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Swallowing After Unilateral Stroke of the Cerebral Cortex: Preliminary Experience

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Abstract. In an attempt to impose an organizational format on the specific effects that isolated stroke syndromes have on swallowing ability, we have developed a systematic approach to investigating the underlying neural control mechanisms in patients with clinical and computed tomographic evidence of unilateral ischemic stroke involving the cerebral cortex. When compared to findings in normal controls, initiation of the pharyngeal response was delayed in all stroke subjects. Left cortical stroke dysphagia was characterized primarily by impaired oral stage function, difficulty initiating coordinated motor activity, and apraxia. Right cortical stroke dysphagia was characterized primarily by pharyngeal pooling, penetration, and aspiration. Thus, these preliminary data indicate distinct patterns of dysphagia after unilateral cortical stroke and challenge the traditional classification of swallowing as a bilateral and brainstem-mediated activity.

Key words: Neurogenic dysphagia – Stroke, unilateral cerebral cortical – Aspiration.

The central nervous system (CNS) pathway for swallowing traditionally is thought to be bilateral and not linked to speech. However, swallowing and speech disorders frequently coexist in neurologically impaired patients. Before a neuromotor relationship beteen the two functions can be defined, the neural control mechanisms for swallowing must be clarified. Systematic examination of temporospatial upper aerodigestive tract activity during swallowing in patients with specific stroke syndromes should offer insight into the underlying neurophysiological processes.

Although upper aerodigestive tract patterns of movement differ for speech production and swallowing, the major upper aerodigestive tract components and their individual movements, synchronized with respiration or its cessation, are much the same. Despite the structural commonality between the two functions, both in terms of neurophysiological mechanisms and final output structures, speech generally is viewed as a voluntary higher cortical function. Swallowing is generally considered a brainstem reflexive behavior [1, 2]. Current evidence points to a coordinating swallow center in the medullary reticular formation [3].

There is little doubt that initiation of the pharyngeal events for swallowing is related to voluntary attempts to swallow. Current studies of reflexes and peripheral input to aerodigestive tract structures suggest a complex interaction of lower level neural systems with cortical activity during deglutition [4–6]. Animal experiments [7] as well as observations of human behavior [8–10] report that subjects with anterior cortical damage must perform compensatory oral behaviors, such as tipping the head backward to initiate swallowing. This suggests that some aspects of swallowing are under control of the cerebral cortex. Therefore, the neural mechanisms of the volitional stages of swallowing may involve some of the same neural structures, and perhaps control mechanisms, as speech production. However, due to the traditional "neural control dichotomy" imposed upon swallowing and speech, which classifies swallowing as primarily a function of the lower centers and speech as a cortical function, little information is available to clarify the relationship between the neural mechanisms for speech production and the supratentorial neural circuitry for swallowing.

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We describe our preliminary findings in patients with clinical and computed tomographic (CT) evidence of unilateral stroke of the cerebral cortex and compare these to the swallowing activity in normal controls. The role of various neurophysiological structures in swallowing is then inferred from range and duration measures of structural movement and motility patterns in subject examined.

Methods

Subjects

To date, 24 right-handed subjects participated in this continuing investigation. All subjects provided their informed consent. Sixteen subjects were recruited after giving a detailed clinical history, undergoing neurologic examination by a board-certified neurologist (R.L.L.), and having cranial CT confine their presenting neurologic disorder to that of a single, unilateral ischemic cerebrovascular accident (CVA). Eight subjects were recruited as normal controls following detailed clinical history and neurologic assessment. Normal subjects did not undergo cranial imaging studies as part of this protocol. The average age for each group was 64 years, ranging from 53 to 81 years. Normal subjects had no prior stroke or swallowing disorders history. Patients with CVA were classified into right cerebral cortical (RCVA) and left cerebral cortical (LCVA) stroke subgroups based on clinical examination and CT. Magnetic resonance imaging (MRI) was not performed on these patients. All CVA patients were studied at 21 ± 2 days following onset of stroke. The first part of the two-part test protocol includes an oropharyngeal motor examination of the lips, tongue, mandible, velum, and larynx at rest, during nonspeech movements, and during speech production. The second part of the protocol was videofluoroscopic examination of the oropharynx during swallowing and speech production.

Videofluoroscopic Protocol

Videofluoroscopy and a video counter-timer were used to record movement patterns during swallowing and speech. Subjects were seated and viewed radiographically in the lateral plane. During the speech studies the patient wore a headband-supported microphone positioned 5 cm from the lips for simultaneous audio recordings. The fluoroscopic tube was focused on the oral cavity from the lips anteriorly to the pharnygeal wall posteriorly, and from the nasopharynx superiorly to just below the larynx inferiorly. Lateral radiographic views were recorded while the subjects swallowed 2 ml radiopaque liquid, 2 ml radiopaque semisolid, and repeated /pamper/ and /aka/ and elevated pitch on /a/ phonation. Small amounts of material were used to minimize potential aspiration. Each task was performed three times.

Data Analysis

The videotapes were examined in slow motion and frame by frame for analysis of each swallow. The data were analyzed in samples without stroke localization noted. The measures and observations listed in Table 1 were made for each swallow. Quantitative measures were made for prepharyngeal response time (PPRT) and pharyngeal response time (PRT). The PPRT was defined operationally as the duration from initiation of bolus movement after the command to swallow to initiation of maximal hyoid excursion. The PPRT is subdivided into oral transit and delay time in subjects who demonstrated a delayed initiation of the pharyngeal stage[1] of the swallow. Delay time is the time from bolus arrival at midvelum (approximately perpendicular to the anterior faucial arches) to initiation of maximal hyoid excursion. The PRT was operationally defined as the time from initiation of maximal hyoid excursion to hyoid return to rest. The PPRT and PRT were defined in an attempt to compare events during the initial volitional stage of swallowing to the more automatic events occurring later in the swallow sequence.

Table 1. Measures and observations for each swallow

Prepharyngeal response time (oral transit and delay)
Oral motility patterns
Pharyngeal response time
Pharyngeal motility patterns
Aspiration
If present
Cause

Descriptive motility characteristics were categorized according to stage of swallowing, oral versus pharyngeal. These were used to describe structural movement relative to impact on bolus propulsion, as well as evidence of residual material in the oral cavity or pharynx.

Results

Duration Measures

A general finding across all groups regarding type of material swallowed is a difference in oral stage duration for liquid and semisolid (paste) swallows. The PPRTs for paste are longer and more variable than for liquids (Fig. 1). Even in the control group, PPRTs for the paste swallows are longer and more variable. In contrast, PRTs are similar for liquid and paste material in all groups (Fig. 2).

With regard to differences in subject groups, a major finding is that PPRTs are longer for CVA subjects than controls, The PPRT and variability for swallowing both liquids and paste are greater in the CVA subjects than the controls (Fig. 1). The PPRT for paste swallows is most increased, with ranges of 16 s for the left CVA group compared to 3 s for the control groups. Group means, standard deviations, and the coefficients of variation are shown in Table 2. For liquid swallows, the mean PPRT values for CVA subjects are three times longer than normal. Standard deviations for

[1] Although swallowing is analyzed in terms of stage and specific time periods, any division of a functionally integrated system is somewhat artificial. Nonetheless, division of swallowing into stages and related durations permits an organized approach that may facilitate better understanding of the process.

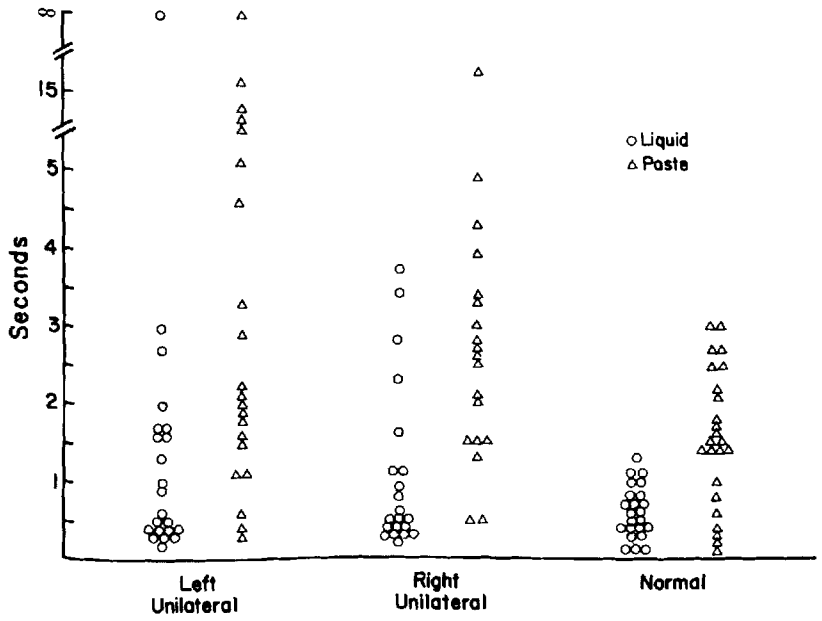

Fig. 1. Prepharyngeal response times for 2 ml liquid and paste swallows in normals and left and right unilateral cerebral cortical stroke subjects.

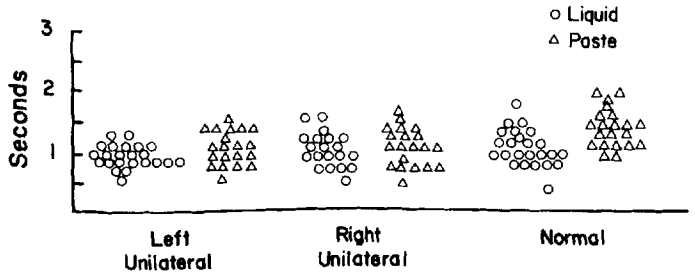

Fig. 2. Pharyngeal response times for 2 ml liquid and paste swallows in normals and left and right unilateral cerebral cortical stroke patients.

Table 2. Group means, standard deviations (SD), and coefficient of variation (CV) for prepharyngeal response times of liquid and paste swallows in normal and left and right cerebral cortical stroke patients

	Left	Right	Normal
Liquid			
Mean	1.5	1.4	0.5
SD	1.2	1.1	0.2
CV	0.8	0.8	0.4
Paste			
Mean	6.5	2.6	1.5
SD	9.2	1.0	0.8
CV	1.4	0.4	0.5

the CVA subjects are considerably greater than for controls, but computation of the coefficient of variation shows obviously longer PPRT values among the stroke subjects.

As compared to either controls or RCVA subjects, the mean PPRT for paste swallows is greatest among LCVA subjects. Computation of the coefficient of variation (Table 2) shows that controls and RCVA subjects had similar durations of paste swallows, while the LCVA subjects are identified by longer PPRTs.

Motility

Motility characteristics are categorized according to stage of swallowing (Table 3). The LCVA subjects had a distinguishing motility abnormality: 50% of this subgroup exhibited "difficulty initiating coordinated movement." Those who demonstrated this behavior also showed oral and verbal apraxia as identified on the oropharyngeal motor examination administered to each participant and the speech production part of the videofluoroscopic protocol. "Anterior lingual motion," not seen in controls, occurred equally in RCVA and LCVA subjects.

"Residual coating" and "residual pooling" in the oral cavity also differentiate the LCVA from the RCVA and control subjects.

Pharyngeal stage motility characteristics are shown in Table 4. "Delayed pharyngeal response" is found in all CVA subjects. "Aspiration" oc-

Table 3. Oral motility characteristics for normal and stroke subjects

Characteristic	Group		
	Left stroke	Right stroke	Normal
Material under tongue	4	3	2
Repeat tongue pumping	5	5	5
Difficulty initiating coordinated movement	4	–	–
Anterior lingual motion	3	3	–
Residual coating			
Tongue	8	8	8
Palate	8	3	5
Velum	5	4	1
Residual pooling			
Tongue	2	1	1
Palate	3	–	1
Velum	2	–	–
Total observations (10 characteristics × 8 subjects = 80/group)	44	27	23

Table 4. Pharyngeal motility characteristics for normal and stroke subjects

Characteristic	Group		
	Left stroke	Right stroke	Normal
Delayed pharyngeal response	8	8	2
Absent pharyngeal response	1	–	–
Aspiration	–	3	1
Before	–	×	×
During	–	–	–
After	–	×	–
Coating			
Vallecula	8	8	8
Pyriform sinus	8	6	5
Posterior pharyngeal wall	6	7	5
Pooling			
Vallecula	4	7	1
Pyriform sinus	5	6	–
Posterior pharyngeal wall	–	1	–
Total observations (10 characteristics × 8 subjects = 80/group)	40	46	23

×, time of occurrence.

curred in 37% and 0% of the RCVA and LCVA subjects, respectively. One control subject also aspirated. "Aspiration" occurred most frequently before initiation of the pharyngeal stage, due to a delay in the pharyngeal response during which the material fell into the open airway. In one RCVA subject, aspiration occurred after the pharyngeal stage was completed, due to inspiration of pooled residual material. "Pooling" in the pharyngeal recesses occurred more often in RCVA subjects.

LCVA Observations

Four of eight LCVA subjects showed a lack of coordination of labial, lingual, and mandibular movement during the oral stage. Various uncoordinated-appearing oral movements were observed in one subject who never did succeed in propelling the paste bolus posteriorly in the oral cavity. His study was terminated during the first paste swallow attempt because the material clung to his palate while he struggled ineffectively for 25 s to elevate his mandible and coordinate placement of his tongue to complete the oral stage. This particular LCVA subject also demonstrated a concomitant oral and verbal apraxia as judged independently by three experienced speech pathologists, to whom this appeared to be "apraxia of swallowing." These observers, however, were aware that this patient had had a LCVA. It is interesting that this patient did not lose weight and appeared to eat well spontaneously. That is, in a natural eating situation in which the patient was not required to swallow upon command, as is the task in the videofluoroscopic swallowing protocol, the apraxic subjects were functional eaters. In fact, all of the LCVA subjects maintained their weight and required no direct swallowing treatment (e.g., nasogastric tube or thermal sensitization) in order to eat safely.

RCVA Observations

Due to the increased frequency of aspiration and less functional swallowing patterns, three of eight RCVA subjects had nasogastric tubes placed and four of eight required treatment for dysphagia. This group demonstrates a more impaired pharyngeal stage of swallowing but did not demonstrate oral or verbal apraxia. Unlike their cortical counterparts with LCVA, the RCVA subjects demonstrated abnormal speech patterns characterized by slightly hoarse, monotonous voice production as judged independently by three experienced speech pathologists. Again, these judges were aware of each patient's RCVA diagnosis.

Discussion

CVA Data Base

A major finding of this preliminary work on a sample of CVA patients is that unilateral cortical CVA does indeed result in disordered swallowing. This is significant in view of the traditional notion that swallowing is a bilaterally controlled function.

A second important finding is that the motility characteristics differ according to the side on which the brain is damaged. Specifically, in this small patient sample, LCVA subjects had more deviant oral stage timing and motility, including residual oral pooling and difficulty in initiating coordinated movement. The RCVA subjects had more pharyngeal dysfunction, including aspiration and reduced peristalsis as indicated by pharyngeal pooling.

The severity of swallowing dysfunction in LCVA subjects appears to correlate with the presence of oral and/or verbal apraxia. In addition, residual material in the mouth could be due to movements that are reduced in force, range, or coordination. The swallowing difficulty seen in RCVA subjects appears to be more clinically significant, since oral intake was determined to be unsafe for some of these patients. Thus, aspiration, as seen in three of eight RCVA and none of eight LCVA patients, may also be a distinguishing feature in stroke subjects. This data base needs to be expanded before conclusions can be made regarding aspiration, especially since one of the eight control subjects also appeared to aspirate on fluoroscopic examination.

Unilateral Cerebral Lesions

Our findings challenge the traditional concept that a unilateral lesion of the cerebrum should exert little, if any, effect on swallowing homeostasis. Patients with unilateral cerebral lesions, particularly those situated in the lowest part of the precentral gyrus or in the posterior portion of the inferior frontal gyrus of either hemisphere, show a considerable hesitancy in deglutition [11]. All of our CVA patients show a delay in the initiation of the pharyngeal response. This is due at least in part to cortical-bulbar and cortical-spinal tract dysfunction, since the primary neurophysiological role of these intertwined tracts is the initiation of voluntary movement. Whether the CVA patients do not receive oral cavity sensory input data properly, whether delayed and hesitant swallowing actions are a motor programming problem, and/or whether cognitive/linguistic factors play a role remain to be clarified.

To date, we have studied swallowing with quantitative techniques in more than 75 CVA patients, each of whom had suffered an acute unilateral CVA as localized by precise clinical history, detailed neurologic examination, and cranial CT. This group includes patients with cerebral cortical lesions, subcortical lesions such as internal capsule, and localized brainstem lesions. Abnormal aspects of deglutition were found in all subjects. We are in the process of expanding our CVA and normal data bases to include cranial CT/MRI comparisons, regional cerebral blood flow analysis using positron emission tomographic scanning (PET), and larger subject populations. The use of MRI, a superiorly sensitive yet nonspecific imaging tool in patients with cerebrovascular disease [12, 13], may help to clarify whether areas of silent, discrete, and/or distant infarctions are present in patients who otherwise appear to have strictly a single, unilateral CVA. Since MRI often uncovers "unidentified bright objects" in the white matter of elderly subjects with identifiable cerebrovascular risk factors [12, 13] and since, to date, these multiple lesions have not proven to be infarcted tissue on pathologic examination, we caution against the overinterpretation of MRI in stroke patients. Although Kim et al. [14] advocate MRI as superior to CT in localizing a stroke, we prefer to await results of clinical, CT, and MRI correlations before accepting MRI as the gold standard of stroke localization.

The central pathway for swallowing has traditionally been thought to be bilateral, and at its simplest may consist of an afferent projection from the periphery to the primary sensory cortex bilaterally, thence to the primary motor cortex and pyramidal tracts, perhaps via the premotor cortex [11]. The first-order estimate of effects of cortical stimulation with respect to mastication and swallowing has yet to be discovered [15]. Woolsey et al. [16] have shown that stimulation of the dorsolateral or anterolateral part of either frontal cortex (premotor areas) causes masticatory and swallowing movements. However, there is normally bilateral representation of cranial musculature in the primary motor cortex, and bilateral lesions are usually required to cause permanent disturbance of speech and swallowing [11]. There has been little study of the sensory inputs to the cortex that eventually lead to mastication and swallowing, except for findings that single cortical neurons cease discharging during deglutition, which suggests that cortical neurons are receiving relatively direct sensory feedback (i.e., 8–12 ms latency) during swallowing [15].

A unilateral cortical CVA may thus interact

with multiple systems when producing dysphagia. Most researchers agree that the cortical swallowing area is probably a sensorimotor area, anterolateral to the orbital gyrus of the frontal lobe, which receives information from the contralateral cortical center and from oral and pharyngeal receptors. We have found that left cerebral injury produces more significant oral stage abnormalities, which might imply that the left premotor cortex is the "dominant" cortical center for the volitional behavior of swallowing. The premotor cortex could contain an asymmetrical innervation ratio or unbalanced neuronal representation for the more volitional stages of swallowing, such that a single, appropriately placed lesion could impair normal deglutition. Alternatively, the premotor cerebral cortex injured by a CVA in one hemisphere may then be functionally disconnected from the healthy hemisphere because of dysfunction of interhemispheric neuronal connection via the corpus callosum. In this fashion a unilateral lesion could alter the normal bilateral homeostasis of swallowing. We are currently studying these possibilites with CT, MRI, and PET imaging in an attempt to confirm the "strictly" unilateral nature of the stroke in similar patients demonstrating dysphagia.

Although several studies in the literature have reported dysphagia in unilateral stroke [11, 17–21], our results are much more homogeneous in terms of timing (all at 21 ± 2 days poststroke), localization of lesion (all cerebral ischemic infarctions), and radiographic confirmation (all with confirmatory CT). Although definitions varied widely, prior series have reported dysphagia in 28% [19], 23% [20], and 43% [21] of patients who had a stroke in a cerebral hemisphere. With detailed videofluoroscopic measures and comparisons to age-matched, neurologically normal individuals, this report further defines dysphagia across all of our CVA subjects.

This is our initial effort to study the relationship between CVA localization and swallowing dysfunction. It appears that unilateral cerebral cortical lesions, as defined on clinical and CT grounds, and involving the premotor cortex, produce swallowing dysfunction. Our findings indicate that RCVA subjects may be more prone to aspiration and thus their dysphagia may be more clinically significant than the dysphagia secondary to LCVA. Expansion of our stroke data base, as well as our confirmatory radiographic data base, will enable us to speculate as to whether specific anatomic areas correlate with specific swallowing abnormalities: whether RCVA patients, for instance, "neglect" swallowing tasks or are more "impulsive" swallowers, leading to higher chances of aspiration, or whether there are various forms of swallowing "apraxia." In addition, longitudinal study will be needed to determine the natural history and consequence, if any, of these abnormalities in swallowing ability. We are studying more subjects in order to validate the generalization of these initial findings to the LCVA and RCVA populations. Nonetheless, the concept of a dominant cortical center for volitional swallowing behaviors is intriguing, as is the physiological phenomenon of interhemispheric neuronal dysconnection, in furthering our understanding of the traditional concept that swallowing is under bilateral cortical control.

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