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Pathophysiology of Post-Stroke Dysphagia

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Abstract

Background— Post-stroke dysphagia (PSD) is impaired swallowing after ischemic or hemorrhagic stroke. Reported prevalence varies widely because studies differ in timing, screening method, stroke severity, lesion location, and whether dysphagia is identified clinically or instrumentally. Contemporary evidence supports PSD as a disorder of a distributed swallowing network rather than a simple problem of throat muscle weakness.

Objective— To provide a detailed, mechanism-based review of the pathophysiology of PSD, emphasizing how lesions in cortical, subcortical, cerebellar, brainstem, sensory, respiratory, and cranial nerve systems produce specific swallowing phenotypes and complications.

Methods— This narrative topic review synthesizes major guideline statements, neurophysiological studies, neuroimaging evidence, and recent reviews of post-stroke swallowing assessment and treatment. The review focuses on normal swallowing control, lesion-location effects, sensory-motor mechanisms, aspiration and pneumonia pathways, recovery and neuroplasticity, and implications for bedside screening, instrumental assessment, and rehabilitation planning.

Neuro-physiological basis— Normal swallowing requires precisely timed coordination of voluntary and reflexive events. The oral phase depends on cortical and corticobulbar control of the lips, jaw, tongue, and buccal musculature. The pharyngeal phase depends on rapid integration of sensory input with patterned motor output through the medullary swallowing central pattern generator (CPG), especially the nucleus tractus solitarius, nucleus ambiguus, adjacent reticular formation, and vagal, glossopharyngeal, trigeminal, facial, and hypoglossal pathways. Supra-medullary regions, including the primary motor and sensory cortices, premotor and supplementary motor areas, insula, anterior cingulate cortex, basal ganglia, thalamus, cerebellum, and connecting white matter tracts, modulate initiation, timing, coordination, attention, and adaptation.

Key pathophysiological mechanisms— Hemispheric strokes may impair bolus preparation, oral transit, swallow initiation, sensorimotor integration, and descending corticobulbar drive. Subcortical and white matter lesions may disconnect cortical swallowing areas from brainstem effectors. Brainstem strokes, particularly medullary lesions, may directly unpair the swallowing CPG and cranial nerve nuclei, producing severe pharyngeal weakness, absent or delayed swallow triggering, impaired laryngeal closure, reduced upper esophageal sphincter opening, and aspiration. Sensory impairment is central because reduced pharyngeal or laryngeal sensation can cause delayed swallow initiation, poor secretion management, blunted cough, and silent aspiration. Impaired respiratory-swallow coordination and weak cough further reduce airway clearance after aspiration.

Clinical implications— The clinical phenotype of PSD may include drooling, oral residue, pocketing, delayed swallow initiation, coughing or choking, wet voice, pharyngeal residue, recurrent fever or chest infection, dehydration, malnutrition, and silent aspiration. Because bedside observation may miss silent aspiration, screening should occur before oral intake and be followed by specialist assessment when abnormal. Fiberoptic endoscopic evaluation of swallowing (FEES) and video fluoroscopic swallow study (VFSS) help define the underlying physiology, including airway invasion, secretion burden, pharyngeal residue, timing, sensation, and upper esophageal sphincter function.

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Conclusions— PSD results from disruption of integrated neural systems that normally coordinate safe and efficient swallowing. Mechanism-based assessment is essential because similar symptoms can arise from different combinations of sensory loss, motor weakness, impaired timing, cognitive inattention, brainstem pattern-generator injury, respiratory compromise, or reduced cough. Early screening, instrumental evaluation when indicated, nutritional support, oral hygiene, compensatory strategies, rehabilitative exercise, and selective neuromodulatory approaches are best understood as interventions aimed at specific points in the pathophysiological cascade.

Keywords— post-stroke dysphagia, swallowing physiology; aspiration, silent aspiration, stroke-associated pneumonia, brainstem stroke, FEES, VFSS, stroke rehabilitation.

1. INTRODUCTION

Post-stroke dysphagia (PSD) is one of the most common and consequential complications after stroke. It occurs when a stroke disrupts the neural systems that coordinate bolus formation, oral propulsion, swallow triggering, pharyngeal clearance, airway closure, upper esophageal sphincter opening, and the timing between swallowing and breathing. PSD is clinically important because it can convert a neurological injury into a multisystem problem involving aspiration, pneumonia, malnutrition, dehydration, medication administration difficulty, prolonged hospitalization, and reduced participation in rehabilitation.

The reported frequency of PSD depends strongly on how and when it is measured. Screening tools identify risk, clinical examinations identify signs and functional limitations, and instrumental tests such as fiberoptic endoscopic evaluation of swallowing (FEES) or video fluoroscopic swallow study (VFSS) identify airway invasion and biomechanical impairment. The European Stroke Organization and European Society for Swallowing Disorders guideline notes that PSD is present in more than half of acute stroke patients in many clinical cohorts and is associated with aspiration pneumonia, malnutrition, dehydration, poor outcome, and mortality.[1] A 2024 meta-analysis estimated an overall prevalence of 46.6%, with the assessment method being a major source of variation.[2] Earlier systematic reviews similarly showed wide variation in reported PSD frequency by assessment method and linked dysphagia and aspiration with pulmonary complications after stroke.[3] More recent meta-analytic data further confirm that PSD is associated with increased risk of pneumonia and mortality in acute stroke patients.[4]

A detailed understanding of pathophysiology matters because dysphagia management is not simply a pass-or-fail decision about oral intake. A patient who aspirates because of delayed swallow initiation may need a different treatment emphasis than a patient who aspirates because of weak pharyngeal contraction, impaired laryngeal vestibule closure, poor sensation, severe residue, or reduced cough. The goal of assessment is therefore to identify the mechanism of impaired swallowing safety and efficiency and to match treatment to that mechanism.

2. Normal Swallowing Physiology and Neural Control

Swallowing is a rapid sensorimotor behavior that must be coordinated with breathing. Although it is often divided into phases, these phases overlap and depend on continuous sensory feedback. The oral preparatory phase includes mastication, saliva mixing, bolus formation, and containment. The oral transit phase propels the bolus posteriorly using tongue-palate pressure. The pharyngeal phase requires near-simultaneous soft palate elevation, tongue base retraction, hyolaryngeal elevation and anterior excursion, laryngeal vestibule closure, vocal fold closure, pharyngeal contraction, epiglottic inversion, and upper esophageal sphincter opening. The esophageal phase then carries the bolus to the stomach. Distributed neural control of swallowing has been illustrated in Figure 1.

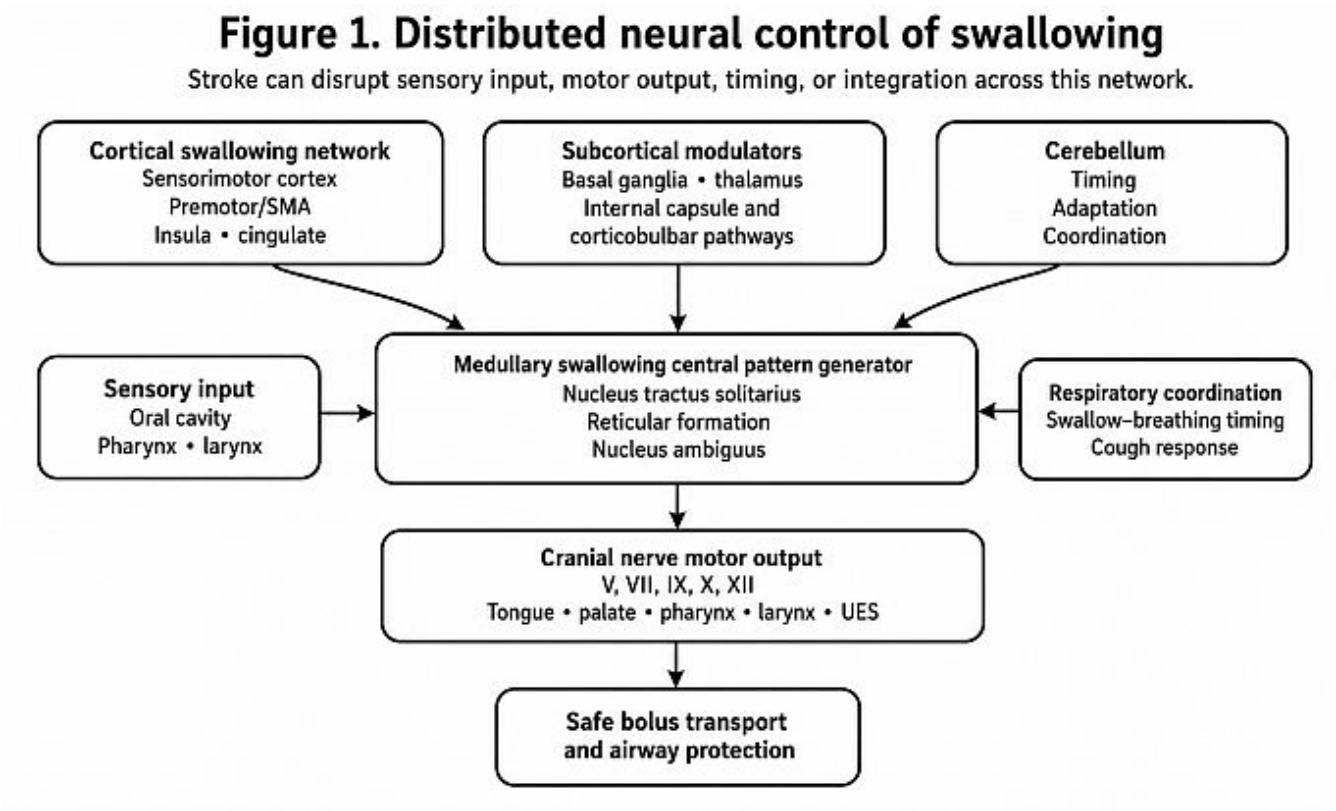
Table 1 shows the major neural structures involved in swallowing control. At the brainstem level, swallowing is organized by a medullary central pattern generator (CPG). Sensory input from the oropharynx, larynx, and upper esophagus reaches the nucleus tractus solitarius through cranial nerve pathways, especially glossopharyngeal and vagal afferents. This information is integrated with inter-neuronal circuits in the reticular formation and translated into motor output through the nucleus ambiguus, dorsal motor nucleus of the vagus, hypoglossal nucleus, trigeminal motor nucleus, and facial motor nucleus. This network generates the stereotyped but adaptable sequence of pharyngeal swallowing.

Swallowing is also under extensive supra-medullary influence. Functional and lesion studies implicate the primary motor cortex, primary somatosensory cortex, premotor cortex, supplementary motor area, insular cortex, anterior cingulate cortex, opercular regions, basal ganglia, thalamus, cerebellum, and white matter projections. These areas help initiate voluntary swallowing, modulate timing and force, process taste and somatosensory feedback, support attention and salience, and adapt motor output to bolus volume, viscosity, temperature, and patient posture. The swallowing network is bilateral but often asymmetric, which helps explain why unilateral stroke may or may not cause clinically significant dysphagia depending on the dominant swallowing representation and the reserve of the unaffected hemisphere.[5–7]

3. Core Pathophysiology of Post-Stroke Dysphagia

The central pathophysiological concept is network failure. The pathophysiology has been illustrated in Figure 2. Stroke may damage one node of the swallowing network, interrupt communication between nodes, reduce sensory input, reduce motor output, disturb timing, or impair arousal and attention. These mechanisms often coexist. For example, a patient with a large hemispheric stroke may have weak tongue propulsion, poor attention to the bolus, delayed pharyngeal initiation, and

FIGURE 1: Distributed neural control of swallowing. The safe swallow emerges from coordinated cortical, subcortical, cerebellar, brainstem, sensory, respiratory, and cranial nerve systems rather than from a single isolated center.



Abbreviations: SMA, supplementary motor area; UES, Upper esophageal sphincter.

TABLE 1: Major neural structures involved in swallowing control.

Structure or pathway	Main role in swallowing	Pathophysiological effect if disrupted
Primary motor cortex and corticobulbar tracts	Voluntary oral control, tongue propulsion, descending drive to cranial nerve motor nuclei.	Poor bolus control, weak tongue movement, delayed initiation, impaired oral propulsion, reduced pharyngeal drive.
Primary somatosensory cortex and sensory pathways	Bolus awareness, oral and pharyngeal feedback, adaptive modulation of timing and pressure.	Reduced awareness of residue or airway invasion, delayed swallow trigger, silent aspiration risk.
Insula and frontal operculum	Integration of taste, visceral sensation, salience, motor planning, and autonomic components of swallowing.	Disorganized swallow initiation, impaired sensorimotor integration, aspiration risk, poor coordination.
Basal ganglia and thalamus	Timing, gating, relay of sensorimotor information, scaling of motor patterns.	Bradykinetic or poorly timed swallow, delayed swallow initiation, reduced coordination.
Cerebellum	Coordination, timing, error correction, motor learning, adaptation to bolus properties.	Ataxic or poorly coordinated swallowing, variable timing, inefficient clearance.
Nucleus tractus solitarius	Major sensory integration hub of the medullary swallowing network.	Delayed or absent swallow triggering, impaired secretion response, poor sensory-motor coupling.
Nucleus ambiguus and cranial nerve motor nuclei	Motor output to pharynx, larynx, palate, and upper esophagus.	Weak pharyngeal contraction, impaired laryngeal closure, vocal fold dysfunction, aspiration.
Respiratory-swallow network and cough pathways	Coordinates swallow apnea, exhalation after swallow, cough initiation, and airway clearance.	Aspiration becomes harder to clear; weak cough and abnormal breathing-swallow timing increase pneumonia risk.

impaired cough. A patient with lateral medullary infarction may have preserved cognition and oral control but severe pharyngeal weakness, absent swallow triggering, vocal fold paresis, secretion pooling, and profound aspiration.

The clinical signs of PSD are produced by two broad categories of impairment: impaired swallowing safety and impaired swallowing efficiency. Safety refers to keeping food, liquid, saliva, and refluxate out of the airway. Efficiency refers to moving the bolus through the oral cavity and pharynx with minimal residue and adequate nutritional intake. A patient can be unsafe, inefficient, or both. For example, thin-liquid aspiration reflects impaired safety, whereas large vallecular or pyriform sinus residue reflects impaired efficiency and can secondarily become unsafe if residue spills into the airway after the swallow.

PSD evolves over time. In the acute stage, dysphagia may reflect direct tissue injury, edema, diaschisis, impaired alertness, fatigue, poor posture, respiratory compromise, and reduced attention. Some patients improve as these reversible factors resolve. Persistent dysphagia reflects more durable injury to sensory-motor networks, corticobulbar pathways, medullary circuits, or cranial nerve outflow, as well as secondary factors such as disuse, sarcopenia, poor oral hygiene, reduced endurance, and inadequate compensatory cortical reorganization.

4. Lesion-Location Mechanisms

4.1 Hemispheric cortical lesions

Cortical lesions can disrupt voluntary oral control, swallow initiation, sensory awareness, motor planning, and descending corticobulbar drive. Table 2 summarizes the lesion location and expected dysphagia phenotype, while Figure 3 illustrates mechanism. Primary motor cortex lesions may reduce tongue, lip, jaw, and pharyngeal motor output. Somatosensory cortex lesions may reduce awareness of bolus position and residue. Insular and opercular lesions may disturb the integration of taste, visceral sensation, salience, and motor planning. Anterior cingulate and supplementary motor areas may affect initiation, attention, and motivational aspects of swallowing.

The effect of unilateral hemispheric stroke depends on the bilateral but asymmetric representation of swallowing. In many individuals, one hemisphere has stronger pharyngeal motor representation. If the stroke injures the more dominant hemisphere for swallowing, dysphagia is more likely. Recovery can occur when the unaffected hemisphere expands or strengthens its swallowing representation, a mechanism supported by classic transcranial magnetic stimulation studies.[6,7]

4.2 Subcortical and white matter lesions

Subcortical stroke may produce dysphagia by damaging the internal capsule, corona radiata, basal ganglia, thalamus, or connecting white matter. These lesions may disconnect cortical swallowing regions from brainstem motor nuclei even when cortical swallowing centers and brainstem nuclei are structurally intact. Internal capsule and corona radiata involvement is especially important because corticobulbar fibers are compactly organized. Basal ganglia and thalamic lesions may impair timing, sensorimotor gating, attention, and motor scaling, leading to delayed initiation or poorly coordinated swallow patterns.

4.3 Brainstem lesions

Brainstem lesions often cause severe PSD because they can directly injure the medullary swallowing CPG, cranial nerve nuclei, and the afferent and efferent pathways required for the pharyngeal swallow. Lateral medullary infarction is a classic cause of profound dysphagia because it can involve the nucleus ambiguus, nucleus tractus solitarius, reticular formation, vestibular connections, and descending autonomic and respiratory-swallow pathways. Clinically, this may produce delayed or absent swallow triggering, impaired vocal fold closure, reduced pharyngeal contraction, severe pyriform sinus residue, cricopharyngeal dysfunction, nasal regurgitation, secretion pooling, and aspiration. Reviews of lateral medullary syndrome emphasize that dysphagia in this setting can be severe, prolonged, and mechanistically related to medullary swallowing network and cranial nerve pathway involvement.[8]

The severity of brainstem PSD also reflects the density of essential swallowing structures in a small anatomical space. A relatively small medullary lesion can disrupt sensory input, motor output, and respiratory-swallow coordination simultaneously. This differs from many cortical lesions, where bilateral representation and compensatory reorganization may provide greater redundancy.

4.4 Cerebellar lesions

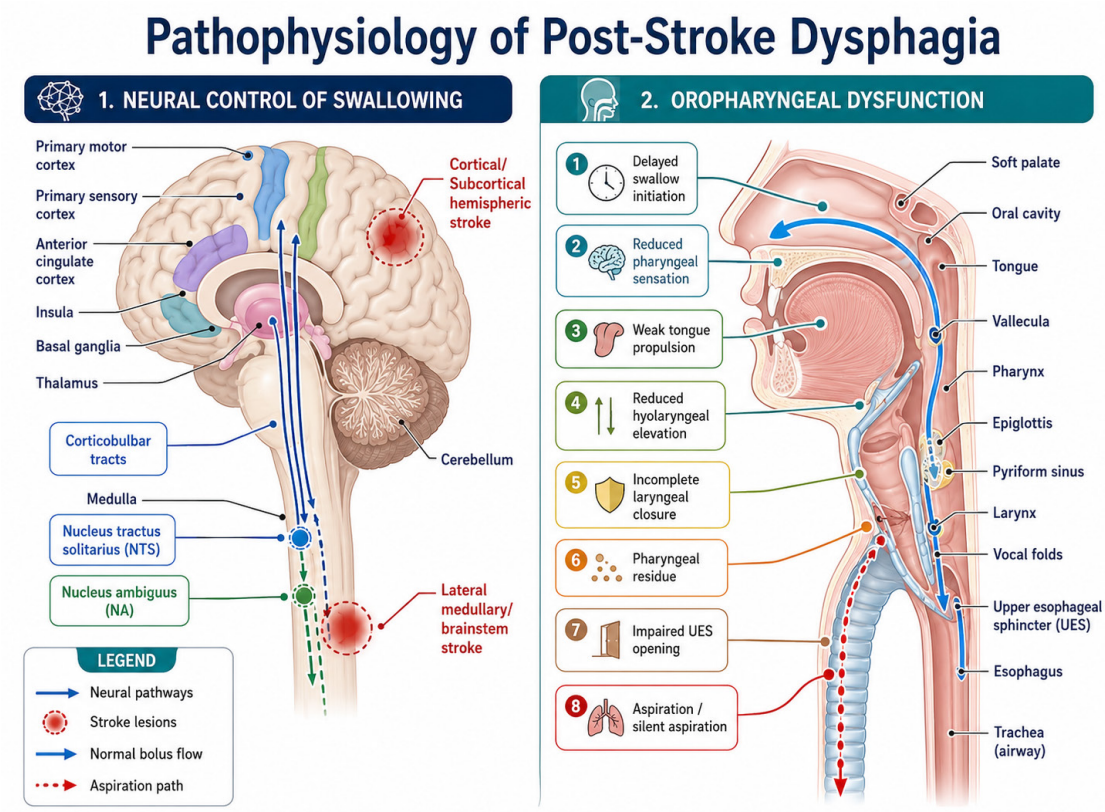
The cerebellum contributes to timing, coordination, error correction, and adaptation. Cerebellar lesions may produce variable swallow timing, reduced coordination between pharyngeal contraction and upper esophageal sphincter opening, and impaired adaptation to changes in bolus size or viscosity. Dysphagia can also arise from network disconnection: a lesion may not destroy the brainstem swallowing generator but may isolate it from cortical, sensory, or cerebellar modulation. This explains why neuroimaging studies identify multiple cortical and subcortical regions rather than one single lesion site for PSD.[9,10]

5. Physiological Swallowing Impairments Produced by Stroke

5.1 Oral-phase impairment

Oral-phase dysphagia results from impaired lip seal, facial weakness, reduced jaw control, impaired mastication, reduced tongue strength, impaired tongue-palate contact, oral apraxia, poor attention, or sensory loss. The bolus may be poorly formed, spill anteriorly, remain pocketed in the buccal sulcus, or fall prematurely into the pharynx before the swallow is triggered. Premature spillage is especially dangerous with thin liquids because material can reach the airway before laryngeal closure is complete.

FIGURE 2: Pathophysiology of post-stroke dysphagia.



Abbreviations: NTS, Nucleus tractus solitarius; NA, Nucleus ambiguus; UES, Upper esophageal sphincter.

TABLE 2: Lesion location and expected dysphagia phenotype.

Lesion pattern	Common physiological impairments	Typical clinical clues
Frontal motor or premotor cortex	Poor voluntary oral control, reduced tongue propulsion, impaired initiation, weak oral-to-pharyngeal transition.	Pocketing, oral residue, prolonged oral transit, need for repeated cues.
Insula or frontal operculum	Impaired sensorimotor integration, poor swallow planning, delayed initiation, reduced coordination.	Disproportionate aspiration or residue relative to limb weakness; poor timing.
Postcentral or sensory pathway lesion	Reduced bolus awareness, impaired pharyngeal sensation, delayed swallow trigger.	Silent aspiration, residue without awareness, poor secretion management.
Internal capsule/corona radiata	Interrupted corticobulbar drive, weakness or poor coordination of oral and pharyngeal musculature.	Dysarthria, facial weakness, tongue weakness, aspiration risk.
Basal ganglia or thalamus	Impaired gating, timing, and sensorimotor relay; delayed or bradykinetic swallow.	Slowed oral transit, delayed trigger, variable coordination.
Lateral medulla or pontomedullary region	CPG disruption, cranial nerve nucleus injury, pharyngeal weakness, impaired laryngeal closure, UES dysfunction.	Severe pharyngeal residue, secretion pooling, vocal fold paresis, aspiration, need for alternative nutrition.
Cerebellum	Impaired timing and coordination; reduced adaptation to bolus characteristics.	Variable performance, poor coordination, inefficient clearance despite preserved strength.
Bilateral, large, or multifocal stroke	Combined oral, pharyngeal, sensory, cognitive, respiratory, and motor impairment.	High aspiration risk, poor endurance, malnutrition, prolonged NPO or tube feeding need.

Abbreviations: CPG, Central pattern generator; UES, Upper esophageal sphincter; NPO, "nil per os" translates to "nothing by mouth". High aspiration risk, poor endurance, malnutrition, prolonged NPO or tube feeding need.

5.2 Delayed or absent swallow initiation

Delayed swallow initiation is one of the most important mechanisms of aspiration. It may arise from cortical initiation failure, reduced pharyngeal sensation, brainstem sensory integration failure, reduced alertness, or impaired attention. When the swallow is delayed, the bolus may pool in the valleculae, pyriform sinuses, or laryngeal vestibule before airway closure. If sensation is impaired, the patient may not cough or may cough too late.

5.3 Sensory impairment and silent aspiration

Sensory impairment is a central mechanism of PSD. Normal swallowing depends on sensory input about bolus volume, texture, temperature, location, and airway invasion. Reduced pharyngeal or laryngeal sensation can delay triggering, reduce the intensity of the swallow response, impair secretion management, and blunt cough. Labeit and colleagues found that pharyngeal hypesthesia was associated with greater dysphagia severity, impaired secretion management, and delayed or absent swallow reflex in acute stroke patients.[11]

Silent aspiration occurs when material enters the airway without an effective outward sign such as coughing. It is particularly dangerous because bedside observation may falsely suggest safe swallowing. Silent aspiration can reflect impaired laryngeal sensation, reduced cough drive, reduced consciousness, weak expiratory muscles, or impaired coordination between airway invasion and protective response. For this reason, an apparently calm bedside swallow does not exclude aspiration in high-risk stroke patients.

5.4 Pharyngeal weakness and residue

Weak tongue-base retraction and pharyngeal constriction reduce bolus clearance. Residue may remain in the valleculae, along the posterior pharyngeal wall, or in the pyriform sinuses. Residue indicates impaired swallowing efficiency and can lead to post-swallow aspiration when material spills into the laryngeal vestibule after the airway has reopened. Residue burden is influenced by pharyngeal strength, sensation, upper esophageal sphincter (UES) opening, bolus viscosity, fatigue, and the ability to perform repeated clearing swallows.

5.5 Impaired laryngeal vestibule closure and vocal fold protection

Airway protection depends on several overlapping events: true vocal fold closure, arytenoid approximation, false vocal fold closure, epiglottic inversion, hyolaryngeal elevation, and closure of the laryngeal vestibule. Stroke may impair these events through corticobulbar weakness, brainstem cranial nerve injury, poor timing, reduced sensation, or reduced hyolaryngeal excursion. Aspiration during the swallow is especially likely when laryngeal vestibule closure is delayed, incomplete, or too brief relative to bolus flow.

5.6 Upper esophageal sphincter dysfunction

UES opening depends on cricopharyngeal relaxation, hyolaryngeal traction, bolus pressure, and pharyngeal contraction. Stroke can impair UES opening by disrupting brainstem pattern, reducing hyolaryngeal excursion, weakening pharyngeal propulsion, or disturbing timing. Clinically, this may appear as pyriform sinus residue, repeated swallows, regurgitation into the pharynx, sensation of food sticking, and aspiration after the swallow.

5.7 Respiratory-swallow coordination and cough impairment

Swallowing normally occurs during a brief pause in breathing and is often followed by exhalation, which helps move residual material away from the airway. Stroke can disturb this timing, especially when there is brainstem involvement, reduced alertness, respiratory disease, weak cough, or poor trunk control. Aspiration becomes more dangerous when the patient cannot generate an effective cough or when aspiration is followed by inhalation rather than exhalation. Table 3 summarizes the physiological swallowing impairments produced by stroke.

6. Why Dysphagia Leads to Complications

Aspiration pneumonia is not caused by aspiration alone. Many healthy adults microaspirate without developing pneumonia. In stroke, pneumonia risk rises when aspiration is combined with impaired cough, reduced laryngeal sensation, poor oral hygiene, pathogenic oral colonization, reduced mobility, reduced consciousness, immune changes, poor nutrition, and comorbid lung disease. This multifactorial model is consistent with evidence showing that dysphagia and aspiration increase the risk of pulmonary complications after stroke, while PSD is also associated with increased pneumonia and mortality risk.[3,4] This explains why prevention requires more than diet modification; oral hygiene, positioning, mobilization, hydration, nutrition, and respiratory care all matter.

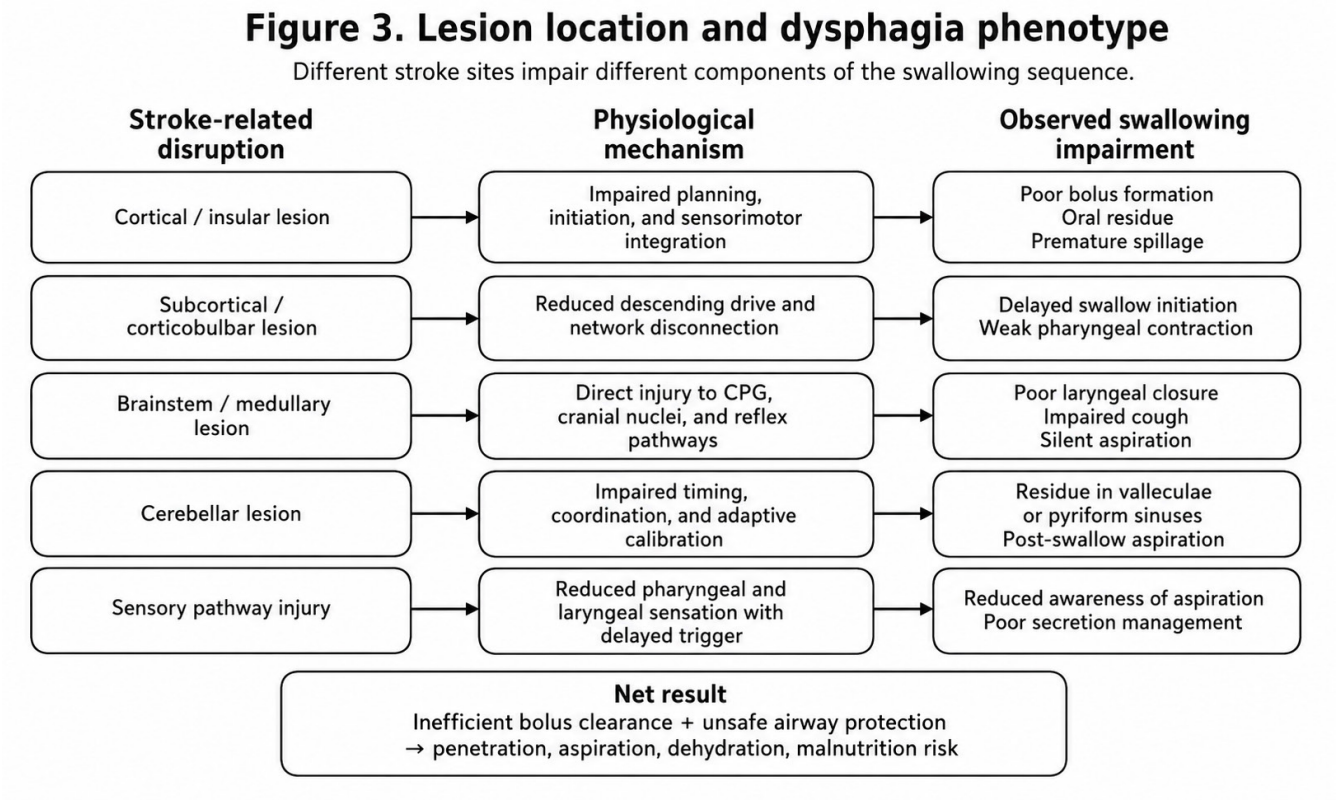
Malnutrition and dehydration arise when patients cannot safely or efficiently meet intake needs. Thickened liquids and modified diets may reduce airway invasion in selected patients, but they can also reduce intake and increase residue if not matched to the swallowing physiology. Medication administration can also be affected because pills may lodge in the pharynx, be aspirated, or require alteration of formulation. Therefore, swallowing plans should include food, liquid, medication, nutrition, hydration, and oral care considerations.

PSD also affects quality of life. Eating is social, cultural, and emotional. Fear of choking, prolonged mealtimes, restrictions on preferred foods, tube feeding, and recurrent respiratory infections may contribute to anxiety, depression, isolation, and reduced rehabilitation engagement. These effects are clinically relevant because adequate nutrition, hydration, and participation support neurological recovery.

7. Recovery and Neuroplasticity

Many patients improve from the first days to weeks after stroke. Early recovery may reflect resolution of edema, improved arousal, restoration of perfusion around the lesion, reduction in functional diaschisis, improved posture and respiratory status, and spontaneous neural reorganization. Later recovery is more dependent on neuroplasticity, practice-dependent learning, strengthening of residual pathways, compensatory recruitment of the unaffected hemisphere, and adaptation of sensory-motor control.

FIGURE 3: Lesion location and dysphagia phenotype.



Abbreviations: CPG, central pattern generator.

TABLE 3: Physiological impairment, assessment target, and management focus.

Impairment	How it may appear clinically or instrumentally	Mechanism-based management focus
Poor oral control	Drooling, pocketing, prolonged chewing, oral residue, premature spillage.	Positioning, cueing, bolus-size control, oral motor and tongue exercises, diet texture adjustment.
Delayed swallow initiation	Bolus pooling before swallow, aspiration before airway closure, delayed laryngeal elevation.	Sensory enhancement when appropriate, pacing, smaller boluses, postural adjustments, targeted swallow timing strategies.
Reduced pharyngeal sensation	Silent aspiration, poor secretion response, absent awareness of residue.	Instrumental assessment, secretion management, oral hygiene, sensory stimulation approaches, close monitoring.
Weak pharyngeal contraction	Vallecular or pyriform sinus residue, multiple swallows, post-swallow aspiration.	Effortful swallow, pharyngeal strengthening tasks, texture modification based on assessment, repeated clearance swallows.
Impaired laryngeal closure	Penetration or aspiration during the swallow, wet voice, cough after swallow.	Airway-protection maneuvers when cognitively feasible, bolus control, posture, rehabilitative airway-closure exercises.
Reduced UES opening	Pyriform sinus residue, post-swallow aspiration, food sticking sensation.	Assess hyolaryngeal excursion and UES opening; consider targeted exercises and specialist management.
Weak cough or respiratory compromise	Unable to clear aspirate, recurrent chest symptoms, fatigue during meals.	Respiratory support, cough-strengthening approaches, meal pacing, upright positioning, oral hygiene, medical optimization.

Abbreviations: UES, Upper esophageal sphincter.

Classic neurophysiology studies showed that recovery of swallowing after unilateral hemispheric stroke is related to increased pharyngeal representation in the unaffected hemisphere.[7] This finding supports current rehabilitation logic: repeated, task-specific swallowing practice and selected neurostimulation strategies aim to enhance excitability and functional connectivity in surviving swallowing networks. However, recovery potential varies with lesion location, lesion size, bilateral involvement, brainstem injury, baseline frailty, cognition, comorbid disease, sarcopenia, and ability to participate in therapy.

Persistent PSD should not be assumed to be static. Swallowing may worsen with fatigue, infection, medication effects, dehydration, reduced alertness, or poor positioning, and it may improve with rehabilitation, medical stabilization, and neuroplastic adaptation. Reassessment is therefore essential, especially when diet is changed, respiratory status changes, or the patient transitions from acute care to rehabilitation or community care.

8. Assessment Implications

Guidelines consistently emphasize early screening because the first oral intake after stroke may occur before dysphagia is obvious. The 2026 American Heart Association/American Stroke Association acute ischemic stroke guideline recommends bedside swallow screening before initiation of liquid or food intake.[12] Canadian Stroke Best Practices recommend screening before any oral intake, including medications, by an appropriately trained professional using a valid screening tool, and recommend referral for comprehensive assessment when screening fails.[13] Delays in dysphagia screening and specialist swallowing assessment after acute stroke have been associated with higher risk of stroke-associated pneumonia, supporting rapid screening pathways and timely referral when screening is abnormal.[14]

A swallow screen is not the same as a full dysphagia assessment. Screening answers a narrow safety question: is the patient likely safe to begin oral intake now? Bedside tests remain useful early tools, but their diagnostic accuracy varies and they may not reliably exclude aspiration, particularly silent aspiration.[15,16] A clinical swallowing assessment describes oral motor function, secretion management, voice, cough, cognition, posture, trial swallows, and functional feeding. Instrumental assessment identifies the physiology behind impairment and is particularly important when aspiration is suspected, pneumonia occurs, findings are inconsistent, or silent aspiration is possible.

FEES provides direct endoscopic visualization of secretions, laryngeal anatomy, pharyngeal residue, penetration, aspiration before and after the swallow, vocal fold movement, and sensory response. It can be performed at bedside and repeated without radiation. VFSS provides dynamic imaging of oral, pharyngeal, and upper esophageal bolus flow, timing of airway closure, hyoid and laryngeal movement, bolus residue, penetration-aspiration events, and UES opening. FEES and VFSS are complementary; the best choice depends on the clinical question, patient stability, local expertise, and availability.

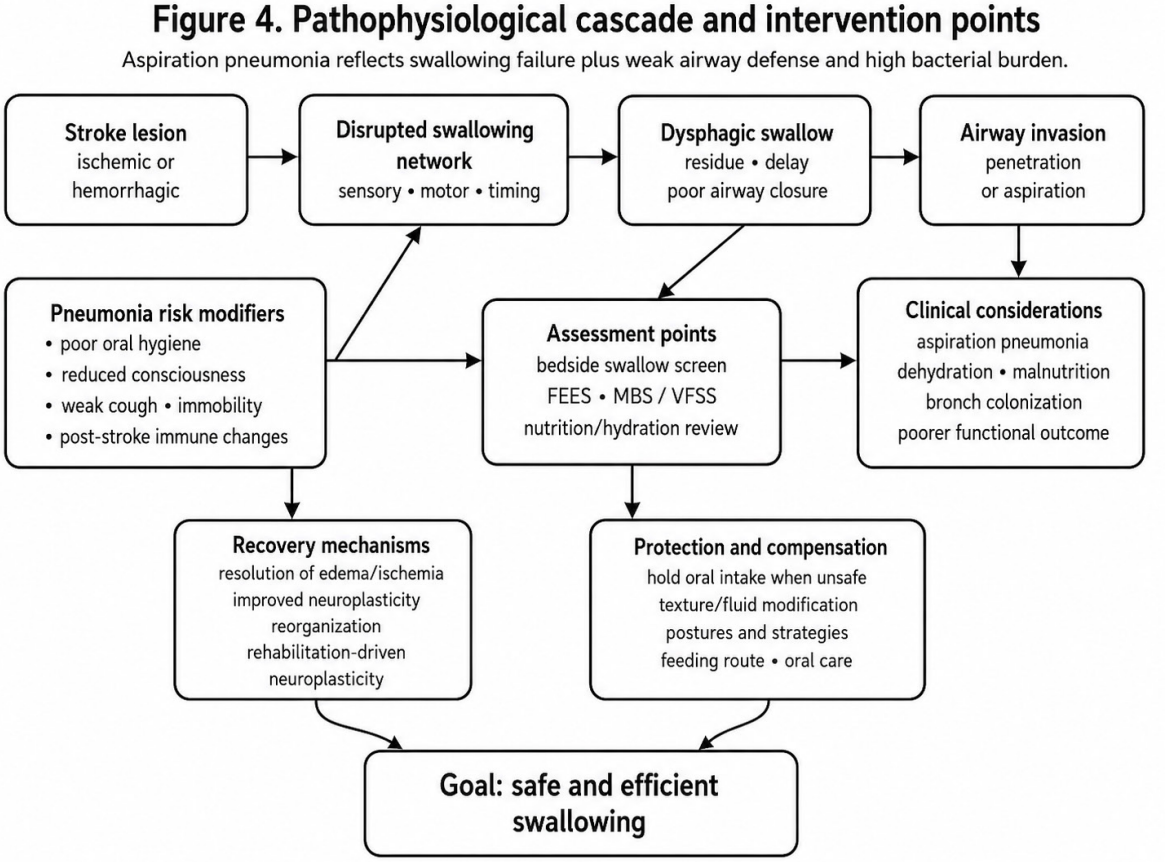
9. Management Linked to Pathophysiology

Effective management begins with identifying the dominant mechanism of impairment. A patient with sensory loss and silent aspiration may need instrumental assessment, conservative oral intake decisions, secretion management, and oral hygiene. A patient with reduced pharyngeal strength may need effortful swallow or other strengthening tasks, texture selection to reduce residue, and repeated clearance swallows. A patient with delayed swallow initiation may benefit from bolus-size control, pacing, sensory enhancement strategies when appropriate, and postural strategies. A patient with severe brainstem dysphagia may require temporary enteral nutrition while rehabilitation and recovery are monitored.

Compensatory strategies aim to improve safety or efficiency during the swallow without necessarily changing underlying physiology. Examples include upright positioning, small boluses, pacing, supervision, diet texture modification, fluid viscosity modification, postural maneuvers, and airway-protection maneuvers. Rehabilitative strategies aim to change swallowing physiology through exercise, task-specific practice, sensory stimulation, respiratory-swallow training, or neurostimulation. Current reviews describe ongoing research into behavioral exercises, sensory receptor agonists, transcranial direct current stimulation, repetitive transcranial magnetic stimulation, and pharyngeal electrical stimulation, but treatment choice must be individualized and guided by evidence, safety, availability, and patient factors.[17,18] Randomized trial data for pharyngeal electrical stimulation in subacute stroke have been mixed, so it should be presented as a selected or investigational strategy rather than routine therapy.[19]

Oral hygiene is a pathophysiological intervention, not merely nursing care. Reducing oral bacterial load reduces the pathogenic burden available for aspiration. Pilot randomized data suggest that intensified oral hygiene care may reduce suspected oral stroke-associated pneumonia pathogens and may lower pneumonia incidence in selected high-risk stroke patients, although larger confirmatory studies are needed.[20] Nutrition and hydration support tissue healing, muscle function, immune response, cognition, and rehabilitation tolerance. Enteral feeding is appropriate when oral intake is unsafe or insufficient, but tube feeding does not eliminate aspiration risk because patients may aspirate saliva, refluxate, or colonized secretions. These realities explain why PSD management requires interdisciplinary coordination among nursing, speech-language pathology, dietitians, medicine, occupational therapy, physiotherapy, respiratory therapy, dentistry or oral care specialists, patients, and caregivers. Pathophysiological cascade and intervention points are illustrated in Figure 4.

FIGURE 4: Pathophysiological cascade and intervention points. Management can target screening, airway protection, oral care, nutrition, rehabilitation, respiratory support, and neuroplastic recovery.



Abbreviations: FEES, Fiberoptic Endoscopic Evaluation of Swallowing; MBS, Modified Barium Swallow; VFSS, Video fluoroscopic Swallowing Study.

10. Mechanism-Based Clinical Pearls

- A normal bedside appearance does not exclude dysphagia; silent aspiration is possible when sensation or cough is impaired.
- Brainstem strokes can produce severe dysphagia even when limb weakness or cognition is relatively preserved.
- Residue is both an efficiency problem and a safety problem because residue can be aspirated after the swallow.
- Thickened liquids may reduce immediate airway invasion in selected patients but must be balanced against hydration, residue, patient preference, and reassessment findings.
- Tube feeding supports nutrition but does not remove the need for oral hygiene, secretion management, positioning, and aspiration prevention.
- Recovery is often driven by compensatory reorganization of intact swallowing networks, especially the unaffected hemisphere after unilateral hemispheric stroke.
- A pathophysiology-focused report should specify the mechanism: delayed trigger, sensory loss, weak pharyngeal contraction, impaired laryngeal closure, UES dysfunction, poor cough, or combined impairment.

11. Conclusion

Post-stroke dysphagia is a complex neurogenic disorder caused by disruption of the distributed swallowing network. Hemispheric lesions commonly disturb oral control, sensorimotor integration, voluntary initiation, and corticobulbar drive. Subcortical and white matter lesions disconnect cortical swallowing areas from brainstem effectors. Brainstem lesions can directly injure the medullary swallowing CPG and cranial nerve nuclei, often causing severe pharyngeal dysphagia. Cerebellar and basal ganglia involvement impairs timing, coordination, and adaptive control. Sensory impairment is especially important because it contributes to delayed swallow initiation, secretion problems, poor cough, and silent aspiration.

The pathophysiology of PSD explains its clinical consequences. Aspiration pneumonia develops from the interaction of airway invasion, impaired airway clearance, pathogenic secretions, poor oral hygiene, reduced mobility, comorbidity, and systemic vulnerability. Malnutrition and dehydration arise when swallowing efficiency and safety are insufficient for oral intake. A mechanism-based approach to screening, clinical assessment, FEES, VFSS, diet planning, rehabilitation, oral hygiene, nutrition, and reassessment is therefore essential to reduce complications and support recovery.

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Simulations were completed and portion of manuscript was written (with author prompts) using ChatGPT, model GPT-5.5 Pro, OpenAI.

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