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Dysphagia. Part 1: General issues

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Abstract

Dysphagia is a common problem among ICU patients. The frequency of dysphagia increases with age and sometimes symptoms can be difficult to recognise. But the consequences of dysphagia can be very serious, including aspiration and subsequently aspiration pneumonia. Therefore, knowing mechanisms and symptoms causing dysphagia is very important and should be well recognised. Proper diagnosis allows one to prevent further complications. However, both the diagnosis and treatment can be very complicated, especially among the patients who do not cooperate. In many cases, the implementation of an appropriate nutrition strategy and proper rehabilitation can alleviate the symptoms of dysphagia and avoid the most severe complications.

Key words: dysphagia, pathophysiology, symptoms, diagnosis, treatment, compensation.

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Swallowing disorders – such as aphagia, odynophagia and dysphagia are increasingly observed among patients in intensive care units (ICU). Aphagia means inability to swallow, and odynophagia means painful swallowing. Their most common causes are inflammatory or neoplastic lesions in the oropharynx, or the consequences of oncological treatment, e.g. radiotherapy [1–3]. Dysphagia is an abnormality in the swallowing process, i.e. ingestion of food, grinding it, and transporting it from the oral cavity through the oesophagus to the stomach. The severity of the pathology may vary depending on the aetiology. It can be caused by structural anomalies in the upper gastrointestinal (GI) tract or functional disturbances of the nervous and/or muscular systems [3]. Both the diagnosis and treatment of dysphagia require the cooperation of specialists in many fields of medicine. It seems that anaesthesiologists should be included in this group [3].

Dysphagia significantly worsens the patients' quality of life. It results in increased morbidity and mortality, mainly due to a higher risk of aspiration and subsequent aspiration pneumonia, as well as to difficulties in the intake of food and/or fluids by mouth, which leads to malnutrition [1].

Diagnosis and determination of the cause of dysphagia is crucial, and in many cases it offers the opportunity to treat and/or compensate for swallowing problems and thus reduce the risk of complications. The final effect of the therapy, however, is difficult to predict and largely depends on the type and degree of abnormality [1–4].

SWALLOWING PHYSIOLOGY

The swallowing reflex involves many cranial nerves and over 50 muscles, whose contractions are coordinated by the central nervous system (CNS) [1, 4].

According to Logemann, the act of swallowing is divided into three phases:
– oral (with the pre-oral phase),
– pharyngeal,
– oesophageal [5].

The oral phase is the only one that is arbitrary and is controlled by the motor cortex; the remaining phases are reflexive.

The swallowing reflex is caused by the swallowing centre located in the reticular system in the brainstem. Its activation starts a bilateral and symmetrical sequence of excitation and inhibition of neurons located in the brainstem [4, 6]. The centripetal route consists of the nerves conducted by the V, IX and X cranial nerves, which conduct impulses from the receptors located in the mouth, pharynx and oesophagus. The centrifugal pathway leads from the swallowing centre in the brainstem through the V, VII, IX, X and XII cranial nerves to the muscles of the pharynx and oesophagus. The duration of individual stages depends primarily on the type of food consumed, the degree of hydration of the mucous membranes and the efficiency of the muscles involved [3, 4, 6, 7].

The oral phase consists of two stages:

– in the first, preparatory stage, food is crushed and chewed by the work of the masseter muscles and mixed with saliva; the contraction of the transverse muscle of the tongue and the genioglossus muscle leads to the formation of a food bolus and its placement on the tongue. The dominant role during this phase is played by the temporomandibular, medial and lateral pterygoid muscles, as well as the cheek and tongue muscles, stimulated by the V, VII, XII cranial nerves.
– in the second, transport stage, the bolus is moved towards the oropharyngeal isthmus, stimulates the sensory receptors, then rises and tightens the soft palate, which separates the nasal cavity, while the palatine arches that get nearer close the oral cavity. At the same time, the larynx moves upwards and closes the epiglottis and the glottis, i.e. the respiratory tract. The throat remains the only way open to the bolus. In this phase, the activity of the muscles involved is controlled by the V, VII, XII nerves [4, 6, 7].

The pharyngeal phase begins when the food bolus exerts pressure on the anterior palatine arches, the back of the tongue and the soft palate. Backward movement of the tongue closes the epiglottis to protect the respiratory tract, and the laryngohyoid complex rises upward, expanding the upper oesophageal sphincter. The shortening of the vocal folds is an additional protection of the respiratory tract. Nerve impulses are conducted to the muscles through the IX, X, XII cranial nerves [2, 4, 8]. The contraction of the muscles of the upper, middle and lower pharyngeal constrictors raises the pressure in the throat and creates a peristaltic wave that moves the food bolus from the throat to the oesophagus, thanks to simultaneous relaxation of the upper oesophageal sphincter. Immediately after the food bolus has passed through, the upper oesophageal sphincter is closed again. The path to the nasal cavity and glottis is then opened, and breathing is restored.

The transport of the bolus within the throat takes place thanks to the force of gravity, pressure exerted by the muscles of the tongue and peristalsis of the pharyngeal constrictor muscles described above. It should be emphasized that the pressure difference between the oropharynx and the inside of the upper oesophageal sphincter plays a crucial role [4, 7–9].

In the oropharynx, positive pressure is generated by the movement of the tongue, which moves like a piston in the space limited by the pharyngeal wall and the soft palate [8, 9]. Negative pressure is generated in the upper oesophageal sphincter because of the upward and anterior movement of the larynx and the relaxation of the muscles forming the upper oesophageal sphincter [8–10]. Maintaining high pressure at the border of the pharynx and oesophagus depends on the correct tone and functioning of the cricopharyngeal muscle, lower pharyngeal constrictor muscle and the circular fibres of the initial oesophagus, which form the functional unit of the upper oesophageal sphincter [6, 8, 9].

The movement of the larynx, which relaxes the upper oesophageal sphincter, is caused by contraction of the suprahyoid and infrahyoid muscles. Moving up, the larynx pulls the cricopharyngeal fibres attached to the cricoid cartilage, causing the lumen of the upper oesophageal sphincter to widen and the pressure in the sphincter to drop. The difference between the positive pressure produced by the tongue and the negative pressure of the dilated upper oesophageal sphincter displaces the food bolus into the oesophagus [7–10]. A delay or lack of pressure drop within the lumen of the upper oesophageal sphincter has severe consequences – it disrupts the transport of the bolus from the laryngeal part of the pharynx to the oesophagus and poses a risk of food aspiration to the respiratory tract [8, 9].

The pharyngeal phase, although lasting a short time, < 1 s, is complicated, and its correct course allows not only the transfer of a food bolus into the oesophagus, but also protects the respiratory tract against aspiration.

The elements determining the proper course of the pharyngeal phase and thus protection of the respiratory tract include closing the nasopharynx and larynx, contraction of the pharyngeal constrictor muscles, and relaxation of the upper oesophageal sphincter. The latter three mechanisms constitute protection against aspiration [6–9].

The protection of the lower respiratory tract also has several levels and is dependent on the contraction of laryngeal structures: the epiglottis, aryepiglottic, vestibular, and vocal folds [9].

The duration of the oesophageal phase is slightly longer, averages 4–20 seconds, and depends mainly on the type of food. The oesophageal phase begins with the expansion of the upper oesophageal sphincter. The work of the muscles is controlled by the X nerve. The passage of food through the oesophagus is determined by the changing pressure inside it. Shortly after the food bolus has passed through the upper oesophageal sphincter, the pressure in the oesophagus increases temporarily and subsequently returns to the baseline value. Later, as the peristaltic wave passes through, an increase in pressure is observed in the body of the oesophagus. When the peristaltic wave reaches the lower oesophageal sphincter, it relaxes and the food passes to the stomach. The pressure value in the relaxation phase of the lower oesophageal sphincter and the duration

of the negative pressure phase are particularly important. When the pressure in the stomach exceeds this value, the gastric contents will be refluxed into the oesophagus; the same is observed when the sphincter is opened for too long. After the passage of food, the pressure of the lower oesophageal sphincter temporarily increases and then returns to its resting value.

The oesophageal sphincters play a dual role:
– they prevent air from entering the oesophagus and stomach during breathing,
– they protect against the reflux of gastric juice into the oesophagus and throat [5, 6, 8].

Two types of peristaltic waves develop in the oesophagus:
– the primary one, which is a continuation of the pharyngeal peristaltic wave, starts below the upper oesophageal sphincter and spreads along the oesophagus to its lower sphincter,
– secondary, which can be initiated anywhere in the oesophagus, begins after the primary wave has passed and is caused by food debris [8–11].

Dysphagia affects about 7% of the population. The percentage increases with age. Among the elderly, the prevalence of dysphagia is estimated at 30–40%, approx. 60% are residents of nursing homes [3, 12, 13].

In some elderly people, about 6–8%, regardless of the underlying cause, dysphagia may be caused by impaired swallowing due to the aging process. It is particularly affected by the reduction of the sensitivity of sensory receptors located in the mouth and throat, lower efficiency of the muscles involved in swallowing, impaired elasticity of the connective tissue in the neck, reduced saliva production, and diminished sense of smell and taste [1–3, 9, 14–16]. The European Society of Geriatric Medicine has recognized dysphagia as a geriatric syndrome [17].

Moreover, in elderly patients, the mobility of the hyoid cartilage changes, as it rises more slowly and remains elevated for a shorter time, but the range of its movement is greater, which is probably a compensatory mechanism. Impairment of laryngeal mobility may cause an excessive pressure difference between the pharynx and the oesophagus, which prevents appropriate transport of the food bolus and causes dysphagia [9, 17–19].

CAUSES OF DYSPHAGIA AND ITS SYMPTOMS

Dysphagia results from morphological damage to the upper GI tract or functional disorders of the nervous and / or muscular systems.

In younger patients, under 60 years of age, morphological and structural causes prevail, while in older patients, over 60 years of age, neurological disorders are the most common, followed by neuromuscular disorders or changes resulting from oncological diseases or their treatment.

Due to the different pathophysiology, dysphagia is divided into upper – oropharyngeal, and lower – oesophageal. About 80% of oropharyngeal dysphagia cases are caused by neurological diseases, less frequently neoplastic lesions in the head and neck area, while in more than 85% oesophageal dysphagia is caused by the gastrointestinal diseases, either structural in nature, such as oesophageal recesses, or functional, such as reflux disease [3, 4, 9, 12].

In oral phase dysphagia, the problems that develop may be associated with grinding of food due to dentition defects, abnormal chewing, forming as well as transport of the food bolus, most often due to weakened muscle tone. Poor lip closure can result in excessive salivation, numbness in the mouth, and dry mouth. As a result, food may accumulate in the vestibule of the mouth and cheeks, excessive salivation and even leakage of saliva from the mouth may be noticeable.

In pharyngeal dysphagia, the protective function of the larynx is impaired, and the cough reflex is weakened. Aspiration is the most serious consequence. Considering the degree of invasion, saliva and/or food may be retained, penetrate, or be aspirated. Retention means pooling of saliva in the epiglottic valleculae or pyriform recesses, penetration takes place when saliva enters the laryngeal vestibule, and aspiration is associated with the presence of saliva below the vocal folds [2, 4, 12].

Aspiration pneumonia may be caused by aspiration of colonized saliva and secretions, aspiration of gastric contents, colonized or not, and food aspiration during feeding [12, 21]. Aspiration and its consequences are among the most severe complications of dysphagia. Diabetes or systemic scleroderma, as well as old age may cause a reduced perception of dysphagia symptoms by the patient, despite confirmed pathologies [2, 3, 9, 12].

The prevalence of aspiration is difficult to assess due to a wide variety of methods available; therefore, some authors assess the incidence of aspiration pneumonia, or the frequency of aspiration pneumonia-associated deaths, considering these data to be more objective [12].

Clinical symptoms of dysphagia in the second phase of swallowing are coughing during meals, choking, regurgitation of food to the nasopharynx, which may lead to choking, lacrimation, sneezing, excessive throat reactivity and retching [2, 4, 6, 9, 12]. The causes of oropharyngeal dysphagia are presented in Table 1.

Up to 85% of oesophageal dysphagia is caused by structural changes, which include organic strictures of the oesophagus (neoplasms, diverticula,

stenosis after burns with corrosive substances, oesophageal rings, oesophageal webs, post-inflammatory and ulcerative strictures caused by drugs), or by motor disorders (cardiac spasm, diffuse oesophageal spasm, gastroesophageal reflux disease).

Motility disorders may take the form of its impairment or intensification. Impaired oesophageal peristalsis may result from neurological or muscular changes, develop during systemic diseases such as diabetes, systemic connective tissue diseases, hypothyroidism or have idiopathic causes. Ineffective peristalsis leads to failure of the lower oesophageal sphincter, resulting in gastroesophageal reflux and inflammatory changes in the oesophageal mucosa.

Oesophageal hypermotility can be caused by insufficient activity of the nerves that inhibit peristalsis. As a result, oesophageal contractions are weak and chaotic, and passage ineffective. The causes may include paraneoplastic syndromes, oesophageal achalasia or its diffuse spasm [2].

Other causes include diseases of the adjacent organs (enlargement of the left atrium, retrosternal goitre, mediastinal and intramedullary tumours, defects of the subclavian artery, paraesophageal hernias), previous thoracic and cardiac surgical procedures, swallowed foreign bodies, prior radiotherapy.

Oesophageal dysphagia manifests mainly as the feeling of a food bolus stuck [2, 4, 12]. The causes of oesophageal dysphagia are presented in Table 2.

NEUROGENIC DYSPHAGIA

The highest incidence of oropharyngeal dysphagia is observed among patients with neurological disorders. Neurogenic dysphagia may be the result of damage to the nervous system at various levels, pathological processes of the neuromuscular junction (myasthenia gravis, myasthenic syndromes), as well as damage to muscle cells (myopathies, muscular dystrophies, mitochondrial myopathies). Other causes include acute cerebral ischaemia of vascular origin, brain tumours and injuries, bulbar and pseudobulbar syndrome, neurodegenerative diseases (amyotrophic lateral sclerosis, multiple sclerosis, Parkinson's disease), tabes dorsalis, multiple system atrophy, peripheral neuropathies (Guillain-Barré syndrome, botulism, Sjögren's syndrome, amyloidosis, diphtheria, sarcoidosis, diabetes, poliomyelitis), as well as collagenoses (visceral lupus, dermatomyositis and polymyositis, scleroderma). The underdevelopment of the nerve cells of Auerbach's plexus is the cause of oesophageal achalasia [3, 12, 13, 20].

Dysphagia is divided into mechanical and functional. The causes of mechanical dysphagia are largely the same as the causes of oesophageal dysphagia. The causes of functional dysphagia are identical with those of neurogenic oropharyngeal dysphagia [1, 2, 4, 9].

Liquid food dysphagia is characteristic of the oropharyngeal phase and solid food dysphagia is characteristic of the oesophageal phase.

The main symptoms of dysphagia that should prompt diagnosis are food retention in the mouth

TABLE 1. Causes of oropharyngeal dysphagia, based on [2, 3, 7, 22]

Neurological causes
• Central nervous system diseases – stroke, neurodegenerative diseases (Parkinson's disease, amyotrophic lateral sclerosis, multiple sclerosis)
• Traumatic damage to peripheral nerves and impaired function of the neuromuscular junction
• Primary damage to the neuromuscular junction (*myasthenia gravis*, Lambert-Eaton syndrome)
• Primary muscle damage (myopathies, post-inflammatory myopathy)
Structural causes
• Structural damage from intubation or disease
• Changes after head and neck surgery
• Changes after cancer surgery (morphological defects in the mouth and throat, damage to the nerves supplying these areas)
• Damage after adjuvant treatment (radiotherapy)
Other
• Presbyphagia
• Phagophobia
• Drug-induced (toxic): cholinolytic drugs (hyoscine butylbromide), opioids, tricyclic antidepressants – intensify xerostomia and consequently difficulties in forming and swallowing a food bolus, muscle relaxants, anxiolytics – may impair the swallowing reflex

TABLE 2. Causes of oesophageal dysphagia, according to [2, 3, 7, 23]

Structural disorders – structural strictures of the oesophagus
• Cancer
• Diverticula
• Acid reflux disease
• Strictures after burns with caustic substances
• Oesophageal rings
• Oesophageal webs
• Post-inflammatory stenoses
• Ulcerative stenoses
Functional disorders – motor skills
• Cardiac spasm
• Diffuse oesophageal spasm
• Acid reflux disease
Other causes
• Collagenoses
• Diabetes
• Drug-induced
• External pressure by adjacent organs (enlargement of the left atrium, retrosternal goitre, mediastinal tumours, intramedullary tumours, defects of the subclavian artery, paraesophageal hernias)
• Previous thoracic and cardiac surgical procedures
• Lying position in patients with cns diseases
• Foreign bodies
• Previous radiotherapy

and throat, difficulties in initiating swallowing, prolonged eating, choking while eating, accompanying cough, increased gag reflex, clearing throat, weight loss, loss of taste and smell. There may also be primitive symptoms, such as sucking or biting the cheeks [2, 4, 9].

Other nonspecific symptoms include burning retrosternal pain, frequent belching, heartburn, drooling, spitting up, no coughing on command, symptoms of dysarthria, dysphoria, and voice change [2–4, 7].

Speech disorders are a common symptom of dysphagia because the same muscle structures are involved in the act of breathing, swallowing and phonation. Dysarthria is caused by a lack of coordination in breathing and phonation. It is characterized by slurred, blurred and slow speech, a change in voice timbre, hoarseness of varying intensity, and increased tension in the neck muscles [3, 4]. Apart from the described specific symptoms in the form of cough and choking associated with a meal, symptoms such as malnutrition, recurrent respiratory infections, low-grade fever and fever of unclear origin should prompt the suspicion of dysphagia and initiation of diagnostics [2–4, 7, 9].

The clinical consequences of dysphagia are serious – the most dangerous are associated with a higher risk of aspiration and aspiration pneumonia. Dysphagia delays oral food intake, which leads to malnutrition, worsens the patients' quality of life, extends hospitalization, increases morbidity and mortality, as well as treatment costs. Malnutrition resulting from dysphagia is an unfavourable prognostic factor, it leads to decreased immunity, increased susceptibility to infections, impaired wound healing, pressure ulcer formation, and worsens physical and cognitive performance of the patient. Dysphagia of liquid foods causes dehydration, which may aggravate the symptoms of comorbidities and cause loss of consciousness and impairment of both physical and cognitive functioning [2–4, 8, 9, 23–25].

DYSPHAGIA DIAGNOSIS

Due to the diversity of etiological factors and often non-specific early symptoms of dysphagia, its diagnosis is complex.

In the case of oropharyngeal dysphagia, diagnostics are most often performed by ENT specialists. In the physical examination the physician looks for features of food retention in the oral vestibule, pyriform recesses and vestibule of the larynx, the condition of the glottis, including the area of the posterior commissure and the posterior parts of the vocal folds, is assessed [3]. It is essential to evaluate the entire act of swallowing, which is possible by means of radiological examinations, such as video fluoroscopy, and more and more popular fibreoptic endoscopic evaluation of swallowing (FESS), which does not require the use of radiation and is a cheaper method. It allows the assessment of the base of the tongue, pharynx, nasopharynx and larynx before and after the act of swallowing. The endoscopic examination looks for the characteristics of retention, penetration and aspiration of saliva and/or food, which allows to classify and determine the degree of aspiration on the basis of the penetration and aspiration scale (PAS) described by Rosenbeck. The FESS test also allows to diagnose the most dangerous symptom of dysphagia, the so-called silent aspiration, i.e. aspiration without triggering the cough reflex [3, 26].

In the diagnosis of dysphagia, ultrasound examinations are also used, thanks to which it is possible to assess the movements of the tongue and the hyoid bone in the oropharyngeal phase of swallowing.

Manometry is used for the diagnosis of pharyngeal dysphagia, and the high resolution manometry (HRM) test allows to assess the pharyngeal phase and the upper oesophageal sphincter. On the other hand, scintigraphy (oral-pharyngo-oesophageal scintigraphy (OPES) allows to analyse the transit time and potential food retention at different stages of the swallowing act. Oesophageal pH-metry and manometry are mainly used by gastrologists in cases of suspected gastroesophageal reflux [27–29].

MANAGEMENT OF DYSPHAGIA

The treatment of dysphagia is mainly aimed at preventing aspiration and its complications. The management of dysphagia depends on the severity of problems with swallowing and is based on adaptive, compensatory and rehabilitation measures. In general, adaptation procedures consisting in changing the structure of food, often in cooperation with a dietician, are sufficient:

– dysphagia of liquid foods – the diet should be concentrated,
– dysphagia of solid foods – mixing, grinding food.

The diet can also stimulate the receptors of the mouth and support the swallowing reflex. Food stimulation means taking food at different temperatures or properly seasoned. By changing the temperature of meals and seasoning them appropriately, one can enhance the taste, which may also alleviate the symptoms of dysphagia [2, 3, 7, 12, 20].

In some cases, dysphagia results from xerostomia, then it is necessary to moisturize the oral mucosa. For this purpose, artificial saliva and moisturizing preparations are used, or traditional methods are applied, such as sucking ice cubes, frozen pine-

apple or cranberry pieces, chewing gum, frequent drinking of cold drinks (apart from lemon-flavoured drinks), avoiding substances that dry out and irritate the mucosa, like cigarettes and coffee [2].

Compensation management generally requires cooperation with a physiotherapist. The swallowing reflex can be supported by changing the body posture and proper positioning of the head in relation to the body during a meal. Food can also be administered deeply into the oral cavity, which additionally provides tactile and thermal stimulation [7, 12, 20].

Rehabilitation carried out by an experienced therapist initially comes down to indirect (without food) exercises of the muscles of the lips, tongue, jaw and larynx. Direct (with food) exercises develop the skills of subglottic or glottic closing of the glottis, effort tongue withdrawal, Mendelsohn's manoeuvre [3, 20, 22–25, 30]. However, rehabilitation requires the patient's cooperation, which may be difficult or impossible in the case of neurological dysphagia.

In some cases, only surgical procedures can prevent the patient from choking on saliva or food: removal of the salivary glands, cutting their innervation, or displacement of the salivary ducts, cutting the cricopharyngeal muscle, resection of the cricoid cartilage, an attempt to increase the volume of the vocal fold [22, 23, 31, 32]. A radical procedure involves separating the airway from the digestive tract by selecting a tracheal fistula, and often also nutritional gastrostomy, which is common in patients with neurogenic dysphagia and a significant reduction in consciousness, as well as among patients in intensive care [3, 22].

Pharmacological treatment of dysphagia is used in the treatment of gastroesophageal reflux, but also of disorders of oesophageal motility and tension [2, 3].

The tension of the lower oesophageal sphincter is reduced by nitrates, calcium channel blockers, muscle relaxants, tricyclic antidepressants, and herbal preparations with peppermint oil. Metoclopramide is used to increase the tone of the lower oesophageal sphincter [2].

Endoscopic methods are applied in the treatment of oesophageal dysphagia due to morphological changes: oesophageal widening or its prosthesis in the case of neoplastic changes, as well as brachytherapy [2, 22, 31, 32].

In selected cases, the increasingly popular neurostimulation or botulinum toxins have recently been used to treat dysphagia [33–35].

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