM, SMA, and prefrontal cortex (PFC) 33. With respect to measures of motor function, past work has found a negative relationship between outcome measures, such as Action Research Arm Test and grip strength, and brain activation in areas such as bilateral PM, dorsolateral PFC, cerebellum, ipsilesional SMA and insular cortex 4. As our subset of stroke participants were mildly impaired, most had near maximal scores on our measure of motor outcome, the Fugl-Meyer. Thus, we did not correlate our Fugl-Meyer scores to brain activation as the majority of stroke participants likely experienced a ceiling effect on the scale with their paretic arm, in addition to the fact that the scale cannot be used for the non-paretic arm or control participants. This may suggest that the use of accelerometers as an outcome measure of arm activity provides a superior way to discriminate the amount of arm use amongst mildly impaired stroke patients with a high degree of motor function that obtain maximal, or near maximal scores on measures of functional ability

Our results showed that some contralesional areas, such as M1 and PM, exhibited increased activation during force production with the paretic hand in stroke participants with less arm activity. The likely reasons for increased recruitment of these motor areas may be based on the similarity of corticospinal projections from these cortical motor areas. A number of motor networks acting in parallel may generate an output to the spinal cord in order to produce movement and such networks can be utilized post-injury 4, 34. If damage occurs in one network, greater recruitment of secondary motor areas can occur to compensate. Increased ipsilateral M1 activity has been demonstrated in healthy adults performing more challenging motor tasks 35, 36. As stroke participants with less paretic arm use likely found our motor task challenging to perform, they may require increased recruitment of the ipsilateral (contralesional) M1 to compensate. A more difficult task might also require increased attention for accurate performance, which is another possible explanation for recruitment of the PM, as this area is thought to process and integrate external visual, attentional, and other information in order to produce motor output 37. Finally, a growing body of literature points to contralesional PM activity as representing a functionally relevant, adaptive response to stroke-related brain damage 8, 38. Our finding is in agreement with other work 38 that has stressed the importance of contralesional PM in mediating an adaptive compensation process in the recovery from stroke.

Past literature examining effects of increased motor task difficulty on stroke patients compared to healthy controls has demonstrated that performance of a more difficult motor task with the paretic hand increases activation in contralesional M139 and PM40. However, as projections from secondary motor areas are less numerous and have an overall lower excitatory effect than those from the primary motor area 41, secondary motor area recruitment is often associated with poorer functional outcome 4, 33. Our novel results show that even mildly affected individuals with stroke who have low levels of hemiparetic arm use are likely to activate secondary motor areas. Conversely, it is possible that these participants have less paretic arm usage due to the reduced ability of secondary motor areas to generate functional movement. Unfortunately with our correlational analysis we cannot determine any single causation effect. It is possible that rather than spontaneous reorganization after stroke affecting the amount of functional paretic arm usage, instead increased or decreased arm use may induce brain reorganization. In this way, participants who use their paretic arm

less over time may produce less primary motor activation and rely more on secondary motor areas. If this is indeed the case, then increased paretic arm usage through rehabilitation techniques may be able to induce more beneficial reorganization of brain activation after stroke. In fact, some small pilot studies have shown that stroke patients with increased paretic arm use over a fixed time period due to rehabilitation training exhibit reorganization of brain activation 13, 14, 42.

In contrast to the relationship between activation and paretic arm use, the significant relationship between arm use and ipsilesional brain activation for the non-paretic arm was not simply due to altered non paretic arm use, as the magnitude of arm use was similar between the non-paretic arm of stroke participants and either arm of the control participants. However, we cannot rule out that the type of activity of the non-paretic arm may differ fundamentally from use of a normal arm due to compensation for the paretic arm. Alternatively, the relationship could be explained by altered interhemispheric inhibition of the ipsilesional cortex. Past research has shown that stimulation of the motor cortex in one hemisphere produces inhibition of the motor cortex in the other hemisphere 43, 44. Moreover, this inhibition is stronger when coming from the dominant motor cortex than the non-dominant motor cortex 45. As the majority of our stroke participants had dominant (right) non paretic hands, increasing use of the non paretic arm would increase activity of the dominant, contralesional hemisphere. Subsequently, this greater activity in the contralesional hemisphere would result in stronger interhemispheric inhibition of the ipsilesional cortex and could result in the relationship that we observed: decreased activation of the ipsilesional cortex in those participants with greater non-paretic arm use.

Limitations

One of the limitations of the study is that the use of accelerometers does not provide information on the specific tasks that the upper limb is performing, and is not able to differentiate between functional movements (i.e. eating) and non-functional movements (i.e. swing movements while walking) 46. On the other hand, non-functional movements may also be contributing to brain plasticity, as any movement would still require brain activation. An additional limitation is that the selected task (squeeze at 40% MVC) only represents one level of force and one common task used in daily activities.

It is also possible that error was introduced into our data analyses as a result of our ROI approach. Past work has shown variability in defining M1 boundaries that are largely a result of ambiguity in defining the anterior border of this region 26. However, we minimized potential error by having the same operator define each region and through a subsequent check of these boundaries by a second investigator. Further, ROI approaches take into account variations in individual anatomy and as such may actually be a more accurate representation of regional activation as compared to the use of a standard-space approach such as normalization in Talairach space 47.

Last, a small number of participants were included in our analysis and we were only powered to detect large effect sizes and could not perform any multi-variate analysis. In addition, we did not provide any multiple comparison correction for the 18 correlations, and

thus, there is a chance that one of these correlations will be significant by chance alone (given an alpha of 0.05).

In summary, the use of accelerometers revealed actual arm use after stroke and these data suggested relationships to brain activation that can be pursued. Future applications of accelerometers appear promising as a complementary outcome.

Acknowledgments

We would like to acknowledge funding from the Canadian Institute for Health Research (MSH-63617, CGR-86829) and the Michael Smith Foundation for Health Research (to JJE), The North Growth Fund, Vancouver Coastal Health Research Institute and Foundation, and Heart and Stroke Foundation of British Columbia (to LAB) and Natural Sciences and Engineering Research Council (trainee award to KJK).

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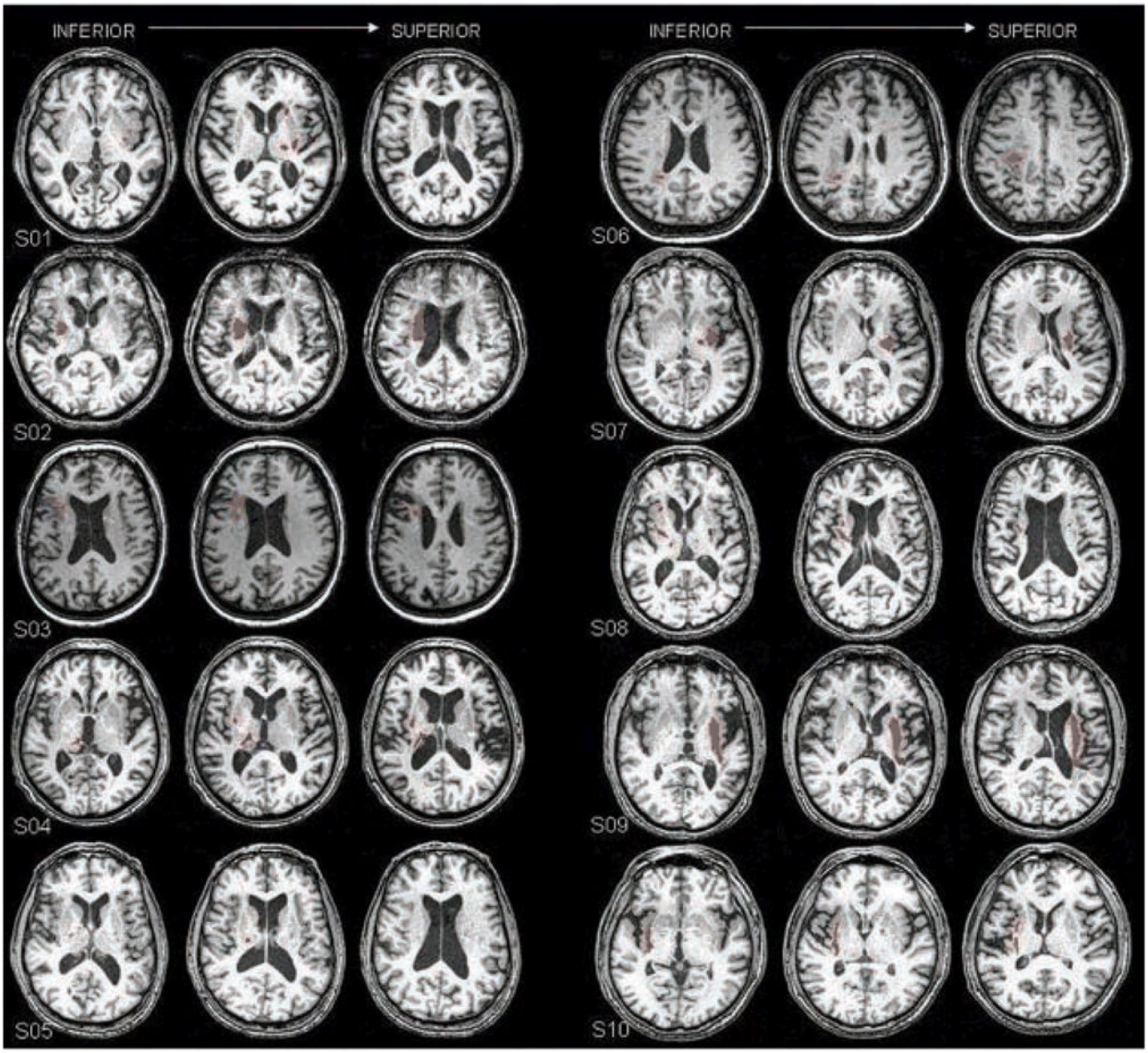

Figure 1.

Illustration of lesion location for each participant. Each individual's lesion location is denoted by three brain slices, 5mm apart. The middle image represents the center of each lesion. Images are in radiological space and presented from superior to inferior.

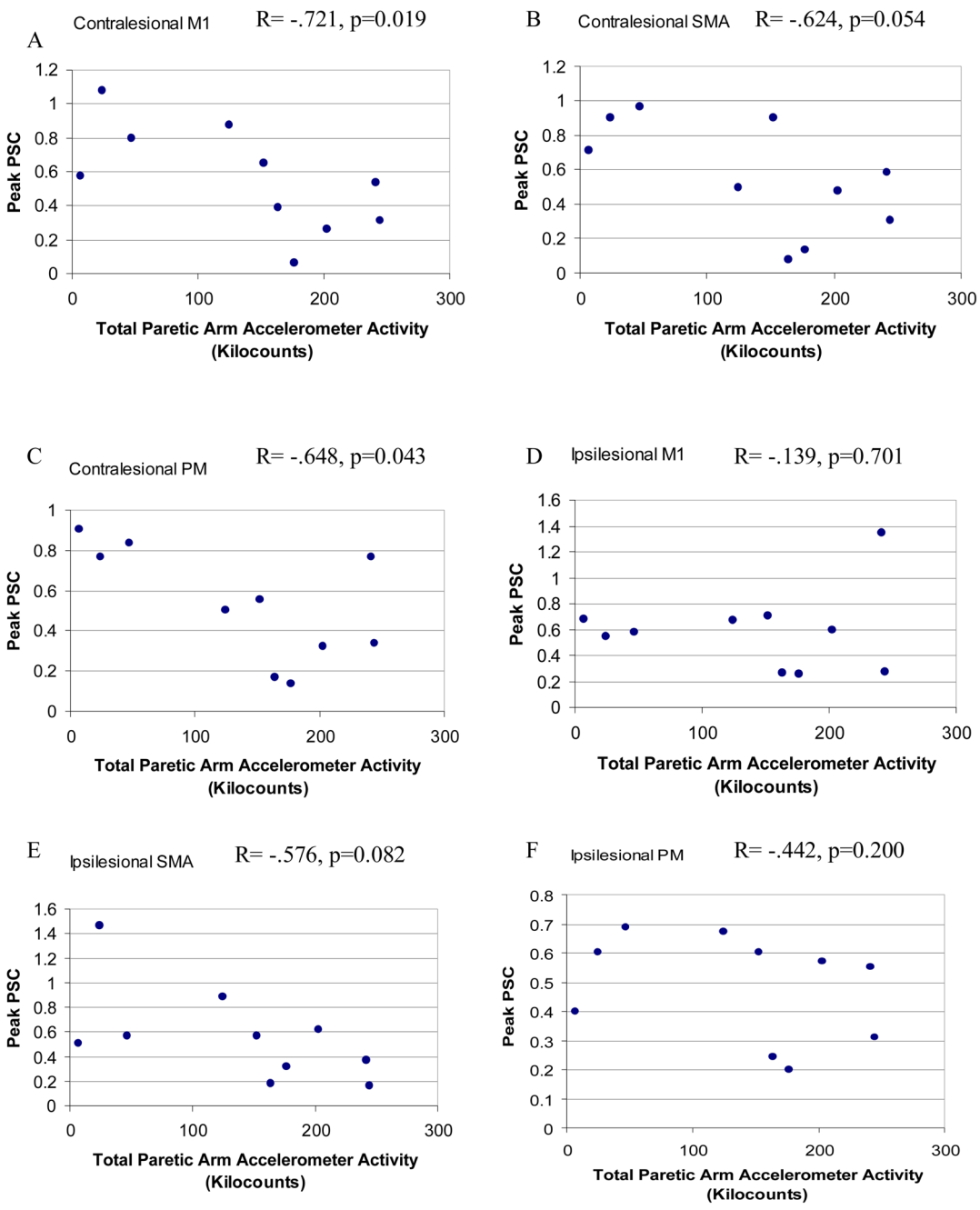

Figure 2.

Graphs of peak PSC vs total accelerometer activity counts for the **paretic** hand of stroke subjects for: (A) contralesional M1; (B) contralesional SMA; (C) contralesional PM; (D) ipsilesional M1; (E) ipsilesional SMA; (F) ipsilesional PM.

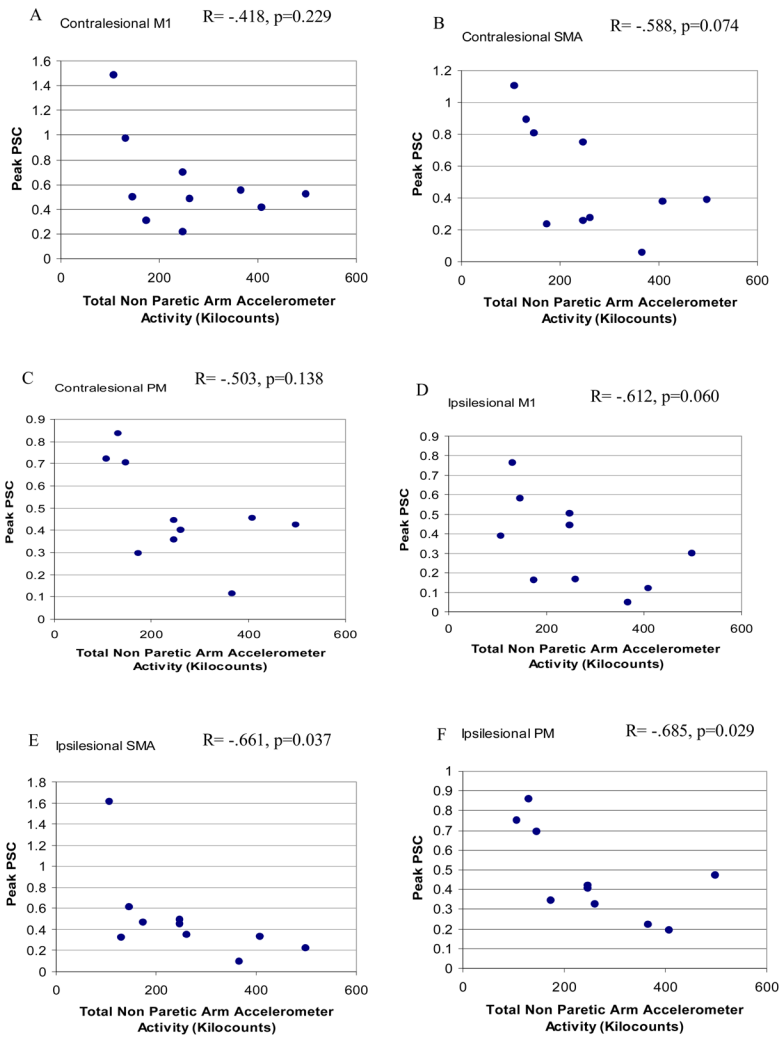

Figure 3.

Graphs of peak PSC vs total accelerometer activity counts for the **non paretic** hand of stroke subjects for: (A) contralesional M1; (B) contralesional SMA; (C) contralesional PM; (D) ipsilesional M1; (E) ipsilesional SMA; (F) ipsilesional PM.

Table 1

Patient Characteristics

Subject	Age	Sex	Time Since Stroke	Fugl Meyer (66)
1	72	F	6 yrs	63
2	80	M	8 months	57
3	60	F	4 yrs	62
4	65	M	2.5 yrs	51
5	63	M	2 yrs	61
6	55	F	2 yrs	57
7	48	M	28 yrs	43
8	62	M	1.5 yrs	57
9	61	M	2 yrs	64
10	57	M	2.5 yrs	15

Table 2

The mean (SD) activity kilocounts per day of the right and left hands for control participants and paretic and non-paretic hands for stroke participants.

Control Participants mean (SD) activity kilocounts/day		Stroke Participants mean (SD) activity kilocounts/day	
Right Hand	**Left Hand**	**Paretic Hand**	**Non Paretic Hand**
295.2 (109.4)	306.4 (134.6)	138.5 (86.4)	259.0 (129.4)

Table 3

The mean (SD) activity peak PSC at 40% MVC force production with the left hand of control participants and paretic and non-paretic hands of stroke participants.

	Contralateral ROIs			**Ipsilateral ROIs**		
	M1	**SMA**	**PM**	**M1**	**SMA**	**PM**
Controls	0.54 (0.23)	0.58 (0.31)	0.44 (0.20)	0.45 (0.15)	0.64 (0.41)	0.53 (0.26)
	Ipsilesional ROIs			Contralesional ROIs		
	M1	SMA	PM	M1	SMA	PM
Stroke P	0.59 (0.32)	0.56 (0.38)	0.49 (0.18)	0.55 (0.31)	0.56 (0.32)	0.53 (0.28)
Stroke NP	0.35 (0.23)	0.49 (0.42)	0.47 (0.23)	0.61 (0.27)	0.51 (0.34)	0.48 (0.22)

Table 4

Spearman correlations between peak PSC in ROIs and total accelerometer activity.

Control	**Non Dominant Hand**		
Contralateral M1	−.333 (.347)		
Contralateral SMA	−.018 (.960)		
Contralateral PM	−.188 (.603)		
Ipsilateral M1	.503 (.138)		
Ipsilateral SMA	.345 (.328)		
Ipsilateral PM	.152 (.676)		
Stroke		Paretic Hand	Non-paretic Hand
Ipsilesional M1		−.139 (.701)	−.612 (.060)
Ipsilesional SMA		−.576 (.082)	−.661 (.037)*
Ipsilesional PM		−.442 (.200)	−.685 (.029)*
Contralesional M1		−.721 (.019)*	−.418 (.229)
Contralesional SMA		−.624 (.054)	−.588 (.074)
Contralesional PM		−.648 (.043)*	−.503 (.138)

Note: r(p value);

* indicates significance at $P < 0.05$.