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 acute 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. Fiberoptic endoscopic evaluation of swallowing (FEES) proved valid for both, assessing oropharyngeal dysphagia pattern and aspiration severity. 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.

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 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. Applying consensus criteria, 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. FEES was performed using a flexible fiberoptic laryngoscope (Olympus BF P-40) with video recording during dry, cream, and liquid swallows. 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.

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

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outcome events and each of the lesion locations present in ≥15 patients were assessed with the χ² 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 univariate analysis, patients with upper motor-neuron, frontal, and parietotemporal (PTI) infarction had increased risk of attention deficit (each, P<0.001), buccofacial apraxia and orofacial paresis (each, P<0.005). Infarction of basal ganglia/capsula interna was associated with attention deficit and buccofacial apraxia (each, P<0.005). Lateral medullary infarction caused impaired UES opening (P=0.003). Table 2 summarizes results of multivariable analysis.

### 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.
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 videofiberoptic 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>
<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>
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<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>
<td></td>
<td></td>
<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.
impairment of vigilance, 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 infarctions cooccurring with MCA infarction.

Earlier studies suggested right-left differences, with prolonged prepharyngeal response time in left-hemispheric and more severe pharyngeal dysfunction in right-hemispheric stroke. 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 hemisphere, because laterality of the latter is independent of handedness and equally right versus left distributed. Our results concur with left-hemispheric dominance in the early stage of volitional swallowing, and key role of left parietal cortex in ideomotor apraxia.

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. Confirming earlier findings, 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.

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 sympathetic hyperactivity. Both conditions may promote pneumonia, the latter via immunosuppression. Further studies are warranted to find out whether dysphagic patients with acute insular stroke might benefit from prophylactic antibiotic treatment.

Disclosures

None.

References

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