Dysphagia in Neurological Disorders

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Abstract

Neurogenic dysphagia is a disorder with impaired swallowing, which is caused by various disorders of the central and peripheral nervous systems, neuromuscular transmission, or muscles. Dysphagia is one of the most common and at the same time the most dangerous symptoms of many neurological disorders. Patients with dysphagia often have severe disability, a higher risk of aspiration pneumonia, and significantly increased mortality rate. Despite the availability of many diagnostic screening methods, clinical scales, questionnaires, and instrumental diagnostic methods, the issue of neurogenic dysphagia is underestimated, especially in the early stages. As a result, patients do not receive timely treatment and prevention of dysphagia and associated complications. Validation of available diagnostic scales, development of international protocols and standards for the diagnosis, treatment, and prevention of dysphagia and associated complications are important to establish a unified and evidence-based approach for patients with dysphagia.

Keywords: *neurogenic dysphagia; oropharyngeal dysphagia; aspiration pneumonia; cachexia*

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Проблема дисфагии в неврологии

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Аннотация

Нейрогенная дисфагия – это расстройство, характеризующееся нарушением акта глотания, вызванным различными заболеваниями центральной и периферической нервной системы, нервно-мышечной передачи или мышц. Дисфагия является одним из наиболее частых и в то же время наиболее опасных симптомов многих неврологических заболеваний. Пациенты, страдающие дисфагией, получают зачастую тяжёлую степень инвалидности, у них выше риск развития аспирационной пневмонии и значительно повышена смертность. Несмотря на большое количество разработанных скрининговых методов диагностики, клинических шкал, опросников и инструментальных методов диагностики проблеме нейрогенной дисфагии, особенно на ранних стадиях, уделяется недостаточное внимание, в результате чего пациенты не получают своевременное лечение и профилактику дисфагии и сопутствующих осложнений. Валидация имеющихся диагностических шкал, разработка международных протоколов и стандартов диагностики, лечения и профилактики дисфагии и ассоциированных осложнений актуальны и важны для создания унифицированного и научно-обоснованного подхода к пациентам, сталкивающимся с дисфагией.

***Ключевые слова**: нейрогенная дисфагия; орофарингеальная дисфагия; аспирационная пневмония; кахексия*

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Introduction

According to the International Statistical Classification of Diseases and Related Health Problems, 10th revision (ICD-10), dysphagia is a disorder characterized by difficulty in swallowing, i.e. disturbed transfer of food and/or liquid from the oral cavity through the pharynx and esophagus into the stomach. Over 30% of hospitalized patients were estimated to have one or another type of dysphagia [2]. A total of 400,000 to 800,000 new cases of dysphagia secondary to neurological disorders are diagnosed annually, and the incidence of neurogenic dysphagia among patients over 60 years of age is 46% [3–5]. Dysphagia is more prevalent in patients with stroke (in 8.1–80% of cases), Parkinson's disease (11–81%), or traumatic brain injury (27–30%) [6]. Dysphagia also occurs in patients with dementia (in up to 85.9% of cases), Huntington's disease (90.5%), multiple sclerosis (25.4%), and in children with neuromuscular disease (47.3%) [7–10]. Chronic dysphagia leads to malnutrition, dehydration, and aspiration pneumonia; it is associated with longer hospital stay, increased anxiety, and risk of death [1]. An informed decision about treatment, rehabilitation, prevention, and improving the quality of life of patients with neurogenic dysphagia involves understanding the physiology and pathophysiology of the act of swallowing, the etiology and clinical features of dysphagia, development of international approaches to diagnosis, and patient's management by an interdisciplinary team of specialists.

Physiology of normal swallowing

International Classification of Functioning, Disability and Health describes swallowing as "functions of clearing substances, such as food, drink and saliva, through the oral cavity, pharynx and oesophagus into the stomach at an appropriate rate and speed" . Current understanding of the swallowing mechanism has been established on the basis of numerous scientific studies that were performed mainly in mammals. Owing to these studies, we know that the act of swallowing is a complex process that consists of three successive phases and involves the organized functioning of approximately 50 pairs of skeletal muscles and 5 pairs of cranial nerves (V, VII, IX, X, XII), which is mediated by the cerebral cortex and the nuclei of the brain stem [3, 11, 12].

The first (i.e. oral transit) phase is a preparatory voluntary step of swallowing, which consists of two stages. First, food is crushed and mixed with saliva due to contraction of the muscles of the tongue, orbicularis oris, masticatory and cheek muscles. This is followed by the stage of holding food or liquid over the lower part of the mouth by stimulating receptors around the soft palate, palatine arches, and root of the tongue. The back of the tongue and the velum palatinum are raised to prevent the bolus from entering the pharynx prematurely. During the second stage, the distal part of the tongue elevates, while its proximal part descends, pushing the bolus along the hard palate toward the oropharynx.

The second (i.e. pharyngeal) phase is a reflex phase, during which the food bolus moves from the oropharynx into the esophagus. When the food bolus reaches the pharynx, the velum palatinum rises, which seals off the nasopharynx and prevents nasal regurgitation, and the tongue rises to the back wall of the pharynx to prevent regurgitation of the bolus into the oral cavity. The oral and nasal cavities are closed, thus creating the pressure to move the bolus through the pharynx. At the same time, the pharyngeal constrictor muscles contract to push the bolus into the esophagus. As the velum palatinum rises, the hypoglossal-laryngeal complex moves upward and forward due to the contraction of the larynx muscles, ensuring closure of the larynx lumen with the help of the epiglottis. The vocal cords close once the adductor muscles of the larynx are activated. With this mechanism, the larynx and lower respiratory tract are protected from aspiration of the bolus passing through the pharynx. The total duration of the oral and pharyngeal swallowing phases is 0.6 to 1.0 seconds.

The muscles of the upper esophageal sphincter (UES) finally relax (their tonic activity is normally constantly maintained outside swallowing), and the third (esophageal) phase of swallowing begins. It is longer (from 10 seconds or longer), is controlled by both the somatic and autonomic nervous systems, and involves transit of the food bolus through the esophagus toward the stomach due to the peristaltic wave caused by contraction of the striated and smooth muscles of the esophagus [3, 12, 13].

Key centers involved in the act of swallowing include the anterior part of the insular cortex and the frontoparietal operculum, including the lower part of the primary motor, somatosensory, and premotor cortex [3, 11]. The primary motor and somatosensory cortex was shown by functional magnetic resonance imaging to be simultaneously activat-

ed during swallowing or oral sensory stimulation, likely functioning synchronically. The primary motor cortex is assumed to initiate and execute swallowing because electrical stimulation of this area causes rhythmic swallowing movements. There are efferent projections from the primary motor cortex to the muscles involved in swallowing, including the mylohyoid, pharyngeal, and esophageal muscles. The somatosensory cortex is activated when various types of sensory information enter the oral cavity, larynx, pharynx, or esophagus [11].

In adults, functional magnetic resonance imaging that was performed during swallowing showed cortical activation of the insula, cingulate cortex, supplementary motor area, premotor cortex, auditory cortex, inferior frontal gyrus, parieto-occipital and prefrontal cortex, tegmentum, putamen, thalamus, globus pallidus, cerebellum, corpus callosum, basal ganglia, caudate nucleus, and inferior parietal lobe [11, 14]. These structures are considered to be interconnected through two main functional circuits, i.e. cerebellar and insular loops. The cerebellar loop includes functional connections between the inferior frontal gyrus, secondary sensory cortex, corpus callosum, basal ganglia, thalamus, and between the sensorimotor cortex and cingulate cortex and cerebellum. The cerebellar loop modulates swallowing movements and coordinates swallowing-related events such as respiration. The insular loop includes connections between the premotor cortex and posterior parietal cortex, the sensorimotor cortex and the cingulate gyrus and insula. The insular loop synchronizes swallowing movements and integrates sensorimotor information in the cerebral cortex. The insula, known as the primary gustatory cortex, is activated during painful and non-painful stimulation of the esophagus and may be involved in the processing of mechanical sensory information. The insula is the primary integrative region for voluntary swallowing, which coordinates visceral sensory and motor information and may play a key role in the initiation of swallowing. The cingulate cortex is a part of the lymbic system, which is involved in the initiation and motivation of goal-directed behaviors, attention, and cognition. The cingulate cortex is involved in higher order cognitive processing of swallowing [11].

The cortical masticatory area plays an important role in swallowing; its repeated stimulation induced rhythmic chewing movements of the jaw in primates [15]. The cortical masticatory area includes the principal part, which is located in the precentral gyrus anterolateral to the primary motor cortex, and the deep part, which is located in the inner face of frontal operculum [11]. This region of the cortex receives projections from the sensory and motor nuclei of the thalamus, intracortical projections from the frontal, parietal, and orbital parts of the cerebral hemispheres, and communicates with the swallowing central pattern generator (SCPG) in the brainstem directly or indirectly through the basal ganglia. While being modulated by sensory feedback, this complex network allows performing a sequence of chewing movements. Located in the medulla oblongata around the solitary tract nucleus, the SCPG consists of two blocks of interneurons of the reticular formation on each side of the medulla oblongata, which regulate the final stage of swallowing, and modulates the swallowing process depending on the size, texture, and temperature of the bolus. The SCPG is connected through the nucleus ambiguus to the muscular complex that is involved in swallowing and to the receptors of the oral mucosa, pharynx and larynx through the trigeminal, glossopharyngeal, and vagus nerves and the solitary tract nucleus [3]. Information about the texture, temperature, taste, and movement of the food bolus is transmitted through transient receptor potential (TRP) receptors, which lead to depolarization of the sensory neurons due to the entry of calcium ions: transient receptor potential vanilloid 1 (TRPV1), which is activated by high temperature (more than 43ºC) and capsaicin, transient receptor potential ankyrin 1 (TRPA1), which is activated by low temperature (less than 17ºC), and transient receptor potential melastatin 8 (TRPM8), which is activated by the temperature of 25 to 28ºC and menthol [16].

Etiology of dysphagia

Dysphagia can occur in any of the three swallowing phases; however, considering that the pathogenesis of oral and pharyngeal dysphagia is similar, dysphagia is most often classified to oropharyngeal and esophageal [17].

The etiology of oropharyngeal dysphagia includes structural, toxic, and neurological disorders. Neurogenic dysphagia is associated with damage to various brain regions (including primary and secondary somatosensory and motor cortex, supplementary motor area, inferior frontal gyrus, anterior cingulate cortex, orbitofrontal cortex, supramarginal gyrus, insula, basal ganglia, corona radiata, thalamus, internal capsule, periventricular white matter and brain stem), damage to the peripheral nervous system, neuromuscular junction, and primary muscle damage [11]. Neurogenic dysphagia occurs mainly in patients with acute stroke, Parkinson's disease, head injury, dementia, amyotrophic lateral sclerosis, myositis, or myasthenia gravis [6, 18, 19]. The swallowing function is also affected by dental disease and decreased saliva production (see Table) [20]. A meta-analysis by F. Rajati et al. showed that the global prevalence of oropharyngeal dysphagia in different populations is 43.8% with a trend toward increasing with age [21]. Oropharyngeal dysphagia may be associated with odynophagia, hypersalivation, heartburn, oral or nasal regurgitation, weight loss, cough or nausea when swallowing [17]. A meta-analysis by KJ. Banda et al. based on 39 studies with 31,488 participants showed that in patients aged 60 years and older, oropharyngeal dysphagia

Etiology of oropharyngeal dysphagia [3, 20]

Nervous system disease	Structural causes	Other causes
• Multiple sclerosis • Spinocerebellar ataxia • Head injury • Brain tumors • Neurodegenerative disease: - Parkinson's disease - progressive; supranuclear palsy - multiple system atrophy - Alzheimer's disease - corticobasal degeneration - frontotemporal dementia - dementia with Lewy bodies - vascular dementia - Huntington's disease - Wilson's disease • Motor neuron disease: - amyotrophic lateral sclerosis - primary lateral sclerosis - spinal muscular atrophy • Neuromuscular disease: - nemaline myopathy - mitochondrial myopathy - inclusion body myositis - dermato-/polymyositis - myasthenia gravis • Peripheral neuropathies: - Guillain–Barre syndrome - polyneuropathy in systemic disease - diabetic neuropathy • Vascular disease: - acute stroke - vascular dementia - congenital cerebral palsy • Iatrogenic causes: - tardive dyskinesia with choreiform movements of the tongue during the treatment with antipsychotics	• Mass lesions of the head and neck • Surgery or radiation for malignant tumors of the head and neck • Chemoradiation mucositis and edema • Zenker's diverticulum • Cervical osteophytes • Lymphadenopathy • Goiter • Cricopharyngeal bar	• Dental disease • Hyposalivation with xerostomia, e.g. of toxic origin (treatment with α- and β-blockers, angiotensin-converting enzyme inhibitors, anticholinergics, antihistamines, anxiolytics, calcium channel blockers, diuretics, muscle relaxants, or tricyclic antidepressants)

is a risk factor for pneumonia, cachexia, and mortality and associated with urinary and fecal incontinence, immobility syndrome, pressure ulcers, sarcopenia, delirium, and frequent falls [4].

Esophageal dysphagia is associated with structural damage to the esophagus and surrounding structures (such as esophagitis of various origin, mass lesions, scleroderma, cardiomegaly, etc.) and with primary and secondary motility disorders of the smooth muscles in the esophagus and esophageal sphincter (such as hyperactive esophageal sphincter syndrome and achalasia). Unlike patients with oropharyngeal dysphagia, who are more likely to report difficulty in food swallowing early in the act of swallowing, patients with esophageal dysphagia typically experience the feeling of "food sticks in the throat or chest" a few seconds after swallowing. Dysphagia for solids is associated with structural abnormalities of the esophagus or oropharynx, while dysphagia for liquid food or liquids is associated with neurogenic causes [17]. Structural or esophageal dysphagia requires gastroenterological examination, which should include qualified examination of the oropharynx, pharyngolaryngoscopy, esophagogastroscopy, and manometry [22]. Esophageal and structural dysphagia is usually managed by gastroenterologists and otolaryngologists, so this review will consider mainly oropharyngeal dysphagia, which is associated with neurological disorders.

Diagnosis of neurogenic dysphagia

Clinical diagnosis

According to the Guidelines of the German Society of Neurology, a survey of the patient or their relatives if neurogenic dysphagia is suspected should include special

questions about changes in eating and drinking behavior; avoidance of certain foods and consistencies; difficulty in taking medications; time needed for a meal; posture during eating; difficulties in chewing; food residues after swallowing in the oral cavity or throat; feeling of "food sticks in the throat" [22].

The clinical assessment of swallow quality includes the following examination protocol [22]:
1) Examining the movement of the jaw and the elevation of the larynx during swallowing. It is recommended to palpate the masticatory muscles and the area above the thyroid cartilage during swallowing.
2) Examination of the soft palate and oral cavity, tongue and lips at rest using a spatula and mirrors with assessment of the pharyngeal and palatal reflexes and the presence of salivary disorders before or after swallowing.
3) Examination of the soft palate, oral cavity, and tongue during phonation, assessment of sound characteristics of the patient's voice.
4) Screening testing for swallowing disorders.

Screening testing should allow quick identification of patients at risk of aspiration to start preventive measures and further diagnosis. Most published testing protocols were evaluated only in stroke patients and have relatively high sensitivity (> 80%) but moderate specificity (up to 60%). However, the optimal testing paradigm have not been defined yet [22]. Three procedures are used as screening methods for diagnosing dysphagia:
1) Water swallow test, which assesses the volume of liquid that the patient can drink without experiencing symptoms of dysphagia [23];
2) Multi-consistency test, which assesses the degree of impairment in swallowing liquids and foods of various consistencies [22];
3) Swallow provocation test, which assesses the involuntary pharyngeal reflex, i.e. only the pharyngeal phase of swallowing [22, 23].

Various clinical scales and questionnaires are used to assess the severity of dysphagia at baseline diagnosis and during follow-up:
- Swallowing Disturbance Questionnaire, which consists of 15 questions on swallowing disturbance to be filled in by the patient [24];
- Swallowing Quality-of-Life Questionnaire, which consists of 10 subscales and a dysphagia symptom battery (14 items assessing symptom severity) to be filled in by the patient [25];
- Eating Assessment Tool, which assesses the severity of dysphagia and its impact on quality of life, with each question rated with a 5-point scale, to be filled in by the patient. Total score of 3 or more is considered abnormality [26];
- Munich Swallowing Score to assess dysfunction of swallowing saliva, food, and liquids [27];
- Gugging Swallowing Screen, a dysphagia screening tool developed for acute stroke patients, which consists of 2 parts: direct and indirect swallow test [28];
- The Functional Oral Intake Scale (FOIS), a 7-point scale used to describe a patient's functional oral intake level with scores ranging from "1" (Nothing by mouth) to "7" (Total oral diet with no restrictions) [26].

Fiberoptic endoscopic evaluation of swallowing

Fiberoptic endoscopic evaluation of swallowing is performed with a fiberoptic flexible endoscope, which is passed transnasally through the middle or lower nasal passages above the velum palatinum into the pharyngeal region. This method allows evaluating the entire pharyngeal phase of swallowing, partially the oral and esophageal phases, including the activity of the velopharyngeal sphincter, pharyngeal and laryngeal reflexes. A colored solution or solid bolus is used to diagnose swallowing disorders and aspiration during swallowing [22, 29].

This method can be used for an objective initial assessment of dysphagia severity, selection of nutrition strategies and food consistency, and assessment of the condition over time during rehabilitation [3, 22]. To assess changes of the condition over time and diagnose latent dysphagia, the following instrumental scales are used: Penetration-Aspiration Scale, Yale Pharyngeal Residue Severity Rating Scale, Murray Secretion Scale, etc. [30–32].

Fiberoptic endoscopy can be used in differential diagnosis of neurological disease or diagnosis of the underlying cause of oropharyngeal dysphagia. A study of T. Warnecke et al. showed that seven dysphagia phenotypes can be identified based on fiberoptic endoscopy findings [18]:
1) "Premature bolus spillage" before the swallowing reflex is triggered: a non-specific phenotype observed in many neurological disorders;
2) "Delayed swallowing reflex": no pharyngeal reflex for more than 3 seconds after the food has reached the valleculae (recesses in the epiglottis), which occurs mainly in stroke patients;
3) "Predominance of residue in the valleculae", which occurs mainly in patients with Parkinson's disease;
4) "Predominance of residue in the piriform sinus", which occurs mainly in patients with myositis, motoneuron disease, or brainstem stroke;
5) "Pharyngolaryngeal movement disorder" (i.e. oropharyngeal "freezing", pharyngeal bradykinesia and pharyngolaryngeal tremor), which occurs mainly in patients with atypical Parkinsonian syndromes or stroke;
6) "Fatigable swallowing weakness", when repeated swallowing attempts result in food residue in the larynx or

increased food residue, which occurs mainly in myasthenia gravis;
7) "Complex disorder" with a heterogeneous dysphagia pattern (i.e. with at least 2 of the mechanisms listed above, another mechanism or with its mechanism that cannot be determined), which occurs mainly in patients with amyotrophic lateral sclerosis.

Videofluoroscopic swallow study

Videofluoroscopic swallow study is an X-ray study of the entire swallowing process, including its oral, pharyngeal, and esophageal phases. The patient swallows a bolus of varying consistency (from solid to liquid) mixed with radiopaque contrast agent. The swallowing process from the formation of a bolus in the oral cavity to the entrance through the UES into the stomach is assessed through the monitor screen in the lateral and anteroposterior projections. This study allows measuring the time needed for the bolus transit in the oral, pharyngeal, and esophageal phases of swallowing, the duration and width of the closure/opening of the velopharyngeal valve and the esophageal sphincter [22, 29]. The following specific scales were developed for this study: Modified Barium Swallow Impairment Profile Scoring, Dynamic Imaging Grade of Swallowing Toxicity, Video Fluoroscopic Swallowing Study for patients with Parkinson's disease, Dysphagia Outcome and Severity Scale [33–36]. The advantage of videofluoroscopic swallow study over fiberoptic endoscopy is that the latter can assess hypertonicity and strictures of the upper esophagus. Videofluoroscopic swallow study is also used to assess the severity of dysphagia and choose a diet in patients after acute stroke or head injury, patients with Parkinson's disease, amyotrophic lateral sclerosis, spinal muscular atrophy, multiple sclerosis or Alzheimer's disease. However, it requires interaction with the patient for correct positioning during the study [17, 22].

High-resolution pharyngeal manometry

Manometry allows measuring pressure in the pharyngeal region and esophagus during the act of swallowing. This method is commonly and most often used for gastroenterological causes of dysphagia to confirm impaired relaxation of the esophageal sphincter and impaired motility of the esophagus with achalasia or diffuse esophagospasm. This method allows evaluating resting pressure, the function of the upper and lower esophageal sphincters, esophageal peristalsis, peak pressure, contraction time of the palatopharyngeal arch and base of the tongue, occlusion pressure in the lumen of the pharynx, hypopharyngeal intrabolus pressure, total swallowing time, wave speed of pharyngeal contraction, and length of active pharyngeal segment [22]. Recently, this method has been used to assess esophageal motility in patients with neurological disease, which is associated with impaired function of the esophageal sphincter and decreased pressure in the lumen of the pharynx, such as Parkinson's disease and atypical parkinsonism, myopathies of various origin, Huntington's disease, and brainstem infarction [17].

Other instrumental methods for diagnosing neurogenic dysphagia

Stimulation electroneuromyography allows evaluating the activation pattern of most muscles involved in the act of swallowing. It is used to assess the orbicularis oris and masseter muscles involved in the oral phase of swallowing, and the suprahyoid and infrahyoid muscles involved in the pharyngeal phase. Needle electrodes are used to record the activation of the cricopharyngeal muscle, which is part of the UES. This method is used in research studies to assess the degree of activation of muscles involved in the oropharyngeal phase of swallowing and to identify target muscles for the administration of botulinum toxin in the treatment of dysphagia [3, 22].

Ultrasound examination is another promising modality for diagnosing and assessing the severity of dysphagia; it allows evaluation of the morphometry of the oropharyngeal muscles and real-time visualization of oral bolus movement, the motor activity of the tongue, larynx movement, and activity of the supraglottic and sublingual muscles. Ultrasound can be used to diagnose structural changes caused by dystrophy or denervation of the muscles involved in swallowing, as well as to detect involuntary movements such as fasciculations and tremor. Advantages of the ultrasound diagnostic method include its non-invasiveness and low cost; however, study protocols and standards should be developed for its use in clinical practice [22, 37].

Dynamic magnetic resonance imaging adopting "Turbo Fast Low Angle Shot (turbo-FLASH) Sequences" at higher field strengths ($\geqslant$ 3 Tesla) provides a series of anatomical images in rapidly acquired consecutive slices. It allows a direct view on the deeper oropharyngeal muscles and soft tissue and tracking of the bolus transit during the swallowing act. Key limitations of this technique in diagnosing dysphagia include the horizontal position of the patient's body during the examination, which is usually not physiological for swallowing and can aggravate the swallowing disorder, and a limited ability to assist during the examination of patients with a high risk of aspiration. Potentially possible methods for diagnosing swallowing disorders include multi-slice computed tomography with high temporal resolution, which can be performed in a semi-sitting position [22, 38].

Treatment and rehabilitation of patients with neurogenic dysphagia

Treatment of patients with neurogenic dysphagia is primarily symptomatic and aims at improving swallowing

safety and efficiency. Treatment of neurogenic dysphagia should be personalized based on a thorough clinical and instrumental diagnosis of the patient, taking into account specific pathophysiological mechanisms of dysphagia. Treatment should be chosen by a multidisciplinary team, which includes a neurologist, speech therapist, physiotherapist, physical therapy instructor, dentist, and gastroenterologist [39].

Three therapy principles have been identified:
- restitution, which is aimed at restoring lost muscle functions;
- compensation, i.e. use of compensatory strategies to replace lost functions;
- adaptation, i.e. use of dietary modifications and other options to ensure safe swallowing [40].

Adaptation measures

Methods aimed at adapting the patient to live with dysphagia include modification of diet and posture during meals [16]. Most common dietary modification strategies include the use of liquid food thickeners and the selection of food bolus size and food consistency based on instrumental findings. It is recommended to calculate the patient's nutritional status, food caloric value, and fluid intake to prevent dehydration and cachexia, and maintain oral hygiene to prevent aspiration pneumonia [22, 38, 41, 42].

Physical methods of compensation and restitution

Physical exercise for patients with dysphagia are chosen individually, taking into account the course of the neurological disorder and the cause of dysphagia. Most common restitution methods include a set of shaker head lift exercises, which is intended for patients with weakness of the suprahyoid muscles and impaired opening of the UES; exercises for training the muscles of the tongue (Masako maneuver); and exercises to strengthen the expiratory and mental muscles. Methods aimed at compensating for impaired swallowing function and preventing complications include various modifications of the head position when swallowing, the "swallowing with effort" technique, which is used in patients with ineffective swallowing, i.e. predominance of food residue in the valleculae and pharynx. The following methods are also used: supraglottic swallowing, which is used as a compensatory maneuver for patients with reduced airway closure, Mendelsohn maneuver (i.e. keeping the larynx in an elevated position while swallowing), and swallowing with blocking the flow of the air to block the access to the glottis and prevent aspiration etc. In many cases, a combination of various adaptive, compensatory, and restorative physical methods is required to improve the quality of swallowing in patients with dysphagia [12, 22].

Medication treatment

Available medications aim at either stimulating the neural pathways in the peripheral or central nervous system that control swallowing or activating the muscles involved in swallowing. Medications that were shown to be effective in improving the swallowing reflex and reducing the incidence of aspiration pneumonia include TRPV1 agonists, TRPA1 agonists, TRPM8 agonists, levodopa and other dopaminergic agents, calcium blockers, dopamine D2 receptor antagonists, angiotensin-converting enzyme inhibitors (ACEIs), β-blockers, nitric oxide donors, and acetylcholinesterase inhibitors [16].

The mechanism of action of TRPV1, TRPA1 and TRPM8 receptor agonists includes stimulation of afferent pathways through the corresponding receptors located in the oropharynx, the activation of which leads to neuroplastic changes in the cerebral cortex. TRPV1 agonists may modulate swallowing through releasing substance P, which enhances cough reflex [16]. The relationship between substance P and swallowing function is not fully understood; however, in patients with Parkinson's disease and dysphagia, increased levels of substance P were shown to be associated with improved swallowing performance and a reduced risk of aspiration pneumonia [43]. A meta-analysis by I. Cheng et al. based on 14 studies including 2186 patients showed that TRPV1, TRPA1, and TRPM8 agonists were significantly superior to placebo in reducing swallowing time and severity of dysphagia [16].

As for other medications, a limited number of randomized clinical trials have been conducted to confirm their efficacy. However, calcium channel blockers (e.g. nifedipine) and dopamine D2 receptor antagonists (e.g. metoclopramide) were shown to be more effective than ACE inhibitors (e.g. lisinopril) and acetylcholinesterase inhibitors (e.g. physostigmine) [16]. The mechanism of action of capsaicin, ACE inhibitors, and β-blockers is thought to be related to increased levels of substance P, while levodopa and dopaminergic agents may improve swallowing efficiency by improving dopamine metabolism. Finally, acetylcholinesterase inhibitors (e.g. physostigmine) may improve swallowing function through cholinergic stimulation [16, 44–46].

Neurostimulation methods

Recently, peripheral neurostimulation methods, such as neuromuscular electrical stimulation (NMES) and pharyngeal electrical stimulation (PES), and central neurostimulation methods, such as rhythmic transcranial magnetic stimulation (rTMS) and transcranial electrical stimulation (TES), have been actively developed for the treatment of neurogenic dysphagia [22].

NMES is electrical transcutaneous stimulation of sensory and motor nerve fibers that are involved in swallowing; it is performed in order to restore and enhance the motor function of weakened muscles and prevent their atrophy. Stimulation is performed using surface electrodes applied to the skin of the chin and/or anterior neck [22, 40]. A meta-analysis by S. Miller et al. based on 14 studies showed that NMES is an effective method for the treatment of dysphagia, especially in combination with conventional rehabilitation options. However, further studies are needed as available stimulation protocols are very heterogeneous, and the effectiveness of the method was studied mainly in stroke patients [40].

PES is based on electrical stimulation of the bottom of the tongue and the posterior wall of the pharynx using a transnasal catheter with bipolar ring electrodes. Unlike NMES, PES is aimed at inducing neuroplasticity of the motor and sensory cortex and restoring sensorimotor integration [22]. PES showed its efficacy in patients with multiple sclerosis or stroke [47–49] but not in patients with amyotrophic lateral sclerosis [50]. Results of meta-analyses by R. Speyer et al., I. Cheng et al. were also controversial, and, therefore, the efficacy of PES requires further confirmation [51, 52].

rTMS and direct current TES are used to modulate cortical activity and cause long-lasting changes in synaptic plasticity [22, 51, 53–55]. In 2018 clinical guidelines, the effect of rTMS in stroke patients is considered unknown due to the heterogeneity of results and treatment protocols [53]. However, a meta-analysis by X. Wen et al. showed that low-frequency and high-frequency rTMS can improve swallowing function in stroke patients. Cortical representations of the muscles involved in swallowing (including the mylohyoid muscle) and the cerebellum were used as targets. The analysis of the studies demonstrated that stimulation of the cerebral cortex was effective in both affected and unaffected sides in comparison with standard physical treatments and placebo [54]. Similar results were shown by a meta-analysis by N. Zhao et al. for direct current TES; a significant positive effect of TES on reducing post-stroke dysphagia was demonstrated [55]. Limited data are available for other neurological disorders, so new randomized clinical studies are needed to confirm rTMS and TES efficacy.

Surgical methods

Minimally invasive surgical procedures are offered for patients with UES hyperactivity or other disorders of its opening. Such methods include open or endoscopic cricopharyngeal myotomy and dilatation of the UES using a balloon. Chemical cricopharyngeal myotomy using endoscopic or percutaneous injection of botulinum toxin is a safer and less invasive option. These methods have been used in patients with inclusion body myositis, muscular dystrophy, multiple sclerosis, amyotrophic lateral sclerosis, stroke, or Parkinson's disease. Surgical interventions may be associated with side effects such as supraglottic edema, mediastinitis, retropharyngeal hematoma, esophageal damage, laryngospasm and bleeding, so they should be administered after the comprehensive diagnosis is established and if conservative treatment is ineffective [22].

If severe dysphagia develops, i.e. if there is a high risk of cachexia and dehydration, insertion of a nasogastric tube or percutaneous endoscopic gastrostomy should be considered. Insertion of a nasogastric tube is indicated for patients with acute conditions, such as acute stroke or head injury, in which dysphagia may resolve within weeks or months. Percutaneous endoscopic gastrostomy is more suitable for patients with chronic progressive disorders such as Parkinson's disease, dementia or amyotrophic lateral sclerosis [3].

Conclusion

Neurogenic dysphagia is a common symptom of many neurological disorders. It significantly impairs patients' quality of life and leads to serious complications such as aspiration pneumonia, cachexia, and death. Despite the availability of relatively simple screening and highly informative instrumental diagnostic methods, treatment and prevention of swallowing disorders in neurological patients, as well as rehabilitation of patients with neurogenic dysphagia, remain insufficiently studied and require the development of unified treatment protocols based on large-scale multicenter clinical studies for medications and high-tech rehabilitation options.

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