Dysphagia Post Subcortical and Supratentorial Stroke

Ping Wan, MD, PhD,* Xuhui Chen, MD,† Lequn Zhu, MD,‡ Shuangjin Xu, MD,* Li Huang, MD,§ Xiangcui Li, MD,† Qing Ye, MD, PhD,‖ and Ruiying Ding, PhD¶

Background: Studies have recognized that the damage in the subcortical and supratentorial regions may affect voluntary and involuntary aspects of the swallowing function. The current study attempted to explore the dysphagia characteristics in patients with subcortical and supratentorial stroke. *Methods:* Twelve post first or second subcortical and supratentorial stroke patients were included in the study. The location of the stroke was ascertained by computed tomography and magnetic resonance imaging. The characteristics of swallowing disorder were assessed by video fluoroscopic swallowing assessment/fiberoptic endoscopic evaluation of swallowing. The following main parameters were analyzed: oral transit time, pharyngeal delay time, presence of cricopharyngeal muscle achalasia (CMA), distance of laryngeal elevation, the amounts of vallecular residue and pyriform sinus residue (PSR), and the extent of pharyngeal contraction. *Results:* Eighty-three percent of the 12 patients were found suffering from pharyngeal dysphagia, with 50% having 50%-100% PSRs, 50% having pharyngeal delay, and 41.6% cases demonstrating CMA. Simple regression analysis showed PSRs were most strongly associated with CMA. Pharyngeal delay in the study can be caused by infarcts of basal ganglia/thalamus, infarcts of sensory tract, infarcts of swallowing motor pathways in the centrum semiovale, or a combination of the three. *Conclusion:* Subcortical and supratentorial stroke may result in pharyngeal dysphagia such as PSR and pharyngeal delay. PSR was mainly caused by CMA. **Key Words:** Subcortical and supratentorial stroke—dysphagia—cricopharyngeal muscle achalasia—pharyngeal delay.

Introduction

Swallow is a complex process of sensation and movement controlled by the brain. It involves multiple levels of the nervous system including the cerebral cortex, the subcortex, and infratentorium as well as coordination of the various muscles. Damage at any levels will result in dysphagia. A clear understanding of the neural control of swallow is vital for designing appropriate evaluation and treatment programs for dysphagia.[1] Extensive research has attempted to correlate the location of brain damage with the specific symptoms of dysphagia. Warabi et al[2] found that damage to swallow-related cortical areas, including caudal sensorimotor cortex (Brodmann areas 3, 4, and 6), lateral premotor cortex, (Brodmann areas 6 and 8), cingular gyrus, insular and temporopolar cortices, amygdala (Brodmann areas 34 and 38), cerebellum, and cortical swallowing motor pathways (such as internal capsule posterior limb and corona radiata), could result

From the *Department of Speech and Hearing Rehabilitation, Shanghai University of Traditional Chinese Medicine, Shanghai, China; †Department of Ear, Nose and Throat, Yueyang Hospital affiliated to Shanghai University of Traditional Chinese Medicine, Shanghai, China; ‡Department of Radiology, Yueyang Hospital affiliated to Shanghai University of Traditional Chinese Medicine, Shanghai, China; §Department of Rehabilitation Medicine, Yueyang Hospital affiliated to Shanghai University of Traditional Chinese Medicine, Shanghai, China; ‖Department of Ear, Nose and Throat, No. 7 Hospital of Shanghai, Shanghai, China; and ¶Department of Communication Sciences and Disorders, Elmhurst College, Elmhurst, Illinois.

Received June 26, 2015; revision received August 13, 2015; accepted August 26, 2015.

The authors have no competing interests. The authors are responsible for the content and writing of the paper.

Address correspondence to Ruiying Ding, Department of Communication Sciences and Disorders, Elmhurst College, 190 S Prospect Avenue, Elmhurst, IL 60126. E-mail: ruiying.ding@elmhurst.edu.

1052-3057/$ - see front matter

http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2015.08.037

in lengthened oral transit time (OTT) and pharyngeal delay time (PDT). In a meta-analysis, Flowers et al[3] investigated the incidence of dysphagia according to the neuroanatomical lesion sites within the infratentorium and found that dysphagia was present in 6% of midbrain damage, 43% of pons damage, 40% of medial medulla damage, and 57% of lateral medulla damage. Because of the high incidence of dysphagia seen in brainstem stroke patients, previous studies have suggested that the involuntary swallowing function should be intact if the brain stem is not affected.[2,4] However, more recent investigations have indicated that the subcortical and supratentorial structures also play an important role in the control of the swallowing function, including the pharyngeal phase of swallow.

Other parts of the central nervous system are also implicated in the control and coordination of the swallowing function by several studies. Suzuki et al[4] studied 11 right-handed volunteers with no history of swallowing problems undergoing functional magnetic resonance imaging (MRI) examination and demonstrated that volitional swallowing involves the cerebellum and basal ganglia as well as cortical structures. Galovic et al[5] recently investigated the correlation between risk of aspiration with the anatomical location of an acute stroke and they found that the main determinants of aspiration in patients with supratentorial strokes were lesions in the frontal operculum and the insular cortex, whereas patients with acute basal ganglia lesions had less clinically significant dysphagia and sooner recovery. In a study conducted by Sundar et al,[6] of 29 subcortical infarct patients, 6 developed dysphagia, which included 2 out of 9 basal ganglia stroke patients, 1 out of 4 thalamic stroke patients, 1 out of 9 internal capsule stroke patients, and 2 out of 3 brainstem stroke patients, but 4 patients with stroke in the corona radiata and centrum semiovale regions demonstrated safe swallowing. Research of 8 subjects identified with a single, small, left basal ganglion/internal capsule infarction indicated that stroke patients exhibited significantly longer OTT and significantly lower oropharyngeal swallow efficiency scores than the age-matched normal subjects.[7] Results from functional MRI studies of swallowing showed nearly all cortical and subcortical gray region structures were activated during the oral and pharyngeal phases of swallowing.[8] Swallowing deficits involving oral control and transmittal might be a marker of subcortical neural axis involvement. Not only several gray matter areas were found to be involved in swallowing function, but also lesions to the left periventricular white matter were found to be more disruptive to swallowing behavior than similar lesions to the right periventricular white matter.[9]

Normal swallowing tasks produce activation in many sensorimotor cortices including cingulate, premotor cortex, parietal cortex, temporal cortex, basal ganglia and thalamus, insula, and cerebellum.[10] The principal component analysis of these regions revealed 5 functional modules, the model of modules organized into parallel loops to effectively coordinate and integrate this highly complex sequentially based motor behavior. Functional MRI studies with 12 right-handed male volunteers demonstrated the key role of cortico-basal ganglia–thalamocortical networks for self-initiated automated motor repertoires. Involvement of the substantia nigra during planning indicated dopaminergic gating of motor sequences.[11]

To sum up, numerous studies recognized that the damage in the subcortical and supratentorial regions that include basal ganglia and the corona radiata/centrum semiovale may affect the voluntary aspect of swallowing function, such as prolonged OTT and PDT, as well as the involuntary aspect of swallowing function. Our current study attempted to investigate the dysphagia characteristics associated with basal ganglia/centrum semiovale infarcts. More specifically, using instrumentation techniques of video fluoroscopic swallowing assessment (VFSS) and fiberoptic endoscopic evaluation of swallowing (FEES), we analyzed the following swallowing variables: OTT, PDT, presence of vallecular residue (VR) and pyriform sinus residue (PSR), presence of cricopharyngeal muscle achalasia (CMA), distance of laryngeal elevation (DLE), and extent of pharyngeal contraction (PC). The results of the present study will allow us to study the function of basal ganglia in swallow control and explore the central mechanism of CMA.

Materials and Methods

Participants

Between March 2009 and July 2014, 105 consecutive patients with dysphagia were seen at the Department of Rehabilitation and Department of Otolaryngology in the Yueyang Hospital. In this retrospective study, a total of 42 patients following dysphasia suffered from stroke mainly in the basal ganglia region and/or the centrum semiovale. The patients who could not undergo VFSS or FEES because of poor consciousness or were unable to perform a 1-step command were excluded. Therefore, a total of 12 patients met the inclusion criteria. The ages of the 9 male patients and the 3 female patients ranged from 54 to 82 (average ± standard deviation: 66 ± 10).

All 12 patients presented either post first stroke or second stroke. Contrast-enhanced computed tomography or MRI was used to identify the location of the fresh infarcts according to the diagnosing criterion of MRI/computed tomography,[12,13] which were reported in Table 1. Nine patients had first stroke; all showed dysphagia symptoms immediately post stroke. Patients 6 and 12 received swallowing therapy immediately after the acute stroke in other hospitals and continued to show dysphagia symptoms when they came to our hospital as outpatients. It is assumed the dysphagia symptoms were still resulted from the initial basal ganglia stroke. Three patients had second cerebral infarcts: Patients 8 and 10 had their first infarct