Mechanisms of Dysphagia in Suprabulbar Palsy With Lacunar Infarct

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Background and Purpose—The objective of the present study was to investigate the neural mechanisms of dysphagia in suprabulbar palsy (SBP) with multiple lacunar infarct.

Methods—We evaluated the swallowing disorders of patients with SBP (n=34) and age-matched healthy control subjects (n=35) by means of an electrophysiological method that recorded the oropharyngeal swallowing patterns. With this method, dysphagia limit, the triggering of voluntarily initiated swallows, duration of laryngeal relocation time, and total duration of oropharyngeal swallowing were recorded and measured. In addition, the EMG behavior of the cricopharyngeal (CP) muscle of the upper esophageal sphincter was also assessed.

Results—In patients with SBP, the dysphagia limit in all except 1 patient was pathological with limits of <20-mL bolus volume, which is contrary to normal subjects, in whom the dysphagia limit exceeds the 20-mL bolus volume. Either triggering of swallowing reflex was delayed (P<0.04), or the swallow could hardly be triggered in 7 patients on the voluntary attempts for 3 mL water. Whenever the reflex swallowing could be triggered, it was slow and prolonged (P<0.01). The CP muscle of the upper esophageal sphincter appeared to have become hyperreflexic and incoordinated with laryngeal movements during swallowing.

Conclusions—It was proposed that the progressive involvement of the excitatory and inhibitory corticobulbar fiber systems linked with the bulbar swallowing center is mainly responsible for the triggering difficulties of the swallowing reflex and for the hyperreflexic/incoordinated nature of the CP sphincter. In addition, the dysfunction of the extrapyramidal system has a specific role in the slowing of oropharyngeal swallowing and the accumulation of saliva in the mouth.

(S. Stroke. 2000;31:1370-1376.)

Key Words: dysphagia ■ electromyography ■ lacunar infarction ■ paralysis, bulbar ■ pharyngeal muscles

Suprabulbar palsy (SBP) is a well known clinical syndrome that results from various pathologies. Multiple lacunar infarcts are the most frequently encountered causes of SBP.1 Problems associated with swallowing are common in patients with SBP. The severity of dysphagia varies from subclinical disorders to advanced clinical manifestations that require nonoral feeding. Despite the high incidence and long-standing course of dysphagia among patients with SBP that result from multiple lacunar infarcts, the mechanisms of swallowing function in these patients have scarcely been studied. On the other hand, many aspects of dysphagia associated with acute stroke have been well documented in recent years.2-8 In acute stroke with an identified location of the infarct or hemorrhage, dysphagia can readily be evaluated due to the restricted and solitary involvement within the central nervous system. However, in patients with SBP with lacunar infarcts, the multiple involvement of various ascending or descending long tracts and local neuronal circuits in the central nervous system has made it difficult to analyze and to understand the nature of swallowing disorders. In the present study, the mechanisms of dysphagia in patients with SBP with lacunar state were investigated clinically, and the electrophysiological methods used to assess deglutition are described.

Subjects and Methods

Thirty-four patients with SBP (24 men and 10 women) and 35 age-matched healthy control subjects (23 men and 12 women) participated in the study. SBP with multiple lacunar state was diagnosed according to the following criteria: (1) dysphagia, (2) dysphonia or dysarthria, (3) forced laughing/crying (emotional lability), (4) brisk brain stem reflexes, including glabellar and mandibular reflexes, (5) paresis of the tongue, (6) saliva accumulation in the mouth, (7) signs of long tract involvement, (8) high blood pressure (arterial hypertension), (9) history of previous cerebrovascular attacks, and (10) CT scan or MRI with positive signs of multiple lacunar infarcts. Although criteria 1, 7, 8, 9, and 10 were unequivocally found in all patients who were investigated, the presence of the other criteria varied from patient to patient. The patients’ ages ranged between 32 to 75 years, with a mean age of 61.9 years. The
average duration of SBP since the onset of incident was 36 months and ranged from 1 month to 9 years. Clinical symptoms and dysphagia were stable in 13 patients, had shown some improvement in 3, and were clinically progressive in the remaining 18 patients. In 11 patients, there was a history of only 1 previous cerebrovascular attack, but in the remaining 23 patients, there had been 2 or more previous attacks. The clinical signs and symptoms associated with dysphagia are documented in Results. For the 35 age-matched control subjects, the mean age was 59.6 and ranged from 30 to 75 years.

Clinically, the degree of dysphagia was graded as follows: 1 indicates no clinical signs and symptoms of dysphagia; 2, very mild dysphagia suspected from clinical examination, but the patient never complained of any swallowing difficulty; 3, patient had complaints of dysphagia that were supported by other clinical signs, but nonoral feeding was not necessary at the time of investigation; and 4, patient had distinct clinical signs and symptoms of dysphagia, including aspiration, and dysphagia was sufficiently severe to necessitate nonoral feeding.

The electrophysiological methods used in the present study were previously described in detail. In brief, during swallowing, the EMG activity was recorded on an EMG apparatus (Medelec MS-20) by means of bipolar silver-chloride EEG electrodes taped under the chin on the mylohyoid-geniohyoid-anterior digastric muscle complex (referred to as the submental EMG, or SM-EMG). The EMG signals were filtered (band pass 0.01 to 20 Hz), amplified, rectified, and integrated. For the detection of laryngeal upward and downward movements, a mechanical sensor that consisted of a simple piezoelectric wafer with a small rubber bulge affixed to it at its center was placed on the coniotomy region between the cricoid and thyroid cartilages on the midline. The output from the sensor was fed into a separate channel of the EMG apparatus before being amplified and band filtered (band pass 0.01 to 20 Hz). During each swallow, the sensor gave 2 deflections of generally opposing polarity. The first deflection was used to trigger the delay line circuitry of the recording apparatus, so that all signals related to swallowing were time locked to the same instant.

In 19 patients (15 men and 4 women, age range 32 to 75 years) and 21 control subjects (10 men and 11 women, age range 30 to 75 years), in addition to the SM-EMG, the EMG activity of the cricopharyngeal (CP) muscle of the upper esophageal sphincter was recorded. The CP-EMG was obtained with a concentric needle electrode (Medelec disposable needle electrode DMC-37) that was inserted through the skin at the level of the cricoid cartilage and was advanced laterally in the posterior medial direction until the CP muscle was reached. The CP-EMG signals were also fed to the EMG apparatus before being filtered, amplified, rectified, and integrated.

Each subject sat on an examination chair and was requested to hold his or her head in a natural upright position. The swallows were initiated by 1 or 3 mL volume of tap water introduced through a disposable syringe. The water was positioned on the tongue while the subject was initiated by 1 or 3 mL volume of tap water introduced through a disposable syringe. The water was positioned on the tongue while the subject was attempting to swallow 10 mL water.

To investigate the “piecemeal deglutition” and dysphagia limit, an identical recording procedure was used with the sweep duration set at 10 seconds and the delay line set at 2 seconds. All subjects were given 1, 3, 5, 10, 15, and 20 mL water, and for each volume, the oscilloscopic traces were initiated at the examiner’s command to “swallow.” During these long sweeps, the laryngeal sensor signals and the integrated SM-EMG activity were collected at the beginning of the sweep, immediately after the command to swallow. Normally, with volumes up to 20 mL, all material could be swallowed in 1 attempt, as seen in the early segments of 8-second-long traces. However, when the amount of water exceeded 20 mL, piecemeal deglutition was usually observed. Any duplication or multiplication of a swallowing attempt with ≤20 mL water within the 8-second period after the first swallow was designated as the “dysphagia limit” and regarded as pathological.

Statistical Analysis
Levene’s F test was used for ANOVA between the patient and control subject groups. The mean and SEM values were calculated for all quantities measured, and the Student’s t test was performed to assess the differences in swallowing-related variables between the groups. The Mann-Whitney U test was used for comparison of normally distributed values. A value of P<0.05 was considered to indicate a significant difference.

The study was approved by the ethics committee of our university hospital, and an informed consent was obtained from each participant.

Results
Clinical Findings Associated With Dysphagia
In 34 patients with SBP, clinical assessment revealed that the degree of dysphagia varied among patients and that the signs and symptoms could be divided into several major categories. The most common complaints and the clinical findings associated with dysphagia are summarized in Table 1.

Clinical assessment of patients suggested that in 7 patients (20.6%), in whom nonoral feeding by means such as nasogastric cannula was necessary, the degree of dysphagia was classified as grade 4. In 3 patients among this severely dysphagic group, voluntarily initiated swallows could not be accomplished at all. For the remaining 4 patients, although small amounts of food could be swallowed from time to time, nonoral feeding was also necessary, because they frequently experienced aspiration. The degree of dysphagia was evaluated as grade 3 in 20 patients (58.8%) and grade 2 in 7 patients (20.6%).

Dysphagia Limit
Although the grading procedure for dysphagia was useful in clinical evaluation of the patients, the method used to measure the dysphagia limit was more sensitive and objective in classifying the severity of clinical problems associated with swallowing. Only 1 patient with grade-2 dysphagia showed a normal dysphagia limit of >20 mL water. The remainder of the patients who were investigated displayed a dysphagia limit of <20 mL, including an additional 6 patients with grade 2 dysphagia. Figure 1 shows the dysphagia limits obtained from a patient (assessed with grade 2 dysphagia) in whom the bolus was divided into 2 or more swallows (as indicated by arrows) while attempting to swallow 10 mL water.
The pattern of dividing the bolus into 2 separate swallows occurred with volumes between 3 and 20 mL. As the volume increased, patients had to divide the bolus into even smaller portions and to perform additional swallows to swallow the entire amount in the mouth. This pattern was never observed in normal subjects unless they were required to swallow volumes exceeding 20 mL. In some cases, the dysphagia limit, at which the patient could swallow in a single attempt, was reduced to 1 to 3 mL. Furthermore, in some instances, coughing, which indicates subglottal aspiration, occurred during an attempt to swallow, and these movements were detected as several consecutive artifactual deflections in the swallowing signal. Overall, in patients with SBP, the degree of dysphagia was well correlated with dysphagia limits (Figure 2). The dysphagia limit was significantly reduced as the degree of dysphagia increased ($r=0.62$, $P<0.0001$).

### Laryngeal Movements and Submental EMG During Swallowing

Figure 3 illustrates the representative traces obtained from a normal control subject (top traces) and a dysphagic patient with SBP (bottom traces) during swallowing of 3 mL water. Measurements of the 0-2 interval, which is the time between the onsets of the upward and downward movement deflections of the larynx and indicates the laryngeal upward and relocation time during a swallow, and the A-C interval, the total duration of SM-EMG that indicates the onset and end of oropharyngeal swallowing, revealed significant differences in the 2 groups studied. These parameters of swallowing, particularly the duration of SM-EMG, were prolonged in patients with SBP (Figure 3B), as shown in the statistical results given in Table 2. The A-0 time interval between the onset of SM-EMG and the onset of laryngeal upward deflection (shown as A-0 in Figure 3 by oblique arrows) was also significantly prolonged in patients with SBP.

In patients with SBP with severe dysphagia, the electrophysiological pattern of swallowing showed further differences. Three of these patients could not produce voluntarily initiated swallows, and any swallows recorded electrophys-
ologically were spontaneous/reflexive in nature. An additional group of 4 patients with grade 4 dysphagia was still able to swallow voluntarily, but their spontaneous swallows occurred more frequently. Figure 4 illustrates that in their attempt to swallow voluntarily, dysphagic patients first demonstrate a prolongation of the A-0 interval (Figure 4A), and in the advanced stages of dysphagia, when the voluntary triggering of swallows is severely reduced or lost and all swallows become spontaneous or saliva swallowing, the A-0 interval is notably shortened (Figure 4B), in most cases down to 50 ms.

### EMG Activity of the CP During Swallowing

In 19 of 34 patients with SBP, the CP-EMG recordings were analyzed and compared with recordings obtained from 21 control subjects. Figure 5 displays the CP-EMG traces obtained from a control subject and a patient with dysphagia. During a swallow, the tonic EMG activity of the CP sphincter is normally switched off. The average pause in the EMG activity during voluntarily initiated swallows was found to be 462.0 ± 17.8 ms in normal subjects and 388.6 ± 17.8 ms in dysphagic patients (Table 3). This difference represents a significant shortening of the CP-EMG pause in patients with SBP during swallowing of 1 to 3 mL water (P < 0.006). In 9 patients with SBP, there were unexpected bursts of motor unit potentials (MUP; arrow).

### Table 2. Summary of Electrophysiological Findings of Patients and Normal Subjects During 3 mL Water Swallowing

<table>
<thead>
<tr>
<th>Swallowing Parameters</th>
<th>Patients With SBP With Dysphagia (n=31)*</th>
<th>Normal Control Subjects (n=35)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–2 Laryngeal relocation time, ms</td>
<td>640.1 ± 20.4</td>
<td>581.1 ± 129.0</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>111.6</td>
<td>74.0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>360–925</td>
<td>420–702</td>
<td></td>
</tr>
<tr>
<td>Submental EMG duration (A-C interval), ms</td>
<td>1188.0 ± 103.2</td>
<td>924 ± 24.5</td>
<td>0.02</td>
</tr>
<tr>
<td></td>
<td>565.5</td>
<td>140.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>500–3440</td>
<td>674–1250</td>
<td></td>
</tr>
<tr>
<td>A-0 interval, ms</td>
<td>423.5 ± 64.6</td>
<td>279.2 ± 19.4</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>353.6</td>
<td>111.7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>18–1440</td>
<td>40–480</td>
<td></td>
</tr>
<tr>
<td>Submental EMG amplitude, µV</td>
<td>68.1 ± 4.8</td>
<td>57.3 ± 4.1</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>26.4</td>
<td>23.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>26–133</td>
<td>22–103</td>
<td></td>
</tr>
</tbody>
</table>

Values are given as mean ± SEM, SD, and range.

*In 3 patients, voluntarily initiated swallows could not be performed.
TABLE 3. CP-EMG Parameters of the Cricopharyngeal Muscle of Upper Esophageal Sphincter During Voluntarily Initiated Swallowing of 3 mL Water

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patients With SBP With Dysphagia (n=19)</th>
<th>Normal Control Subjects (n=21)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>CP sphincter EMG pause, ms</td>
<td>388.6±17.8</td>
<td>462.0±17.8</td>
<td>0.006</td>
</tr>
<tr>
<td></td>
<td>77.8</td>
<td>81.8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>250–600</td>
<td>300–630</td>
<td></td>
</tr>
<tr>
<td>Time from onset of laryngeal upward movement to onset</td>
<td>137.9±23.2</td>
<td>113.1±17.7</td>
<td>NS</td>
</tr>
<tr>
<td>of CP-EMG pause, ms</td>
<td>92.9</td>
<td>70.8</td>
<td></td>
</tr>
<tr>
<td></td>
<td>20–380</td>
<td>36–256</td>
<td></td>
</tr>
<tr>
<td>Time from end of CP-EMG pause to onset of laryngeal</td>
<td>166.5±15.7</td>
<td>100.4±19.0</td>
<td>0.006</td>
</tr>
<tr>
<td>downward movement, ms</td>
<td>56.8</td>
<td>60.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>60–232</td>
<td>18–190</td>
<td></td>
</tr>
</tbody>
</table>

Values are given as mean±SEM, SD, and range.

In SBP with multiple lacunar state, dysphagia is a very common disorder, and for many patients, this causes serious management problems. This was also reflected in our sample, because 79.4% of 34 patients with SBP who were included in the study had severe dysphagia and 20.6% required nonoral feeding. Another important point was that in the majority of patients, dysphagia persisted for weeks to months without any improvement. Thus, dysphagia is common, severe, and persistent in majority of patients with SBP. This is probably due to the presence of lesions in multiple loci, including the brain stem and both cerebral hemispheres. Furthermore, the basal ganglia and associated neural pathways should also play a role in the pathogenesis of dysphagia, as in most cases the involvement of these structures was demonstrated by MRI.

In the present study, some clinical findings were frequently associated with dysphagia. Among these were the weak or slow elevation of larynx during swallows (87%), dysarthria (83%), and saliva accumulation in the mouth (80%). Dysarthria/dysphonia are reported to be common clinical characteristics of stroke patients with dysphagia and aspiration. However, in previous stroke studies, the weak or slow elevation of larynx and saliva accumulation in the mouth either have not been found in a significant proportion of patients or were not mentioned at all. Nevertheless, according to our clinical experience, these findings also appear to be frequently associated with dysphagia of patients with SBP.

With the electrophysiological tests described in our earlier studies for the evaluation of oropharyngeal swallow, the dysphagia limit in all except 1 patient with SBP was found to be pathological, with limits of <20-mL bolus volume. On the other hand, in all normal subjects, the dysphagia limit exceeded the 20-mL bolus volume. Furthermore, the measurements of dysphagia limit also correlated with the degree of clinical dysphagia in these patients (Figure 3). Therefore, the measurement of dysphagia limit is sensitive for the assessment of neurogenic dysphagia and as a simple, quick, reproducible, and cost-effective method, it can readily be used in patients with SBP and the time course of dysphagia can be followed objectively.

In addition to dysphagia limit assessment, other electrophysiological methods, namely recordings of laryngeal movement, submental EMG, and CP-EMG of the upper esophageal sphincter, also provided valuable findings in patients with dysphagia. These additional findings can be summarized as follows:

1. In patients, the triggering of a voluntarily initiated swallow was delayed compared with control subjects, as demonstrated by the prolongation of the A-0 interval (see Figure 3), which is the time difference between the onset of SM-EMG and the onset of upward movement of the larynx and provides indirect information about the voluntary triggering of swallowing reflex.

2. The laryngeal upward movement and relocation time of the larynx during swallowing were significantly prolonged in patients with SBP, as revealed by the prolongation of 0-2 time interval, which reflects the pharyngeal phase of swallowing reflex.

3. During swallowing, the duration of activation of submental suprahyoid muscles was longer in patients than in
control subjects. These muscles have 2 principal roles in swallowing: the protection of the larynx by elevation, and the transportation of bolus by providing a secondary support to the tongue for its pumping action.\textsuperscript{18,25,26} Therefore, the duration of the SM-EMG is a parameter that provides considerable information about the duration of the pharyngeal phase of swallowing that was prolonged in dysphagic patients with SBP with lacunar state.

(4) The CP-EMG recordings made during swallowing revealed that the striated sphincter muscle of the upper esophageal sphincter was hyperreflexic and presumably dysinhibited like other brain stem reflexes, such as mandibular and palatopharyngeal reflexes. The emotional lability observed in these patients may also have been associated with dysinhibition at the brain stem level. The hyperreflexic nature of CP sphincter was apparent in the shorter EMG pause period during swallowing and its premature closure before the larynx descended.\textsuperscript{9,14}

Three pathophysiological mechanisms can be proposed for the manifestation of dysphagia in SBP with multiple lacunar state:

Because corticobulbar fibers are involved in the triggering of voluntarily initiated swallows and have facilitatory effects on the bulbar swallowing center,\textsuperscript{26–29} in SBP, the extreme prolongation of excitation of the submental muscles for triggering of the swallowing reflex observed with the A-0 interval should be associated with the involvement of the corticobulbar fiber system. In the advanced stages of corticobulbar descending motor system involvement, a voluntary swallow cannot be initiated, and therefore it would be impossible for a patient with SBP to swallow voluntarily, although reflex swallowing can still be performed with the bulbar swallowing center.\textsuperscript{26} These reflex swallows can be detected clinically as well as electrophysiologically\textsuperscript{9,14} (Figure 4).

The second mechanism affected in swallowing disorders may be associated with suprasegmental inhibitory influences on the bulbar center. These influences appear to have been removed through some peculiar pathological involvement of corticobulbar fibers. The abnormal EMG patterns recorded from the CP sphincter is similar to those obtained from patients with amyotrophic lateral sclerosis with corticobulbar involvement.\textsuperscript{9} There is further evidence in the literature for the presence of such pyramidal inhibition on the swallowing reflex.\textsuperscript{30} Therefore, it appears that the involvement of corticobulbar pathways in SBP not only alters the direct excitatory influences but also results in dysinhibition by affecting the inhibitory neural circuits. Consequently, the swallowing reflex, particularly the CP sphincter reflex pattern, is dysinhibited (ie, released from inhibitory control). Further support for this mechanism comes from observations in our patients of increased mandibular and palatopharyngeal reflexes and emotional lability. Likewise, it has also been suggested that in amyotrophic lateral sclerosis, the possible involvement of suprabulbar inhibitor control may be accountable, through a release from inhibitory influences, for the increase in brain stem reflexes.\textsuperscript{9,14,31–33}

Because the bulbar swallowing center is presumed to be intact in patients with SBP, it is difficult to explain the prolongation of the laryngeal relocation time of the swallowing reflex as measured with the 0-2 time interval. Furthermore, the duration of submental muscle contraction was also prolonged, as demonstrated by prolongation of the A-C interval. These findings may indicate that the extrapyramidal influences on the bulbar center have somehow been affected. This assumption is based on similar findings observed in patients with Parkinson’s disease with dysphagia.\textsuperscript{14} Similarly, in patients with SBP, the accumulation of saliva in the mouth and drooling may also be related to the involvement of the extrapyramidal system, in addition to the disturbance associated with the corticobulbar system.

Acknowledgments

This work was supported by a grant from the Turkish Scientific and Technological Research Council (TÜBİTAK) (project SBAG-1739).

References


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*Stroke*. 2000;31:1370-1376
doi: 10.1161/01.STR.31.6.1370
*Stroke* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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