Swallowing Disturbance Pattern Relates to Brain Lesion Location in Acute Stroke Patients

Volker Steinhagen, MD; Annette Grossmann, MD; Reiner Benecke, MD; Uwe Walter, MD

Background and Purpose—The relationship of brain lesion location and swallowing disturbance pattern has been poorly studied in acute stroke patients.

Methods—Sixty patients with first-ever ischemic stroke at clearly assessed location and clinical signs of dysphagia were studied. Swallowing-related parameters rated clinically and fiberendoscopically were attention deficit, buccofacial apraxia, orofacial paresis, gag reflex, delay of pharyngeal swallow, pharyngeal contraction, larynx elevation, function of upper esophageal sphincter (UES), and aspiration severity.

Results—Attention deficit was independently predicted only by parietotemporal infarction, buccofacial apraxia by left-sided parietotemporal infarction, orofacial paresis by infarction encompassing upper motor neuron of cranial nerves, and impaired UES opening by lateral medullary infarction. Other swallowing parameters were not related to lesion topology. On posthoc analysis, pneumonia within 21 days after stroke was predicted only by insular lesion.

Conclusions—Distinct acute brain lesion locations result in characteristic swallowing disturbance patterns. Dysphagic patients with insular stroke appear to have even higher risk of pneumonia suggesting a further associated factor promoting infection in these subjects. (Stroke. 2009;40:1903-1906.)

Key Words: dysphagia ■ ischemic stroke ■ brain injuries

Dysphagia affects up to half of stroke patients and promotes pneumonia and fatal outcome.1,2 Fiberoptic endoscopic evaluation of swallowing (FEES) proved valid for both, assessing oropharyngeal dysphagia pattern and aspiration severity.1,3 Few studies examined the relationship between videoscopic dysphagia pattern and roughly classified brain lesion location and suggested differences comparing supratentorial versus brain stem infarctions, frontal versus parietotemporal and right versus left middle cerebral artery (MCA) infarctions.1,4–7 Here, we explored whether brain lesion topology relates to specific dysphagia patterns.

Patients and Methods

Study Sample

Sixty dysphagic patients (58% women) with first-ever acute ischemic stroke were prospectively studied. For reasonable stroke categorization and accurate FEES, exclusion criteria were: bilateral stroke were prospectively studied. For reasonable stroke categorization and accurate FEES, exclusion criteria were: bilateral infarctions, supratentorial infarction diameter <15 mm, severe leuencephalopathy, dementia, insufficient language comprehension, impaired vigilance, mechanical ventilation, or concomitant diseases causing dysphagia. Dysphagia was considered present at inability to swallow, food stuck in mouth/throat, cough after swallowing, impaired voice, or abnormal oximetry result on water swallowing test.2 Applying consensus criteria,2 pneumonia within 21 days after stroke was diagnosed (n=39; median latency, 3 [1–16] days). Decision on gastrostomy (n=17) was based on FEES. Brain lesion location was assessed on MRI (n=28, including all infratentorial infarctions) or CT (n=32) by a senior radiologist (A.G.) blinded to clinical findings (Table 1). MCA infarction size was rated: <33% (n=11), 33% to 66% (n=17); and >66% (n=13) of ipsilateral territory. All investigations were part of standardized routine diagnostic workup.

Assessment of Swallowing

Dysphagia was assessed by an experienced investigator (V.S.) aware of stroke location as supratentorial or infratentorial but unaware of exact lesion location and size. Clinical parameters scored (0=no, 1=mild, 2=moderate, 3=severe abnormality/absent function) were attention deficit, buccofacial apraxia, paresis of orofacial muscles involved in the oral phase of swallowing, and gag reflex. Attention deficit was considered present if the patient repeatedly delayed or failed to comply with the command to swallow, despite principal ability to swallow and absence of severe buccofacial apraxia or orofacial paresis. Buccofacial apraxia was identified by impaired performance of symbolic and nonsymbolic movements to command.3 FEES was performed using a flexible fiberoptic laryngoscope (Olympus BF P-40) with video recording during dry, cream, and liquid swallows.1,3 The rating of the most impaired swallowing with either consistency was used for further analysis. Parameters scored (0–3) were delayed or absent pharyngeal swallow, impaired (asymmetrical or reduced) pharyngeal contraction, impaired larynx elevation, and impaired opening of the upper esophageal sphincter (UES). Aspiration severity was assessed using validated scales.3,8

Statistical Analyses

Patients were classified as having abnormality of a distinct swallowing parameter at individual score of ≥1 (≥2) if mean score of all patients was <1 (≥1). Crude associations between the categorical
outcome events and each of the lesion locations present in ≥15 patients were assessed with the \( \chi^2 \) test. Because multiple comparisons were performed for 5 lesion locations, a \( P<0.01 \) was taken here as being statistically significant. Subsequently, a multivariable logistic regression model, controlling for possible confounding covariates, was fitted by forward stepwise selection from the lesion locations significant on univariate analysis to identify lesion locations independently predicting an outcome event. Analyses were performed with SPSS 15.0.

**Results**

**Swallowing Disturbance Patterns**

Buccofacial apraxia was exclusively found in left-sided MCA infarctions. Other swallowing parameters, aspiration severity, and lesion location were not related to lesion laterality. MCA infarction size did not significantly influence any swallowing parameter (Spearman test, each, \( P>0.05 \)). The Figure shows dysphagia patterns of different brain lesion locations. On

**Other Outcome Measures**

Aspiration severity and need of gastrostomy were independent of brain lesion location. PTI, however, was associated with increased risk of pneumonia (\( P=0.009 \)). On post hoc analysis subdividing PTI topology, insular infarction (n=30) predicted pneumonia (\( P=0.003 \)). Pneumonia was not related to any swallowing parameter, aspiration severity, lesion size, or laterality.

| Table 1. Demographic and Clinical Features of 60 Dysphagic Acute Stroke Patients |
|-------------------------------------------------|-----------------|-------------|-------------|-------------|-------------|-------------|-------------|
| Patient Characteristics                        | UMI             | FRI         | PTI         | BCI         | POI         | LMI         | CBI         |
| No. *                                           | 43              | 33 (5)      | 31 (4)      | 20 (4)      | 4 (3)       | 16 (7)      | 9 (1)       |
| Sex (F/M), n                                    | 28/15           | 21/12       | 20/11       | 14/6        | 2/2         | 7/9         | 4/5         |
| Age, y                                          | - Mean          | 74.6        | 74.6        | 74.4        | 72.9        | 75.8        | 59.3        | 62.7        |
|                                               | - SD            | 11.4        | 12.0        | 11.1        | 9.9         | 16.5        | 10.2        | 13.0        |
| Laterality of brain infarction R/L, n           | 16/27           | 14/19       | 9/22        | 6/14        | 1/3         | 8/8         | 5/4         |
| NIHSS score                                    | - Mean          | 14.4        | 14.3        | 15.6        | 18.1        | 11.8        | 4.9         | 4.4         |
|                                               | - SD            | 6.0         | 6.3         | 5.9         | 2.9         | 2.9         | 2.3         | 1.9         |
| Latency stroke–FEES, d                         | - Mean          | 9.3         | 9.5         | 9.9         | 10.6        | 5.5         | 8.4         | 9.1         |
|                                               | - SD            | 5.0         | 4.9         | 4.8         | 5.2         | 3.3         | 3.8         | 3.5         |
|                                               | - Range         | 1–21        | 1–21        | 2–21        | 4–21        | 2–10        | 1–13        | 2–13        |
| PAS score                                      | - Mean          | 4.4         | 4.2         | 4.4         | 4.6         | 6.0         | 4.0         | 3.9         |
|                                               | - SD            | 2.7         | 2.7         | 2.7         | 2.8         | 1.4         | 2.7         | 2.9         |
|                                               | - Range         | 1–8         | 1–8         | 1–8         | 4–7         | 1–8         | 1–8         |
| SMS score                                      | - Mean          | 1.3         | 1.1         | 1.2         | 1.4         | 2.3         | 1.3         | 1.3         |
|                                               | - SD            | 1.5         | 1.4         | 1.4         | 1.5         | 1.3         | 1.5         | 1.6         |
|                                               | - Range         | 0–4         | 0–4         | 0–4         | 0–4         | 1–4         | 0–4         | 0–4         |
| Pneumonia,† n                                  | 30 (70)         | 23 (70)     | 25 (81)     | 16 (80)     | 3 (75)      | 9 (56)      | 5 (56)      |
| Latency stroke–pneumonia, d                    | - Mean          | 3.6         | 3.5         | 3.4         | 3.5         | 5.0         | 5.4         | 6.2         |
|                                               | - SD            | 2.4         | 2.7         | 2.6         | 2.0         | 1.7         | 4.5         | 5.6         |
|                                               | - Range         | 1–12        | 1–12        | 1–12        | 1–9         | 3–6         | 1–16        | 2–16        |

UMI indicates middle cerebral artery or pontine infarction affecting the upper motor neurons of cranial nerves involved in swallowing; FRI, frontal infarction; PTI, parietotemporal infarction; BCI, infarction of basal ganglia or capsula interna; POI, pontine infarction; LMI, lateral medullary infarction; CBI, cerebellar infarction; NIHSS, National Institute of Health Stroke Scale; FEES, fiberoptic endoscopic evaluation of swallowing; PAS, Penetration-Aspiration Scale; SMS, Schröter-Morasch Aspiration Scale.

*No. of patients with combined (isolated) infarction in the referring brain region.
†No. (percentage proportion) of patients with pneumonia in that group.
Discussion

Data obtained in this study show that distinct acute brain lesion locations result in characteristic swallowing disturbance patterns. To our knowledge, this is the first study comparing clinical and videoendoscopic dysphagia patterns in acute stroke patients with a number of predefined lesion locations. Even though we missed patients with subclinical dysphagia and patients with extended infarctions causing

**Table 2. Multivariate Prediction Models: Logistic Regression Analysis**

<table>
<thead>
<tr>
<th>Outcome Event/Brain Lesion Location</th>
<th>OR (95% CI)</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attention deficit (33 patients)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parietotemporal*</td>
<td>44 (9.7–207)</td>
<td>85%</td>
<td>89%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Buccofacial apraxia (23 patients)</td>
<td></td>
<td></td>
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<tr>
<td>Parietotemporal left-sided†</td>
<td>378 (32–4424)</td>
<td>91%</td>
<td>97%</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Orofacial paresis (53 patients)</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Upper motor neuron</td>
<td>26 (2.8–239)</td>
<td>81%</td>
<td>86%</td>
<td>0.004</td>
</tr>
<tr>
<td>Impaired UES opening (9 patients)</td>
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<tr>
<td>Lateral medulla oblongata‡</td>
<td>8.2 (1.7–39)</td>
<td>67%</td>
<td>80%</td>
<td>0.008</td>
</tr>
<tr>
<td>Pneumonia (39 patients)</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Parietotemporal involving insula§</td>
<td>5.7 (1.7–19)</td>
<td>64%</td>
<td>76%</td>
<td>0.004</td>
</tr>
</tbody>
</table>

UES indicates upper esophageal sphincter.

*Frequency of attention deficit in isolated and combined parietotemporal lesion: 100%, 90%.
†Frequency of buccofacial apraxia in isolated and combined left parietotemporal lesion: 100%, 95%.
‡Frequency of impaired UES opening in isolated and combined lateral medullary lesion: 43%, 38%.
§Result of post hoc analysis.
impaired degree of dysphagia, we feel confident that the usually moderate
degree of dysphagia in our cohort promoted recognizability of
lesion-specific swallowing disorder patterns. Because in 32
patients no MRI but only CT was performed, we cannot
entirely exclude that we have overlooked infratentorial in-
farctions cooccurring with MCA infarction.

Earlier studies suggested right-left differences, with pro-
longed prepharyngeal response time in left-hemispheric and
more severe pharyngeal dysfunction in right-hemispheric stroke.8,9 Here, buccofacial apraxia was related to lesion
laterality but no other swallowing parameter. This does not
contradict the fact that pharyngeal and esophageal muscles
are represented asymmetrically between the two hemispheres,
with most individuals having a “dominant” swallowing hemi-
sphere, because laterality of the latter is independent of
handedness and equally right versus left distributed.1 Our
results concur with left-hemispheric dominance in the early
stage of volitional swallowing,9 and key role of left parietal
cortex in ideomotor apraxia.10

The found association of PTI (on any side) with attention
deficit underscores significance of bilateral parietal cortex in
control of attention, even in tasks involving neither spatial nor
visual cognition.11 Confirming earlier findings,6 all patients
with lateral medullary infarction had pharyngeal dysfunction
which, however, was also frequent and more severe in
hemispheric infarctions. In contrast, we found a specific
association of lateral medullary infarction with impaired UES
opening. This agrees with previous case reports and can be
explained by affection of the dorsal nucleus ambiguus that
contains premotor neurons regulating the pharyngolaryngeal
and esophageal motor system.12

Interestingly, pneumonia in our cohort was predicted by
insular stroke which implies association with a factor other
than formal dysphagia, further increasing risk of pneumonia.
Insular infarction can prolong dysphagia and cause sympa-
thetic hyperactivity.13,14 Both conditions may promote pneu-
monia, the latter via immunosuppression.15 Further studies
are warranted to find out whether dysphagic patients with
acute insular stroke might benefit from prophylactic antibi-
otic treatment.

Disclosures

None.

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