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Review article

Dysphagia in Lateral Medullary Syndrome

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SUMMARY

Introduction. The lateral medullary syndrome is a neurological disease caused by ischemia in the lateral part of the medulla oblongata and is the most common form of brainstem infarction. Dysphagia is a common and clinically significant symptom of this disease because it is closely associated with the risk of recurrent pneumonia, malnutrition, dehydration, and an increase in mortality and prolonged hospital treatment.
Aim. This paper aims to review and analyze data on the correlation between swallowing disorders and lateral medullary syndrome. We intend to present the symptoms, diagnostic and therapeutic procedures of dysphagia in patients with this syndrome in a comprehensive way.
Methodology. The following databases were used to search the literature: KoBSON-Consortium of Serbian Libraries for Unified Procurement, PubMed, Science Direct.
Results. Based on the results of the reviewed studies, it was determined that patients with the lateral medullary syndrome often have swallowing disorders. They are often fed through a nasogastric tube a few months after the stroke, which significantly impairs their quality of life. To overcome swallowing disorders and create conditions for safe swallowing function, most patients need treatment for a longer period. In addition to the available screening tests, instrumental diagnostic methods provide insight into the biomechanical aspects of swallowing disorders, determine the risk of aspiration, and provide a starting point for selecting treatment strategies.
Conclusion. Treatment of dysphagia depends on the mechanisms of occurrence and the predictors of recovery of swallowing function. When conducting treatment, among other things, it is very important to know the pathological mechanisms of neural connections of the medulla oblongata.

Keywords: **lateral medullary syndrome, dysphagia, diagnosis, treatment**

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INTRODUCTION

Lateral medullary syndrome (LMS), also called Wallenberg syndrome and posterior inferior cerebellar artery syndrome (PICA), is a neurological disease caused by ischemia in the lateral part of the medulla oblongata. It occurs due to occlusion in the vertebral artery or the posterior inferior cerebellar artery. The syndrome was first described in 1895 by Adolf Wallenberg, a German neurologist, to whom this syndrome is often referred. Clinical studies show that it most often occurs in men with an average age of 55.6 years (1).

As LMS usually occurs due to occlusion of the arteria vertebralis (VA), which is the first lateral branch of the subclavian artery, the following is an overview of the provision of this artery to understand its connection with swallowing. After separation from the subclavian artery (art. subclavia (AS), the vertebral artery extends vertically upwards, and then crosses the anterior side of the transverse extension of the seventh cervical vertebra and enters the opening of the transverse extension (foramen transversarium) of the sixth cervical vertebra, then extends upwards). Passing through the openings of the transverse extensions of the atlas, it then enters its bony groove (sulcus arteriae vertebralis) on the posterior arch of the atlas, and then penetrates the posterior atlantooccipital membrane, the dura mater, passes through the large occipital foramen and enters the cranial cavity, through the large occipital opening. Finally, this artery ascends along the clivus to meet the eponymous artery of the opposite side with which it merges into one arterial tree-arteria basilaris (AB) (2). AV damage in any part of its pathway can lead to ischemia in the medulla oblongata and thus cause swallowing disorers. According to empirical data, lesions in the medulla oblongata can manifest as median, dorsolateral, inferodorsolateral, and paramedical (3).

The clinical picture of the lateral medullary syndrome depends on the location of the lesion. The patient manifests dysphagia, the sensory deficit on the ipsilateral side of the face, contralateral trunk, and extremities. Ataxia, dizziness, Horner's syndrome (unilateral ptosis, miosis, and facial anhydrosis) also occur (4).

Dysphagia includes difficulty swallowing and controlling saliva, as well as difficulty feeding. In the broadest sense, dysphagia encompasses a disorder of all behavioral, sensory, and preliminary motor actions in preparation for swallowing, including awareness of an impending feeding situation and visual recognition of food. It also includes an increase in the amount of saliva as a physiological response to food (5). Depending on the place of occurrence of the disorder, there are two basic types of dysphagia: 1. oropharyngeal and 2. esophageal dysphagia (6).

To understand the nature of dysphagia and implement adequate therapy, a good knowledge of the physiology of swallowing is important. Eating and swallowing are complex motor actions that include voluntary and reflex activities of as many as 30 muscles and five cranial nerves. These motor actions have two biological roles: 1) food transfer from the oral cavity to the stomach and 2) respiratory protection (7). Different authors point to different divisions of swallowing phases. According to Logeman (8), swallowing has four phases: a) preparatory oral, b) oral, c) pharyngeal and e) esophageal. Matsuo and Palmer (9) further break down the oral phase into three more levels: food transfer through the oral cavity, food processing by chewing and saliva, and food transfer to the oropharynx. In the literature, a division into only three phases can be found: oral, pharyngeal, and esophageal (7).

The patterns of movement in the preparatory oral phase vary depending on the viscosity of the food, its quantity as well as the degree of pleasantness (the subjective sense of taste). The moment the fluid is placed in the mouth, the lips close, which requires nasal breathing, and the soft palate and tongue prevent premature leakage of fluid into the pharynx. In case of solid food, the tongue rotates, placing food on the teeth laterally due to chewing. At this stage, the food bolus is softened by saliva. During the oral phase, the tip of the tongue is raised, touches the alveolar ridge, the posterior part is lowered and opens the passage to the pharynx. The dorsal surface of the tongue moves upwards, expanding the area of contact with the palate and pressing the fluid against the palate. When taking solid food, in the oral phase the food is completely prepared and thus facilitates the pharyngeal phase of swallowing. The pharyngeal phase begins with the trigger of the pharyngeal swallowing reflex. The velopharyngeal sphincter rises and closes the path to the epipharynx, the suprahyoid muscles push the larynx up and forward, and the epiglottis closes. Pushing the larynx forward and under the base of the tongue also causes the adduction of the vocal

cords, which closes the glottis and thus prevents the penetration of food into the larynx and airways. These actions achieve separation of the digestive and respiratory tract, filling of the pharynx, its passive emptying and active peristalsis of the pharyngeal muscles. Thus, the sequences of swallowing in the pharyngeal phase are lifting of the larynx, anterior movement of the chiode bone, movement of the epiglottis, closing of the larynx, movement of the pharyngeal wall, contraction of the pharynx, and opening of the pharyngoesophageal segment. A lesion in the lateral part of the medulla oblongata disrupts the order of swallowing. Patients cannot begin to contract the suprachiodine muscles, which disrupts the movements of the pharynx and the base of the tongue. Without sufficient pressure on the base of the tongue, the epiglottis loses its ability to close completely (10). The esophageal phase begins in the esophagus, which consists of striated and smooth muscles. This phase allows the transfer of food to the stomach. With peristaltic movements, the food goes down. The esophageal phase is an involuntary phase of swallowing and is slower than the pharyngeal phase (9).

Eating and swallowing are closely related to breathing. In healthy adults, breathing is interrupted during swallowing, both due to the physical closure of the airways by raising the soft palate and closing the epiglottis and due to the neural control of respiration in the brainstem. Understanding the physiology and pathophysiology of eating and swallowing are key to the evaluation and treatment of swallowing disorders (8).

NEURAL SWALLOWING CONTROL

Supratentorial regions are crucial for modulation and initiation of swallowing itself, and brainstem structures are recognized as the basic motor plan for the pharyngeal response. The first evidence of a swallowing center was presented by Miller and Sherrington in 1915. Additionally, Miller (11) spoke of electrostimulation of specific nuclei of the brainstem that do not elicit the pharyngeal phase of swallowing, although muscles are innervated from these motor nuclei. Such an observation suggested that there is a complex interdependent cycle of the swallowing process. Some studies have highlighted the importance of the medulla oblongata in the coordination between swallowing and respiration (12). Ongoing considerations of the swallowing mechanism within the brainstem bring to the fore a central pattern generator (CCE) that can be considered a functionally connected set of neurons capable of producing a rhythmic, predictable output in the absence of afferent sensory input (13). The central pattern generator can also be found in other systems such as chewing (14), movement (15), breathing (16). Many components of the neural network for swallowing are not intended only for swallowing, but the swallowing pattern is closely related to chewing and breathing through the common bases of neurons (17).

Sensory information from the pharynx is transmitted to the afferent fibers V, VII, IX, and X of the cranial nerve and ends in the nucleus tractus solitarius in the medulla oblongata, in the brainstem. Through afferent fibers, information reaches the cerebral cortex where the cortical response to swallowing begins (18).

The posterior cerebellar artery is the largest branch of the vertebral artery and begins on the anterolateral side of the medulla oblongata near the lower cranial nerves. Swallowing is affected by the complex relationship of this artery with the n. facialis, n. vagus, n. glossopharyngeal, n. hypoglossus (19).

PATHOPHYSIOLOGICAL MECHANISMS OF DYSPHAGIA IN LMS

Risk factors for LMS include hypertension, smoking, and diabetes. A significant cause of LMS is the dissection of the vertebral artery, which can be caused by: neck injury, Marfan's syndrome, Eichler Danlos syndrome, and fibromuscular dysplasia. In young people, vertebral artery dissection is the most common cause of LMS (20).

Swallowing disorder, to some degree, is present in between 50 and 100% of patients with lateral medullary infarction (21). Dysphagia after medullary infarction is more common in patients with lesions in the upper and middle level, as well as in the dorsolateral level of the medulla oblongata. Videofluoroscopy in these patients shows a disorder of the opening of the esophageal sphincter, food debris in the piriform sinuses due to weakness of the muscles that make up the pharyngeal wall, as well as multiple attempts to swallow to move the bolus from the pharynx to the esophagus. It has been shown that more precise localization and level of the lesion can

represent a significant predictor of dysphagia and aspiration in lesions in the medulla oblongata (3, 21).

Lesions in the medulla oblongata usually cause disorders of the oropharyngeal phase of swallowing because they are located in important centers for swallowing: nucleus tractus solitarius and nucleus ambiguous. Patients with a unilateral medullary lesion have mostly intact oral bolus control but significant trigger damage and neuromotor control of the pharyngeal phase of swallowing. If a trigger for the pharyngeal swallowing reflex exists, a delay of 10 to 15 seconds is observed. When pharyngeal swallowing is triggered, the following is observed in these patients: 1) reduced lifting of the larynx, which leads to reduced opening of the cricopharyngeal region and collection of food debris in the piriform sinuses; 2) unilateral weakness of the pharynx, which again leads to the collection of food debris in the piriform sinuses. Unilateral paresis or vocal cord paralysis occurs in many patients. Symptoms of laryngeal weakness and occasional hiccups, as well as velopharyngeal incompetence in LMS, indicate damage to the nucleus ambiguous (22). Lesions involving the nucleus tractus solitarius cause decreased sensory function of the pharynx, base of the tongue, and epiglottis. Decreased sensory function leads to a higher risk of aspiration, as well as food residues in the pharynx. Some studies focus on the ability to open the upper esophageal sphincter. Lesions in the medulla oblongata affect the ability of the cricopharyngeal muscle to relax. In patients with lateral medullary infarction, the upper esop hageal sphincter is damaged ipsilaterally (3, 8).

Two bilateral centers in the brainstem are thought to represent the anatomical structure for the central pattern generator (CCE). The dorsal part of the medulla oblongata is anatomically located 1.5 to 4 mm rostrally from the obex and consists of the area that surrounds and includes the nucleus tractus solitarius and the adjacent reticular formation. The nucleus tractus solitarius is the primary sensory nucleus for the n.facialis, n.glossopharyngeus, and n.vagus. Afferent pathways from the pharynx and larynx travel through these cranial nerves to the nucleus tractus solitarius and it also receives impulses from the trigeminal sensory nucleus of the pons. Mucosal receptors in the pharynx respond to touch, water, pressure and facilitate movement and reactivation of swallowing (23). The dorsal neurons of the medulla oblongata lack a connection with the n. hypoglossus and n.trigeminus and are directly connected in the nucleus ambiguous and the adjacent reticular formation. That is why the dorsal neurons of this region are considered to be programmatic interneurons that set up sequential patterns of neuronal activation that are transmitted to the ventral parts for motor activation (24).

At the neurological level, bilateral swallowing centers in the medulla oblongata function as one integrated center, and the lesion of part of the center is sufficient to cause a complete loss of swallowing function. Dysphagia within the LMS is more severe and has a longer duration compared to dysphagia with hemispherical stroke (3). In most patients with lateral medullary infarction in the acute phase, dysphagia is a severe symptom, which is why the patient requires non-oral feeding. However, swallowing disorders in these patients often spontaneously restitute within two months after the stroke. In hemispherical stroke, the frequency of symptoms in the oral swallowing phase is higher, while the symptoms in LMS are related to the pharyngeal swallowing phase. In other words, swallowing disorder in LMS occurs as a result of contraction of the proximal pharynx with the absence of motor activity of the upper esophageal sphincter and proximal esophagus during swallowing (23).

Empirical studies indicate variability in the duration and severity of dysphagia in patients with lesions in the dorsolateral part of the medulla oblongata. Although the lesion is unilateral, its effect on swallowing is mutual. It is assumed that this occurs because the premotor neurons in the ambiguous nucleus and their connections are damaged. Consequently, damage or disruption of the connection of cranial motor neurons that participate in swallowing with the contralateral ambiguous nucleus leads to swallowing disorders. Preserved ipsilateral premotor neurons and contralateral centers in the medulla oblongata may affect the degree and duration of dysphagia (25).

DIAGNOSTIC PROCEDURES FOR ASSESSMENT OF DYSPHAGIA IN LMS

Early detection of risk through a post-stroke aspiration screening test is crucial because it allows for rapid intervention, reduces mortality, length of hospital treatment, and overall treatment costs (26). Accordingly, the screening test is the first step in evaluating swallowing in the acute phase of a stroke. Screening involves performing a minimally invasive

procedure that is not a diagnostic procedure. The purpose of swallowing screening is to detect the risk of aspiration and dysphagia. If screening indicates the presence of risk, diagnostic procedures are performed. The validity of the screening, through the sensitivity and specificity of the test, implies the degree to which the test measures the risk of aspiration. Sensitivity is the probability that some of the clinical signs of aspiration (cough, choking) will be present. There are different variations of the screening test, but they all refer to the use of water, the volume of which ranges from 3 ml (27) to 90 ml (28). When performing the screening method, there are signs of aspiration, cough during or after swallowing, choking, change of voice.

Instrumental diagnostic procedures

Before any instrumental diagnostic methods are performed, the medical documentation is evaluated and, if possible, heteroanamnestic data are taken from the caregiver. The history of the disease, possible diseases that may be associated with dysphagia, previous strokes, the presence of other neurological diseases, data on the possible presence of head or neck cancer, as well as data on possible surgical interventions are considered; application of pharmacological agents as well as the functional status of the patient concerning independence before admission to the hospital, cognitive status, ability to communicate are analyzed, too (29). A team of experts, including a speech therapist, gastroenterologist, otorhinolaryngologist, radiologist, nurse, and physiotherapist, participates in the diagnostic procedure. The speech therapist detects swallowing disorders and monitors the patient through all diagnostic procedures and later through treatment. The gastroenterologist performs diagnostic procedures related to the esophageal segment. The otorhinolaryngologist diagnoses oral, pharyngeal, laryngeal and pathology at the level of the trachea and, together with the speech therapist, cooperates with videofluoroscopy. The radiologist applies radiological procedures by which, together with the speech and language pathologist, they assess the swallowing disorder. A nurse trained in swallowing disorders as well as a physiotherapist who assists in posture adequate for performing indicative procedures also participate in the diagnostic procedure (30). Before instrumental diagnostics, an evaluation of anatomical structures and structural integrity is performed, which implies the existence of hypersalivation. Patients with lateral medullary syndrome generally cannot swallow saliva. Non-control of saliva can be a symptom of the weakness of the facial nerve. Therefore, the assessment of cranial nerves is performed by placing orders on the patient to perform certain motor actions or to send sensory manifestations. A more comprehensive assessment involves assessing the cough reflex (31, 32).

Videofluoroscopy (VFSS) or modified barium ingestion occupies a special place in the diagnostic procedure of dysphagia. Videofluoroscopy is treatment-oriented and allows real-time insight and visualization of the bolus through the oral cavity, oropharynx, hypopharynx, and esophagus using modified barium. This method is otherwise considered the gold standard in diagnosing dysphagia (7). VFSS provides insight into the manipulation of the bolus in the oral cavity (chewing, bolus formation, directing the bolus to the posterior part), evidence of aspiration/penetration into the airways (before, during, and after swallowing), the amount and localization of food residues in the oral cavity and pharynx (29). VFSS results determine treatment strategies. A significant part of this diagnostic finding is the lateral section of the swallowing process, which enables the observation of tongue base movement, velopharyngeal sphincter, chiolaryngeal elevation, laryngeal closure, contraction of the pharyngeal constrictor, and opening of the upper esophageal sphincter (7).

The next diagnostic method is video endoscopy. This method allows the specialist to formulate an effective plan for swallowing therapy and is a safe instrumental method of diagnosis for the patient. It is a minimally invasive technique that is well tolerated and in which the patient is not exposed to radiation. It is performed using a flexible laryngoscope that is placed through the nose and extends through the pharynx to see the pharyngeal and laryngeal structures during swallowing (32). Video endoscopy allows static and dynamic assessment of structures in the upper respiratory tract and upper digestive tract. This method provides information on pathophysiological deficits of the soft palate, pharynx, and larynx, and provides direct insight into superficial anatomy, mucosal abnormalities, effects of altered structures on bolus flow, and airway protection, glottis occlusion, bolus pathway, and bolus location in the hypopharynx. This method assesses

the patient's ability to swallow a bolus of different textures and consistency (7).

Although video endoscopy and videofluoroscopy provide insight into the pathophysiology of swallowing, their application limits visualization in three dimensions, and therefore today dysphagias are approached from the angle of a new perspective. For that purpose, computerized tomography is used, which enables 3D imaging, as well as quantitative measurements. Using computed tomography, the dynamics of the movement of structures, which participate in the act of swallowing, is simultaneously assessed (29).

The methods of quantitative analysis of motion are mainly used to collect kinematic and kinetic data. This group of methods includes high-resolution manometry, which provides insight into kinetic parameters such as the pressure of the base of the tongue and the narrowing of the pharynx. As the pharyngeal phase of swallowing is very complex and requires high coordination of muscle contractions and the creation of pressure to efficiently transfer the bolus to the esophagus, the high-resolution method provides information on the pressure in the pharynx and upper esophageal sphincter during swallowing (33).

TREATMENT OF DYSPHAGIA IN LMS

Many patients with LMS who have swallowing disorders require appropriate treatment. Given the complexity of the problem, a team of experts usually participates in the treatment. The professional team for the treatment of dysphagia includes doctors, dentists, speech therapists, nurses, physiotherapists, and nutritionists. In Japan, studies were conducted on the participation of various team members in the rehabilitation of dysphagia and the results indicated that the participation of speech therapists was 34%, dentists 20%, nurses 14%, doctors 8%, nutritionists 8%, while the rest of the participation was related to occupational therapists. and physiotherapists (7). As a rule, the team leader in the treatment of dysphagia in LMS is a speech therapist. The traditional approach to the treatment of swallowing in patients with lateral medullary syndrome includes exercises for the pharyngeal muscles, which include: Shaker exercises, tongue base exercises, and Masako maneuver, as well as expiratory muscle exercises (34). Treatment of swallowing disorders is classified as direct and indirect. Indirect treatment focuses on exercise without the inclusion of food or fluids, while direct treatment refers to the intake of food and fluids. This classification was performed from the aspect of aspiration risk (29). Exercises in the treatment of dysphagia refer to the integration of interdependent elements in the act of swallowing. As the deficit of the pharyngeal phase of swallowing is most often noticed in the lateral medullary syndrome, therapeutic exercises should be based on initiating and strengthening the movements of the base of the tongue, hyoid, pharynx, larynx, and upper esophageal sphincter. In this way, it acts on hyolaryngeal elevation, closure of the larynx, constriction of the pharynx, and dilatation of the upper esophageal sphincter (25).

Shaker exercise involves a combination of isometric and isokinetic exercises to strengthen the suprahyoid muscles (m. geniohyoid, m. mylohyoid, and m. digastric) and allow shortening of m. thyrohyoid. This exercise results in a reduction of residues in the piriform sinuses and a reduction in hypopharyngeal intrabolic pressure. It is performed three times a day for six weeks (35, 36).

Tongue base retraction exercises allow pressure to be created to trigger the pharyngeal bolus. The base of the tongue passes the bolus through the pharynx, retracting and making full contact with the pharyngeal wall and thus applying pharyngeal pressure to the bolus. The exercise is designed to improve the maximum range of motion of the base of the tongue, establishing the power to push the bolus and ensure the passage of the entire bolus through the pharynx (37).

The Masako maneuver or contraction of the posterior part of the pharynx together with the gradual movement of the base of the tongue during swallowing provides the driving force necessary to move the bolus through the upper part of the pharynx. The "swallowing tongue" swallowing exercise is aimed at contracting the pharynx, physiologically increasing the anterior movement of the pharyngeal muscles (m. superior constrictor), and thus improving the contact between the base of the tongue and the posterior wall of the pharynx. The goal of the exercise is to strengthen the contractions of the pharynx (38, 39).

Thermotactile stimulation also plays an important role in the treatment of dysphagia. It is a sensory stimulation technique used to improve the trigger of the pharyngeal reflex in patients in whom the swallowing reflex is delayed and who have a

high risk of aspiration. Cold stimulation is a sensory stimulus that goes to the brain stem and induces an improvement in swallowing physiology that requires the ability to swallow voluntarily without stimulation. Alveolar arches are especially recommended for mechanical thermal stimulation because they are sensitive to activate the swallowing reflex (8, 40).

The method of neuromuscular electrostimulation (NMES) is also used. This therapeutic technique stimulates the neck muscles to activate the sensory swallowing pathways. NMES implies strengthening the musculature that participates in the act of swallowing. It originated from many years of use in physical therapy. Since 2002, the VitalStim device has been used in the United States in the treatment of dysphagia resulting from stroke and neuromuscular diseases (41, 42).

CONCLUSION

Having insight into the scientific literature on dysphagia and research of neural connections of the medulla oblongata, we have concluded that in patients with LMS, the velopharyngeal sphincter, retraction of the base of the tongue, as well as the pharyngeal phase of swallowing is most often damaged. In the diagnosis of dysphagia in LMS, a special place is occupied by instrumental diagnostics, which enables insight into which phase the act of swallowing is most damaged. In addition to detecting a swallowing damage, instrumental diagnostics directs us to choose the appropriate treatment. Since speech therapy occupies a central place in the treatment of dysphagia, and the treatment of swallowing disorders is based on good diagnostics, it is extremely important to educate speech therapists about the existing diagnostic procedures, their scope, and limitations. Since dysphagia in lateral medullary syndrome is a significant and often the only persistent symptom, the treatment of swallowing disorders in patients with this syndrome is a real challenge for speech therapists and other professionals. As the deficiency of the pharyngeal phase of swallowing is most often noticed within the lateral medullary syndrome, the program of therapeutic exercises should be based on initiating and strengthening the movements of the base of the tongue, hyoid, pharynx, larynx, and upper esophageal sphincter. In that way, it acts on hyolaryngeal elevation, closing of the larynx, constriction of the pharynx, and dilatation of the upper esophageal sphincter. The ability to establish neural control of swallowing and the location of the lesion is considered an important predictor of swallowing disorders in patients with LMS.

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Disfagija kod lateralnog medularnog sindroma

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SAŽETAK

Uvod. Lateralni medularni sindrom je neurološko oboljenje izazvano ishemijom u bočnom delu produžene moždine i predstavlja najčešći oblik infarkta moždanog stabla. Disfagija je čest i klinički značajan simptom ovog oboljenja jer je usko povezana sa rizikom od ponovljene pneumonije, malnutricije, dehidracije, te sa povećanjem procenta mortaliteta i produženim bolničkim lečenjem.
Cilj. Cilj ovog rada bio je pregled i analiza podataka o korelaciji poremećaja gutanja i lateralnog medularnog sindroma. Namera nam je da na sveobuhvatan način prikažemo simptome, dijagnostičke i terapijske procedure disfagije kod bolesnika sa ovim sindromom.
Metodologija. Za pretraživanje literature korišćene su sledeće baze podataka: KoBSON – Konzorcijum biblioteka Srbije za objedinjenu nabavku, *PubMed*, *Science Direct*.
Rezultati. Na osnovu rezultata pregledanih studija utvrđeno je da bolesnici sa lateralnim medularnim sindromom često imaju poremećaje gutanja. Oni se neretko hrane putem nazogastrične sonde i po nekoliko meseci nakon moždanog udara, što značajno narušava njihov kvalitet života. U cilju prevazilaženja smetnji u gutanju i stvaranja uslova za bezbednu funkciju gutanja, većina bolesnika ima potrebu za tretmanom u dužem vremenskom intervalu. Pored dostupnih skrining testova, instrumentalne dijagnostičke metode omogućavaju uvid u biomehaničke aspekte poremećaja gutanja, determinišu rizik od aspiracije i predstavljaju polaznu osnovu za odabir strategija tretmana.
Zaključak. Tretman disfagije zavisi od mehanizama nastanka i faktora predikcije oporavka funkcije gutanja. Pri sprovođenju tretmana, pored ostalog, veoma je važno poznavanje patoloških mehanizama neuralnih veza produžene moždine.

***Ključne reči*: lateralni medularni sindrom, disfagija, dijagnostika, tretman**