Relevance of Subcortical Stroke in Dysphagia

Monique G. Cola, PhD; Stephanie K. Daniels, PhD; David M. Corey, PhD; Lisa C. Lemen, PhD; Maryellen Romero, PhD; Anne L. Foundas, MD

Background and Purpose—Unilateral cortical lesions are associated with dysphagia in ischemic stroke. It is unclear, however, whether acute subcortical stroke is associated with a similar risk of dysphagia. The aim of this study was to determine the occurrence of dysphagia in purely subcortical stroke and identify dysphagia characteristics.

Methods—Between 2003 and 2005, videofluoroscopic swallow studies (VFSSs) were completed in 20 consecutive ischemic stroke patients with purely subcortical lesions (right hemisphere damage [RHD]=10, left hemisphere damage [LHD]=10) and 25 age-matched controls. Individuals were classified with dysphagia when at least 2 swallowing measures were 2 standard deviations above mean scores for the control group. Lesion volume, hemisphere, and location were determined from diffusion-weighted magnetic resonance imaging scans.

Results—Seven subcortical stroke patients (35%) met VFSS criteria for dysphagia (LHD=5, RHD=2); 4 patients presented with clinically significant dysphagia. A significant interaction between hemisphere and lesion location was identified. Whereas 3 of 5 patients with dysphagia (60%) had lesions to the left periventricular white matter (PVWM), LHD patients without dysphagia did not have PVWM lesions. In contrast, no RHD patients with PVWM lesions had dysphagia, and 6 of 8 patients without dysphagia (75%) had PVWM lesions. Oral transfer was significantly slower in patients with subcortical stroke compared with the healthy adults.

Conclusions—Lesions to the left PVWM may be more disruptive to swallowing behavior than similar lesions to the right PVWM. Swallowing deficits involving oral control and transfer may be a marker of subcortical neural axis involvement. (Stroke. 2010;41:482-486.)

Key Words: stroke ■ dysphagia ■ periventricular white matter

Dysphagia occurs in ≈50% of stroke patients1–3 and is a major source of disability after stroke. One half of stroke patients with dysphagia become malnourished,4 and many develop pneumonia regardless of the severity of dysphagia or presence of aspiration.5 Length of hospitalization is increased in stroke patients with dysphagia, and these individuals are more likely to be discharged to nursing homes compared with stroke patients without dysphagia.6

Although dysphagia is commonly observed in acute stroke patients, the neural control of swallowing remains unclear. Functional and anatomic imaging studies have identified several sites important to swallowing, including the primary sensorimotor cortices, insula, anterior cingulate, internal capsule, basal ganglia, and thalamus.7–10 Daniels and Foundas7 have developed an anatomic model of swallowing defined as a distributed neural network involving bilateral input from the sensorimotor cortex with descending input to the brainstem medullary swallowing center. Disruption of cortical-cortical and cortical-subcortical white matter connections, specifically periventricular white matter (PVWM) lesions, seems to increase the risk of dysphagia and aspiration by lowering the threshold of input to the medullary swallowing center. Contributions of specific cortical sites, such as the anterior insula, to precise timing and coordinated evocation of the pharyngeal swallow remain unclear. The unique contributions of subcortical regions and white matter pathways to swallowing behaviors have not been studied in acute stroke patients.

Identifying and treating stroke patients at risk for dysphagia are extremely important. This information, combined with our observation that dysphagia is commonly associated with PVWM lesion location in combination with cortical lesions, motivated this study of swallowing behaviors in patients with acute purely subcortical stroke. The use of the “gold standard” videofluoroscopic swallow study (VFSS) to examine swallowing behaviors within the same time frame as the magnetic resonance imaging (MRI) scan to examine lesion location permitted us direct examination of structure-function relationships. Specifically, we were interested in determining whether (1) dysphagia was associated with isolated subcortical stroke, (2) hemisphere and specific subcortical lesion sites impact swallowing, and (3) specific patterns of dysmotility occur in subcortical dysphagia. We hypothesized that the
stroke patients would be impaired relative to controls on VFSS, although we were unsure whether individuals with right hemisphere lesions would be more impaired than individuals with left hemisphere stroke. Furthermore, we were not sure how common subcortical dysphagia would be. On the basis of our previous research, we posited that PVWM involvement would be related to dysphagia, but we did not know whether different lesion locations would relate to different disorders of swallowing.

Subjects and Methods

Participants
Participants included individuals with subcortical stroke (N = 20; 10 right hemisphere damaged [RHD] and 10 left hemisphere damage [LHD]) from consecutive patients with an acute unilateral ischemic stroke admitted to the Southeast Louisiana Veterans Healthcare System in New Orleans, LA (June 2003 to August 2005) and healthy, age-matched controls (n = 25). Participants had no history of neurologic disease (except the stroke group), head and neck structural damage, or dysphagia unrelated to the stroke. The inclusion criterion in the stroke group was a unilateral subcortical ischemic infarct confirmed by a diffusion-weighted imaging MRI sequence. A lesion was classified as subcortical when it was limited to the white matter and/or subcortical gray matter structures without extension to adjacent cortical gray matter. National Institutes of Health Stroke Scale (NIHSS) scores were obtained from the admission note on all individuals with stroke.

The study was approved by the institutional review board at Tulane University Health Sciences Center and by the Veterans Affairs Medical Center in New Orleans, and all participants provided written consent before participation.

VFSS Acquisition and Analysis

Swallowing was evaluated with VFSS. Lateral radiographic views of swallowing were obtained with the fluoroscopic tube focus encompassing the oral cavity and the pharynx. Participants completed 2 swallows of 3 mL of liquid barium (E-Z-Paque, E-Z-EM, Westbury, NY; diluted 2:1, water to barium). VFSS samples were recorded on a Super-VHS videocassette recorder (AG-1980 Panasonic, Secaucus, NJ); a counter timer (Thalner Electronics Laboratories, VC 436; Ann Arbor, Mich) encoded digital time (0.01 second) on each video frame. Videofluoroscopic recordings were obtained at a resolution of 30 frames per second. The VFSS was completed an average of 1.57 days from admission (range, 0 to 4 days) for the participants with acute stroke. The examiners completing the analysis of the recorded VFSS data were blinded to group, hemisphere, and lesion location.

Three domains of bolus flow were evaluated: (1) bolus timing, measured as oral transit time (OTT), stage transit duration (STD), and pharyngeal response time (PRT); (2) bolus direction measured by the Penetration-Aspiration Scale (PAS); and (3) bolus clearance measured by vallecular retention (VR) and pyriform sinus retention (PSR). OTT was measured from initiation of bolus movement in an anterior or posterior direction until arrival of the leading edge of the bolus at the posterior angle of the ramus of the mandible. STD was measured from the arrival of the leading edge of the bolus at the posterior angle of the ramus of the mandible to initiation of maximum superior movement of the hyoid bone. PRT was measured with the initiation of maximum superior movement of the hyoid and ending with passage of the bolus tail through the upper esophageal sphincter.

The PAS, a validated ordinal scale, was used to measure bolus direction. A score of 1 indicated no airway invasion (ie, no laryngeal penetration or aspiration). Scores 2 to 5 indicated laryngeal penetration, with depth and clearance to determine the score. Scores 6 to 8 indicated aspiration, with response and clearance to determine the score. Bolus clearance was rated on an ordinal scale of 1 to 3 for both the vallecular (VR) and pyriform (PSR) sinuses. A score of 1 indicated no to minimal bolus residual. A score of 2 indicated moderate bolus retention with up to half of the recess filled with postswallow residual. A score of 3 indicated severe bolus retention with more than half of the recess filled with postswallow residual. Interrater and intrarater reliabilities for each of the swallowing measures according to the intraclass correlation coefficient was previously obtained in a larger dataset that contained the data currently reported. Reliability was established by the second author (S.K.D.) who has extensive research and clinical experience in examining VFSS. For all measures, the intraclass correlation coefficient was >0.85, except interrater PAS (0.78).

Durations and scores were determined in each participant for each swallow across the 2 trials by slow-motion and frame-by-frame analysis, with scores collapsed across trials for a single score for each measure. Individuals were classified with dysphagia when at least 2 of the swallowing measures were 2 standard deviations above mean scores for the control group. Clinically significant dysphagia was examined and defined when the patient received diet modification and/or swallowing therapy. Recommendations for diet alteration and treatment were based on the results of the VFSS and determined by the clinician, who was blinded to the results of bolus flow measurement and MRI results.

MRI Acquisition and Analysis

MRI brain scans were acquired and used for lesion localization. A multislice, isotropic, single-shot echoplanar imaging sequence with a bmax of 1000 seconds/mm2 was used. Imaging parameters included echo time = 118 seconds, field of view = 240 mm, and a matrix size of 256×256 pixels, as a gapless series of 6-mm axial images. The diffusion gradient was applied along the x, y, and z axes. An average of all 3 diffusion directions was computed to minimize the effects of diffusion anisotropy. The examiner mapping the lesions was blinded to clinical information.

Lesion volumes were mapped out on diffusion-weighted imaging images by manually tracing lesions with the Image J image software program (http://www.rsb.info.nih.gov/ij). This method is similar to that used in our previous lesion studies when localizing lesions with conventional computed tomography images. The full extent of the lesion was mapped by using a semiautomated threshold method to trace the margin of the lesion in consecutive images. Lesions were traced by hand with a mouse-driven cursor on each MRI slice. Lesion volume was then computed by summing the area traced on each slice and multiplying by slice thickness. Lesions were defined by hemisphere (left, right) and specific subcortical location. Location was further categorized as (1) including PVWM or (2) excluding PVWM (ie, other white matter sites or subcortical gray matter structures).

Statistical Analyses

Stroke groups (LHD, RHD) were compared with controls in all analyses. Group (RHD, LHD, control) differences in sex (male, female) and race (white, black) distributions were assessed by separate χ2 analyses. Group differences in stroke severity and lesion volume were tested by ANOVA with group (RHD, LHD, control) entered as a grouping factor. NIHSS score and lesion volume were entered as dependent variables (DVs) in separate analyses.

Where necessary, associations between group and a demographic variable were controlled for by including the demographic variable as a covariate in univariate and multivariate ANCOVA. The ANCOVA homogeneity of regression assumption was tested by including independent variable×covariate interactions in an initial analysis step. Absent violation of the assumption, independent variable×covariate interactions were removed from the model.

Between-group differences in OTT, STD, PRT, PAS, and VR were tested by MANOVA with group entered as a grouping factor and with swallowing characteristics entered as DVs. PSR was not entered as a DV, because the subjects in all 3 groups demonstrated PSR scores of 1. Significant MANOVA was followed by univariate ANOVA for each DV. Significant ANOVA results were followed by Newman-Keuls pairwise comparisons.
Table 1. Lesion Location and Dysphagia Classification for Stroke Subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Hemisphere</th>
<th>PVWM</th>
<th>Other Subcortical Areas</th>
<th>Dysphagia</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>RHD</td>
<td>No</td>
<td>Thalamus</td>
<td>Yes</td>
</tr>
<tr>
<td>2</td>
<td>RHD</td>
<td>No</td>
<td>Deep white matter</td>
<td>Yes</td>
</tr>
<tr>
<td>3</td>
<td>RHD</td>
<td>Yes</td>
<td>Thalamus, internal capsule</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>RHD</td>
<td>Yes</td>
<td>Caudate, internal capsule</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>RHD</td>
<td>Yes</td>
<td>Thalamus, internal capsule</td>
<td>No</td>
</tr>
<tr>
<td>6</td>
<td>RHD</td>
<td>Yes</td>
<td>Caudate</td>
<td>No</td>
</tr>
<tr>
<td>7</td>
<td>RHD</td>
<td>Yes</td>
<td>Frontal white matter</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>RHD</td>
<td>No</td>
<td>Thalamus</td>
<td>No</td>
</tr>
<tr>
<td>9</td>
<td>RHD</td>
<td>Yes</td>
<td>. . .</td>
<td>No</td>
</tr>
<tr>
<td>10</td>
<td>RHD</td>
<td>No</td>
<td>Thalamus</td>
<td>No</td>
</tr>
<tr>
<td>11</td>
<td>LHD</td>
<td>No</td>
<td>Thalamus, internal capsule</td>
<td>Yes</td>
</tr>
<tr>
<td>12</td>
<td>LHD</td>
<td>Yes</td>
<td>Deep white matter</td>
<td>Yes</td>
</tr>
<tr>
<td>13</td>
<td>LHD</td>
<td>No</td>
<td>Putamen, internal capsule</td>
<td>Yes</td>
</tr>
<tr>
<td>14</td>
<td>LHD</td>
<td>Yes</td>
<td>External capsule</td>
<td>Yes</td>
</tr>
<tr>
<td>15</td>
<td>LHD</td>
<td>Yes</td>
<td>. . .</td>
<td>Yes</td>
</tr>
<tr>
<td>16</td>
<td>LHD</td>
<td>No</td>
<td>Deep white matter</td>
<td>No</td>
</tr>
<tr>
<td>17</td>
<td>LHD</td>
<td>No</td>
<td>Basal ganglia, caudate</td>
<td>No</td>
</tr>
<tr>
<td>18</td>
<td>LHD</td>
<td>No</td>
<td>Thalamus</td>
<td>No</td>
</tr>
<tr>
<td>19</td>
<td>LHD</td>
<td>No</td>
<td>Parietal white matter</td>
<td>No</td>
</tr>
<tr>
<td>20</td>
<td>LHD</td>
<td>No</td>
<td>Thalamus, internal capsule</td>
<td>No</td>
</tr>
</tbody>
</table>

The ability of the affected hemisphere (left, right) and PVWM involvement (present, absent) to predict dysphagia (present, absent) was evaluated by binary logistic-regression analysis with hemisphere, PVWM, and the hemisphere×PVWM interaction entered as predictors and with dysphagia entered as the dichotomous DV. The independent contribution to the model for each effect was evaluated by complete versus reduced model testing.

Results

Lesion characteristics and dysphagia diagnoses are shown in Table 1. Seven of 20 (35%) subcortical stroke patients met the VFSS criteria for dysphagia, with 4 patients presenting with clinically significant dysphagia. No control participants were classified with dysphagia.

Five (71%) of the individuals with stroke and dysphagia had LHD and 2 (29%) had RHD. Sixty percent of the individuals with LHD and dysphagia had lesions to the PVWM; participants with LHD without dysphagia did not have PVWM lesions. Seventy-five percent of the participants with RHD without dysphagia had PVWM lesions. No individuals with right-hemisphere PVWM lesions had dysphagia. A significant PVWM×hemisphere interaction, $\Delta \chi^2(1)=9.85$, $P=0.002$, was found. Whereas a perfect relation between hemisphere and dysphagia was observed in those with PVWM damage (100% dysphagia for LHD, 0% dysphagia for RHD; $\chi^2(1)=9.00$, $P=0.003$), no such association was detected in those without PVWM damage (29% for LHD, 50% for RHD; $\chi^2(1)=0.505$, $P=0.477$). Of the 4 patients with clinically significant dysphagia, 3 had LHD, 1 had RHD, and 2 had a lesion of the PVWM.

Group demographics are presented in Table 2. No significant associations with group were observed for sex, NIHSS score, or lesion volume. Blacks were overrepresented among stroke groups relative to controls. Race was, therefore, included as a covariate in subsequent analyses.

Group means for all measures of swallowing characteristics are shown in Table 3. In testing the homogeneity of regression assumption associated with ANCOVA, a significant multivariate group×race interaction was observed for the DVs of OTT, STD, PRT, PAS, and VR (Wilks’ $\Lambda=0.284$, $F_{10,76}=6.14$, $P<0.001$), requiring the retention of the group×race interaction in the model for affected DVs. Univariate ANOVA indicated that the multivariate effect was driven solely by PAS. The white LHD group (mean $\lambda=3.75$, SD=1.06) scored significantly higher on PAS than all other groups (mean=1.33, SD=0.80). The LHD group included only 2 whites, and these were the 2 individuals with the highest PAS averages (4.5 and 3) among all participants. PAS >1 was observed for only 3 other cases (PAS=2).

Significant group×race interactions were not observed for any other DVs. Race was, therefore, treated as a standard covariate in the remaining analyses. MANCOVA indicated significant group differences in swallowing, Wilks’ $\Lambda=0.575$, $F_{8,78}=3.35$, $P=0.004$. Subsequent ANCOVA indicated that this multivariate effect was associated with group differences in OTT and VR but not in STD (Table 3). For OTT, stroke group means were significantly higher than those in controls but were not significantly different from each other. Both control and RHD groups scored significantly lower on VR than did the LHD group. Control and RHD groups did not differ.

Discussion

This study was designed to examine swallowing deficits after unilateral subcortical ischemic stroke. There were 3 major results from this study. First, dysphagia occurred in approximately one third of the cases with acute subcortical stroke, with one half of these individuals demonstrating clinically significant dysphagia. Swallowing disorders were more com-
mon in the cases with left hemispheric stroke. The left hemispheric PVWM was a major site of acute brain injury associated with dysphagia, whereas brain injury to the right PVWM was never associated with swallowing disorders in our sample. Finally, a specific swallowing impairment, longer OTT, was associated with subcortical dysphagia. This work expands on findings from prior studies by objectively defining dysphagia by VFSS, including the identification of disordered bolus flow patterns, and by providing lesion analysis on diffusion-weighted imaging MRI scans in acute stroke patients with purely subcortical lesions.

Subcortical lesion sites have been implicated in poststroke dysphagia; however, small sample sizes13 and lack of lesion location description13 have limited the impact of these studies. Significant VFSS-identified swallowing changes have been noted 3 to 4 weeks after stroke in individuals with dysphagia. Dysphagia was not defined by VFSS criteria but included 14 cases with dysphagia and 15 cases without dysphagia. Dysphagia was not defined by VFSS criteria but was based on a clinical evaluation by a speech-language pathologist who determined that the symptoms were clinically significant. The internal capsule was the most common anatomic region of interest associated with dysphagia. Unfortunately, left and right hemisphere locations and the PVWM region were not examined in this study.

The PVWM, which is the white matter adjacent to the body of the lateral ventricles, is important in the neural control of swallowing. Ascending somatosensory and descending motor fibers as well as intrahemispheric corticocortical pathways are segregated within the PWMN.17 Descending corticospinal fibers from the mouth/face representation within the ventrolateral precentral gyrus (motor cortex) are located anterolaterally in the PVWM. Ascending sensory and descending motor pathways cross these levels and interconnect with specific cortical, subcortical, and brain stem regions involved in swallowing. The current results in conjunction with our earlier study support the notion that discrete lesions to the PVWM can induce dysphagia in acute stroke. Patients identified with a small subcortical stroke must therefore be considered at risk for dysphagia, especially when the lesion involves damage to white matter connections. These data support the notion that all patients with acute stroke, regardless of lesion size or location, should be screened for dysphagia.

Swallowing impairment involving the tongue appears to be associated with acute subcortical stroke. This was reflected by a longer OTT and VR, which are generally associated with dysphagia than the right PVWM, or perhaps the right hemisphere may better compensate after disruption of white matter pathways. These findings replicate and extend results from our earlier study of dysphagia in consecutive subacute stroke patients. In our earlier study, we found that lesions involving the PVWM with extension to adjacent cortical gray matter were significantly associated with dysphagia compared with lesions restricted to subcortical gray matter structures.7 A recent study of acute stroke was conducted to examine clinically significant dysphagia in a sample of ischemic stroke patients with lesions identified on diffusion-weighted and perfusion-weighted MRI scans.16 The sample included 14 cases with dysphagia and 15 cases without dysphagia. Dysphagia was not defined by VFSS criteria but was based on a clinical evaluation by a speech-language pathologist who determined that the symptoms were clinically significant. The internal capsule was the most common anatomic region of interest associated with dysphagia. Unfortunately, left and right hemisphere locations and the PVWM region were not examined in this study.

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Table 3. Means, SDs, and SEs for All Measures of Swallowing Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>RHD</th>
<th>LHD</th>
<th>Control</th>
<th>F</th>
<th>df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>OTT, mean (SD), ms*</td>
<td>0.84 (0.44)</td>
<td>1.01 (0.45)</td>
<td>0.52 (0.31)</td>
<td>6.68 (2, 41)</td>
<td>0.003</td>
<td></td>
</tr>
<tr>
<td>OTT, mean adj. (SE), ms</td>
<td>0.80 (0.12)</td>
<td>0.98 (0.12)</td>
<td>0.40 (0.14)</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>STD, mean (SD), ms</td>
<td>0.21 (0.51)</td>
<td>0.43 (0.44)</td>
<td>-0.01 (0.36)</td>
<td>1.44 (2, 41)</td>
<td>0.249</td>
<td></td>
</tr>
<tr>
<td>PRRT, mean (SD), ms</td>
<td>0.86 (0.25)</td>
<td>0.82 (0.17)</td>
<td>0.91 (0.17)</td>
<td>1.19 (2, 41)</td>
<td>0.313</td>
<td></td>
</tr>
<tr>
<td>VR, mean (SD)*</td>
<td>1.00 (0.00)</td>
<td>1.20 (0.42)</td>
<td>1.00 (0.00)</td>
<td>4.76 (2, 41)</td>
<td>0.014</td>
<td></td>
</tr>
<tr>
<td>VR, mean adj. (SE)</td>
<td>0.97 (0.07)</td>
<td>1.18 (0.06)</td>
<td>0.93 (0.07)</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>PRS, mean (SD)</td>
<td>1.00 (0.00)</td>
<td>1.00 (0.00)</td>
<td>1.00 (0.00)</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>PAS, mean (SD)</td>
<td>1.10 (0.32)</td>
<td>1.65 (1.20)</td>
<td>1.04 (0.20)</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

Adj indicates adjusted; H, hemisphere; and R, race.
*Significant between-group differences.
†Significant group×race interaction.
decreased base of the tongue retraction. Tongue base retraction involves the pharyngeal tongue and occurs after evocation of the pharyngeal swallow; thus, it is considered part of the pharyngeal stage of swallowing. Prior research has suggested that deficits in the pharyngeal stage of swallowing either occur with RHD or are not hemisphere specific. Increased OTT was found in our patients with LHD and RHD, consistent with our earlier studies. Others have found that deficits in the oral stage of swallowing are associated with LHD. Parkinson disease is a neurodegenerative disease of subcortical neural systems including the substantia nigra and cortical-subcortical white matter connections. Dysphagia in Parkinson disease is associated with disturbed lingual motility resulting in increased OTT and reduced base of tongue retraction with VR. Findings from these studies as well our current results suggest that swallowing deficits involving the tongue, particularly disordered oral transit, may be a marker of subcortical neural axis involvement.

Limitations of our study include the small sample size and the lack of longitudinal data. Longitudinal studies with a larger population are warranted to confirm these preliminary results and to determine whether dysphagia persists in some individuals with purely subcortical lesions. Moreover, we did not obtain an MRI in the healthy subjects, nor did we analyze white matter disease in stroke subjects. Future research should investigate white matter disease on semiquantitative scales or diffusion tensor imaging to determine the impact of white matter burden on swallowing.

Conclusions

Acute purely subcortical stroke is associated with clinically important dysphagia. Disordered oral transit may be a distinct hallmark of subcortical involvement. Lesions to the left PVWM may be more disruptive to swallowing behavior than similar lesions to the right PVWM. Findings support previous research suggesting that subcortical white matter connections are important in swallowing and that disruption of cortico-subcortical connectivity may result in dysphagia.

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Disclosures

None.

References

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