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Dysphagia

Oropharyngeal Swallowing after Stroke in the Left Basal Ganglion/Internal Capsule

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Abstract. One of the foci of Martin Donner's work was the neural control of swallowing. This present investigation continues that work by examining oropharyngeal swallowing in 8 patients identified with a single, small, left-basal ganglion/internal capsule infarction and 8 age-matched normal subjects. Stroke patients were assessed with a bedside clinical and radiographic swallowing assessment, and normal subjects received only the radiographic study. Results revealed disagreement between the bedside and radiographic assessments in one of the 8 stroke patients. Stroke and normal subjects differed significantly on some swallow measures on various bolus viscosities, but behaved the same as normal subjects on a number of measures. Differences in swallowing in the stroke subjects were not enough to prevent them from eating orally. The significant differences seen in the basal ganglia/internal capsule stroke subjects may result from damage to the sensorimotor pathways between the cortex and brainstem. These differences emphasize the importance of cortical input to the brainstem swallowing center in maintaining the systematic modulations characteristic of normal swallowing physiology.

Key words: Fluoroscopy — Swallowing — Basal ganglia — Stroke — Deglutition — Deglutition disorders.

In the late 1960s and early 1970s, Martin Donner and colleagues were among the first clinicians to recognize and study the effects of damage to the central nervous system (CNS) on oropharyngeal swallow physiology [1,2]. These early investigations described swallowing abnormalities in groups of patients with a variety of neurologic impairments including stroke, Parkinson's disease, and amyotrophic lateral sclerosis. Although these studies included patients with a range of neurologic diagnoses and, therefore, damage at varying levels of the CNS, this work established that CNS damage at a variety of levels does create swallowing abnormalities. These investigations formed the foundation for more recent studies of the effects of specific CNS lesions on oropharyngeal deglutition [3,4]. In the past 10 years, the research of Martin Donner and others on neurologic dysphagia has focused on specifying patient populations more carefully, i.e., patients with lesions in particular locations within the CNS and at specific points in time after onset of the damage [4–6].

This present investigation continues this focus on dysphagia in carefully defined neurologic patient groups by examining swallowing differences between patients with a single, small, left basal ganglion/internal capsule infarct studied at 3 weeks post-ictus and normal, age-matched control subjects.

Methods

Eight subjects aged 36–84 years (mean age 59 years) were identified with a single, small, left basal ganglion/internal capsule infarction (hemorrhagic or multifocal neurovascular lesions were excluded) as indicated on a computerized tomographic (CT) scan performed between 21 and 28 days post-ictus and interpreted by both a neurologist and a neuroradiologist. Eight volunteers with normal swallowing function (i.e., no neurologic diagnosis and no history of dysphagia) who were within 2 years of age of the stroke subjects were also studied. The study protocol was approved by the Northwestern University Institutional Review Board, and informed consent was obtained from all participants.

Concurrent with the CT scan, each stroke subject received a modified barium swallow (MBS) and a clinical dysphagia exam admin-

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istered by certified speech-language pathologists [7,8]. The clinical and radiographic swallowing studies were completed within the same day or within 3 days of each other for each patient. Each normal subject received only an MBS study.

The MBS consisted of a standard protocol of two swallows each of 1, 3, 5, and 10 ml volumes of thin liquid, 1 ml paste, and 1/4 of a Lorna Doone cookie which each subject was asked to chew and swallow. A videotimer was used to encode timing information onto each frame in order to facilitate slow motion and frame-by-frame analysis. Each swallow was analyzed to determine the following temporal measures:

1. oral transit time (OTT)—onset of bolus movement in the mouth until the head of the bolus reached the point where the lower rim of the mandible crosses the tongue base;
2. pharyngeal delay time (PDT)—bolus head arrival at the point where the lower rim of the mandible crosses the tongue base until first laryngeal elevation;
3. pharyngeal transit time (PTT)—bolus head arrival at the point where the lower rim of the mandible crosses the tongue base until the bolus tail passes through the cricopharyngeal (CP) region;
4. pharyngeal response time (PRT)—pharyngeal transit time minus pharyngeal delay time;
5. cricopharyngeal opening duration (DCPO)—onset to termination of cricopharyngeal opening [9];
6. laryngeal closure duration (DLC)—onset to termination of closure of laryngeal vestibule [10];
7. velopharyngeal closure duration (DVC)—onset to termination of velar contraction to the posterior pharyngeal wall;
8. duration of hyoid movement (DHM)—onset to termination of hyoid motion;
9. duration of laryngeal elevation (DLE)—onset to termination of laryngeal elevation;
10. time from first crycopharyngeal (CP) opening (time 0) to first closure of the laryngeal vestibule (LCPO).

In addition to these measures, judgments were made from each radiographic study regarding percentage aspiration, oral residue, and pharyngeal residue for each swallow. Oropharyngeal swallow efficiency (OPSE) was then calculated by dividing percentage of the bolus swallowed (minus percentage oral residue (ORES) and pharyngeal residue and aspiration) by oral plus pharyngeal transit time [11]. These data were subjected to two 3-way analyses of variance (ANOVA). These analyses examined the volume and group differences as well as the viscosity and group differences. Each analysis accounted for the repeated measures within individuals. When tests for main effects were significant, pairwise comparisons were done using *t*-tests.

The clinical dysphagia examination consisted of evaluation of movements of the lips, tongue, and soft palate during voluntary nonspeech and speech movements with rating as "adequate," "reduced," "inadequate/absent," or "could not test." Labial and lingual function, as well as hyoid and laryngeal movement, were also rated during swallows of thin liquid, thick liquid, and pureed and solid foods. Any clinical signs of aspiration were recorded (i.e., coughing and/or wet vocal quality related to swallows). A clinical judgment was made regarding degree of dysphagia (mild, moderate, severe, or no dysphagia).

Results

Clinical vs. Radiographic Swallow Disorders in the Stroke Subjects

The clinical dysphagia examination yielded clinical judgments that swallowing function was within normal limits for 6 of the 8 stroke subjects. Subject 7 was judged to have severe dysphagia characterized by reduced oral initiation and tongue functioning, and reduced hyoid and laryngeal movement, with aspiration clinically indicated by a cough after the swallow. This patient was being fed through a gastrostomy tube. Subject 8 was judged clinically to exhibit mild dysphagia related to reduced lingual strength resulting in pocketing of food in the buccal cavity on the weak side. A mild pharyngeal swallow delay with no aspiration was also noted. This patient was on an unrestricted oral diet.

The MBS performed the same day as the clinical dysphagia examination revealed functional swallows, i.e., no aspiration, and only minimal oral or pharyngeal residue and mild pharyngeal swallow delays (1–3 sec) for 7 of the 8 stroke subjects. The fluorographic study confirmed a mild pharyngeal swallow delay in subject 8, but no aspiration was indicated. A full oral diet was recommended for all 8 subjects as a result of the radiographic study.

Measures of Swallow Physiology

Volume Effects

Results of the 3-way ANOVA for volume effects for liquid boluses in normal and stroke subjects revealed no significant interaction, indicating that the normal and stroke subjects exhibited the same pattern of change across volumes. When stroke and normal subjects were pooled, significant volume effects were seen for five swallow measures (Table 1). Pharyngeal transit times and pharyngeal delay times were significantly longer for 1 ml than for 3, 5, or 10 ml volumes. Cricopharyngeal opening duration increased significantly from 1 ml to 3, 5, and 10 ml. Oropharyngeal swallow efficiency increased significantly from 1 to 5 ml and from 1 to 10 ml. Oral residue increased significantly for 10 ml vs. 1, 3, and 5 ml. No other measures exhibited significant volume effects.

When the means for all liquid volumes were combined for each subject group and the groups were compared, two measures were significantly different ($p < 0.0001$) between stroke and normal subjects: OTT and OPSE. Stroke subjects exhibited significantly longer OTT (mean ($\pm$SEM) = 0.73 sec ($\pm$0.08)) than the age-matched normal subjects (mean ($\pm$SEM) = 0.42 sec ($\pm$0.03)) and significantly lower OPSE scores [mean ($\pm$SEM) = 68 ($\pm$4)) than the normal subjects (85 ($\pm$3)].

Viscosity Effects

For assessment of viscosity effects, all liquid bolus volumes were combined. Examination of viscosity effects revealed that stroke and normal subjects differed in viscosity effects on pharyngeal transit times and pharyngeal

Table 1. Liquid volume effect on mean (± SEM) PTT, PDT, DCPO, OPSE, and percentage oral residue (ORES) for the combined stroke and normal subjects

	1 ml	3 ml	5 ml	10 ml	ANOVA p	Pairwise comparisons[a]
PTT (sec)	1.13 ± 0.13	0.86 ± 0.07	0.79 ± 0.04	0.84 ± 0.06	0.006	1,2,3
PDT (sec)	0.30 ± 0.13	−0.04 ± 0.08	−0.06 ± 0.04	−0.03 ± 0.08	0.01	1,2,3
DCPO (sec)	0.41 ± 0.03	0.49 ± 0.02	0.51 ± 0.02	0.52 ± 0.02	0.001	1,2,3
OPSE	66 ± 4	75 ± 4	83 ± 5	83 ± 6	0.01	2,3
ORES (%)	0.78 ± 0.33	1.88 ± 0.49	2.81 ± 0.59	7.42 ± 2.03	<0.0001	3,5,6

See text for abbreviations.
[a]1: $p < 0.05$ 1 ml vs. 3 ml; 2: $p < 0.05$ 1 ml vs. 5 ml; 3: $p < 0.05$ 1 ml vs. 10 ml; 4: $p < 0.05$ 3 ml vs. 5 ml; 5: $p < 0.05$ 3 ml vs. 10 ml; 6: $p < 0.05$ 5 ml vs. 10 ml.

Table 2. Differences in mean (± SEM) PTT and PDT in the stroke and normal subjects across bolus consistencies

	Liquid	Paste	Cookie	ANOVA p	Pairwise comparisons[a]
PTT					
Stroke	0.96 ± 0.06	2.13 ± 0.41	2.77 ± 0.59	<0.0001	1,2
Normal	0.85 ± 0.05	0.91 ± 0.09	1.20 ± 0.23	0.04	2
PDT					
Stroke	0.11 ± 0.07	1.29 ± 0.40	2.02 ± 0.57	<0.0001	1,2,3
Normal	−0.04 ± 0.06	−0.10 ± 0.09	0.26 ± 0.24	0.08	NA

NA: not applicable since ANOVA $p < 0.05$. See text for other abbreviations.
[a]1: $p < 0.05$, liquid vs. paste; 2: $p < 0.05$, liquid vs. cookie; 3: $p < 0.05$, paste vs. cookie.

Table 3. Mean (± SEM) oropharyngeal swallow measures on which stroke and normal subjects behaved similarly across bolus consistencies

	Liquid	Paste	Cookie	ANOVA p	Pairwise comparisons[a]
OTT	0.57 ± 0.05	0.82 ± 0.12	1.25 ± 0.23	<0.0001	1,2,3
DCPO	0.48 ± 0.01	0.48 ± 0.02	0.42 ± 0.03	0.04	2
DLC	0.54 ± 0.02	0.50 ± 0.02	0.46 ± 0.02	0.04	2
ORES	3.19 ± 0.58	4.22 ± 0.60	10.78 ± 2.92	<0.0001	2,3
OPSE	77 ± 3	54 ± 5	52 ± 7	<0.0001	1,2

[a]See Table 2 for pairwise comparisons.

delay times (Table 2). Stroke patients exhibited significantly increased pharyngeal transit times for paste and cookie as compared with liquid, and pharyngeal delay times from liquid to paste to cookie. In contrast, normal subjects showed a significant increase (of substantially less magnitude) only for pharyngeal transit times on liquid vs. cookie and no significant change in delay time.

When pooled, normal and stroke subjects exhibited longer oral transit times as bolus viscosity increased from liquid to paste to cookie, reduced CP opening for cookie vs. liquid, shorter duration of laryngeal closure on cookie vs. liquid, increased oral residue on cookie vs. liquid and paste swallows, and reduced oropharyngeal swallow efficiency on paste and cookie vs.liquid (Table 3). No other measures showed significant viscosity effects.

When all bolus viscosities were combined for each of the two subject groups and compared, stroke subjects and age-matched normal subjects differed significantly ($p \leq 0.005$) on three measures: OTT, OPSE, and PRT. Stroke subjects exhibited longer OTT [mean (±SEM) = 0.93 (±0.09)] than normal subjects [mean (±SEM) = 0.52 (±0.05)], lower OPSE (mean (±SEM) = 59 (±3)] than normal subjects [mean (±SEM) = 79 (±3)], and shorter PRT [mean (±SEM) = 0.84 (±0.03)] than normal subjects [mean (±SEM) = 0.91 (±0.03)].

Discussion

This study examined differences in results of clinical bedside assessments and radiographic studies in 8 pa-

tients with basal ganglion/internal capsule infarcts, and compared measures of swallow physiology on specified bolus volumes and viscosities in these stroke subjects and 8 age-matched normal subjects. Despite the fact that the clinical bedside and radiographic assessments were completed on the same day, their results disagreed in 1 of the 8 patients. The differences seen may have been physiologic and related to varying levels of fatigue or alertness. Or, the cough and other symptoms observed in 1 stroke patient may have been unrelated to swallow physiology. The radiographic studies demonstrated mild swallowing differences in these basal ganglion stroke patients, but no serious swallowing abnormalities that would keep them from eating orally. The measures of swallowing confirmed some mild and statistically significant differences in swallowing in these stroke vs. normal subjects.

Stroke and normal subjects exhibited the same systematic changes in five swallow measures with liquid bolus volumes. Pharyngeal transit time and pharyngeal delay time were longer for 1 ml than other volumes, making that volume, on average, the slowest moving bolus for all subjects. CP opening was shortest for 1 ml, a result that has been previously reported [9]. Oral residue was greatest after 10-ml swallows, indicating slightly less efficient clearance from the mouth on this large volume.

When all liquid bolus volumes were combined, the stroke subjects exhibited significantly slower oral transit and reduced oropharyngeal swallow efficiency, indicating that the stroke subjects were slower and less efficient swallowers than the age-matched normal subjects, though only mildly so.

Stroke subjects differed significantly from normals on two swallow measures during modulation of bolus viscosity. Stroke subjects exhibited longer pharyngeal transit times and pharyngeal delay times on thicker boluses (paste and cookie) than normal subjects. These differences may indicate reduced sensory recognition of the bolus or reduced motor control to compensate for increased viscosity as a result of the unilateral stroke in the basal ganglia/internal capsule. These differences in the stroke vs. normal subjects in reaction to bolus viscosity may reflect mild unilateral damage in the sensorimotor pathways from the cortex to the brainstem which pass through the basal ganglia and internal capsule. The changes in swallow physiology in these patients with unilateral lesions in the CNS above the brainstem swallowing center emphasize the importance of neural transmission to and from the cortex in the systematic modulations of normal oropharyngeal swallowing [12,13].

All subjects (stroke and normals) exhibited significant differences between bolus viscosities in five swallow measures. Slower oral transit times and reduced OPSE as bolus viscosity increases concur with earlier studies [14]. Slightly, but significantly reduced CP opening and laryngeal closure durations on cookie may reflect a slightly smaller volume on the cookie than liquid and paste boluses.

Overall, the stroke subjects swallowed slower and less efficiently than the normal subjects, though only mildly so.

Summary

Although the unilateral basal ganglia internal capsule stroke patients and normal subjects exhibited some of the same systematic changes in swallow measures in relation to bolus volume and viscosity, the stroke subjects also exhibited several significant differences from the normal subjects. These differences are likely to result from damage to sensorimotor pathways from the cortex to the brainstem which pass through the basal ganglia internal capsule. Much additional research is needed on swallowing physiology in patients with specific lesions in the CNS before we will completely understand normal neural control of deglutition and the effects of CNS damage on oropharyngeal swallowing. Martin Donner's work in this area provided a foundation for these current and future efforts.

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