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Size Doesn't Matter: Cortical Stroke Lesion Volume is Not Associated with Upper Extremity Motor Impairment and Function in Mild, Chronic, Hemiparesis

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Abstract

Objective—To determine: (a) the relationship between lesion volume and upper extremity (UE) motor impairment using the UE section of the Fugl-Meyer (FM); and (b) the relationship between lesion volume and UE functional outcomes using the Arm Motor Ability Test (AMAT) Functional Ability (FA) and Time scales.

Design—Secondary, retrospective analysis of randomized controlled trial data

Setting—Not applicable

Participants—139 subjects with chronic stroke (83 males; mean age of all subjects = 56.7 ± 11.2 years; mean time since stroke onset = 59.6 ± 65.6 months; 90 subjects with right hemiparesis) and stable, active, distal UE movement.

Intervention—Data were collected related to subjects' lesion volum and UE movement prior to their participation in a multicenter randomized controlled trial.

Main Outcome Measures—The FM and the AMAT.

Results—Neither age nor lesion volume was related to FM performance. The p-value for the regression coefficient of lesion volume was 0.045 in the AMAT FA model and 0.016 in the AMAT Time model. Lesion volume accounted for only an additional 1.7% (AMAT FA) to 3.1% (AMAT Time) of the variability in motor function, and was not clinically meaningful.

Conclusions—Data suggest no relationship between lesion volume and UE impairment, and a small, clinically insignificant relationship between lesion volume and UE motor function. Stroke affects metabolic changes in intact regions, and causes diffuse structural loss in anatomically remote regions from the infarction. These other factors may account for variance in motor outcomes following stroke.

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Upper extremity (UE) hemiparesis remains one of the most common and devastating stroke-induced impairments.[1] Several rehabilitative approaches targeting stroke survivors with active, distal movement in their paretic UE's are efficacious.[2,3,4] However, the factors that predispose individuals with this level of movement to benefit from such approaches are not fully understood.

It has been suggested that lesion characteristics – including its volume - influence stroke recovery,[5] but equivocal evidence[6,7,8] supports this precept. In the acute phase (< 1 month post ictus), weak relationships are reported between lesion volume and UE outcome.[9,10,11] Furthermore, the ongoing and erratic nature of neurological recovery during this phase diminishes validity of using a single measurement point to characterize UE recovery. Since lesion volume remains relatively constant during the chronic phase (> 6 months post ictus),[12] others have examined the associations between lesion volume and response to UE interventions administered chronically.[13,14,15] However, conclusions from chronic stroke intervention studies are also limited, due to relatively small subject numbers (e.g., Sterr et al:[13] n = 10; Riley et al:[14] n = 23), and varied treatment parameters (e.g., treatment session duration) and study criteria used in these trials. Other variations during the intervention period (e.g., differences in subject compliance; variances in how the treatments are administered) may also affect treatment response and, thus, the validity of conclusions made by chronic UE intervention studies.

To overcome the above shortfalls and better understand factors affecting UE motor status, the purposes of this study were to determine: (a) the relationship between lesion volume and UE motor impairment using the UE section of the Fugl-Meyer (FM); and (b) the relationship between lesion volume and UE functional outcomes using the Arm Motor Ability Test (AMAT). This study enrolled a well-defined cohort of 139 subjects with stable, mild, chronic UE hemiparesis. We enrolled people with mild UE hemiparesis given the proliferation of efficacious therapies targeting subjects exhibiting this level of UE motor impairment.[e.g.,2–4] To our knowledge, this was the largest study examining relationships among lesion volume and chronic UE motor status.

Method

Study Design

This study was a secondary analysis of data from the Everest randomized controlled trial of implanted cortical stimulation for UE movement in chronic stroke.[16] Outcome measures had been administered before and after intervention as part of the above trial. However, the current study focused solely on lesion volume and values obtained on the FM and AMAT prior to randomization, or to any interventions taking place. The study had been approved by all participating centers' institutional review boards, or by outside institutional review boards.

Subjects

Subjects were recruited for the intervention trial from across the United States using several recruitment strategies, including print advertisements (e.g., pamphlets) placed in clinics near enrolling sites, radio advertisements in the markets of enrolling sites, and print advertisements placed in national magazines whose primary subscribers were survivors of stroke. As volunteers came forward, the following screening criteria were applied: Inclusion criteria: (1) Subjects must have had an ischemic vascular lesion (i.e., stroke), as documented by computerized tomography or magnetic resonance imaging, and occurring above the level of the midbrain; (2) stroke occurred > 4 months prior to study enrollment; (3) Subjects were medically and neurologically stable, as determined by medical history and documented

neurological examination; (4) score on the upper extremity section of the Fugl-Meyer scale (described later) ≥ 28 ≤ 50, and active extension in the affected wrist of at least 5°; (5) Age ≥ 21 years or older at time of enrollment; (6) For women of childbearing potential, a negative serum βhCG pregnancy test within 2 weeks of study entry, and willingness to practice adequate contraception during the study, due to unknown potential impact of the surgery used in the trial on the fetus; (7) Ability to comply with the study rehabilitation protocol. Exclusion criteria: (1) hemorrhagic stroke (2) > 1 stroke; (3) any neurologic condition (beyond the stroke) or physical condition that impaired function of the affected arm; (4) History of seizure disorder or a spontaneous seizure that had occurred one month or longer from stroke, due to the possible impact of cortical stimulation on seizure activity; (5) Neurological condition that would likely reduce the safety of study participation, including central nervous system vasculitis, intracranial tumor, intracranial aneurysm, multiple sclerosis or arteriovenous malformations; (6) Moderate to severe hemispatial neglect and/or anosognosia involving the affected arm, due to the potential impact of this condition on ability to participate in the rehabilitative therapies provided during this trial; (7) Severe sensory deficit, including, but not limited to, a score of 2 on part 8 of the National Institutes of Health Stroke Scale (NIHSS); (8) Inability to understand, cooperate, or comply with the study procedures; (9) Severe spasticity, defined as an Ashworth score of 4 in any region of the affected arm; (10) Change in oral spasticity medications occurring 2 weeks prior to enrollment, or Botulinum toxin A injections in the affected arm 4 months prior to enrollment; (11) Major active psychiatric illness that may interfere with treatment; (12) Untreated or inadequately treated depression defined by a score of 19 or greater (out of 63) on the 21-question version of the Beck Depression Inventory; (13) Modified Rankin Score ≥ 4; (14) A substantial cardiopulmonary or metabolic disorder. This includes a current serum creatinine>3.0 mg/dL, a total serum bilirubin >2.0 mg/dL, or advanced chronic obstructive pulmonary disease; (15) Increased risk for myocardial infarction or other major medical complications of general anesthesia or surgery. (16) Terminal illness associated with survival < 12 months, due to the projected duration of the study; (17) Inability to discontinue antithrombotic therapy (e.g., antiplatelet agents or anticoagulants) perioperatively for device implantation and removal; (18) Introduction in the 2 months prior to enrollment of a potentially confounding central nervous system drug (e.g., amphetamines, antiepileptics, anxiolytics, and antidepressants); (19) History of spinal cord injury, traumatic brain injury, or spontaneous subdural or epidural hematoma that has resulted in a neurologic deficit; (20) Current abuse of alcohol or drugs, as this may have an influence on study compliance; (21) Contraindication to magnetic resonance imaging (e.g., implanted metallic or electrical devices); (22) Nursing a child, pregnancy, or intent to become pregnant during the study, given the unknown impact of cortical stimulation on the fetus and on breastmilk; (23) Participating in another trial within 30 days of enrollment in this study; (24) any other condition that, in the opinion of the investigators, would interfere with study compliance or safety.

Using the aforementioned study criteria, 139 subjects were included in the current analysis (83 (57.6%) males; mean age of all subjects = 56.7 ± 11.2 (standard deviation) years; mean time since stroke onset for subjects in sample= 59.6 ± 65.6 months; 90 subjects with hemiparesis affecting their right UEs; 80 (57.6% subjects with hemiparesis affecting their dominant UE's) (see Table 1 for subject demographics).

Outcome Measures

Lesion volume was obtained from the original medical record and verified by at least 2, independent, neurologist-observers for each subject by manually tracing the perimeter of the area of abnormal low attenuation on each CT or MRI slice showing the infarct, as described

elsewhere.[17,18] This has been shown to be the most reproducible method of measuring lesion volume.[18]

Lesion volume may affect different domains of function in varied ways. Thus, we administered measures discerning the constructs of UE impairment (i.e., active movement in each UE joint) and function (i.e., intersegmental movements put together in sequential fashion to accomplish a functional goal). The measures were administered by a blinded rater at one of the participating centers at which the trial was being conducted. All raters were certified and re-certified on the outcome measures every 3 months using standardized, live and video based inter-rater reliability checks at the main study center: (a) The upper extremity motor portion of the *Fugl-MeyerScale(FM)*[19] was used to assess upper extremity impairment. Data arise from a 3-point ordinal scale (0=cannot perform; 1=can partially perform; 2=can perform fully) applied to each item, and the items are summed to provide a maximum score of 66. The FM's scores have been shown to offer high test-retest reliability (total=.98–.99; subtests=.87–1.00), interrater reliability, and construct validity in similar contexts to that which was tested herein (i.e., mildly impaired, chronic stroke).[20,21] (b) the *Arm Motor Ability Test*,[22] used to determine whether changes occur in activity limitation. The AMAT is a 13-item test in which ADLs are rated according to a functional ability scale (FA) that examines paretic limb use (0 = does not perform with paretic upper extremity; 5 = does use arm at a level comparable to less affected side) and a scale in which subjects are timed on each item (Time). Many of the test items are further subdivided into sub-activities to be rated, include use of a knife and fork, eating with a spoon; combing hair; and tying shoelaces. The AMAT is a valid, stable, and reliable scale, and correlates positively with other stroke specific functional scales.

Statistical Analyses

Preliminary stepwise regression analyses were performed to identify variables with potential relationships with one or all of the outcomes variables (FM, AMAT FA, AMAT Time) from among 3 possible covariates: age, lesion volume and months since stroke Lesion volume (variable of interest) and subject age(nuisance covariate) were identified as having potential relationships with one or all of the outcomes variables, and were thus retained for subsequent analyses. A statistical alpha level of 0.05 was used. Table 2 contains the correlations of the three covariates with the outcome variables.

A two-step multivariate regression analysis was then used to capture the additional variance explained by lesion volume, after accounting for effects of age. Two multivariate general linear models were fit (dependent variables = FM, AMAT FA, AMAT Time); the first with age as the only predictor and the second with lesion volume serving as a second predictor. All analyses were performed using SPSS version 19.0.

Results

Outcomes of Correlation Analyses

Multivariate tests of significance of independent linear combinations of the outcome variables were performed to assess the response variables that were related to predictor variables. Lesion volume was not related to FM performance ($p = 0.77$).

Age and lesion volume were significantly related to performance on the AMAT FA and the AMAT Time scales. Their combined influence was small, however, accounting for only 6% of the variance in AMAT FA and AMAT Time ($R^2 = .07$, Adjusted $R^2 = .06$) (Table 2). When the two general linear models with and without lesion volume as a predictor were compared, lesion volume was found to significantly improve the predictive value of the AMAT model. The p-value for the regression coefficient of lesion volume was 0.045 in the

AMAT FA model and 0.016 in the AMAT Time model. However, it accounted for only an additional 1.7% (AMAT FA) to 3.1% (AMAT Time) of the variability in motor function and was, thus, not clinically meaningful. Table 3 shows the adjusted R^2 when only age is included in the model and the adjusted R^2 including both age and lesion volume in the model.

Discussion

UE hemiparesis remains one of the most common and disabling stroke sequelae. Yet, while association between lesion volume and general neurologic (e.g., NIHSS) or functional (e.g., Functional Independence Measure) status has been examined, no large studies have determined the relationships between lesion volume and functional UE motor outcomes using stroke specific, validated, functional measures and a large, well defined cohort of stroke survivors. This study addressed these limitations by examining associations between cortical lesion volume and UE motor outcomes in a well-defined cohort of 139 stroke survivors with chronic, ischemic stroke. To our knowledge, this was the largest study examining the associations among lesion volume and UE motor recovery using UE motor outcome measures in chronic stroke.

We found that lesion volume did not significantly predict UE impairment in our cohort. Moreover, although lesion volume significantly predicted the extent of motor function as measured by the AMAT, this effect was not clinically significant and accounted for only 2–3% of variance in outcomes on this measure. These findings are consistent with other, smaller studies examining the relationships between lesion volume and motor recovery in the chronic phase.[5,13–15,23] The absence of a strong relationship between lesion volume and UE movement may be considered counterintuitive, given that the motor deficit is a direct result of the lesion. However, there is mounting evidence suggesting that neurologic factors other than lesion volume(e.g., lesion location; structural integrity of spared tissue) are stronger predictors of UE motor recovery than is lesion volume. For example, centrum semiovale infarcts located at the intersection of the corpus callosum and corticospinal tract have been shown to be particularly detrimental to motor function.[24,25] Lesions that disrupt major axonal pathways to a greater extent (e.g., corticospinal projections) are also associated with poorer recovery.[26,27] Thus, relatively small lesions that focally affect major tracts would be expected to produce larger deficits than more superficial lesions of similar volume,[9] diminishing the predictive value of lesion volume alone.[24] Although often considered a relatively focal event, stroke is also known to affect metabolic changes in intact regions,[28,29,30] and to produce bilateral diffuse structural loss in brain areas that are anatomically remote from the infarction,[31,32] the extents of which are associated with degree of motor recovery.[e.g.,27–29,33] In sum, these other factors likely account for a substantial portion of the variance in UE motor outcomes following stroke, whereas lesion volume appears to be of secondary importance. Thus, our current findings may be explained by the relative importance of other factors in UE recovery versus lesion volume. Future studies may wish to examine not only lesion volume, but place it in a regression model that takes into account the above factors, to better understand their relative importance on UE motor outcome.

It should be noted that results of this study, which is specific to the motor domain, do not imply that lesion volume is a poor predictor of general neurological recovery. The results of several studies suggest that lesion volume is predictive of post-stroke outcomes; however, these studies employed bedside measures of general neurologic status(e.g., NIHSS score) that concurrently assess several domains of recovery, and that do not purely examine UE motor outcome. Our results, when interpreted in the context of the larger literature, suggest that small as well as large lesions have the potential to be detrimental to particular functions,

whereas larger lesions often result in more complex neurological presentations that can affect multiple domains of neurological function.

Limitations and Future Directions

While our findings are likely valid, the study has limitations that should be noted. For example, a carefully selected group of individuals with relatively mild UE motor deficits, limited comorbidities, and chronic, stable, ischemic infarcts was enrolled. It could be argued that the relatively strict selection criteria and limited range of motor impairment that this group represents may limit generalizability of the results. Concurrently, though, the well-defined, large nature of the sample makes it likely that findings are likely valid for people with mild UE hemiparesis. The volume and nature of the sample also constitutes an advancement over smaller studies attempting to define neurologic substrates of recovery.[13,14] Moreover, a growing number of promising restorative UE therapies have used criteria similar to those used for this study.[2–4] Our results are expected to assist in understanding what factors contribute to the efficacy of such therapies, by identifying the patients most likely to benefit from them. A second limitation is that brain volume was not controlled in this study. This shortfall is likely to produce some heterogeneity and would undermine the validity of findings in smaller studies. However, it is likely that these small variances are inconsequential given the large number of subjects enrolled in this trial.

Conclusions

This was the first study to determine whether lesion volume is significantly associated with UE motor status in a well-defined, sizable cohort of chronic stroke survivors. Data suggest a small, clinically insignificant relationship between lesion volume and UE impairment and motor function.

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ABBREVIATIONS

UE	Upper extremity
UE FM	upper extremity Fugl Meyer
AMAT	Arm Motor Ability Test

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Table 1

Subject Demographics

	N	Mean	Standard Deviation
Age (years)	139	56.7	11.2
% male	139	57.6%	
Time since stroke (months)	139	59.6	65.6
% Affected side same as dominant	139	57.6%	
Lesion volume (cm^3)	139	26.3	38.7
FM Total Score (LOCF)	139	37.4	5.9
AMAT FA Mean (LOCF)	139	3.0	0.7
Affected Hand 1-minute Test (LOCF)	139	19.0	11.4

Table 2

Correlations of Covariates with Outcome Measures

Potential Predictors	FM Total Score	AMAT FA Mean	AMAT Time Test Mean
Age			
Pearson Correlation	.083	.206	.181
Sig. (2-tailed)	.332	.015	.033
N	139	139	139
Strkmos			
Pearson Correlation	−.037	.012	−.025
Sig. (2-tailed)	.663	.887	.767
N	139	139	139
Lesion size			
Pearson Correlation	.025	−.171	−.205
Sig. (2-tailed)	.772	.045	.015
N	139	139	139

Table 3

Multivariate GLM Results

Dependent Variable	Adjusted R^2 Age	Adjusted R^2 Age and Lesion Volume (cm^3)	Change in Adjusted R^2	P-value of Lesion Size Effect in General Linear Model
FM Total Score	0.000	0.000	0.000	0.331
AMAT FA Mean	0.040	0.057	0.017	0.045
AMAT Time	0.029	0.060	0.031	0.016