

Effect of Visuospatial Neglect on Electrophysiological Parameters of Oropharyngeal Swallowing

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Abstract

Objective: The aim of this study was to determine the effect of visuospatial neglect on the electrophysiological parameters of oropharyngeal swallowing.

Material and Methods: Twenty-six healthy volunteers and 42 stroke patients with complaints of dysphagia were included in the study. The patients were grouped as left hemiplegic patients with visuospatial neglect, left hemiplegic patients without visuospatial neglect, and right hemiplegic patients without visuospatial neglect. Submental electromyographic activity and laryngeal vertical movements were electrophysiologically recorded while each subject swallowed water.

Results: The time required for triggering the pharyngeal phase of swallowing (A–0) and the duration of the pharyngeal phase of oropharyngeal swallowing (A–C) were significantly prolonged in left hemiplegia patients with neglect compared with the patients in the other groups (p<0.05). The time necessary for the elevation, closure, and upward relocation of the larynx (0–2 interval) was prolonged, and the dysphagia limit was decreased in the left hemiplegia patients with neglect compared with the patients in the other groups. The prolongation of larynx movement and decrease in dysphagia limit in patients with neglect were significantly different to the relative measures in the healthy volunteer group (p<0.05) but not to those in the patients without neglect (p>0.05). **Conclusion:** Perceptual deficit is apt to play a significant role in the development of dysphagia via reduced stimulus detection and discriminative capacity in the oropharynx. Thus, we propose that stroke patients with neglect may require a swallowing evaluation, regardless of whether they have complaints of dysphagia.

Keywords: Swallowing, neglect, stroke, electrophysiology, ENMG

Introduction

Sensory inputs can be initiated and sustained either by mucosal receptors of the oropharynx and/or by lingual and/or palatopharyngeal mechanoreceptors during swallowing (1-3). It is believed that sensory feedback originates from the oropharyngeal mucosa and that deeper receptors in the region may modify the central pattern generator (CPG) of the bulbar swallowing center (4). Sensory deficit in the laryngopharyngeal mucosa has been proven as an important cause of aspiration and dysphagia in stroke patients (5,6). Visuospatial neglect can be defined as the tendency for decreasing motor behaviors toward orienting to, searching for, and finding stimuli on the opposite hemispace of brain damage, as well as error tendency in reacting to or reporting orally and/or not being aware of stimuli on the left visual hemispace (7,8). Visuospatial neglect syndrome is frequently seen in patients with right cerebral hemisphere paralysis related

Address for Correspondence: Barın Selçuk, MD, E-mail: barinselcuk@yahoo.com Received: January/Ocak 2014 Accepted: June/Haziran 2014 ©Copyright 2015 by Turkish Society of Physical Medicine and Rehabilitation - Available online at www.ftrdergisi.com Cite this article as: Selçuk B, Uysal H, Kurt M, Kurtaran A, İnanır M, Sade I, Akyüz M. Effect of Visuo-Spatial Neglect on Electrophysiological Parameters of Oropharyngeal Swallowing. Turk J Phys Med Rehab 2015;61:203-10. to cerebral hemorrhage or cerebral infarct (9). The frequency of visuospatial neglect syndrome varies between 13% and 81% in patients with right cerebral hemisphere damage depending on the test for the assessment of the neglect and the location of the lesion (10-12).

In visuospatial neglect syndrome, a decrease in motor behaviors towards the perception and swallowing of food at the beginning of oral swallowing is the cause of serious dysphagia and therefore aspiration. However, a systematically designed study of the effects of visuospatial neglect on deglutition does not currently exist in literature. Therefore, the first aim of this study was to identify the existence of abnormalities in oropharyngeal swallowing in stroke patients with neglect. The second aim was to consider the mechanisms leading to oropharyngeal dysphagia in neglect patients, and the third aim was to determine the electrophysiological parameters of oropharyngeal swallowing affected by neglect in stroke patients. All aspects of swallowing were studied using previously described electrophysiological methods (13-15).

Material and Methods

Participants

Twenty-six healthy volunteers and 42 stroke patients were included in the study. The 26 healthy volunteers (16 men, 10 women, aged 50–78; mean age: 56.8) had no complaint of dysphagia or other neurological, otolaryngological, and/or respiratory problems. The following criteria were used to screen volunteers via a self-report: 1) Good health; 2) No history of any neurological, otolaryngological, and/or respiratory disorders; 3) No history of dysphagia; 4) Not taking any medications that could affect swallowing function; 5) No history of smoking within one year of study; and 6) No ingestion of caffeine for 6 h before the study.

All 42 stroke patients had a history of ischemic cerebrovascular accident and were inpatients being treated at the rehabilitation department of the hospital. Exclusion criteria included having a history of previous stroke, diabetes mellitus, alcoholism, aphasia, dementia, or any other kind of mental disorder (previous or existing); local oropharyngeal disease; and a lack of cooperation. The patients were divided into three groups. Group 1 consisted of 19 left hemiplegic patients (LHPs) with visuospatial neglect (11 men, 8 women, aged 51–77; mean age: 60.6). Group 2 consisted of 12 LHPs without visuospatial neglect (7 men, 5 women, aged 52–74; mean age: 62.1). Group 3 consisted of 11 right hemiplegic patients (RHPs) without visuospatial neglect (7 men, 4 women, aged 55–72; mean age: 63.0). The time between the onset of the medical-neurological problem and the investigation ranged from 35 to 156 days for group 1, 40 to 144 days for group 2, and 29 to 162 days for group 3. Informed consent was obtained from all patients, and the study was approved by the local ethics committee of our hospital. The investigators and data analysis were not blinded.

Procedures

The visuospatial neglect was evaluated using the Turkish Version of the Cancellation Test from which the psychometric

studies for the Turkish culture were comprehensively performed (16,17). The cancellation test has been comprehensively studied in relation to the Turkish culture by Karakaş et al. (17,18). It is sensitive to right hemisphere damage and is widely used to assess behavioral symptoms of visuospatial neglect syndrome (19). All patients were evaluated using this test and were classified in groups 1, 2, or 3.

The subjects were asked to sit on an examination couch and were instructed to hold their heads in a natural upright position. Electrophysiological measurements were then taken. The methods used for the evaluation of swallowing function have been described previously (13-15). In brief, mechanical upward and downward laryngeal movements during swallowing were detected by means of a piezoelectrical sensor. A mechanical laryngeal sensor that consisted of a single piezoelectric wafer with a rubber bulge fixed at its center was placed over the cricothyrotomy region between the cricoid and thyroid cartilages on the midline. The sensor was secured with a rubber band tied around the neck, and its output was connected to the first channels of the electromyographical (EMG) apparatus (Neuropack µ; Nihon Kohden Corp, Tokyo, Japan). The sensor amplifier output was also band-pass filtered (cut-off frequencies: 0.01-20 Hz). The sensor detected two deflections of generally opposing polarity during each swallow. The first deflection of the laryngeal sensor signals represents the upward movement of larynx, whereas the second deflection represents the downward movement. The upward and downward deflections of the laryngeal sensor were sometimes diphasic or triphasic. The first deflection from the baseline was accepted as the point of onset. The leading or trailing edge of the first deflection was used to trigger the delay line circuitry of the recording apparatus so that all signals were time locked to the same instant.

We recorded EMG activity [or submental EMG (SM-EMG)] on the second channel of the EMG apparatus using bipolar silver chloride electroencephalographic (EEG) electrodes taped under the chin and over the mylohyoid–geniohyoid–anterior digastric muscle complex. The EMG signals were pass filtered (100 Hz to 10 kHz), amplified, rectified, and averaged. Because the SM-EMG activity coincided with the laryngeal upward movement, the rectified-integrated SM-EMG activity was also time locked to the laryngeal sensor signals. The total analysis time was adjusted to 2 s, and at least five successive sensor and SM-EMG traces were recorded. The individual traces were examined, superimposed, and then averaged. At least five successive sensor and EMG traces were recorded for each type of swallow.

We performed two analyses during this testing method: single bolus analysis and dysphagia limit analysis. For single bolus analysis, electrophysiological recordings were taken for swallows initiated with 3 ml of water while the tongue tip was touching the upper incisors. The onset of two deflections in the laryngeal sensor signal recordings are denoted as "0" and "2". The interval between the onsets of two deflections (0–2 interval) is thought to reflect the time necessary for the elevation, closure, and upward relocation of the larynx (Figure 1) (13,15). The onset and duration of pharyngeal swallowing were recorded from the SM-EMG activity of the mylohyoid–geniohyoid–anterior digastric

Table 1. Average values of electrophysiological parameters ob- tained from each group				
	Healthy volunteers	LHP with neglect	LHP without neglect	RHP without neglect
0-2 (ms)*	569.4±89.7	694.7±121.0	673.9±107.9	681.1±107.1
A-O (ms) **	123.4±69.4	302.0±57.5	219.5±85.6	225.4±95.5
A-C (ms) ***	707.5±175.3	1139.2±245.1	916.3±179.0	888.5±179.6
DL (mL) ****	27.7±7.1	9.0±7.1	10.0±3.7	9.6±3.5

* Time for the pharyngeal phase of swallowing

** Time for the triggering of the pharyngeal phase of swallowing

*** Duration of SM-EMG **** Dysphagia limit

LHP: Left hemiplegic patients; RHP: Right hemiplegic patients

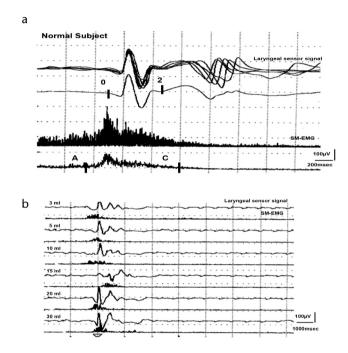


Figure 1. a, b. Electrophysiological assessment of oropharyngeal swallowing in a normal subject: Laryngeal sensor signal (upper traces) and integrated submental EMG activity (lower traces) obtained from a normal subject during swallowing of 3 mL water. The upper two traces in each pair are superimpositions of five responses, whereas the lower two traces in each pair are averages (a). Laryngeal sensor signal (upper traces in each pair) and integrated submental EMG activity (lower traces in each pair) obtained from a normal subject during swallowing of 3 mL water; swallowing of different amounts of water increased from 3 mL to 30 mL. Note that all volumes were swallowed in one attempt up to 30 mL (b)

muscle complex. The total duration was denoted by the "A–C" interval, and the peak amplitude of SM-EMG was measured from averaged traces. The SM-EMG or A–C interval gives a considerable amount of information about the onset and duration of oropharyngeal swallowing (Figure 1) (4,20-22). Oral and pharyn-

geal periods of swallowing are included in the SM-EMG duration (15,20). We were able to simultaneously use the laryngeal sensor and SM-EMG traces to measure the triggering of the pharyngeal phase of swallowing, which was determined by the time interval between the onset of SM-EMG and the first deflection of the signal of the laryngeal sensor. This deflection is one of the first events of the pharyngeal phase of swallowing (4,20). Therefore, the "A–0" time interval between the onset of SM-EMG and the instended on set of the first deflection of the laryngeal sensor provided information about the temporal relationship between the instant of the voluntary activation of SM-EMG and the instant of reflex triggering of the swallowing response (Figure 1) (23,24).

In the second analysis in this method, we measured the dysphagia limit, which is also known as "piecemeal deglutition". The phenomenon of dysphagia limit has previously been investigated using the same measuring technique applied here (14,15,24). The dysphagia limit is based on the detection of a physiological phenomenon that occurs when an oral bolus of large liquid volume is divided into two or more pieces that are then successfully swallowed. We investigated the dysphagia limit with the sweep time set at 10 s and the delay line started for 2 s. Therefore, after a certain amount of liquid was consumed, the effect of the bolus was followed for 8 s. All subjects were given 3, 5, 10, 15, 20, and 30 mL of water, and oscilloscope traces were started at the examiner's order to swallow. If there was no recurrence of EMG and laryngeal activity with these amounts of water, 40 mL and 50 mL of water were given until two or more swallows occurred. The laryngeal sensor traces and the rectified and averaged activities of SM-EMG were recorded from the beginning of these long sweeps of the oscilloscope (Figure 1). It was requested that the patient swallow all the liquid given in a single effort. Any swallowing-related recurrence of the EMG activity and the laryngeal sensor signal within 8 s after the onset of the sweep was accepted as piecemeal deglutition or as a sign of the dysphagia limit. However, because the piecemeal deglutition was physiologically observed in the normal subjects when swallowing more than 20 mL of water, duplication or multiplication at or below 20 mL of water is referred to as the "dysphagia limit" (14).

Statistical Analysis

We calculated the mean ± standard error of the mean for all measured parameters. Variance and correlation analysis were used to assess the differences in swallowing parameters as appropriate. Paired t tests were also used to make statistical comparisons. A univariate one-way analysis of variance and post hoc Tukey's honest significant difference test were applied to the data obtained for the groups using Statistical Package for the Social Sciences for Windows release 10.0 (SPSS, Inc., Chicago, IL, USA).

Results

The average values of electrophysiological parameters obtained from the normal subjects and the patients for each group are shown in Table 1. The duration of the pharyngeal phase of swallowing (calculated from the 0–2 interval) was slightly prolonged in LHPs with neglect compared with the patients in

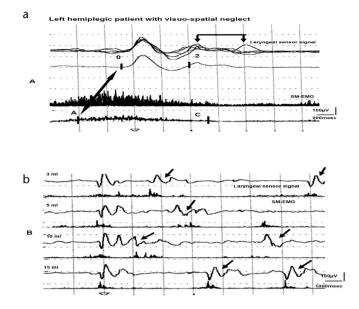


Figure 2. a, b. Electrophysiological assessment of oropharyngeal swallowing in a left hemiplegic patient with visuospatial neglect: Laryngeal sensor signal (upper traces) and integrated submental EMG activity (lower traces) obtained from a left hemiplegic patient with visuospatial neglect during the swallowing of 3 mL water. The upper two traces in each pair are superimpositions of five responses, whereas the lower two traces in each pair are averages (a). Laryngeal sensor signal (upper traces in each pair) and integrated submental EMG activity (lower traces in each pair) obtained from a left hemiplegic patient with visuospatial neglect during swallowing of 3 mL water; swallowing different amounts of water increased from 3 to 15 mL. The A-0 interval, A-C intervals, 0-2 interval, and jitter are prolonged in the left hemiplegic patient with neglect compared with these measures in normal subjects, and A-0 and A-C intervals are prolonged in left hemiplegic patients with neglect compared with these measures in patients from each of the other groups. The dysphagia limit was 3 mL of water in this patient; hence, the bolus divided into two separate swallows during swallowing \geq 3 mL water (note the traces) (b)

each of the other groups (Figure 2). This prolongation in LHPs with neglect was significantly different to the duration of the pharyngeal phase of swallowing in the healthy volunteer group (p=0.001) but not in LHPs without neglect or RHPs without neglect (p=0.950 and p=0.998, respectively). This variable was also prolonged in all patient groups (groups 1, 2, and 3) compared with the relative measurement in the healthy volunteers (p=0.001, p=0.029, and p=0.022, respectively) (Figure 3,4).

The time necessary for the triggering of the pharyngeal phase of swallowing (calculated from the A–0 interval) was significantly prolonged in LHPs with neglect compared with the patients in each of the other groups, i.e., healthy volunteers and groups 2 and 3 (p=0.02, p=0.019, and p=0.049, respectively) (Figure 2-4). This variable was also prolonged in all patient groups (groups 1, 2, and 3) compared with the relative

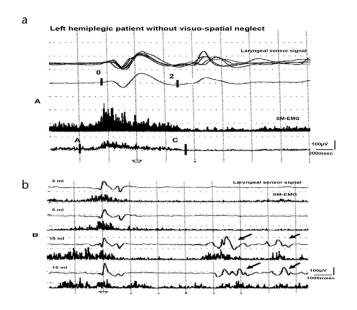


Figure 3. a, b. Electrophysiological assessment of oropharyngeal swallowing in a left hemiplegic patient without visuospatial neglect: Laryngeal sensor signal (upper traces) and integrated submental EMG activity (lower traces) obtained from a left hemiplegic patient without visuospatial neglect during swallowing of 3 mL water. The upper two traces in each pair are superimpositions of five responses, whereas the lower two traces in each pair are averages (a). Laryngeal sensor signal (upper traces in each pair) and integrated submental EMG activity (lower traces in each pair) obtained from a left hemiplegic patient without visuospatial neglect during swallowing of 3 mL water; swallowing different amounts of water increased from 3 to 15 mL. The 0-2 and A-0 intervals were prolonged in the left hemiplegic patient without visuospatial neglect compared with respective measures in a normal subject. The dysphagia limit was 10 mL of water in these subjects; thus, the bolus divided into two separate swallow sequences when swallowing 10 mL water (note the traces at 10 mL) (b)

measurement in the healthy volunteers (p=0.001, p=0.002, and p=0.002, respectively).

The total duration of SM-EMG (calculated from the A–C interval) was significantly prolonged in LHPs with neglect compared with the patients in each of the other groups, i.e., healthy volunteers and groups 2 and 3 (p=0.01, p=0.017, and p=0.008, respectively) (Figure 2). This variable was also prolonged in all patient groups (groups 1, 2, and 3) compared with the relative measure in the healthy volunteers (p=0.001, p=0.019, and p=0.064, respectively) (Figure 3,4).

The dysphagia limit was >20 mL of water in all the normal (control) volunteers investigated, whereas it was clearly pathological and <20 mL in all stroke patient groups. Additionally, the dysphagia limit decreased in LH with neglect patients compared with the limit observed in patients from each of the other

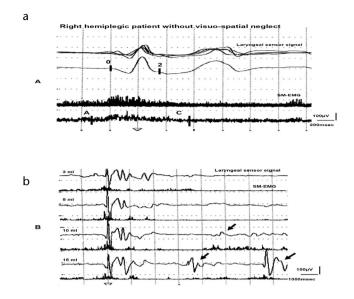


Figure 4. a, b. Electrophysiological assessment of oropharyngeal swallowing in a right hemiplegic patient without visuospatial neglect: (A) Laryngeal sensor signal (upper traces) and integrated submental EMG activity (lower traces) obtained from a right hemiplegic patient without visuospatial neglect during swallowing of 3 mL water. The upper two traces in each pair are superimpositions of five responses, whereas the lower two traces in each pair are averages (a). Laryngeal sensor signal (upper traces in each pair) and integrated submental EMG activity (lower traces in each pair) obtained from a right hemiplegic patient without visuospatial neglect during swallowing of 3 mL water; swallowing different amounts of water increased from 3 to 15 mL. The 0-2 and A-0 intervals were prolonged in the left hemiplegic patient without visuospatial neglect compared with these respective measures in a normal subject. The dysphagia limit was 10 mL of water in these subjects; thus, the bolus divided into two separate swallow sequences during the swallowing of 10 mL water (note the traces at 10 mL) (b)

groups. The decrease in the dysphagia limit observed in LHPs with neglect differed significantly with the limit observed in the healthy volunteers (p=0.001) but not the limit in LHPs without neglect or RHPs without neglect (p=0.995 and p=0.998, respectively). The dysphagia limit also decreased in all patient groups (groups 1, 2, and 3) compared with that measured in healthy volunteers (p=0.001, p=0.001, and p=0.001, respectively) (Figure 2-4).

Discussion

One of the most devastating and common complications of stroke is dysphagia, which occurs in approximately 30–45% of all stroke patients (25-27). The etiology of dysphagia after stroke is multifactorial, but one of the most common reasons is motor dysfunction, which includes the absence or delay in the triggering of the swallowing reflex, cricopharyngeal dysfunctions, re-

duced lingual control, reduced gag reflex, and reduced strength of pharyngeal contraction (24-26). Although motor dysfunction of the laryngopharynx clearly plays a major role in dysphagia in stroke patients, sensory dysfunction in this region also impairs laryngeal reflexes and the voluntary laryngopharyngeal protective mechanism (5-6,28-31). According to Aviv et al. (5), the sensory deficit in stroke patients is related to the disruption of an ascending protection pathway transmitting sensory information from the laryngopharynx mucosa to the diencephalon and cortex.

Visual neglect is a common deficit after unilateral brain injury, particularly following strokes centered on the right superior and inferior parietal lobes; however, other influential areas also exist, including the frontal lobes, anterior cingulate cortex, frontal eye fields, basal ganglia, and thalamus (32-38). Falsetti et al. (38) showed that hemispatial neglect is characteristically associated with cortical right (nondominant) hemispheric damage but not internal capsule (subcortical) and lacunar right-sided damage. Patients with visuospatial neglect typically fail to appropriately report or orient for visual stimuli presented contralaterally to the damaged hemisphere. They may also neglect food on the left part of their plate because of perceptual dysfunction. Patients with perceptual dysfunction have problems discriminating among sensory stimuli and organizing to give meaning to stimuli (37).

The A-0 interval reflects voluntary attempts to swallow until the swallowing reflex is initiated, and therefore, it may be related to the triggering of the swallowing reflex (9,39-40). In the present study, the A-0 interval was prolonged in all stroke groups compared with healthy volunteers, and it was also prolonged in LHPs with neglect compared with LHPs and RHPs without neglect. The prolonged A-0 time interval is probably related to the abnormal triggering mechanism of the swallowing reflex. Normally, this interval is under cortical control either directly or via the brainstem CPG, and this phase of swallowing is presumably initiated purely by sensory inputs arising from the oropharyngeal cavity (20,23,40-45). At the brainstem level, all afferent nerve fibers from the oral cavity are involved in initiating or facilitating swallowing convergence in CPG, especially in NTS, together with cortical drive. Therefore, the brainstem CPG receives the main sensory input from the oropharyngeal region, and cortical descending inputs reach similar areas of CPG. Hence, some sensory inputs that initiate swallowing are transmitted to the region of the cortex that facilitates the initiation of swallowing (20).

In classical neglect syndrome, there is an absence of normal attention and response to events in the visual field contralateral to the lesion. In addition, according to the sensory–perceptual hypothesis, conceptualized neglect involves attenuation of sensory input to the right hemisphere from the contralateral side of the body and space (46). A few studies have specifically examined the relationship between lateralized neurocognitive deficits (hemispatial neglect and aphasia) and dysphagia. Schroeder (47) showed that hemispatial neglect is associated with dysphagia, and that it is associated more strongly with dysphagia than with aphasia. Although cognitive deficits are frequently as-

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sociated with specific hemispheres, they can occur with deficits in the neural systems that underlie these cognitive functions, which are not strictly hemispheric in nature. Therefore, we suggest that sensory inputs necessary for the perception of bolus and viscosity are not propagated to the central nervous system and that the triggering of swallowing reflex is delayed in neglect patients because of perceptual dysfunction, problems discriminating among sensory stimuli, problems organizing to give meaning to stimuli, and problems with bolus formation in the mouth. The SM-EMG duration, denoted as the A-C interval in this study, was significantly prolonged in LHPs with neglect compared with the relative measurements observed in LHPs without neglect and RHPs without neglect. SM-EMG also demonstrates the onset and the end of the oropharyngeal stage of swallowing because these muscles fire concurrently to initiate the swallowing response and then stop firing eventually as the larynx begins to descend. Movements that occur from the beginning of submental muscle contraction to the elevation of the larynx are important for the safe passage of bolus to the pharyngoesophageal segment without escaping into the other cavities. The contraction of the submental muscles continues until the completion of the oropharyngeal swallowing process (20,23). Thus, submental muscles have two actions during swallowing: the protection of the larynx by elevation and the transportation of the bolus by a secondary support to the tongue to pump (48). We found that in LHPs with neglect, the submental muscles were activated for an abnormally long period during swallowing and that the duration of SM-EMG (A-C interval) was prolonged. In stroke patients, the increase in duration of SM-EMG exceeds the upper limit of normal values obtained from healthy volunteers. The prolongation of SM-muscle excitation is related to either muscular weakness or a central effect. The prolonged activity of SM-muscle probably serves to overcome aspiration during swallow. It may also be related to the difficulties in triggering the swallowing reflex (40).

In neglect patients, there is diminution of sensory capacity in the laryngopharynx (5,6). Because discriminating among sensory stimuli and organizing to give meaning to stimuli are disturbed. So, submental muscles activation was prolonged. The duration of the swallowing reflex (0–2 time interval of laryngeal relocation time) was prolonged in LH with neglect patients compared with that observed in patients from each of the other groups. Although this prolongation was not statistically significant in either LH without neglect or RH without neglect patients, it was significantly prolonged in all stroke patients compared with healthy volunteers. Muscle weakness in the jaw, buccal, tongue, submental, and pharyngeal constrictor muscles, as well as perceptual dysfunction, may be responsible for the problems of bolus formation in the mouth and slow transit of the bolus along the pharynx in stroke patients.

All normal subjects in this study easily tolerated swallowing 20 mL (or a little more) water in one attempt. However, stroke patients, especially LH with neglect patients, exhibited dysphagia limits of 9–10 mL water and two or more swallows. Piecemeal deglutition and lowered dysphagia limits, together with duplication or multiplication of the bolus, have frequently been observed in patients with neurogenic dysphagia and after topical anesthesia of oropharyngeal mucosa (14,40-41,43,48). When oropharyngeal swallowing is impaired but compensated, patients can change their eating habits, i.e., by frequently eating small meals, and thereby reduce the individual bolus size. Swallowing for a second time with each bolus helps to clear retained material from the pharynx (14). Besides the voluntary compensations for impaired swallowing of which the patients may be aware, the compensation is also involuntary, i.e., it takes place through adjustments in the swallowing apparatus itself (14,41).

It is suggested that mucosal receptors of the oropharynx are necessary for normal swallowing physiology and that the diminution of sensory input, discriminating among sensory stimuli, and problems organizing to give meaning to stimuli due to neglect cause swallowing dysfunction in stroke patients with neglect. The re-positioning time (0-2 interval) of the larynx during a swallow was measured from the onset of laryngeal sensor signals, which reflects the time necessary for the elevation and upward relocation of the larynx. In a previous study, it was found that laryngeal relocation time during swallowing is prolonged in stroke patients, probably due to weakness in laryngeal elevators (40). Swallowing jitter can be regarded as the variability in the swallowing apparatus under a given swallowing condition. It can also be an important parameter for the safety of deglutition during swallowing (15). Swallowing jitter is prolonged in stroke patients; this is another type of compensation for swallowing when weakness exists in oral and pharyngeal muscles (15,40).

The present study demonstrated that dysphagia exists in stroke patients with neglect. The perceptual deficit is apt to play a significant role in the development of dysphagia by reducing stimulus detection and discriminative capacity in the laryngopharynx. We suggest that stroke patients with neglect are at the greatest risk of developing aspiration because of these issues. Neglect in LHPs can be treated by cognitive rehabilitation, and this form of management could also contribute to improving dysphasia in these patients. Further studies on neglect rehabilitation in these patients, which aim to specifically assess the decrease in time necessary to trigger the pharyngeal phase of swallowing and identify the duration of the pharyngeal phase of oropharyngeal swallowing, will be necessary to confirm this suggestion.

One should note that we neither identified precise lesion locations or size in these patients nor studied the effect of clinical characteristics of stroke on swallowing. Thus, whether lesion size, lesion location, or a combination of these factors in conjunction with clinical and neurocognitive deficits are related to poor swallowing outcome remains unclear.

Conclusion

Based on our results, we propose that stroke patients with neglect, whether or not they have complaints of dysphagia, require an evaluation of swallowing.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Ankara Physical Medicine and Rehabilitation Training and Research Hospital.

Informed Consent: Written informed consent was obtained from patients and patients' parents who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - B.S.; Design - B.S., H.U., M.A.; Supervision - B.S., H.U., M.A.; Resource - A.K., M.İ., I.S.; Materials - B.S., H.U.; Data Collection and/or Processing - B.S., M.K.; Analysis and/or Interpretation - B.S., H.U., M.A., M.K.; Literature Review - B.S., A.K., M.İ., I.S.; Writer - B.S.; Critical Review - B.S., H.U., M.A., M.İ.

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References

- Månsson I, Sandberg N. Effect of surface anaesthesia on deglutition in man. Laryngoscope 1974;84:427-37. [CrossRef]
- Månsson I, Sandberg N. Oro-pharyngeal sensitivity and elicitation of swallowing in man. Acta Otolaryngol 1975;79:140-5. [CrossRef]
- Miller AJ. Significance of sensory inflow to the swallowing reflex. Brain Res 1972;43:147-59. [CrossRef]
- 4. Miller AJ. Deglutition. Physiol Rev 1982;62:129-84.
- Aviv JE, Martin JH, Sacco RL, Zagar D, Diamond B, Keen MS, et al. Supraglottic and pharyngeal sensory abnormalities in stroke patients with dysphagia. Ann Otol Rhinol Laryngol 1996;105:92-7. [CrossRef]
- Aviv JE, Sacco RL, Thomson J, Tandon R, Diamond B, Martin JH, et al. Silent laryngopharyngeal sensory deficits after stroke. Ann Otol Rhinol Laryngol 1997;106:87-93. [CrossRef]
- Rizzolatti G, Berti A. Neglects as a neural representation deficit. Review of Neurology 1990;146:626-34.
- Bisiach E, Capitani E, Porta E. Two basic properties of space representation in the brain: Evidence from unilateral neglect. J Neurol Neurosurg Psychiatry 1985;48:141-4. [CrossRef]
- Robertson IH, Halligan PW. Spatial Neglect: A Clinical Handbook for Diagnosis and Treatment. Hove, United Kingdom: Psychology Press Ltd, 1999:6.
- Stone SP, Wilson BA, Wroot A, Halligan PW, Lange LS, Marshall JC, et al. The Assessment of Visuospatial Neglect After Acute Stroke. J Neurol Neurosurg Psychiatry 1991;54:345-50. [CrossRef]
- Sunderland A, Wade DT, Langton-Hewer R. The natural history of visual neglect after stroke: indications from two methods of assessment. J Int Disabil Stud 1987;9:55-9. [CrossRef]
- Stone SP, Patel P, Greenwood RJ, Halligan PW. Measuring visual neglect in acute stroke and predicting its recovery: the visual neglect recovery index. J Neurol Neurosurgery Psychiatry 1992;55:431-6.
 [CrossRef]
- Ertekin C, Pehlivan M, Aydoğdu I, Ertaş M, Uludağ B, Celebi G, et al. An electrophysiological investigation of deglutition in man. Muscle Nerve 1995;18:1177-86. [CrossRef]
- Ertekin C, Aydoğdu I, Yüceyar N. Piecemeal deglutition and dysphagia limit in normal subject and in patients with swallowing disorders. J Neurol Neurosurg Psychiatry 1996;61:491-6. [CrossRef]
- Selçuk B, Uysal H, Aydogdu I, Akyuz M, Ertekin C. Effect of temperature on electrophysiological parameters of swallowing. J Rehabil Res Dev 2007;44:373-80. [CrossRef]
- Weintraub S, Mesulam MM. Mental state assessment of young and elderly adults in behavioral neurology. In: Mesulam MM, ed. Principles of Behavioral Neurology. Philadelphia: F.A. Davis Company, 1985.

- 17. Karakaş S. BILNOT Battery: Research and Development of Neuropsychological Tests. Ankara (Turkey): Dizayn Ofset, 2004.
- Kurt M, Karakaş S. The Effect of an original attention-related training program on visuospatial neglect. Int J Psychophysiol 2004;54:152-3.
- Lezak M. Neuropsychological Assessment. (3.Ed), New York: Oxford University Press, 1995.
- 20. Ertekin C, Aydogdu I. Neurophysiology of swallowing. Clin Neurophysiol 2003;114:2226-44. [CrossRef]
- 21. Miller AJ. The neuroscientific principles of swallowing and dysphagia. San Diego, CA/London, Singular Publication Group, 1999.
- 22. Doods WJ, Stewart ET, Logemann JA. Physiology and radiology of the normal oral and pharyngeal phase of swallowing. AJR Am J Roentgenol 1990;154:953-63. [CrossRef]
- Ertekin C, Kiylioglu N, Tarlaci S, Turman AB, Secil Y, Aydogdu I. Voluntary and reflex influences on the initiation of swallowing reflex in man. Dysphagia 2001;16:40-7. [CrossRef]
- Logeman JA. Evaluation and treatment of swallowing disorders.
 2nd edition Austin: TX, Pro-ed Inc, 1998.
- 25. Veis SL, Logemann JA. Swallowing disorders in persons with cerebral vascular accident. Arch Phys Med Rehabil 1985;66:372-5.
- Flowers HL, Silver FL, Fang J, Rochon E, Martino R. The incidence, co-occurence, and predictors of dysphagia, dysartria, and aphasia after first-ever acute ischemic stroke. J Commun Disord 2013;46:238-48. [CrossRef]
- 27. Daniels SK, Brailey K, Priestly DH, Herrington LR, Weisberg LA, Foundas AL. Aspiration in patients with acute stroke. Arch Phys Med Rehabil 1998;79:14-9. [CrossRef]
- 28. Johnson ER, Mckenzie SW, Sievers A. Aspiration pneumonia in stroke. Arch Phys Med Rehabil 1993;74:973-6.
- 29. Aviv JE, Sacco RL, Mohr JP, Thompson JL, Levin B, Sunshine S, et al. Laryngopharyngeal sensory testing with modified barium swallow as predictors of aspiration pneumonia after stroke. Laryngoscope 1997;107:1254-60. [CrossRef]
- Kumar S, Doughty C, Doros G, Selim M, Lahoti S, Gokhale S, et al. Recovery of swallowing after dysphagic stroke: An analysis of prognostic factors. J Stroke Cerebrovasc Dis 2014;14:56-62. [CrossRef]
- 31. Kidd D, Lawson J, Nesbitt R, Macmahon J. Aspiration in acute stroke: a clinical study with videofluroscopy. Q J Med 1993;86:825-9.
- Vallar G. The anatomical basis of spatial hemineglect in humans. In: IH Robertson, JC Marshall (Eds), Unilateral neglect: Clinical and experimental studies. Hove: Lawrence Erlbaum Ass, 1993, pp.27-59.
- Pavani F, Husain M, Ládavas E, Driver J. Auditory deficits in visuospatial neglect patients. Cortex 2004;40:347-65. [CrossRef]
- Fujii T, Fukatsu R, Kimura I, Saso S, Kogure K. Unilateral spatial neglect in visual and tactile modalities. Cortex 1991;27:339-43.
 [CrossRef]
- 35. Vallar G, Perani D. The anatomy of unilateral neglect after right hemisphere stroke lesions. A Clinical/CT-scan correlations study in man. Neuropsychologia 1987;24:609-22. [CrossRef]
- 36. Kastner S, Ungerleider LG. Mechanisms of visual attention in the human cortex. Annu Rev Neurosci 2000;23:315-41. [CrossRef]
- 37. Wyness MA. Perceptual dysfunction: nursing assessment and management. J Neurosurg Nurs 1985;7:105-10. [CrossRef]
- Falsetti P, Acciai C, Palilla R, Bosi M, Carpinteri F, Zingarelli A, et al. Oropharyngeal dysphagia after stroke: incidence, diagnosis, and clinical predictors in patients admitted to a neurorehabilitation unit. J Stroke Cerebrovasc Dis 2009;18:329-35. [CrossRef]
- 39. Ertekin C. Clinical diagnosis and electrodiagnosis of swallowing disorders. In Swenson M.R. (Ed), Disorders of Speech and Swallowing.

AAEM 19th Annual Continuing Education Courses. Johnson, Minneapolis, MN, 1996, pp. 23-33.

- Ertekin C, Aydogdu I, Yüceyar N, Tarlaci S, Kiylioglu N, Pehlivan M, et al. Electrodiagnostic methods for neurogenic dysphagia. Electroencephalogr Clin Neurophysiol 1998;109:331-40. [CrossRef]
- 41. Ertekin C, Kiylioglu N, Tarlaci S, Keskin A, Aydogdu I. Effect of mucosal anaesthesia on oropharyngeal swallowing. Neurogastroenterol Motil 2000;12:567-72. [CrossRef]
- 42. Doods WJ, Stewart ET, Logemann JA. Physiology and radiology of the normal oral and pharyngeal phase of swallowing. AJR Am J Roentgenol 1990;154:953-63. [CrossRef]
- Ertekin C, Aydogdu I, Yüceyar N, Kiylioglu N, Tarlaci S, Uludag B. Pathophysiological mechanisms of oropharyngeal dysphagia in amyotrophic lateral sclerosis. Brain 2000;123:125-40. [CrossRef]

- 44. Perlman AL, Palmer PM, McCulloch TM, Vandaele DJ. Electromyographic activity from human laryngeal, pharyngeal and submental muscles during swallowing. J Appl Physiol 1999;86:1663-9.
- 45. Jean A. Brain stem control of swallowing: neuronal network and cellular mechanisms. Physiol Rev 2001;81:929-69.
- MijovicD. Mechanism of visual spatial neglect. Brain 1991;114:1575-93. [CrossRef]
- 47. Schroeder MF, Daniels SK, McClain M, Corey DM, Foundas AL. Clinical and cognitive predictor of swallowing recovery in stroke. J Rehabil Res Dev 2006;43:301-10. [CrossRef]
- Ertekin C, Yüceyar N, Aydogdu I. Clinical and electrophysiological evaluation of dysphagia in myasthenia gravis. J Neurol Neorosurg Psychiatry 1998;65:848-56. [CrossRef]