Videofluoroscopic Assessment of Pharyngeal Stage Delay Reflects Pathophysiology After Brain Infarction

Hideaki Miyaji, MD; Toshiro Umezaki, MD, PhD; Kazuo Adachi, MD; Motohiro Sawatsubashi, MD, PhD; Hideyuki Kiyohara, MD; Takashi Inoguchi, MD; Satoshi To, MD, PhD; Shizuo Komune, MD, PhD

Objectives/Hypothesis: The pathophysiology of dysphagia caused by brain infarction varies with the site of the lesion in the brain. Patients with suprabulbar lesions have demonstrated delayed triggering of pharyngeal stage including delayed laryngeal elevation. Patients with severe pharyngeal stage delay have a high risk of intractable aspiration to the lower respiratory tract. Despite this, few studies have compared the pharyngeal stage delay with the lesion site. We defined a new temporal parameter of the pharyngeal stage delay to assess laryngeal elevation delay against the bolus inflow into the pharyngeal space. This study aimed to elucidate whether this parameter of pharyngeal stage delay is clinically useful to assess the pathophysiology of brain lesions after brain infarction.

Study Design: Case-control study.

Methods: Videofluoroscopic assessment of swallowing examinations was performed from January 7, 2000 to March 29, 2011 at Kyushu University Hospital. We evaluated the pharyngeal stage delay using motion analysis on videofluoroscopic swallowing examination in patients with normal swallowing and brain infarction patients divided into pathophysiologic lesion groups. Laryngeal elevation delay time and pharyngeal delay time were analyzed.

Results: Significant differences in laryngeal elevation delay time were observed between each pathophysiologic lesion group. However, pharyngeal delay time remained similar among groups. Brain infarctions of corticobulbar tract and basal ganglion were significantly associated with laryngeal elevation delay time prolongation.

Conclusions: Laryngeal elevation delay time with low-viscosity contrast medium is a recommended parameter to discriminate the corticobulbar tract and the basal ganglion lesion.

Key Words: Swallowing, laryngeal elevation delay time, pharyngeal delay time, central pattern generator, deglutition, deglutition disorders.

Level of Evidence: 3b

INTRODUCTION

Suprabulbar impairment has been observed to cause a delay in triggering the swallowing reflex, which results in delayed laryngeal elevation and a prolonged pharyngeal stage.1–4 Additionally, subjects with suprabulbar lesions have increased recognition thresholds in the laryngopharyngeal mucosa and displayed aspiration.5 As severe delay of the pharyngeal stage causes critical problems such as intractable aspiration to the lower respiratory tract, some previous studies have investigated the relationship between prolonged pharyngeal stage time and presence or absence of aspirations from videofluoroscopic examinations (VFSE).3 However, few studies have examined the relationship between the temporal parameters of pharyngeal stage delay and brain lesions after brain infarction.2,6 Pharyngeal delay time (PDT) has been used to measure the delay of the pharyngeal stage in previous reports.2,6 Using a parameter PDT, it was previously reported that unilateral basal ganglia and internal capsule stroke subjects exhibited a longer PDT on thicker boluses (paste and cookie) than on thin liquid,2 whereas normal subjects exhibited no significant difference between PDT on thicker boluses and thin liquid. They suggested that these differences in the stroke versus normal subjects regarding the bolus viscosity may reflect mild unilateral damage in the sensorimotor pathways from the cortex to brainstem, which pass through the basal ganglia and internal capsule. However, the concept of PDT did not conform to the clinical fact that high-viscosity liquids decrease the risk of aspiration in the stroke patients with dysphagia. Because the position of the larynx was easy to range as described in the Mendelson maneuver,7 PDT was not defined considering the rapid laryngeal elevation stage, which is directly linked to swallowing reflex. Therefore, PDT did not appear to assess the delay of the pharyngeal stage.

Consequently, we defined a new parameter—laryngeal elevation delay time (LEDT). LEDT was defined to

From the Department of Otorhinolaryngology, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan.

Editor’s Note: This Manuscript was accepted for publication June 20, 2012.

This study was supported by Grant-in-Aid for Scientific Research (C) (22591909).

The authors have no other funding, financial relationships, or conflicts of interest to disclose.

Send correspondence to Hideaki Miyaji, MD, Department of Otolaryngology, Faculty of Medicine, Graduate School of Medical Sciences, Kyushu University, 3-1-1 Maidashi, Higashi-ku, Fukuoka 812-8582, Japan. E-mail: miyaji1023@gmail.com

DOI: 10.1002/lary.23588

Laryngoscope 122: December 2012
TABLE I.

Patient Groups Based on Magnetic Resonance Imaging Findings (N = 43).

<table>
<thead>
<tr>
<th>Infarctions</th>
<th>Suprabulbar Group (n = 27)</th>
<th>Bulbar Group (n = 6)</th>
<th>Mixed Group (n = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral cortex (5, 2, 1)</td>
<td>Medulla oblongata (2, 3, 1)</td>
<td>Medulla oblongata (5, 5, 0)</td>
<td></td>
</tr>
<tr>
<td>Internal capsule (1, 3, 0)</td>
<td>Cerebellum (2, 0, 3)</td>
<td>Cerebral cortex (1, 0, 0)</td>
<td></td>
</tr>
<tr>
<td>Basal ganglion (1, 3, 19)</td>
<td>Internal capsule (1, 0, 1)</td>
<td>Basal ganglion (1, 1, 6)</td>
<td></td>
</tr>
<tr>
<td>Thalamus (1, 1, 14)</td>
<td>Thalamus (1, 0, 2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pons (2, 5, 9)</td>
<td>Pons (1, 3, 4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cerebellum (6, 4, 1)</td>
<td>Midbrain (0, 1, 0)</td>
<td>Cerebellum (3, 2, 1)</td>
<td></td>
</tr>
</tbody>
</table>

(A, B, C) indicate (right hemisphere, left hemisphere, bilateral hemisphere). Twenty-seven patients in the suprabulbar group were affected by infarctions of cerebral cortex, internal capsule, basal ganglion, thalamus, pons, and cerebellum. Six patients in the bulbar group demonstrated infarctions of the medulla oblongata and cerebellum. Ten patients in the mixed group presented both suprabulbar and bulbar infarctions. In this study, the cerebral cortex lesions included precentral gyrus, supplementary motor area, somatosensory cortex, parietal cortex, cingulate cortex, temporal cortex, insula, and frontal operculum lesions. As there was no incidence of dysphagia in patients with cerebellar lesions in a previous systematic review and meta-analysis study,7 we ignored cerebellar lesions.

TABLE II.

Dysphagia Severity According to Presence or Absence of Aspiration, State of Consciousness, and Nutrition Management.

<table>
<thead>
<tr>
<th></th>
<th>Normal-Swallowing Patients (n = 15)</th>
<th>Suprabulbar Group (n = 27)</th>
<th>Bulbar Group (n = 6)</th>
<th>Mixed Group (n = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean ± SD), yr</td>
<td>69.5 ± 10.8</td>
<td>71.1 ± 6.8</td>
<td>56.8 ± 13.8</td>
<td>68 ± 9.3</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>10/5</td>
<td>20/7</td>
<td>6/0</td>
<td>8/2</td>
</tr>
<tr>
<td>Stroke phase in MRI</td>
<td>10/5</td>
<td>20/7</td>
<td>6/0</td>
<td>8/2</td>
</tr>
<tr>
<td>Presence or absence of aspiration (no aspiration/aspiration)</td>
<td>15/0</td>
<td>21/6</td>
<td>6/0</td>
<td>2/8</td>
</tr>
<tr>
<td>State of consciousness</td>
<td>15/0</td>
<td>21/6</td>
<td>6/0</td>
<td>9/1</td>
</tr>
<tr>
<td>Nutrition management</td>
<td>15/0/0</td>
<td>22/4/1</td>
<td>2/0/4</td>
<td>3/1/6</td>
</tr>
</tbody>
</table>

**Subjects**

A case-control study of videofluoroscopic assessment of swallowing examinations was performed from January 7, 2000 to March 29, 2011, at Kyushu University Hospital. The total number of VFSEs was 3,180, of which 63 were brain infarction patients. Twenty of these 63 patients were excluded from the study because they did not have brain MRI performed and/or had clouded consciousness, Japan Coma Scale ≥10 at the time of VFSE and/or had been assessed by VFSEs in acute phase (<8 days), and/or had nasal feeding tubes at the time of VFSE and/or had a tracheostomy tube via the tracheostomy orifice at the time ofVFSE. Patients were also excluded from the study if they had a history of neurological disease or an intercurrent illness that could cause dysphagia. The cohort included 43 subacute and chronic brain infarction patients with dysphagia (34 male, 9 female; mean age, 68.4 years; age range, 36–83 years). All brain infarction patients had infarctions confirmed by their cranial MRI and diffusion weight imaging (DWI) scan by radiologists. The cohort also included 15 patients with normal swallowing (10 male, 5 female; mean age 69.5 years; age range, 39–83 years) who had been assessed for organic lesions in the oral cavity, pharynx, and/or larynx as part of a preoperative inspection. These patients were recruited as normal controls demonstrating no difficulties swallowing, no neurologic disorders, no lesions in the oropharyngeal space, and normal findings in VFSE; radiologists also confirmed no cerebrovascular accident in both their cranial MRIs and DWI scan. We ignored leukoaraiosis in this study. All procedures in this study were approved by the ethics committee of Kyushu University.

In this study, the pathophysiology of dysphagia caused by brain infarction was divided into three categories: 1) bulbar, caused by impairment of the bulbar neural structures including the CPG; 2) suprabulbar, caused by impairment of the cortico-bulbar tract and the basal ganglion; and 3) mixed, bulbar + suprabulbar. We divided all 58 patients into four groups: normal swallowing (n = 15), suprabulbar (n = 27), bulbar (n = 6), and mixed (n = 10) on the basis of MRI findings (Table I). As there was no incidence of dysphagia in patients with cerebellar delay in VFSE using both parameters—PDT and LEDT—in patients with normal swallowing and brain infarction patients divided into pathophysiologic lesion groups by magnetic resonance imaging (MRI) findings.
lesions in a previous systematic review and meta-analysis study, we ignored cerebellar lesions. We checked the severity of dysphagia for all participants according to state of consciousness, presence or absence of aspirations, and nutrition management (Table II).

**Videofluoroscopic Protocol**

Each study was recorded at 30 frames/second with a HR-VT700 (Victor Company of Japan, Ltd., Yokohama, Japan). All subjects swallowed 10 mL of a thin liquid (iohexol: nonionic watersoluble iodine-containing contrast medium) twice and were viewed radiographically in the lateral plane by four to five reviewers. This thin liquid was chosen because its tissue invasion is considerably less than that of barium, and it is less likely to cause pneumonia and acute inflammation of the bronchial wall.

**Definition of Terms**

LEDT and PDT are quantitative measurements of the duration of the pharyngeal stage. We defined LEDT as the time from arrival of the bolus head at the bottom of either side of the pyriform sinus until the first highest position of laryngeal elevation (Fig. 1A). PDT was defined as the time from arrival of the bolus head at the point where the lower rim of the mandible crosses the tongue base until the first laryngeal elevation (Fig. 1B,C).

**Data Analysis**

The series of swallowing in videotapes were digitized for further analysis. The data were analyzed in samples without brain infarction localization noted. All liquid swallows were analyzed using DIPP-Motion Pro 2D (DITECT Corp., Tokyo, Japan). This software package includes tools that allow time and distance to be measured across image sequences.

First, we identified three points (anterior superior margin of vertebra C3, anterior inferior margin of vertebra C5, and vocal cord level) on some frames of the sequence. Then computer-assisted measurement tracked these points across all frames of the image sequence using spline interpolation. Processing time for an entire image sequence of about 100 frames was less than 5 seconds. Second, we determined the laryngeal elevation curve, with the line from C3 to C5 as the y-axis and the line perpendicular to the y-axis at C5 as the x-axis. The time course of the distance of the vocal cord from its resting position more than 2 seconds before the onset of the laryngeal elevation were calculated with reference to the x-axis and y-axis to exclude head movement as a cause of low reproducibility. Third, we identified four points on the laryngeal elevation curve: arrival of the bolus head at the point where the lower rim of the mandible crosses the tongue base (A point), beginning of laryngeal elevation (B point), arrival of the bolus head at the bottom of either side of the pyriform sinus (P point), and the first highest position of laryngeal elevation (M point). Finally, times for the above-mentioned points were used to calculate LEDT and PDT.

![Diagram of laryngeal elevation curves](image-url)
TABLE III. Variables of Each Brain Lesion Between the Normal LEDT Group and Prolonged LEDT Group.

<table>
<thead>
<tr>
<th>Variables of Each Brain Lesion</th>
<th>LEDT &lt; 0.33 s (n = 18)</th>
<th>LEDT ≥ 0.33 s (n = 40)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean ± SD)</td>
<td>66.4 ± 14.0</td>
<td>69.7 ± 7.3</td>
<td>.866</td>
</tr>
<tr>
<td>Sex (male/female)</td>
<td>14/4</td>
<td>30/10</td>
<td>.819</td>
</tr>
<tr>
<td>Aspiration (no aspiration/aspiration)</td>
<td>15/3</td>
<td>25/15</td>
<td>.113</td>
</tr>
<tr>
<td>State of consciousness (clear sensorium/10 &gt; JCS &gt; 1)</td>
<td>17/1</td>
<td>34/6</td>
<td>.307</td>
</tr>
<tr>
<td>Nutrition management (oral intake/oral intake + supplemental nutrition/parenteral alimentation)</td>
<td>15/0/3</td>
<td>27/5/6</td>
<td>.288</td>
</tr>
<tr>
<td>Corticobulbar tract*</td>
<td>17/1/0</td>
<td>11/6/23</td>
<td>.000</td>
</tr>
<tr>
<td>Parietal cortex*</td>
<td>17/1/0</td>
<td>33/6/1</td>
<td>.222</td>
</tr>
<tr>
<td>Cingulate cortex*</td>
<td>18/0/0</td>
<td>39/1/0</td>
<td>.502</td>
</tr>
<tr>
<td>Insular cortex*</td>
<td>17/1/0</td>
<td>37/3/0</td>
<td>.789</td>
</tr>
<tr>
<td>Temporal cortex*</td>
<td>17/1/0</td>
<td>39/1/0</td>
<td>.559</td>
</tr>
<tr>
<td>Basal ganglion*</td>
<td>17/0/1</td>
<td>10/6/24</td>
<td>.000</td>
</tr>
<tr>
<td>Thalamus*</td>
<td>17/0/1</td>
<td>22/3/15</td>
<td>.004</td>
</tr>
<tr>
<td>Cerebellum*</td>
<td>14/3/1</td>
<td>21/15/4</td>
<td>.082</td>
</tr>
<tr>
<td>Medulla oblongata*</td>
<td>14/3/1</td>
<td>28/12/0</td>
<td>.634</td>
</tr>
</tbody>
</table>

*No lesion/unilateral hemisphere/bilateral hemisphere. LEDT = laryngeal elevation delay time; SD = standard deviation; JCS = Japan Coma Scale.

The Mann-Whitney U test was used to compare the patients’ ages, nutrition management, and the degree of each brain lesion between the normal LEDT group and prolonged LEDT group. Pearson’s χ² test was used to analyze differences in the proportion of sex, no aspiration/aspiration, and clear sensorium/10 > Japan Coma Scale ≥ 1 between normal LEDT group and prolonged LEDT group. As the bilateral lesions of swallowing-related cortex result in severe dysphagia, each infarction lesion was divided into no lesion, unilateral hemisphere lesion, or bilateral hemisphere lesion. The independent variables—aspiration, corticobulbar tract, parietal cortex, basal ganglion, thalamus, and cerebellum—had P value < .25. In this study, the corticobulbar tract lesions included precentral gyrus, supplementary motor area, frontal operculum, internal capsule, midbrain, and pons.

The Mann-Whitney U test was used to compare the patients’ ages, nutrition management, and the degree of each brain lesion between the normal LEDT group and prolonged LEDT group. Pearson’s χ² test was used to analyze differences in the proportion of sex, no aspiration/aspiration, and clear sensorium/10 > Japan Coma Scale ≥ 1 between normal LEDT group and prolonged LEDT group. As the bilateral lesions of swallowing-related cortex result in severe dysphagia, each infarction lesion was divided into no lesion, unilateral hemisphere lesion, or bilateral hemisphere lesion. The independent variables—aspiration, corticobulbar tract, parietal cortex, basal ganglion, thalamus, and cerebellum—had P value < .25. In this study, the corticobulbar tract lesions included precentral gyrus, supplementary motor area, frontal operculum, internal capsule, midbrain, and pons.

The Mann-Whitney U test was used to compare the patients’ ages, nutrition management, and the degree of each brain lesion between the normal LEDT group and prolonged LEDT group. Pearson’s χ² test was used to analyze differences in the proportion of sex, no aspiration/aspiration, and clear sensorium/10 > Japan Coma Scale ≥ 1 between normal LEDT group and prolonged LEDT group. As the bilateral lesions of swallowing-related cortex result in severe dysphagia, each infarction lesion was divided into no lesion, unilateral hemisphere lesion, or bilateral hemisphere lesion. The independent variables—aspiration, corticobulbar tract, parietal cortex, basal ganglion, thalamus, and cerebellum—had P value < .25. In this study, the corticobulbar tract lesions included precentral gyrus, supplementary motor area, frontal operculum, internal capsule, midbrain, and pons. calculate the two parameters: PDT = B − A, LEDT = M − P. Any up or down movements of the larynx before the onset of the rapid laryngeal elevation and spillage, defined as the head of the bolus entering the bottom of either side of the pyriform sinus for more than 1 second before the swallowing response occurred, were ignored. The worse of the two swallows was used for further analysis.

**Statistical Analysis**

A P value < .05 was considered to indicate statistical significance. Descriptive and comparative analyses between LEDT and PDT were made using IBM SPSS version 19 (SPSS Inc., IBM, Somers, NY). The Mann-Whitney U test was used to compare patient ages between the normal-swallowing and brain infarction groups. Fisher exact test was used to explore differences in the proportion of aspirations and subacute and chronic infarctions among each brain infarction group, and sex distribution between the normal-swallowing and brain infarction groups. Differences in age, LEDT, and PDT among the normal-swallowing group, suprabulbar group, bulbar group, and the mixed group were evaluated with one-way analysis of variance (ANOVA).

Further, power analysis of LEDT and PDT was performed between the normal-swallowing and brain infarction groups and the powers of test were calculated by two-sided test to determine the smallest difference for which the available number of subjects would show statistically significant results. The receiver operating characteristic (ROC) curve was used to assess LEDT and PDT diagnostic accuracy.13 An optimal cutoff point to identify suprabulbar lesions in the normal-swallowing group and the suprabulbar group was determined mainly on the basis of the maximum values of the Youden index, calculated by (sensitivity + specificity − 1) and the minimum values of the

![Fig. 2. (A) Box plots of laryngeal elevation delay time (LEDT) with swallowing 10 mL of a thin liquid in each group. LEDT ranged from 0.20 to 0.97 seconds. Significant differences were found between the normal-swallowing group (n = 15) and the suprabulbar group (n = 27) (P < .017) and between the normal-swallowing group (n = 15) and the mixed group (n = 10) (P < .008), between the suprabulbar group (n = 27) and the bulbar group (n = 6) (P = .002), and between the bulbar group (n = 6) and the mixed group (n = 10) (P = .017). LEDT remained similar between the normal-swallowing group (n = 15) and the bulbar group (n = 6) (P = .849) and between the suprabulbar group (n = 27) and the mixed group (n = 10) (P = .280). (B) Box plots of pharyngeal delay time (PDT) with swallowing 10 mL of a thin liquid in each group. PDT ranged from −0.43 to 0.7 seconds. Significant differences among the normal-swallowing group, suprabulbar group, bulbar group, and the mixed group were evaluated by using one-way analysis of variance. Unlike LEDT, no significant differences in PDT were observed between any two groups. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]
square root of \((1 - \text{sensitivity})^2 + (1 - \text{specificity})^2\), which indicates the minimum distance from the upper left corner to the point on the ROC curve.\(^\text{14}\) Univariate analyses were performed to identify the independent variables between the normal LEDT group and prolonged LEDT group. The variables of each brain lesion were defined as 0, no lesion; 1, unilateral hemisphere; and 2, bilateral hemisphere (Table III). Then the independent variables with \(P < .25\) were used in following multiple logistic regression analyses. Multiple logistic regression analyses using a stepwise technique were performed to identify the independent variables significantly associated with LEDT prolongation. Odds ratio (OR) was calculated with 95% confidence interval (CI).

RESULTS

Intergroup Analyses

**Laryngeal elevation delay time.** Significant differences among the normal-swallowing group, suprabulbar group, bulbar group, and the mixed group were evaluated by using one-way ANOVA. LEDT ranged from 0.20 to 0.97 seconds. Significant differences were observed between the normal-swallowing group and the suprabulbar group and mixed group, between the suprabulbar group and the bulbar group, and between the bulbar group and the mixed group (all \(P < .05\)) (Fig. 2A).

**Pharyngeal delay time.** PDT ranged from -0.43 to 0.7 seconds and remained similar across groups (Fig. 2B).

**Power analysis of LEDT and PDT between the normal-swallowing and brain infarction groups.** The power of the test between the normal-swallowing group (\(n = 15\)) and the suprabulbar group (\(n = 27\)) were 0.99 and 0.36, respectively. The power of the test between the suprabulbar group (\(n = 27\)) and the bulbar group (\(n = 6\)) were 0.77 and 0.34, respectively. The power of the test between the normal-swallowing group (\(n = 15\)) and mixed group (\(n = 10\)) were 0.95 and 0.03, respectively. The power of the test between the bulbar group (\(n = 6\)) and the mixed group (\(n = 10\)) were 1.00 and 0.06, respectively.

Characteristic Analysis of LEDT and PDT

Figure 3A shows the ROC curve and area under the curve (AUC) of LEDT and PDT to identify the suprabulbar group from among the normal-swallowing group and the suprabulbar group. The AUC of LEDT was 0.922,
whereas the AUC of PDT was 0.657. The cutoff value of LEDT to identify the patients with suprabulbar lesions from among the normal-swallowing and suprabulbar groups was 0.31667 seconds. Figure 3B shows the ROC curve and AUC of LEDT and PDT to identify the bulbar group from among the normal-swallowing and bulbar group. The AUC of LEDT and PDT were 0.644 and 0.506, respectively. Figure 3C shows the ROC curve and AUC of LEDT and PDT to identify the mixed group from among the normal-swallowing and mixed group. The AUC of LEDT and PDT were 0.990 and 0.487, respectively.

### Univariate Analyses and Multiple Logistic Regression Analyses

After univariate analyses, the independent variables—aspiration, corticobulbar tract, parietal cortex, basal ganglion, thalamus, and cerebellum—associated with LEDT prolongation (≥0.33 seconds) had \( P < .25 \) (Table III). According to multiple logistic regression analyses, brain infarction of the corticobulbar tract and basal ganglion was significantly associated with LEDT prolongation (OR, 12.871, 4.985; \( P = .020, .019; 95\% \) CI, 1.493-110.976, 1.304-19.052).

### DISCUSSION

To our knowledge, this study is the first to compare the videofluoroscopic assessment of pharyngeal stage delay with a low-viscosity contrast medium between age- and sex-matched patients with normal swallowing and with brain infarction, divided into four pathophysiologic lesion groups confirmed by MRI findings. This is also the first study in which videofluoroscopic assessment was done with computer assistance to negate head movement. Significant differences in LEDT were identified between the normal-swallowing group and the suprabulbar group and mixed group, the suprabulbar group and the bulbar group, and the bulbar group and the mixed group. According to multiple logistic regression analyses, brain infarction of corticobulbar tract and basal ganglion was significantly associated with LEDT prolongation. These results suggest that LEDT with a low-viscosity contrast medium is a recommended parameter to discriminate the corticobulbar tract and the basal ganglion lesion.

Although PDT has been used as a parameter of pharyngeal stage delay, we did not find PDT to significantly differ between groups. This may be because the starting point of PDT is far from the laryngeal epithelium, whereas the starting point of LEDT is near the arytenoid region. In a previous study, Shin et al. proposed that highly sensitive mechanoreceptors, chemoreceptors, and polymodal receptors are present in the laryngeal mucosa and particularly rich on the laryngeal surface of the epiglottis and arytenoid region.\(^\text{15}\) As Palmer et al. reported, the position of the leading edge of the barium at the onset of a rapid hyoid elevation was altered from oral cavity to valleculae by volition\(^\text{16}\); this may be because the starting points of PDT were altered...
CONCLUSION

This videofluoroscopic assessment with a new temporal parameter of pharyngeal stage delay is the first study to demonstrate multiple logistic regression analyses of each infarction lesion using only consistency of the contrast medium. Laryngeal elevation delay time with low-viscosity contrast medium is a recommended parameter to discriminate the corticobulbar tract and the basal ganglion lesion.

BIBLIOGRAPHY