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# Swallowing management of post-stroke dysphagia

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## Abstract

Post-stroke dysphagia is a common and serious complication of cerebrovascular accidents, affecting up to 80% of patients in the acute phase and significantly increasing the risk of aspiration pneumonia, malnutrition, dehydration, and mortality. Among swallowing disorders, delayed or absent initiation of the swallowing reflex represents one of the most severe forms, particularly following brainstem or bilateral cortical lesions.

This article provides an integrative review of the physiological mechanisms of swallowing and their neurological control, with a specific focus on post-stroke dysphagia associated with swallowing areflexia. It synthesizes current knowledge on the anatomy and neurophysiology of swallowing, the central and peripheral neural networks involved, and the pathophysiological mechanisms underlying impaired reflex initiation after stroke. Clinical manifestations, lesion-related etiologies, recovery patterns, and prognostic factors are discussed.

The paper further reviews current assessment and rehabilitation strategies for post-stroke dysphagia, emphasizing the importance of early screening and individualized speech-language therapy. Conventional compensatory and restorative interventions are examined alongside emerging neurostimulation and manual therapy approaches aimed at enhancing sensory afferent input and promoting neuroplasticity.

In conclusion, effective management of post-stroke dysphagia requires early, multidisciplinary intervention and continued development of evidence-based therapeutic strategies for restoring swallowing reflex function.

## Keywords

Post-stroke dysphagia; Swallowing reflex; Swallowing areflexia; Neurophysiology; Rehabilitation.

## 1. Introduction

Swallowing is a spontaneous and complex mechanism that enables the transfer of the food bolus, liquids, and salivary secretions from the mouth to the stomach, via the esophagus, while protecting the airways (Merrot et al., 2011). This function encompasses a sophisticated physiological sensorimotor mechanism, which unfolds in three stages: the oral phase, the pharyngeal phase, and the esophageal phase (Bleeckx, 2001). When one of these stages is impaired, the condition is referred to as dysphagia. Indeed, dysphagia involves all physiopathological mechanisms that may

affect the three phases of swallowing (Lacau St Guily et al., 2005) and is defined as a "temporary or permanent, partial or total inability to swallow food or liquids by mouth for nutritional purposes" (Brin et al., 2011) .

Stroke (Cerebrovascular Accident, CVA) is the most frequent cause of oropharyngeal dysphagia (Ickenstein et al., 2012), affecting 40% to 80% of patients during the acute phase of stroke, depending on the study (Flamand-Roze et al., 2012). Within the first two weeks, 50% of these patients reportedly recover this function spontaneously (Flamand-Roze et al., 2012). However, according to Auzou (2007), certain factors may prolong this recovery time, such as an extensive lesion, bilaterality, pre-existing brain injury, or involvement of the brainstem. Consequently, 10% of patients still present with dysphagic sequelae six months later (Bleeckx, 2001).

In neurological dysphagia, it is common to observe a delayed or absent swallowing reflex (Schwarz et al., 2018). When it is completely absent, the condition is referred to as swallowing areflexia, characterized by the absence of laryngeal closure, pharyngeal contraction, and laryngeal elevation (Guatterie & Lozano, 2005). This impairment can lead to numerous consequences. Indeed, persistent swallowing disorders particularly expose patients to risks of complications, such as aspiration pneumonia, malnutrition, and dehydration, which may compromise the recovery of physical functions (Woisard-Bassols & Puech, 2011). Moreover, dysphagia leads to prolonged hospital stays and increases the risk of mortality (Flamand-Roze et al., 2012).

It therefore appears essential to manage dysphagia as early as possible (Oujamaa et al., 2012), by conducting a formal swallowing assessment and developing a specific and individualized rehabilitation program. Classical intervention is generally organized around two main approaches: adaptive or compensatory strategies, and specific rehabilitation protocols (Woisard-Bassols & Puech, 2011).

Since swallowing is a sensorimotor act, the speech-language pathologist may provide sensory and motor stimulation. Various facilitation techniques or specific maneuvers can be used to promote swallowing (Bleeckx, 2001). However, these practices are not intended to specifically stimulate an absent swallowing reflex and are not always suitable for the acute phase, as they require a certain degree of patient cooperation (Woisard-Bassols & Puech, 2011).

New rehabilitation techniques, such as electrical or magnetic stimulation, either central or peripheral, can also be applied passively. Nevertheless, current research remains insufficient to determine their efficacy (Bath et al., 2018; Cabib et al., 2016). Thus, to date, there are few conclusive approaches for restoring an abolished swallowing reflex in acute stroke patients, who may sometimes present with reduced vigilance.

## 2. The Physiological Mechanisms of Swallowing and Its Neurological Control

### Anatomophysiology of Swallowing

### 2.1Anatomical Structures Involved in Swallowing

According to current international research, a set of anatomical structures is engaged in swallowing: the oral cavity, the pharynx, the larynx, and the esophagus. The anatomical and neuromuscular integrity of these structures is indispensable for the proper execution of swallowing (Sasegbon & Hamdy, 2017; Rosero Salazar et al., 2024).

Concerning the **oral cavity**, the lips form the **labial sphincter** via the orbicularis oris muscle. The teeth and the mandible together constitute the **mandibulo-dental sphincter**. The pterygoid, temporal, and masseter muscles allow lowering of the mandible. The muscles of the oral cavity confer mobility to its structures. The **suprahyoid muscles** (mylohyoid, geniohyoid, and the anterior belly of the digastric muscle) form the floor of the mouth. Serving as a support for the base of the tongue, the **hyoid bone** is connected to the larynx via the thyrohyoid muscle and ligament. The **tongue**, divided into two parts (oral portion and base of tongue), comprises seventeen muscles that enable it to move in all directions. The **vallate papillae** (or lingual V) are located anterior to the **valleculae** (the space between the epiglottis and the base of tongue), between the anterior pillars of the soft palate formed by the **palatoglossus muscle**, which closes the isthmus of the throat. Finally, thanks to the **levator (internal palatal muscles)** and **tensor (external palatal muscles)**, the **soft palate (velum)** allows, in particular, the closure of the **velopharyngeal sphincter**.

The **epiglottis** is one of the cartilages of the larynx (together with the cricoid, thyroid, and arytenoid cartilages) that provide its rigidity. Located beneath the root of the tongue and at the entrance of the larynx, it is attached to the thyroid cartilage and connected to the hyoid bone. The **larynx**, positioned anterior to the pharynx and the esophagus, houses the **vocal folds**, which lie below the ventricular folds and are anchored anteriorly to the midline of the larynx and posteriorly to the arytenoid cartilages. On each side of the larynx, the mucosa of the hypopharynx folds to form a groove known as the **piriform sinus**.

The **pharynx** is a musculo-membranous conduit classically divided into three regions: the **nasopharynx**, the **oropharynx**, and the **hypopharynx**. The **pharyngeal constrictor muscles** (superior, middle, and inferior), which line the posterior and lateral pharyngeal walls, are the primary muscles involved in swallowing. Posteriorly, these muscles are anchored to the **pharyngeal raphe**, which itself is attached to the base of the skull. Anteriorly, their attachments include the **skull base, the tongue base, and the mandible** (superior constrictor), the **hyoid bone** (middle constrictor), and the **thyroid and cricoid cartilages** (inferior constrictor).

The **esophagus**, also a musculo-membranous conduit, is closed at both ends by two sphincters: the **upper esophageal sphincter (UES)** superiorly and the **lower esophageal sphincter (LES)** inferiorly. The **cricopharyngeal muscle**, which forms part of the lower pharynx, constitutes the main component of the UES.

### 1.2 The Phases of Swallowing

Conditioned by both physical and neurological integrity, **swallowing** is a spontaneous mechanism that enables the transfer of the food bolus, liquids, and

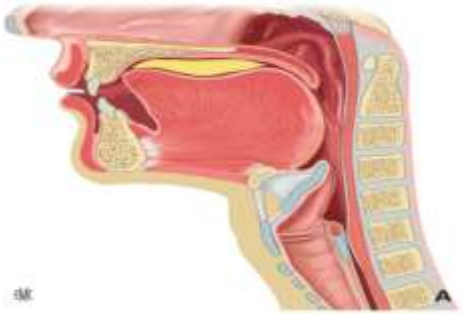
salivary secretions from the mouth to the stomach through the esophagus, while simultaneously protecting the airway. This function represents a sophisticated **sensorimotor process**, classically described in **three distinct but interrelated phases**: the **oral phase**, the **pharyngeal phase**, and the **esophageal phase**. Importantly, swallowing should be considered as an integrated process rather than isolated stages.

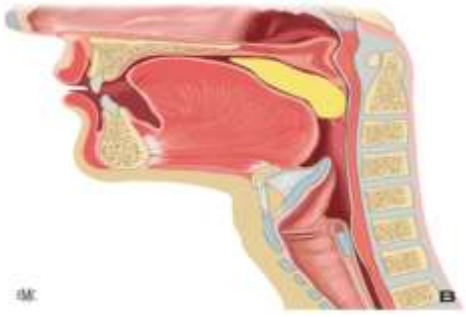
**Oral Phase**

The **oral phase** represents the first stage of swallowing and is the only voluntary phase. It can be divided into two successive steps: the **oral preparatory phase** (introduction of food into the mouth and preparation of the bolus) and the **oral transport phase** (propulsion of the bolus toward the pharynx) (Matsuo & Palmer, 2008; Steele & Miller, 2010).

During the **oral preparatory phase** (Figure 2A), the bolus is introduced into the oral cavity through adequate mouth opening and food intake, which may involve suction in the case of liquids. Sensory receptors of the tongue assess the characteristics of the bolus, including texture and volume, allowing appropriate modulation of movements (Dodds, 1989). The bolus is lubricated by salivary secretions (Matsuo & Palmer, 2008) and masticated through rhythmic mandibular movements coordinated by the masticatory muscles (Hiiemae & Palmer, 1999). The tongue plays a key role in positioning the bolus onto the molar teeth, while the buccinator muscles assist in controlling food within the oral cavity during mastication (Palmer, 1997). For liquids, negative intraoral pressure facilitates suction and propels them toward the pharynx.

The **oral transport phase** (Figure 2B) begins once the bolus, sealed between the dorsum of the tongue and the soft palate, is ready to be swallowed. At this stage, the tongue assumes a grooved shape, with its apex positioned just behind the upper incisors, and positions the bolus onto its dorsal surface (Matsuo & Palmer, 2008). The tongue then generates coordinated anterior-to-posterior pressure waves, which actively propel the bolus toward the oropharynx (Palmer, 1998; Steele & Miller, 2010). This phase is completed when the bolus passes beyond the anterior faucial pillars, triggering the pharyngeal stage of swallowing (Dodds, 1989; Logemann, 1999).



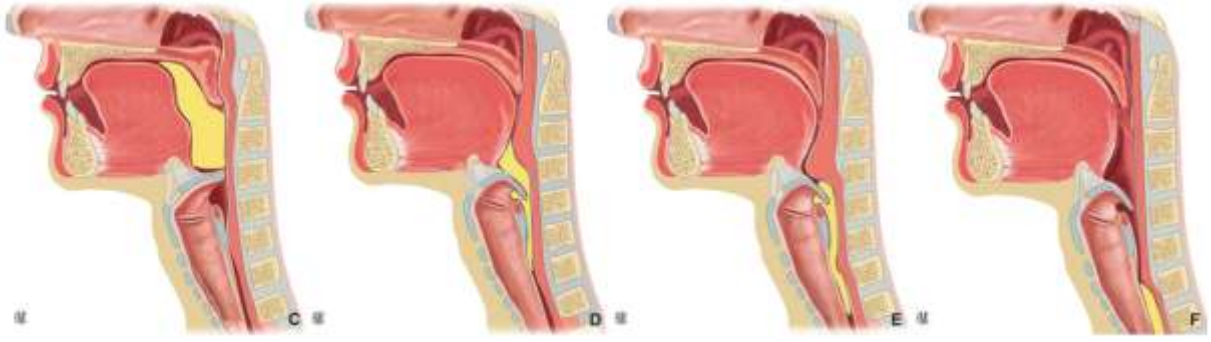

**Pharyngeal Phase**

The **pharyngeal phase** of swallowing is an automatic, reflexive stage and represents the second step of deglutition. Sensory receptors located at the faucial isthmus, the oropharynx, and the laryngeal vestibule trigger the swallow reflex, which initiates

this phase (Matsuo & Palmer, 2008; Dodds, 1989). It involves anterior-superior elevation of the larynx, along with propulsive and protective mechanisms that allow safe bolus passage from the oropharynx to the upper esophageal sphincter (UES) (Logemann, 1999; Humbert & German, 2013). This process requires multiple synchronized neuromuscular events that occur in rapid sequence (Logemann, 1999).

The first events include **velopharyngeal closure** and **hyoid elevation**. The soft palate, initially in a low resting position, elevates against the posterior pharyngeal wall to increase intrapharyngeal pressure and prevent nasal regurgitation (Panara & Padalia, 2020). Concurrently, the base of the tongue retracts forcefully against the posterior pharyngeal wall, contributing to bolus propulsion and increased pharyngeal pressure (Logemann, 1999). This posterior tongue movement initiates **pharyngeal peristalsis**, defined as the sequential top-to-bottom contraction of the superior, middle, and inferior pharyngeal constrictors, which compress the bolus downward toward the UES (Matsuo & Palmer, 2008; Kahrilas et al., 2003). During this stage, the pharynx also shortens vertically to reduce its volume, further facilitating bolus transit (Matsuo & Palmer, 2008).

Simultaneously, **airway protection** mechanisms take place. Contraction of the suprahyoid muscles elevates the hyoid bone, leading to the anterior-superior displacement of the larynx (Miller, 2008). This movement tucks the larynx beneath the tongue base and mechanically facilitates UES opening by pulling on the cricopharyngeus fibers (Panara & Padalia, 2020). Airway closure occurs in a bottom-to-top sequence: adduction of the true vocal folds via lateral cricoarytenoid muscle contraction, approximation of the arytenoids to the base of the epiglottis, and posterior tilting of the epiglottis to seal the laryngeal vestibule (Logemann, 1999; Matsuo & Palmer, 2008). Epiglottic inversion, driven by tongue base retraction, hyolaryngeal elevation, bolus pressure, and pharyngeal contraction, primarily directs the bolus laterally into the pyriform sinuses (Matsuo & Palmer, 2008). Importantly, during bolus transit from the oropharynx to the esophagus, **respiration is transiently inhibited**, preventing aspiration (Humbert & German, 2013).




**Esophageal Phase**

The **esophageal phase** of swallowing is an involuntary, reflexive process that begins once the bolus has passed through the upper esophageal sphincter (UES) and continues with esophageal peristalsis. UES opening results from relaxation of the

cricopharyngeal muscle, combined with laryngeal elevation produced by the suprahyoid and thyrohyoid muscles, as well as intrabolus pressure exerted during descent (Matsuo & Palmer, 2008; Shaker et al., 1992). After bolus passage, the larynx descends back to its resting position, and the cricopharyngeus regains its baseline tonic contraction, preventing air entry and reflux (Shaker et al., 1992).

Bolus transit through the esophagus toward the lower esophageal sphincter (LES) is facilitated by both **gravity** and a coordinated wave of **primary peristaltic contractions** generated by the circular and longitudinal smooth muscle layers (Kahrilas & Dodds, 1989; Pandolfino et al., 2003). Upon reaching the LES, relaxation of sphincteric tone allows bolus entry into the stomach, after which basal pressure is restored to prevent gastroesophageal reflux (Dodds, 1989; Pandolfino et al., 2003).

## 2. Neurological Control of Swallowing

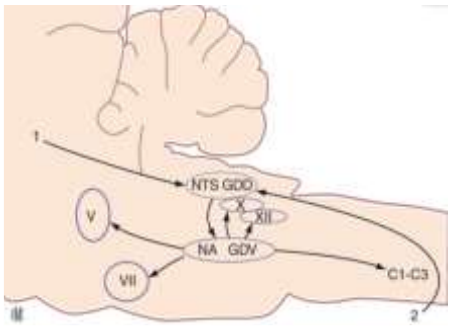
The neurological control of swallowing is governed by peripheral **afferent inputs** (sensory and somatosensory) and **efferent outputs** (motor), their **integration within the brainstem** (particularly the medullary and pontine centers), and a **cortical and subcortical network** that initiates and modulates the process (Kahrilas & Logemann, 1993; Lowell et al., 2008). Swallowing is considered one of the most **complex motor functions**, requiring precise synchronization and interaction between these hierarchical levels of control (Miller, 2008; González-Fernández et al., 2013).

Although understanding of the roles of both the **central nervous system (CNS)** and the **peripheral nervous system (PNS)** in swallowing has advanced considerably in recent decades, particularly with the advent of neuroimaging and neurostimulation studies (Ertekin & Aydogdu, 2003; Michou & Hamdy, 2009), our knowledge of these mechanisms remains **incomplete**, and many aspects of the neurophysiology of swallowing are still under investigation (González-Fernández et al., 2013).

### 2.1 The Swallowing Center

The **swallowing center**, located within the **pons and medulla oblongata**, is functionally organized into three main components: a **sensory (afferent) level**, a **motor (efferent) level**, and an **integrative level** (Jean, 2001; Ertekin & Aydogdu, 2003). This central pattern generator (CPG) for swallowing receives **peripheral afferents** from anatomical structures involved in deglutition via multiple **cranial nerves**—including the trigeminal (V), facial (VII), glossopharyngeal (IX), vagus (X), accessory (XI), and hypoglossal (XII)—as well as **cervical spinal inputs** (Miller, 2008).

In addition, it receives **central inputs** from cortical and subcortical areas, particularly through the **corticobulbar tract**, which allows voluntary initiation and cortical modulation of swallowing (Lowell et al., 2008; Michou & Hamdy, 2009). Once sensory information is integrated within the medullary swallowing CPG, the center coordinates the **motor output** distributed to more than **thirty muscles** of the oral cavity, pharynx, larynx, and esophagus to generate the sequential motor pattern of swallowing (Martin & Sessle, 1993; Jean, 2001).




**1 : Central regulatory afferents  2 : Peripheral afferents  NA : Nucleus ambiguus**

**  NTS : Nucleus tractus solitarius (Nucleus of the solitary tract)  GDD : Dorsal swallowing group (DSG)  GDV : Ventral swallowing group (VSG)**

**  V : Trigeminal nerve  VII : Facial nerve**

**  X : Vagus nerve  XII : Hypoglossal nerve**

Peripheral afferents and efferents pass through the nuclei of cranial nerves located in the brainstem. Direct sensorial afferents to the brainstem, or secondary afferents projecting to cortex, reach the **trigeminal nerve nucleus** (responsible for oral and deep somatic sensation) and the **nucleus tractus solitarius** (which receives sensory fibres from cranial nerves IX and X, including the internal branch of the superior laryngeal nerve, providing sensation from the base of tongue, the pharynx and larynx, as well as taste fibres carried by VII, IX and X) (Sensory Input Pathways & Mechanisms, 2010; Cranial Nerves and Swallowing, n.d.).

Moreover, the oral cavity, larynx, pharynx and esophagus contain receptors sensitive to touch, muscle pressure, cartilaginous displacements, pain, chemical and thermal changes which, when stimulated, can trigger swallowing via direct projections onto the nucleus tractus solitarius (Sensory Input Pathways & Mechanisms, 2010; Brainstem Organization of the Swallowing Network, 1988).

Motor efferents originate from the motor level, which contains the cell bodies of motoneurons involved in swallowing. These include the nuclei of cranial nerves V, VII, and XII, which control the hyolaryngeal muscles, facial muscles and the tongue, and the **nucleus ambiguus** (with IX, X, XI), which innervates the palate, the larynx and the pharynx (Brain Stem Organization of the Swallowing Network, 1988; StatPearls “Nucleus Ambiguus”, 2024).

The connection between sensorial-sensory afferents and motor efferents, referred to as the **reflex arc**, enables the triggering of the swallowing reflex. This interaction occurs at the **integrative level**, composed of a network of interneurons responsible for programming the motor command for the automatic-reflexive stages of swallowing (Jean, 2001; Steele & Miller, 2010). This network is organized into two distinct bilateral clusters:

- The **dorsal swallowing group (DSG)**, which receives both peripheral and central afferents and thus plays a sensory and organizational role during the pharyngeal and esophageal phases (i.e., in adaptation and the initiation of swallowing) (Jean, 2001; Steele & Miller, 2010).
- The **ventral swallowing group (VSG)**, which performs an effector role by distributing the required action pattern for swallowing to the various motor nuclei of the cranial nerves. It also receives peripheral and cortical inputs that allow modulation of the motor sequence depending on the characteristics of the bolus (volume, texture, etc.) (Jean, 2001; Steele & Miller, 2010).

### 2.2 Cortical and Subcortical Control of Swallowing

The cerebral cortex plays a crucial role in both voluntary and automatic-reflexive phases of swallowing. On one hand, the frontal motor areas and temporal sensory regions, predominantly in the left hemisphere, are implicated in the oral phase by contributing to the learning and regulation of masticatory and manipulative motor skills (Martin et al., 2004; Lowell et al., 2008). Moreover, the decision to initiate or inhibit swallowing appears to involve the cingulate cortex and the insula, regions associated with sensorimotor integration and volitional control (Hamdy et al., 1999; Lowell et al., 2008).

On the other hand, clinical studies in stroke patients, together with findings from functional imaging and non-invasive brain stimulation (electrical or magnetic), demonstrate that cortical regions exert both facilitatory and inhibitory influences on the reflexive phases of swallowing (Hamdy et al., 1998; Michou & Hamdy, 2009). For the pharyngeal phase, evidence suggests the existence of a short pathway, directly via the brainstem, and a long cortical pathway, which interacts with peripheral afferents to regulate swallowing depending on sensory feedback (Jean, 2001; Lowell et al., 2008).

The sensorimotor cortex is particularly engaged during the pharyngeal stage. Studies indicate that the cortical swallowing representation lies in the inferior precentral gyrus, near the Rolandic operculum (Hamdy et al., 1996; Martin et al., 2004). A subtle somatotopic organization has been identified in the primary and premotor cortices, which are implicated in movement initiation and execution (Hamdy et al., 1998). Interestingly, direct stimulation of the primary motor cortex does not reliably elicit swallowing, whereas stimulation of the premotor cortex does, suggesting that the primary motor cortex may not be critical for pharyngeal phase control (Miller, 2008).

Furthermore, the sensorimotor cortical representation of pharyngeal swallowing is bilateral yet asymmetric (Hamdy et al., 1996; Martin et al., 2004). One hemisphere generally shows functional dominance, independent of

handedness, and this dominance correlates with the cortical representation of the pharynx (Hamdy et al., 1998). This bilateral representation is essential, as it provides a degree of redundancy and may facilitate recovery after unilateral cortical lesions (Hamdy et al., 1996; Michou & Hamdy, 2009).

## 2. Post-Stroke Dysphagia and Swallowing areflexia

### 2.1 Definitions

Dysphagia is a primary swallowing disorder involving any pathophysiological mechanisms that may impair one or more of the three phases of swallowing. It is defined as a temporary or permanent, partial or total inability to swallow food or liquids by mouth for nutritional purposes (United European Gastroenterology & European Society for Neurogastroenterology & Motility [UEG/ESNM], 2025; Sasegbon & Hamdy, 2017). High (oropharyngeal) dysphagia refers to impairment of the first two phases of swallowing (oral preparatory and pharyngeal), whereas low (esophageal) dysphagia refers to impairment of the esophageal phase (WGO, 2014; UEG/ESNM, 2025).

Moreover, in medical terminology, **areflexia** is defined as the "absence of a reflex, i.e., of a motor response to stimulation in a given reflexogenic zone, due to inhibition of the reflex arc." In the case of **swallowing areflexia**, the swallowing reflex fails to be triggered when the bolus passes over the lingual V. This dysfunction compromises the **entire pharyngeal phase**, thereby giving rise to what is termed *high dysphagia*. It manifests as a defect in **airway protection** (absence of velopharyngeal closure, supraglottic and glottic closure) and an impairment in **pharyngeal bolus transport** (absence of tongue-base retraction, pharyngeal contraction, laryngeal elevation, and UES opening).

The symptoms produced by this impairment can be **primary** or **secondary**. Primary symptoms derive directly from disruption of bolus transit and manifest as **drooling, residue/stasis, aspiration, choking or blockage, and/or reflux**. Secondary symptoms, on the other hand, reflect the **severity, tolerance, and systemic impact** of the swallowing disorder. They directly affect the patient's **quality of life**, **oral feeding** (prolonged meal times, diet modification or restriction), **nutritional status** (malnutrition and dehydration), and **pulmonary health** (aspiration pneumonia and bronchial congestion) (Oropharyngeal Dysphagia in Older Persons, 2016; Management of Dehydration in Patients Suffering Swallowing Disorders, 2019).

### General Considerations and Prevalence

Swallowing control is a complex process involving multiple neurological mechanisms. Consequently, most neurological disorders can impair swallowing and lead to **dysphagia** (Baijens & Clavé, 2021). Among these, **stroke** is the most frequent cause of **oropharyngeal dysphagia** (Ickenstein et al., 2012). Indeed, swallowing disorders affect **40–80% of patients in the acute phase of stroke**, although prevalence estimates vary depending on study design and assessment methodology, as well as lesion location and severity (Martino et al., 2005; Lakshminarayan et al., 2010).

Rofes et al. (2013) further reported that **unilateral strokes cause dysphagia in about 40% of cases, bilateral hemispheric lesions in 56%, brainstem lesions in 67%, and combined lesions in 85%**. Moreover, in neurological dysphagia, it is common to observe a **delayed or absent swallowing reflex** (Schwarz et al., 2018), although no precise epidemiological data are currently available.

### 2.3 Clinical Manifestations and Etiologies

Post-stroke dysphagia can involve **all three phases of swallowing** (oropharyngeal and esophageal). The **oral phase** may be impaired, presenting with difficulties in labial closure, ineffective clearance of the buccal sulci, reduced oral sensitivity, and abnormalities in bolus formation and posterior propulsion (Martino et al., 2005; Bath et al., 2018).

The **pharyngeal phase** may also be affected by a partially or completely insufficient swallowing reflex, resulting in impaired laryngeal elevation and altered supraglottic and glottic closure (Rofes et al., 2013). Additionally, dysfunction of **upper esophageal sphincter (UES) opening** can further aggravate dysphagia (Kawai et al., 2013).

In this section, we will focus specifically on the **alterations of the pharyngeal phase**, and more precisely on the **clinical manifestations and etiologies of abolished swallowing reflex**.

#### Clinical Manifestations

Since it prevents the initiation of the **pharyngeal phase**, swallowing areflexia can manifest in several ways. In most cases, it leads to **residue (stasis)**, corresponding to the accumulation of saliva or food within the mucosal folds of the pharynx, due to impaired bolus propulsion. In swallowing areflexia, residue may appear along the **pharyngeal wall** (due to impaired pharyngeal transport), within the **pyriform sinuses** (resulting from inadequate upper esophageal sphincter [UES] opening), in the **laryngeal vestibule or supraglottic region** (caused by incomplete supraglottic or glottic closure), and in the **valleculae** (due to reduced tongue base retraction and impaired bolus propulsion) (Logemann, 1998; Martin-Harris et al., 2008; Shaker & Leon, 2019).

When the bolus cannot be efficiently propelled into the pharynx, residue may also persist in the **oral cavity**. Critically, these stases may migrate into the **upper airways**, even minutes after the completion of a meal, increasing the risk of **aspiration pneumonia** (Martino et al., 2005; Langmore et al., 1998; Takizawa et al., 2016).

#### Clinical Manifestations: Obstruction and Aspiration

Swallowing areflexia may also lead to **bolus obstruction**, defined as a complete halt in bolus progression. Such blockages can occur at various levels—**oral cavity, valleculae, or pharynx**—when the pharyngeal phase fails to initiate (Logemann, 1998; Shaker & Leon, 2019).

Furthermore, the absence of a swallowing reflex gives rise to a particular form of **aspiration**, which may occur **without an actual swallow attempt**. In these cases, the bolus slides along the base of the tongue but

fails to trigger the pharyngeal phase. As a result, the **larynx remains open in a respiratory state**, and the bolus directly and massively enters the airway (Robbins et al., 1999; Ertekin & Aydogdu, 2003).

In addition, **residue-related aspiration** may occur when pharyngeal stasis is later inhaled into the trachea, with timing that varies depending on the type of food or liquid consumed (Martin-Harris et al., 2008; Martino et al., 2005).

Critically, when the **cough reflex**—the primary airway protective mechanism—is absent or ineffective, aspiration becomes **silent**, increasing the risk of **airway obstruction, hypoxemia, or aspiration pneumonia** (Smith Hammond & Goldstein, 2006; Langmore et al., 1998; Takizawa et al., 2016).

As observed in stroke dysphagia research, although individual variability is substantial, dysphagia tends to be **less severe in hemispheric strokes** compared to lesions in the posterior fossa, where direct involvement of the brainstem swallowing centers confers more persistent deficits requiring prolonged enteral nutrition (Martino et al., 2005; Sasegbon & Hamdy, 2024). Because swallowing control is bilateral, **bilateral lesions** generally produce more profound dysphagia than **unilateral lesions**. Consequently, the clinical presentation will vary depending on the lesion site.

To date, the literature does not provide specific etiological data on **swallowing areflexia**. Therefore, we focus below on how various stroke lesions (cortical, subcortical, brainstem) implicated in dysphagia might disrupt the swallowing reflex. The **cerebral cortex** plays a key role in regulating both voluntary and reflexive phases of swallowing (Teismann et al., 2011; Sasegbon et al., 2024). Thus, both unilateral and bilateral cortical or subcortical strokes commonly lead to dysphagia and may partially or completely abolish the swallowing reflex (Khedr et al., 2021; Qiao et al., 2022).

Epidemiological data show that **60% to 80%** of patients with a **unilateral hemispheric stroke** experience swallowing dysfunction, and about **25%** of those develop **aspiration pneumonia** (Martino et al., 2005). In many cases, a **delay in swallowing reflex initiation** is observed; once the reflex begins, the subsequent pharyngeal phase is often preserved and functional (Martino et al., 2005; Sasegbon & Hamdy, 2024). **Silent aspiration** occurs in roughly 20% to 40% of cases (Martino et al., 2005; Sasegbon & Hamdy, 2024).

Some studies report that dysphagia is more severe when the hemisphere dominant for swallowing is affected (Khedr et al., 2008). For example, patients with **right-sided hemispheric strokes** may demonstrate more marked delays in reflex initiation and higher rates of airway penetration than left-sided strokes (Yang et al., 2015; Sasegbon et al., 2024). Thanks to **neuroplasticity**, the unaffected hemisphere may help compensate, enabling functional recovery in many cases (Macrae & Humbert, 2013; Sasegbon & Hamdy, 2024).

When a stroke is **bilateral** and affects both corticobulbar motor fibers, the condition is referred to as **pseudobulbar syndrome**. This syndrome is characterized by impaired automatic-voluntary pathways, which results in the relative release of **bulbar and**

**pontine reflex activity** from cortical control (Urban et al., 2001; Kumar et al., 2011).

Several swallowing-related disturbances have been described in pseudobulbar palsy. First, **cervico-cephalic motor dysfunction** may compromise the efficiency of the suprahyoid and infrahyoid muscles, impairing laryngeal elevation. Second, patients often present with **facial, buccal, and lingual paralysis**, which may be severe and prevent voluntary mobilization of the corresponding muscles. This in turn disrupts the **oral phase of swallowing**, impairing bolus preparation and propulsion (Ertekin & Aydogdu, 2003).

Additionally, the **velopharyngeal reflex** may be abolished, leading to **nasal regurgitation**. The swallowing reflex itself is frequently **delayed or diminished**, reflecting impaired transmission of sensory input from the tongue base. In some cases, this may manifest clinically as an **absent swallowing reflex**, requiring strong and repeated stimulation to initiate. This **delayed triggering** is a hallmark of pseudobulbar involvement and, when severe, results in **pre-deglutitive aspiration** (Kumar et al., 2011; Ertekin et al., 2000).

Nevertheless, once initiated, the **pharyngeal phase**—although slowed—usually proceeds in a coordinated manner, reflecting a dissociation between preserved automatic reflex activity and impaired voluntary control. Patients may also exhibit compromised **respiratory functions**, particularly impaired **cough** and **apneic regulation**, further increasing the risk of aspiration pneumonia (Dziewas et al., 2007; Warnecke et al., 2009)

In a study evaluating swallowing disorders according to the site of **infratentorial stroke**, Flowers et al. (2011) reported a dysphagia incidence of **0% in cerebellar strokes**, **6% in midbrain strokes**, **43% in pontine strokes**, **40% in medial medullary strokes**, and **57% in lateral medullary strokes**. When stroke involves the **brainstem**, the lesion directly affects the **motor nuclei of the pharynx**, as well as **pyramidal, sensory, and motor pathways**, leading to a high frequency of swallowing impairments. At the acute stage, dysphagia is often **severe and life-threatening** (Martino et al., 2005; Dziewas et al., 2008).

Although **brainstem infarcts** are relatively less common (Saver et al., 2009), they often cause **alternating syndromes**, with cranial nerve involvement ipsilateral to the lesion and long tract (sensory and/or motor) signs contralateral to the lesion. The most frequent clinical presentation is the **lateral medullary infarction (Wallenberg's syndrome)** (Kim et al., 1997; Kumral et al., 2002).

In this condition, lesions involving the **nucleus ambiguus** (cranial nerves IX, X, and XI) result in **hemiparalysis of the soft palate, pharynx, and larynx**, impairing **pharyngeal propulsion** and causing inadequate relaxation of the **upper esophageal sphincter (UES)** (Prosiegel et al., 2005). **Laryngeal elevation and closure** are often markedly reduced or absent (Dziewas et al., 2004). Furthermore, **delayed triggering of the pharyngeal swallow** has been observed in more than 90% of patients with lateral medullary infarction (Horner et al., 1991).

These pathophysiological alterations lead to both **primary and secondary aspirations**. On the contralateral side of the lesion, patients often present with **thermoalgesic sensory loss** (Kim et al., 1997). Consequently, **Wallenberg's syndrome** is associated with **severe dysphagia**, in which aspiration can occur even with **saliva, nasal secretions, and gastric reflux** (Kumral et al., 2002; Prosiegel et al., 2005). In most cases, it necessitates **suspension of oral feeding** and initiation of enteral nutrition.

Thus, **delayed or absent initiation of the swallowing reflex** can occur not only in **unilateral and bilateral hemispheric strokes**, but also in **brainstem lesions**, particularly those involving the medulla.

**Recovery and Consequences**

The presence or persistence of swallowing disorders during the acute post-stroke phase has been associated, in the medium and long term, with poorer functional outcomes, increased risk of institutionalization, and higher mortality (Outcomes of Dysphagia Following Stroke, 2022; Dysphagia and Tube Feeding After Stroke, 2019). In the first two weeks after stroke, about **50% of patients recover swallowing function spontaneously** (Development and Validation of a Prognostic Model, 2019). The majority of recovery occurs during the **first month** following stroke. Beyond that period, oral feeding is at least partially restored in the majority of patients; subsequent improvements tend to be smaller.

Certain factors are associated with poorer reorganization of swallowing function: higher stroke severity (as measured by NIHSS), older age, bilateral lesions, large lesion volume, and brainstem involvement (Development and Validation of a Prognostic Model, 2019; Dysphagia and Tube Feeding After Stroke, 2019). Use of intubation and/or presence of aspiration also predict worse outcomes (Predictors of Complete Oral Intake After Tracheostomy, 2023).

Persistent dysphagia at six months occurs in a minority of patients (approximately **10-18%**) with ischemic stroke in current studies (Development and Validation of a Prognostic Model, 2019). The persistence of these disorders places patients at elevated risk for complications such as **aspiration pneumonia**, **malnutrition**, and **dehydration**, which can impede recovery of physical function (Dietary Intervention for Post-Stroke Dysphagia, 2024; Effect of Malnutrition After Acute Stroke on Clinical Outcome, 1996). Moreover, dysphagia is associated with prolonged hospital stays and increased mortality risk (Dysphagia and Tube Feeding After Stroke, 2019). Early management of post-stroke dysphagia is thus essential.

**Assessment and Rehabilitation of Post-Stroke Swallow Reflex Impairment**

As highlighted in international guidelines, the prevention and management of swallowing disorders are critical components of post-stroke care, as they significantly reduce the risk of complications such as aspiration pneumonia, malnutrition, and increased mortality (Winstein et al., 2016; Hebert et al., 2016). Early detection of dysphagia is therefore considered a standard of care, and structured swallowing assessments are recommended for all patients in the acute

phase of stroke (Gates et al., 2021; Dziewas et al., 2021).

Speech and language therapy has been shown to play a central role in the prognosis of patients with post-stroke dysphagia. Several studies have demonstrated that targeted interventions improve swallowing safety, functional recovery, and overall quality of life (Carnaby et al., 2006; Bath et al., 2018). The evaluation typically includes a clinical swallowing assessment combined, when necessary, with instrumental examinations such as videofluoroscopic swallow study (VFSS) or fiberoptic endoscopic evaluation of swallowing (FEES), which allow for accurate identification of aspiration risks and impaired swallowing physiology (Terré & Mearin, 2012; Dziewas et al., 2021).

Based on these findings, individualized rehabilitation programs are designed, integrating compensatory strategies (e.g., postural adjustments, dietary texture modification) and restorative exercises aimed at improving swallowing biomechanics and sensorimotor control (Crary et al., 2012; Carnaby-Mann & Crary, 2008). International consensus emphasizes that early initiation of speech and language therapy interventions, ideally within the first days after stroke, is associated with better recovery outcomes and reduced morbidity (Bath et al., 2018; Dziewas et al., 2021).

### 1.1 Assessment

The evaluation of swallowing function is a crucial step preceding any rehabilitation intervention. It constitutes the foundation for therapeutic decision-making, enabling the speech-language pathologist (SLP) to identify specific impairments, determine the urgency of intervention, and design an individualized treatment plan through case history, clinical interview, and structured assessments (Gates et al., 2021; Dziewas et al., 2021).

In cases of suspected post-stroke dysphagia, clinical swallowing screening should be conducted as early as possible—ideally within the first 24 hours of hospital admission and before the initiation of oral feeding—to prevent aspiration and related complications (Winstein et al., 2016; Hebert et al., 2016). Early and systematic screening by trained healthcare professionals has been shown to reduce the risk of pneumonia, malnutrition, and mortality (Gershon et al., 2013; Beavan et al., 2010).

When swallowing impairment is confirmed, daily reassessment during the acute phase is strongly recommended to monitor recovery, adjust compensatory strategies, and guide the initiation of rehabilitation (Martino et al., 2005; Carnaby et al., 2006). The comprehensive swallowing assessment aims to establish an analytical and functional profile of the patient's swallowing abilities, evaluate the risks of oral feeding, and assess the nutritional and pulmonary consequences of dysphagia (Terré & Mearin, 2012; Dziewas et al., 2021).

Prior to conducting the evaluation, the SLP should gather relevant information from the patient's medical record and care team, including global medical status, comorbidities, mode of symptom onset, stroke severity, and prognostic indicators. This ensures that the diagnostic process accounts for fatigue, weight loss,

respiratory congestion, and overall clinical stability before progressing to direct swallowing assessment.

**Case History (Anamnesis)**

Conducted by the clinician with the patient, caregivers, and healthcare staff, the clinical interview provides essential information regarding swallowing-related functions, including level of alertness and autonomy, cognitive abilities, phonation and respiratory functions, awareness of deficits, hygiene, and rehabilitative potential (Carnaby et al., 2006; Martino et al., 2005). It also explores both primary and secondary symptoms of swallowing impairment in order to characterize the nature of the disorder, its clinical impact (nutritional status, pulmonary risks, and overall health), and the patient's eating context (Smithard, 2016; Clavé et al., 2012).

In approximately 70–80% of cases, a structured case history can provide a presumptive diagnosis of dysphagia and guide further instrumental evaluation, although it does not allow for precise grading of severity (Martino et al., 2005; Dziewas et al., 2021).

**Clinical Examination**

The next step consists of a clinical examination, whose purpose is to objectively confirm or rule out the swallowing impairments reported during the interview, to identify potential anatomical and neuromuscular abnormalities, to assess functional abilities and learning capacity, and to better understand the pathophysiological mechanisms underlying the disorder (Carnaby & Hankey, 2003; Mann et al., 2000). This analysis relies on an analytical and functional observation of the sensory-motor subsystems involved in swallowing, performed either directly (at rest and during tasks) or indirectly (during phonation, respiration, and swallowing).

Analytical observation at rest focuses on aspects directly related to swallowing, such as head and trunk posture, global muscle tone, oral and dental status, level of alertness, presence of abnormal movements (e.g., tremor, spasticity), facial paralysis, use of non-oral feeding methods (nasogastric tube, gastrostomy), tracheostomy, bronchial congestion, laryngeal edema, and salivary production (Warnecke et al., 2009; Martino et al., 2005).

Dynamic evaluation, on the other hand, investigates motor, sensory, and sensorimotor abilities of the muscles engaged in swallowing. Using voluntary and imitative praxis, the clinician assesses the performance of movements of the mandible, cheeks, lips, tongue, and soft palate, considering their speed, amplitude, strength, and tone. Comprehension difficulties, dissociation between automatic and voluntary movements, initiation, coordination, and motor control are also taken into account (Carnaby et al., 2006).

Additional tactile, thermal, and gustatory stimulations may be applied to explore sensory function (Logemann, 1999; Clavé et al., 2012). Finally, the evaluation of reflexes provides insight into possible compensatory strategies: the gag reflex, which is not always reliable even in healthy individuals; the swallowing reflex, which is most often assessed during actual swallowing tasks rather than through

isolated stimulation; the velopalatal reflex; the cough reflex; and the persistence of primitive reflexes (Ramsey et al., 2003; Dziewas et al., 2021).

**Functional Observation**

Functional observation consists of evaluating the oral and pharyngeal phases of swallowing. Pulse oximetry can be used as a supportive tool, as a drop of ≥2% in oxygen saturation during swallowing has been suggested as a potential indicator of aspiration, although its sensitivity and specificity remain debated (Colodny, 2000; Ramsey et al., 2003). This clinical assessment helps to identify the underlying pathophysiological mechanisms involved.

The clinician first observes dry swallowing (saliva swallowing), both spontaneously and on command (Warnecke et al., 2008). If the patient demonstrates adequate alertness, preserved sensorimotor abilities, the ability to initiate spontaneous or voluntary swallowing, and an intact cough reflex, a food trial may be conducted (Logemann, 1999; Carnaby et al., 2006). However, this trial should not be performed in cases of absent swallowing reflex, as it would place the patient at significant risk.

In bedridden or drowsy patients, the evaluation of the swallowing reflex relies on careful clinical observation of saliva swallowing, often complemented by palpation of the thyroid cartilage to detect laryngeal elevation (Smithard et al., 1998; Daniels et al., 2012).

**Adaptations, Rehabilitation, and Specific Methods**

Information regarding the patient's cognitive abilities and physiological and sensory impairments collected during the initial assessment is used to design an individualized rehabilitation program (González-Fernández et al., 2013). Clinical guidelines emphasize that all patients with dysphagia should benefit from targeted interventions, including food texture modification, postural adjustments, oropharyngeal exercises, and planned swallowing maneuvers supervised by a speech-language pathologist in collaboration with the multidisciplinary team (Hebert et al., 2016; Bath et al., 2018). Early intervention, starting immediately after the initial evaluation, is crucial as it improves medium-term functional outcomes (Carnaby et al., 2006; Warnecke et al., 2014).

The principles of neuroplasticity are highly relevant to swallowing rehabilitation: training may reshape cortical and subcortical representations of swallowing function, whereas lack of activity hinders reorganization and long-term recovery (Hamdy & Cohen, 2016). Furthermore, muscle disuse can lead to structural changes such as loss of mass and strength (Shune & Moon, 2016). Swallowing therapy is therefore indicated for all patients with post-stroke dysphagia, whether hemispheric or brainstem in origin (Miller et al., 2014). However, brainstem structures exhibit less plasticity compared with the cortex, and recovery strongly depends on the specific site and extent of the lesion (Malandraki & Robbins, 2012).

Conventional dysphagia rehabilitation generally includes both compensatory approaches, aimed at reducing symptoms without modifying the underlying

physiology, and restorative approaches, designed to improve swallowing physiology itself (González-Fernández et al., 2013; Bath et al., 2018). The overall goal is to ensure safe and adequate nutrition and hydration while maximizing quality of life and reducing the risk of aspiration pneumonia (Carnaby et al., 2012; Bath et al., 2018). The choice of intervention depends on the therapeutic indications derived from the assessment, the patient's medical condition, prognosis, etiology, and rehabilitative potential (Carnaby et al., 2006; Warnecke et al., 2014).

In this section, we will focus specifically on the management of **absent swallowing reflex (areflexia of swallowing)**, where the primary goal is to stimulate reflex recovery.

**Adaptive Strategies**

These strategies aim to reduce or suppress symptoms when the underlying anatomical and neurological impairments, as well as the associated pathophysiological mechanisms, cannot be corrected (Logemann, 1999; Carnaby et al., 2006). They act during feeding by targeting:

- **The patient's feeding environment**, by eliminating distracting elements that may interfere with swallowing control, and by adapting seating, utensils, and bolus characteristics to the identified physiological deficits.
- **The patient's behavior**, by modifying swallowing techniques (e.g., laryngeal protective maneuvers, clearing swallows) or head positioning strategies (chin-tuck, head rotation) during swallowing (Robbins et al., 2008).

Although these strategies are widely accepted in clinical practice, the evidence supporting their effectiveness remains limited (Bath et al., 2018; Steele et al., 2015). Moreover, in cases of **absent swallowing reflex (areflexia)**, neither postural adjustments nor texture modifications have demonstrated efficacy in restoring the swallowing response, as they do not modify the altered pathophysiological mechanisms nor promote cortical plasticity (Carnaby et al., 2012; Bath et al., 2018).

In addition, such strategies are not always suitable in the acute phase of stroke, as they often require preserved cognitive function and active patient cooperation (Steele et al., 2015).

When areflexia of swallowing persists, **nasogastric tube (NGT) placement** is frequently indicated. It is recommended in cases where dysphagia prevents safe oral feeding during the first days post-stroke (Intercollegiate Stroke Working Party, 2016; Dziewas et al., 2014). However, prolonged NGT use has been associated with maintenance or worsening of swallowing dysfunction, including reduced swallowing reflex sensitivity (Kataoka et al., 2017).

**Specific Methods**

Several stimulation-based approaches aimed at restoring physiological swallowing after stroke have emerged over the past decades (Bath et al., 2018):

- **Peripheral methods**, including *pharyngeal electrical stimulation* (PES) and *neuromuscular electrical stimulation* (NMES), which are

designed to strengthen the swallowing-related muscle groups, enhance sensory input, and facilitate recruitment of alternative neural networks (Fraser et al., 2002; Carnaby et al., 2012).

- **Central stimulation methods**, also referred to as *non-invasive brain stimulation (NIBS)*, such as *transcranial magnetic stimulation (TMS)* and *transcranial direct current stimulation (tDCS)*. These techniques modulate cortical excitability in swallowing-related areas, either ipsilateral or contralateral to the lesion, by increasing or decreasing neuronal excitability thresholds (Khedr et al., 2009; Jefferson et al., 2009).
- **Combined approaches**, such as *paired associative stimulation (PAS)*, which couples peripheral stimulation of the target muscle with stimulation of its contralesional cortical motor representation. This combined input is thought to promote Hebbian-like plasticity, thereby enhancing cortical reorganization of swallowing networks (Jayasekeran et al., 2010).

These methods are hypothesized to act directly on **neuroplasticity**, thereby contributing to swallowing recovery when combined with conventional therapy (González-Fernández et al., 2013).

However, the **quality of evidence** supporting these techniques in post-stroke dysphagia remains limited (Bath et al., 2018; Pisegna et al., 2016). With respect to **swallowing reflex abolition (areflexia)**, findings are inconsistent: while some studies report improvements in laryngeal elevation, others raise concerns about increased aspiration risk (Suntrup-Krueger et al., 2015).

Given that their clinical efficacy is not yet fully validated, attention has recently shifted toward **manual therapeutic techniques**, which are rapidly developing in speech-language pathology and may be applied to post-stroke dysphagia.

## 2. Manual Therapy

### 2.1 General Principles

Manual therapy, often defined as *"the art of healing through the hands"*, is grounded in a precise diagnostic process and in the fundamental osteopathic principles stating that the body is a functional and biological unit with inherent self-regulatory and defensive mechanisms, and that structure and function are closely interrelated through the concept of mobility (van Dun & Perquin, 2014; Licciardone, 2011).

As discussed previously, and according to established neurophysiological models, two main neural pathways are distinguished: the **afferent (sensory) pathway**, which conveys information from peripheral receptors to the central nervous system (CNS), and the **efferent (motor) pathway**, which transmits neural signals from the CNS to peripheral effectors. Depending on the pathway involved, the therapeutic approach and expected clinical outcomes may differ (Brodal, 2016; Kandel et al., 2021).

Indeed, conventional techniques, such as praxis-based approaches, which follow a centrifugal pathway (efferent route), aim to enhance awareness through verbal

instruction in order to coordinate the motor response. This type of intervention primarily targets voluntary, intentional, and conscious movements that determine function (Kandel et al., 2021).

In contrast, manual techniques, which adopt a centripetal pathway (afferent route), allow for the modulation of ascending sensory input and, consequently, improve physiological function either immediately or after a period of neural integration (Brodal, 2016). These approaches stimulate an increase in afferent proprioceptive input, thereby fostering neuromuscular re-education and sensorimotor integration (Beinert & Taube, 2013).

Both types of approaches are complementary. Through action on sensory, muscular, articular, and cutaneous receptors, which contributes to the regulation of unconscious activities, manual therapy techniques enhance traditional rehabilitative strategies that primarily target voluntary motor activities (Licciardone, 2011; van Dun & Perquin, 2014).

### 2.3 Contributions of Manual Therapy for Swallowing

As previously discussed, swallowing is a complex function involving multiple structures. Conventional post-stroke dysphagia rehabilitation generally relies on environmental adaptations, behavioral modifications, and specific analytical and functional exercises. Most of these approaches require voluntary control from the patient and may not be suitable for swallowing areflexia (Bath et al., 2018).

In contrast, manual therapy techniques may allow for direct stimulation of the impaired structure, thereby activating reflexive or adaptive responses, as well as working on muscular tone (Beinert & Taube, 2013).

Laryngeal manual therapy focuses on therapeutic manipulations of the laryngeal and peri-laryngeal structures (Mathieson et al., 2009; Van Houtte et al., 2011). Within dysphagia management, its goals include reducing mechanical restrictions of the structures involved in swallowing, enhancing proprioceptive feedback (Aranha et al., 2019), restoring motor imagery through neurosensory reprogramming, and preventing loss of body schema (Behrman et al., 2008).

These interventions aim to normalize mobility deficits using appropriate manual techniques, optimize kinesthetic and proprioceptive sensations through tactile input to guide movement, and assist or substitute movement in cases of structural immobility (Van Houtte et al., 2011; Watts et al., 2015).

According to Miller (2008), muscular pressure or cartilaginous displacement at the level of the larynx can trigger the swallowing reflex. Indeed, oropharyngeal receptors are highly sensitive to contact, pressure, and stretch (Jean, 2001; Lang, 2009). Certain maneuvers, such as external manual stimulation of the suprahyoid muscles, can facilitate the initiation of the swallowing reflex (Shaker et al., 2002; Wheeler-Hegland et al., 2009).

When the floor-of-mouth musculature is stimulated by manual pressure, this action—due to its attachment to the posterior aspect of the hyoid bone—elevates the floor of the mouth and

consequently raises the larynx, promoting the triggering of the swallow.

Other passive mobilization techniques have also been described to stimulate swallowing reflex activity. For example, transverse mobilization of the laryngeal cartilages, trachea, and hyoid bone, achieved by applying bidigital pressure on both sides of these structures, has been reported to facilitate reflexive responses. Another technique consists of manually reproducing laryngeal elevation by applying upward pressure with the index fingers at the superior border of the thyroid cartilage and downward counterpressure with the thumbs at the inferior border of the cricoid cartilage (Shaker et al., 2002; Carnaby-Mann & Crary, 2008).

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