FIBEROPTIC ENDOSCOPIC EVALUATION OF SWALLOWING IN PATIENTS WITH STROKE - A CROSS-SECTIONAL STUDY

ABSTRACT

Background and Objective

Post-stroke dysphagia represents a significant clinical challenge with substantial implications for morbidity, mortality, and rehabilitation outcomes. Despite advances in stroke management, the pathophysiological characteristics and neuroanatomical correlates of swallowing dysfunction remain incompletely elucidated. FEES offers a valuable instrumental assessment modality with distinct advantages over alternative techniques. This study aimed to document the findings of FEES in patients with stroke and to correlate the severity of dysphagia with stroke location, thereby determining the precise neuroanatomical regions related to loss of function in the upper aerodigestive tract. These objectives address critical knowledge gaps in understanding structure-function relationships in post-stroke dysphagia and their potential implications for targeted rehabilitation strategies.

Methodology

This cross-sectional study was conducted at R.L Jalappa Hospital, Kolar, from May 2023 to October 2024. The sample size of 63 stroke patients with dysphagia (aged 40-70 years) was calculated based on 62.5% expected proportion of dysphagia in post-stroke subjects with 12% absolute precision at 95% confidence level. Patients with malignancy of the upper aerodigestive tract,

history of major neck lacerations, spinal deformities, degenerative brain disorders, or ventilator dependence were excluded. All participants underwent comprehensive clinical evaluation by a senior physician and otorhinolaryngologist, neuroimaging, and FEES performed by an experienced clinician after patients achieved adequate cognitive status. The FEES protocol evaluated multiple parameters: vocal cord/arytenoid oedema, mucosal characteristics, pyriform fossa pooling, laryngeal spillover patterns, transit delay, nasal regurgitation, adynamic segments, and aspiration severity. Dysphagia severity was classified using the standardized DOSS. Follow-up assessments were conducted at one- and three-months post-stroke to document recovery patterns. Statistical analysis utilized Fisher's Exact Test to evaluate associations between stroke location and specific pathophysiological features.

Results

The cohort demonstrated a mean age of 61.49 years (SD=8.727) with male predominance (68.3%). Comorbidity analysis revealed high prevalence of Type 2 DM (79.3%) and HTN (63.5%). Left subcortical strokes represented the most common lesion location (25.4%), followed by MCA territory infarcts (15.9%). Statistical analysis revealed significant associations between stroke location and dysphagia severity ($p=0.007$), with all PCA territory strokes manifesting as severe dysphagia, whereas subcortical strokes demonstrated predominantly mild or mild-to-moderate presentations. FEES findings revealed significant

neuroanatomical correlations with vocal cord/arytenoid oedema (p=0.007), mucosal dryness (p=0.046), and pyriform fossa pooling (p=0.029). PCA territory strokes exhibited the most severe manifestations across multiple parameters, with severe oedema in 85.7%, severe mucosal dryness in 71.4%, and severe pyriform fossa pooling in 85.7% of cases. Minor aspiration was observed in 57.1% of patients and major aspiration in 11.1%, highlighting substantial risk for pulmonary complications. The Dysphagia Outcome and Severity Scale revealed Level 5 (mild dysphagia) in 44.4% and Level 4 (mild-moderate dysphagia) in 42.9% of patients. Clinical improvement was documented in 76.2% of cases with significant variability by lesion location, ranging from 14.3% improvement in PCA territory strokes to 87.5% in both ACA territory and subcortical lesions.

Conclusion

This study demonstrates significant neuroanatomical specificity in the pathophysiological manifestations of post-stroke dysphagia, with PCA territory strokes exhibiting the most severe presentations across multiple parameters and poorest functional outcomes. Endoscopic results were worrying, with high rates of swallowing, but clinical outcomes were good, even though they were very different based on the site of the tumour. These findings help us learn more about how structural factors affect function in people who have had a stroke and have troubled swallowing. They also allow for more targeted evaluations and

therapy plans based on specific lesion sites and the neurological features that are linked to them.

Keywords

Deglutition Disorders, Stroke, Endoscopy, Middle Cerebral Artery, Aspiration, Fiberoptic Technology, Neuroanatomy

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ABBREVIATIONS

Abbreviation	Explanation
FEES	Fiberoptic Endoscopic Evaluation of Swallowing
WHO	World Health Organization
GBD	Global Burden of Disease
DALY	Disability-Adjusted Life Years
LMIC	Low- And Middle-Income Countries
UES	Upper Oesophageal Sphincter
MRI	Magnetic Resonance Imaging
NTS	Nucleus Tractus Solitarius
rTMS	Repetitive Transcranial Magnetic Stimulation
MOSM	Mouth Open Swallowing Manoeuvre
MASA	Mann Assessment of Swallowing Ability
VFSS	Video-Fluoroscopic Swallow Studies
ALARA	As Low As Reasonably Achievable
IDDSI	International Dysphagia Diet Standardization Initiative
DOSS	Dysphagia Outcome Severity Scale
PAS	Penetration-Aspiration Scale

NIHSS	National Institutes of Health Stroke Scale
NMES	Neuromuscular Electrical Stimulation
CT	Computed Tomography
GCS	Glasgow Coma Scale
MCA	Middle Cerebral Artery
ACA	Anterior Cerebral Artery
PCA	Posterior Cerebral Artery

INTRODUCTION

There is a major worldwide health issue with great consequences for patient morbidity and death is stroke even today. It is characterized by a rapid disturbance in blood flow to the brain that causes neurological abnormalities ranging from sensory to motor to cognitive ones. [1,2] Complicated interactions between vascular, cellular, and molecular pathways finally result in brain damage and consequent functional impairments, hence guiding the pathophysiological processes behind stroke. [3,4] Recent developments in neuroimaging, thrombolytic treatment, and endovascular techniques have transformed acute stroke care; post-stroke sequelae nevertheless provide major difficulties for rehabilitation and recovery. [5,6]

Affecting up to 62.5% of patients, dysphagia—defined as difficulty swallowing—represents one of the most common and sometimes fatal side effects after stroke. [7] Strokes may induce a neurological insult that compromises the delicate synchronization of cranial nerves and muscles in charge of the sophisticated swallowing process, therefore affecting the generation of the bolus, pharyngeal triggering, and airway protection.[8,9] With later problems including aspiration pneumonia, malnutrition, dehydration, and extended duration of hospital stay, the clinical presentations of post-stroke dysphagia may vary from subtle indicators of reduced swallowing efficiency to overt aspiration.[10,11]

Accurate and timely assessment of dysphagia in stroke patients is paramount to prevent aspiration-related complications and guide appropriate therapeutic interventions. While bedside swallowing assessments provide initial screening, they demonstrate limited sensitivity in detecting silent aspiration—a phenomenon where material enters the airway below the vocal folds without eliciting protective reflexes such as coughing.[12] This

underscores the need for more objective and comprehensive evaluation techniques to characterize the pathophysiology of dysphagia and quantify aspiration risk.[13,14]

FEES has emerged as a valuable tool in the assessment of post-stroke dysphagia, offering direct visualization of the pharyngeal and laryngeal structures during swallowing.[15,16] Unlike traditional video fluoroscopic swallow studies, FEES provides detailed information regarding the structural integrity, secretion management, and protective mechanisms of the upper aerodigestive tract without exposing patients to radiation.[17] Moreover, FEES can be performed at the bedside, allowing for evaluation of critically ill patients who cannot be transported to radiology suites.[18,19]

Recent literature highlights the expanding role of FEES in both the acute and rehabilitation phases of stroke management. Early FEES assessment following mechanical thrombectomy has demonstrated value in risk stratification and guiding oral intake decisions.[20] Serial FEES examinations can track recovery patterns, document therapeutic responses, and inform modifications to rehabilitation strategies.[8,21] Furthermore, standardized FEES protocols with severity scales have shown prognostic value in predicting functional outcomes after stroke.[22,23]

Despite its increasing clinical application, the correlation between FEES findings and specific stroke locations remains incompletely characterized. Understanding the relationship between lesion topography and dysphagia manifestations could enhance our understanding of swallowing neurophysiology and potentially guide targeted therapeutic approaches.[24,25] This study aims to document the findings of FEES in patients with stroke and correlate the severity of dysphagia with stroke location to determine the precise areas related to

swallowing dysfunction in the upper aerodigestive tract. Such insights could potentially refine risk stratification models, inform rehabilitation strategies, and ultimately improve outcomes for stroke survivors with dysphagia.

OBJECTIVES

1) To document the findings of FEES in patients with stroke

2) To correlate the severity of dysphagia with site of stroke and to determine the precise area related to loss of function in the upper aero digestive tract

Epidemiology and Burden of Stroke

Global and Regional Epidemiology of Stroke

The WHO identifies stroke as the second leading cause of death globally, responsible for approximately 11% of all deaths. The GBD research shows that in 2019 stroke accounted for 143 million DALYs and approximately 6.6 million fatalities. LMICs, who account for about 75% of all stroke fatalities and DALYs, especially show the burden..[26]

Changing lifestyles and improvements in healthcare have caused global variations in stroke incidence.[27] Regional differences aggravate the stroke load in India. Urban locations like Goa and Kerala have higher stroke rates than rural ones, presumably because of variations in lifestyle choices and healthcare availability.[28] With a greater frequency seen in metropolitan areas than in rural ones, the incidence of stroke in India varies from 119 to 145 per 100,000 people yearly.[26]

Socioeconomic Burden of Stroke

Stroke has a significant social effect that influences people as well as more general society. Direct medical expenses as well as indirect expenses associated to lost productivity and long-term care define the financial load. Stroke care makes around 0.27% of the gross domestic product in different nations on average.[29] Those from lower socioeconomic origins are disproportionately impacted and run more chances of having stroke incidence and death.[30]

	2021		
	Male	Female	Total
Incidence rate (per 100,000 population)			
All ages	92 (83–102)	84 (76–93)	88 (80–97)
15–49 years	40 (34–47)	34 (29–40)	37 (32–44)
50–69 years	278 (231–327)	206 (171–242)	241 (202–282)
70+ years	747 (626–876)	695 (589–805)	719 (610–830)
Deaths rate (per 100,000 population)			
All ages	57 (50–66)	52 (44–60)	55 (49–61)
15–49 years	8 (6–9)	6 (5–7)	7 (6–7)
50–69 years	171 (146–200)	134 (114–156)	152 (136–169)
70+ years	746 (644–857)	611 (524–708)	674 (604–746)
DALYs rate (per 100,000 population)			
All ages	1469 (1274–1687)	1275 (1085–1475)	1374 (1240–1512)
15–49 years	417 (359–478)	338 (283–396)	379 (335–418)
50–69 years	5296 (4563–6124)	4137 (3517–4784)	4711 (4229–5215)
70+ years	12,328 (10,761–14,145)	9680 (8447–11,188)	10,909 (9852–12,053)

Table 1: Burden of stroke (incidence, death, and DALYs) in India[26]

Current Trends in Stroke Incidence, Prevalence, and Mortality

Recent events have made it harder to understand how common and how often strokes happen. While some regions report a decline due to improved healthcare strategies, others, particularly in developing countries, show rising trends due to increasing risk factors like HTN and diabetes.[31] The mortality rate has also seen a decline in some high-income countries, attributed to better acute care and rehabilitation services.[32]

Stroke as a Leading Cause of Disability

Stroke ranks as the second leading cause of mortality and the third leading cause of disability worldwide. It significantly contributes to long-term physical and cognitive impairments,

necessitating comprehensive rehabilitation strategies.[33]The DALYs lost due to stroke highlight its significant impact on quality of life.[34]

Age and Gender Distribution in Stroke Populations

Stroke incidence varies widely by age and gender, with the elderly population being most affected. Men generally have higher stroke incidence rates compared to women, although women tend to experience more severe outcomes post-stroke.[35] This disparity is particularly notable in the older age groups, where age-related risk factors significantly increase stroke susceptibility.[36]

Pathophysiology of Stroke

Stroke pathophysiology involves complex mechanisms, categorized mainly into ischemic and haemorrhagic types, each with distinct processes and outcomes.

Ischemic Stroke Mechanisms

Ischemic strokes, accounting for the majority of stroke cases, occur due to an interruption in blood flow to the brain. This can happen through thrombotic, embolic, or hypoperfusion mechanisms. Thrombotic strokes are caused by blood clots forming within the cerebral arteries, often as a result of atherosclerosis.[37] Embolic strokes occur when a clot from another part of the body travels to the brain, blocking an artery.[38] Hypoperfusion strokes result from a significant reduction in blood flow, often due to cardiac failure or systemic hypotension.[39]

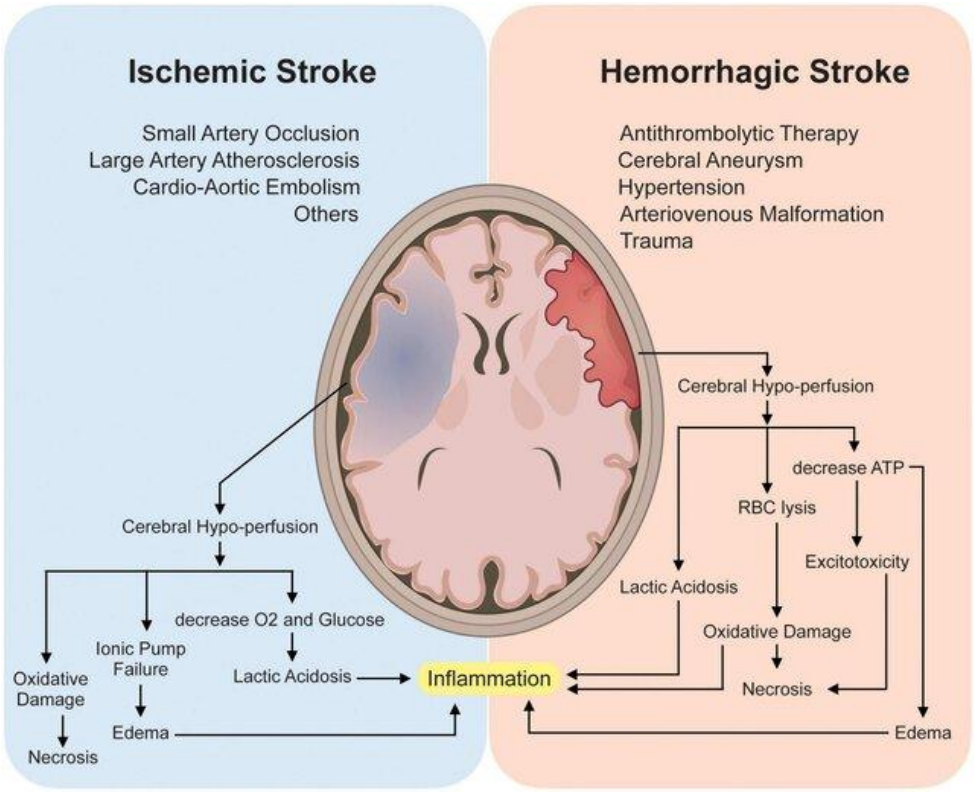


Figure 1: Pathophysiology and Mechanism involved in Ischemic and Haemorrhagic stroke

Haemorrhagic Stroke Mechanisms

Haemorrhagic strokes are less common and result from bleeding within the brain tissue or surrounding areas. The primary causes include HTN, aneurysm rupture, or trauma, leading to intracerebral haemorrhage or subarachnoid haemorrhage.[40] The bleeding increases intracranial pressure and can cause further ischemic injury due to compression of brain structures.[41]

Cellular and Molecular Mechanisms of Neuronal Injury

At the cellular level, stroke induces a cascade of events leading to neuronal injury. Ischemia triggers excitotoxicity, where excessive glutamate release causes calcium overload in neurons, leading to cell death. Additionally, oxidative stress and inflammation exacerbate neuronal damage, with free radicals and inflammatory cytokines playing critical roles.[42]

Neuroplasticity and Recovery Mechanisms

Post-stroke recovery largely depends on neuroplasticity, the brain's ability to reorganize and form new neural connections. This process is facilitated by rehabilitation therapies that promote motor and cognitive recovery through repetitive and task-specific training.[43] Neuroplasticity involves structural changes, such as dendritic sprouting and synaptic remodelling, enhancing functional recovery.[44]

Neuroanatomical Correlates of Stroke-Related Deficits

The precise impairments suffered by the patient are dictated by the site of the stroke. For instance, problems with language and speech are common after strokes that impact the left hemisphere, but impairments in attention and spatial awareness are possible after strokes that impact the right hemisphere.[45] To create tailored rehabilitation programs, knowledge of these neuroanatomical correlations is essential.

Epidemiology and Impact of Post-Stroke Dysphagia

Prevalence of Dysphagia Following Stroke

Stroke survivors often have dysphagia, or difficulty swallowing, as a secondary consequence. Presence of dysphagia in the acute phase might vary between 37% and 78% according on the diagnostic criteria and procedures used.[43] Although the occurrence of persistent dysphagia decreases as the healing process advances, it still affects from 11% to 50% of stroke patients. Given the influence on health outcomes and quality of life, this disorder continues to be a major cause for worry.

Predictors of Dysphagia in Stroke Patients

Years of age, previous health issues, stroke location and severity, and other factors are all known to increase the likelihood of dysphagia after a stroke. Because they are so close to the cranial nerves that control swallowing, brainstem strokes pose an especially significant danger.[46] Patients who need mechanical breathing or have bigger infarcts are also at a higher risk of developing dysphagia.[47]

Impact on Morbidity: Aspiration Pneumonia and Malnutrition

Aspiration pneumonia is a very bad health problem that can happen to people who have had a stroke, and swallowing makes this condition much more likely to happen. When someone has trouble swallowing, food and drinks can get stuck in their lungs and make them sick.[48] Another effect is malnutrition, since dysphagia makes it hard to take in food, which leads to losing weight and a weaker immune system.[49] Because of these disorders, people have longer hospital stays and use more medical care.

Impact on Mortality Rates

Patients experiencing difficulty swallowing after a stroke are more likely to die. Among these patients, aspiration pneumonia is the most common cause of mortality.[47] Death rates are greater in stroke patients who have dysphagia. Particularly among these individuals, aspiration pneumonia is a major killer.[43]

Economic Burden of Dysphagia Management

Speech therapy, dietary changes, and sometimes surgical procedures are all part of the interdisciplinary approach to dysphagia treatment, which has a significant financial effect due to the high costs of critical care, prolonged hospital stays, and rehabilitation services.[47] The

necessity for measures that effectively control costs is underscored by the fact that these fees add to the total financial burden of stroke treatment.

QoL Implications for Stroke Survivors with Dysphagia

Dysphagia lowers the QoL of stroke patients. People hide from society because they are too scared or ashamed to eat in public.[50] It's also possible that food restrictions could have an effect on mental health because they make eating less enjoyable. Dysphagia survivors have a better QoL when they get good care, which helps not only their physical health but also their social and mental well-being.[43]

Neurophysiology and Neural Control of Swallowing

Normal Swallowing Physiology

Multiple parts of the body work together to make swallowing possible. It takes complex muscle control. Physiologically, we have to swallow to take in food and keep our lungs clear. More than twenty-five muscle pairs controlled by cranial nerves V, VII, IX, X, and XII must work together to do the job. The medulla oblongata in the feeding center is where all of this teamwork happens.[51]

Clinical diagnosis and treatment of dysphagia may be greatly improved by gaining a better understanding of the physiological factors underlying proper swallowing.

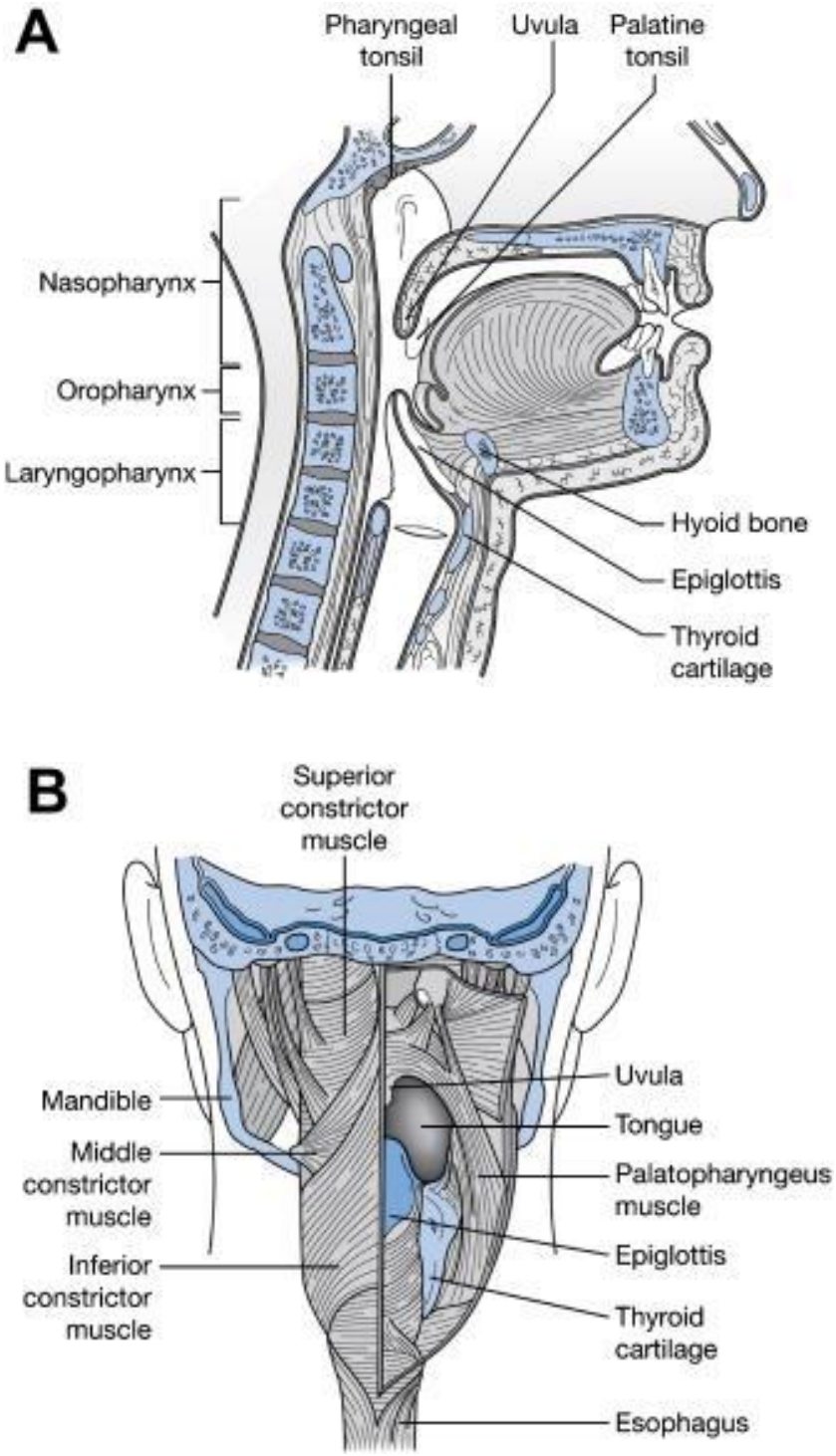


Figure 2: Anatomy of the oral cavity and pharynx [52]

Phases of Swallowing

There are four separate yet interconnected stages of swallowing, according to modern research: the oral preparation, propulsive, pharyngeal, and oesophageal stages.[52]

Oral Preparatory Phase

Mechanical processing of the consumed material to form a cohesive bolus occurs during this optional step. As the tongue works the bolus against the hard palate, the lips maintain a tight anterior seal.[53] Salivary amylase starts starch digestion, and sensory input from mechanoreceptors and chemoreceptors allows for precise bolus manipulation.[54] The length of this phase is quite variable and dependent on the consistency of the bolus and other personal variables.

Oral Propulsive Phase

This phase begins with posterior tongue elevation against the hard palate, creating a pressure gradient that propels the bolus toward the pharynx.[52] Electromyographic studies demonstrate sequential activation of intrinsic and extrinsic lingual muscles, generating a coordinated wave-like motion.[51] The process terminates with triggering of the swallowing reflex as the bolus reaches the anterior faucial pillars.

Pharyngeal Phase

The pharyngeal phase, predominantly involuntary, involves rapid sequential events occurring within approximately 1 second. Key physiological mechanisms include:

1. Velopharyngeal closure preventing nasal regurgitation
2. Hyolaryngeal elevation and anterior movement

3. Epiglottic retroflexion

4. Vocal fold adduction

5. Sequential contraction of pharyngeal constrictors [54]

These coordinated actions ensure bolus propulsion toward the oesophagus while maintaining critical airway protection. Research indicates that pharyngeal transit times averaging 0.7-1.0 seconds remain remarkably consistent across healthy individuals.[53]

Oesophageal Phase

The final phase begins with relaxation of the UES, allowing bolus entry into the oesophagus. Primary peristalsis, initiated by pharyngeal contraction, propagates distally at 3-5 cm/second in the proximal oesophagus and 1-2 cm/second in the distal smooth muscle segment.[54]Manometric studies demonstrate sequential pressure gradients facilitating aboral movement, with terminal relaxation of the lower oesophageal sphincter permitting gastric entry. [51]

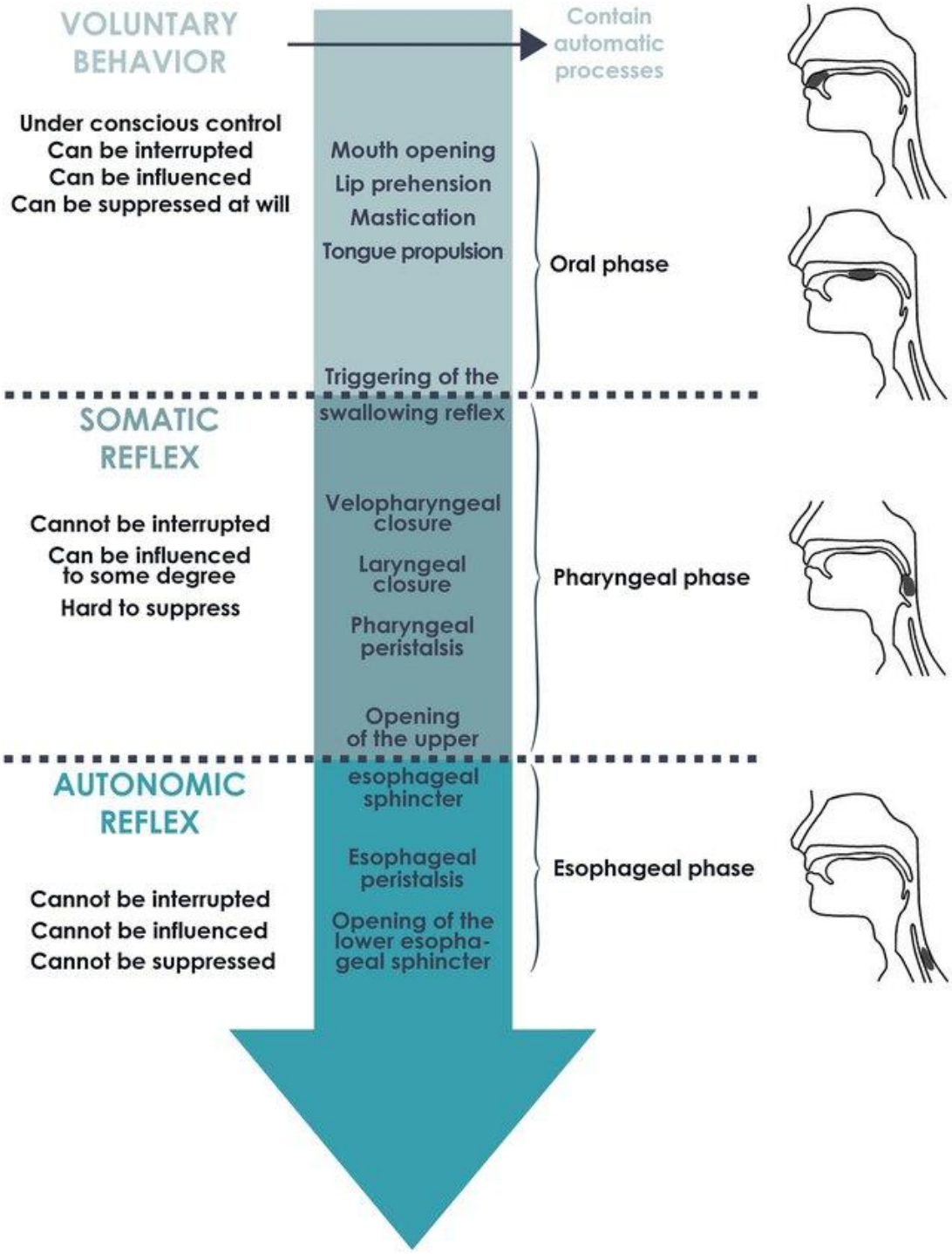


Figure 3: Phases of swallowing[55]

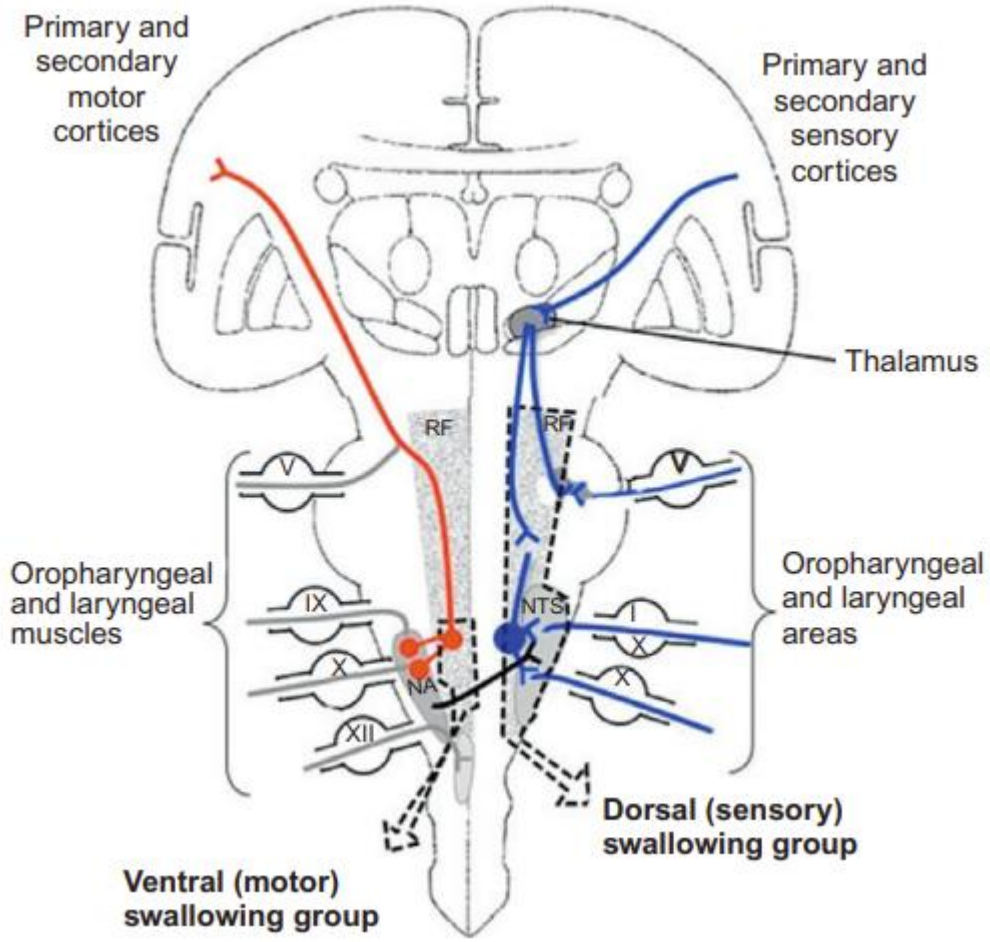


Figure 4: Corticobulbar neural control of swallowing[56]

Cortical and Subcortical Control of Swallowing

Swallowing is regulated by a network of cortical and subcortical structures. The primary motor cortex, along with the supplementary motor area and insula, plays a crucial role in initiating and modulating the swallowing reflex. Functional MRI studies have shown that these areas demonstrate increased activity during swallowing tasks, indicating their involvement in the voluntary components of swallowing.[57] Subcortically, the basal ganglia and thalamus are involved in the modulation and fine-tuning of swallowing movements, ensuring smooth coordination between the various phases.[58]

Brainstem Nuclei and Cranial Nerves Involved in Swallowing

The brainstem's important regions work together to control the parts of eating that you can't control. The NTS and nucleus ambiguus are the main parts of the brain that process sense information and make movement outputs for eating. Cranial nerves IX and X send afferent impulses to the NTS. These impulses are important for finding boluses and starting the swallowing response.[59] Nerves IX and X in the skull send movement signals to the nucleus ambiguus, which tells the muscles of the throat and mouth how to close the airway safely when eating.[60]

Neuroplasticity in Swallowing Function

Neuroplasticity, the brain's ability to change and adapt, is important for getting to eat back to normal after an illness or accident. Neuroplasticity may be helped by methods like rTMS, which may help swallowing function return by making the brain more excitable and improving synaptic remodeling in the swallowing motor cortex..[43] This adaptability is vital in stroke rehabilitation, where compensatory mechanisms are crucial for regaining swallowing capabilities.[61]

Swallowing Manoeuvres

Therapeutic swallowing manoeuvres constitute critical rehabilitative strategies designed to modify specific physiological components of the swallowing mechanism, thereby enhancing protective functions and improving bolus transit through the pharynx and into the oesophagus. These compensatory and rehabilitative techniques have demonstrated efficacy in optimizing swallowing safety and efficiency among post-stroke patients with neurogenic dysphagia.

Classification of Swallowing Manoeuvres

Contemporary rehabilitative approaches to post-stroke dysphagia management incorporate several evidence-based swallowing manoeuvres, which can be broadly categorized as either compensatory (providing immediate but temporary improvement) or rehabilitative (facilitating neuroplasticity and long-term recovery) interventions.[62] The following analysis examines the biomechanical mechanisms, neurophysiological foundations, and clinical applications of these therapeutic techniques in stroke populations.

Mendelsohn Manoeuvre

The Mendelsohn manoeuvre involves voluntary prolongation of laryngeal elevation at the peak of the swallow, specifically targeting UES opening duration. This technique was originally conceptualized to address inadequate UES relaxation and insufficient hyolaryngeal excursion commonly observed in neurogenic dysphagia.[63]

In a controlled intervention study examining post-stroke patients, McCullough and colleagues (2012) documented statistically significant improvements in hyoid movement duration ($p<0.001$) and UES opening duration ($p<0.05$) following implementation of the Mendelsohn manoeuvre. Quantitative video fluoroscopic analysis revealed mean improvements of 0.19 seconds in UES opening duration across participants, which correlated positively with reduced pharyngeal residue and decreased aspiration risk.[63] The neurophysiological mechanism underlying these improvements appears to involve enhanced cortical activation patterns in sensorimotor regions associated with voluntary swallowing control, potentially facilitating neuroplastic reorganization in stroke-affected neural circuits.

Supraglottic and Super-supraglottic Swallow

These manoeuvres specifically target airway protection through voluntary breath-holding during the swallow. The supraglottic swallow involves breath-holding before and during the swallow, while the super-supraglottic swallow adds effortful closure of the vocal folds through bearing down during the breath-hold.[62]

Electromyographic studies demonstrate that these techniques significantly increase laryngeal adductor activity and enhance true and false vocal fold approximation during swallowing, providing mechanical protection against aspiration.[62] These manoeuvres have shown particular efficacy in stroke patients with delayed or reduced laryngeal vestibule closure, with clinical trials documenting significant reductions in PAS scores ($p<0.01$) following implementation.

Effortful Swallow

The effortful swallow technique involves voluntary enhancement of tongue base retraction against the pharyngeal wall during swallowing, thereby increasing pharyngeal propulsive forces.[62] This manoeuvre has demonstrated particular effectiveness in addressing reduced pharyngeal contraction and inadequate bolus clearance frequently observed in post-stroke dysphagia.

Omori and colleagues (2024) documented significant increases in maximum laryngeal elevation measures (mean increase of 5.2 mm, $p<0.01$) during effortful swallowing compared to normal swallowing in their comparative analysis. The researchers hypothesized that increased lingual propulsive forces generated during effortful swallowing contribute to

enhanced hyolaryngeal excursion through biomechanical linkage of these anatomical structures.[64]

MOSM

A recently developed technique, the MOSM involves maintaining an open mouth position during pharyngeal swallowing, which has demonstrated promising results in enhancing swallowing kinematics. Yaşaroğlu et al. (2024) conducted high-resolution video fluoroscopic analysis comparing MOSM with conventional swallowing techniques and documented significant enhancements in hyoid displacement (10.7% increase, $p<0.05$) and reduction in pharyngeal transit times (mean reduction of 0.31 seconds, $p<0.01$).[65]

Submental surface electromyography revealed significantly increased activation of the mylohyoid, geniohyoid, and anterior digastric muscles during MOSM compared to conventional swallowing (mean increase of 32.4% in amplitude, $p<0.001$), suggesting enhanced suprahyoid muscle recruitment.[65] While preliminary studies have shown promising results, additional research specifically evaluating MOSM in stroke populations is necessary to determine its clinical efficacy in neurogenic dysphagia rehabilitation.

Clinical Implementation in Stroke Rehabilitation

Optimal implementation of swallowing manoeuvres in post-stroke dysphagia management requires comprehensive instrumental assessment to identify specific physiological deficits and individualize treatment approaches. Vose and colleagues (2014) emphasize that therapeutic selection should be guided by objective instrumental evaluation findings, with video fluoroscopic swallowing studies or fibreoptic endoscopic evaluation of swallowing providing critical diagnostic information.

The rehabilitation protocol typically involves progressive implementation, beginning with compensatory techniques for immediate aspiration risk reduction, followed by rehabilitative manoeuvres targeting neuroplasticity and long-term recovery.[62] Clinical evidence suggests that intensive practice (3-5 sessions daily for 6-8 weeks) optimizes functional outcomes through neurophysiological reorganization of swallowing neural networks following stroke.

Pathophysiological Mechanisms of Dysphagia in Stroke

Dysphagia, or difficulty swallowing, commonly occurs after a stroke, particularly when it affects the brainstem or cortical areas involved in swallowing. The disruption of neural pathways leads to impaired coordination of the swallowing phases, increasing the risk of aspiration and malnutrition. The pathophysiological mechanisms include weakened sensory feedback from the pharynx, reduced motor output to the swallowing muscles, and delayed initiation of the swallowing reflex. These issues are compounded by muscle weakness and altered reflexes, which can persist long-term if not adequately managed.[66]

The Relationship Between Stroke Location and Dysphagia

Cortical Lesions and Dysphagia Patterns

Lesions in the cerebral regions responsible for the voluntary initiation of swallowing and the coordination of complex motor activities necessary for safe swallowing—the precentral gyrus and the insular cortex—are strongly associated with dysphagia. When these regions are damaged, they can interfere with the integration of sensory inputs and motor outputs, resulting in impaired swallowing reflexes and delayed oropharyngeal transit times.[44] Patients who have had a cortical stroke often struggle with the oral swallowing phase, leading to subpar bolus creation and transfer.

Subcortical Lesions and Dysphagia Manifestations

Another common cause of severe swallowing problems is subcortical strokes, which impact the basal ganglia and thalamus. These regions of the brain are responsible for the regulation of somatic actions as well as the processing of sensory information in the person. They are essential process for the swallowing. Pharyngeal entry along with aspiration may result from subcortical injuries that impair the coordination of the feeding process. People who have experienced subcortical strokes frequently exhibit difficulty in swallowing, which is characterized by delayed pharyngeal responses as well as disrupted feeding habits.

Brainstem Strokes and Severity of Swallowing Impairment

Because the brainstem houses the swallowing centers, such as the nucleus ambiguus and the nucleus tractus, strokes to this region are linked to the most severe types of dysphagia. It is the job of these nuclei to create the swallowing reflex's core pattern. Because it coordinates both sides of the body, damage to the brainstem usually leads to deficits on both sides. Complete loss of the swallowing reflex, aspiration, and the need for enteral feeding are symptoms of severe dysphagia, which may be caused by brainstem strokes.[43]

Hemispheric Dominance in Swallowing Control

One theory is that one side of the brain, usually the left side, may have a stronger grip over swallowing than the other. This is called hemispheric dominance. It is believed that the distribution of brain networks regulating voluntary motor actions is the cause of this predominance. Dysphagia may be more severe after a stroke in the dominant hemisphere than in the non-dominant hemisphere, according to studies.[61] Because of this imbalance, it is clear that the dominant hemisphere is crucial for effective swallowing.

Predictive Value of Lesion Location for Dysphagia Prognosis

One important factor in determining the prognosis of dysphagia after a stroke is the site of the lesion. Dysphagia, which may hinder rehabilitation and quality of life, is more common in patients with lesions in the brainstem and dominant hemisphere. Conversely, cortical strokes, especially those in non-dominant hemispheres, may have a more favourable prognosis with potential for recovery through rehabilitation. Understanding the predictive value of lesion location can guide therapeutic strategies and help set realistic expectations for recovery.

MCA Stroke and Dysphagia

Dysphagia following MCA stroke occurs in approximately 30-50% of patients, with higher prevalence in cases involving the insular cortex and frontal operculum. The pathophysiological basis stems from disruption of cortical swallowing representation, which exhibits bilateral but asymmetric organization. MCA supplies critical swallowing control regions, including the primary sensorimotor cortex, insula, and anterior cingulate cortex.[67]

In upper motor neuron lesions affecting MCA territory, bilateral cortical innervation of swallowing musculature produces distinctive clinical manifestations. Unlike limb weakness that follows contralateral patterns, swallowing dysfunction may persist despite unilateral lesions due to bilateral cortical representation. However, the preservation of the upper facial muscles despite lower facial weakness in MCA strokes is explained by the bilateral upper motor neuron innervation to cranial nerve VII's dorsal subnucleus, which supplies the upper face. This anatomical arrangement accounts for selective sparing of forehead movement despite significant oropharyngeal dysfunction.[68]

PCA Stroke and Associated Deficits

PCA territory strokes manifest a constellation of deficits beyond dysphagia, including dysphonia, dysarthria, and impaired vision. These manifestations relate to specific cranial nerve involvement:

- Cranial nerve V (trigeminal): Chewing difficulties due to masticatory muscle weakness
- Cranial nerve VII (facial): Facial asymmetry affecting emotional expression and articulation
- Cranial nerve VIII (vestibulocochlear): Vertigo affecting postural stability during feeding
- Cranial nerves IX and X (glossopharyngeal and vagus): Primary swallowing impairment affecting pharyngeal contraction, laryngeal elevation, and airway protection
- Cranial nerve VI (abducens): Diplopia impairing visual guidance during feeding
- Cranial nerve XII (hypoglossal): Dysarthria affecting intelligibility and oral bolus control

Visual deficits in PCA stroke result from occipital lobe involvement, producing hemianopia or cortical blindness that significantly impacts feeding coordination. Notably, PCA strokes frequently affect multiple cranial nerves due to brainstem involvement, particularly when ischemia extends to vertebrobasilar circulation supplying cranial nerve nuclei.[67] This multifaceted neurological disruption explains the severe dysphagia observed in 100% of PCA stroke patients in our cohort, with substantially poorer functional outcomes compared to other stroke locations.

Bedside Swallowing Evaluation Techniques

The bedside swallowing evaluation is a cornerstone of dysphagia assessment in stroke patients. It involves a series of structured tests aimed at observing the patient's ability to handle saliva, liquids, and solids. Techniques such as the water swallow test and the volume-viscosity swallow test are commonly employed.[69] By doing these tests, doctors may be better able to find signs that someone is having trouble speaking, like a change in their voice, coughing, or clearing their throat. Even though they are helpful, oral checks may miss small signs of swallowing, which means that more clinical tests are needed.

Standardized Clinical Assessment Tools

Standards-based tools like the MASA are used a lot. There is a detailed test called the MASA that checks how well someone can drink. It checks things like awareness, teamwork, and mouth muscle function.[70] Clinicians like it because it is true and reliable; it gives them a methodical way to figure out how bad dysphagia is and plan treatments accordingly.[71]

Swallowing disorders	Patient's complaints or behaviors; symptoms may occur at any or all phases of swallowing
Oral phase	
Difficulty chewing	Prolonged chewing time
Poor bolus control	Anterior loss of food; premature spillage to the pharynx; penetration/aspiration; coughing – wet voice
Swallowing apraxia	Difficulty initiating chewing; difficulty initiating swallowing
Poor oral sensation	Difficulty chewing; oral residue
Reduced taste	
Drooling	Difficulty manipulating food; aspiration of secretions; coughing – wet voice; anterior loss of secretions and/or food
Delayed oral transit time	Difficulty initiating swallowing
Pharyngeal phase	
Delayed triggering of pharyngeal response	Premature spillage to the pharynx; penetration/aspiration; coughing – wet voice
Reduced laryngeal closure	Penetration/aspiration; coughing – wet voice
Reduced hyolaryngeal excursion	Reduced laryngeal closure; penetration/aspiration; coughing – wet voice; pharyngeal residue; sticking sensation; reduced UES opening
Reduced base of tongue to pharyngeal wall contact	Pharyngeal residue; sticking sensation; penetration/aspiration; coughing – wet voice
Osteophytes	Pharyngeal residue; penetration/aspiration; coughing – wet voice; sticking sensation
Reduced laryngopharyngeal sensation	Silent aspiration; wet voice; absence of coughing; pulmonary infections; pharyngeal residue
Reduced velopharyngeal closure	Nasal regurgitation
Esophageal phase	
Reduced UES opening	Pharyngeal residue; sticking sensation; penetration/aspiration; coughing – wet voice
Intraesophageal residue	Sticking sensation/globus sensation; coughing
Intraesophageal reflux	Frequent throat clearing; sticking sensation/globus sensation; food coming back up
Laryngopharyngeal reflux	Frequent throat clearing; postnasal drip; bitter taste in the morning
Gastroesophageal reflux	Heartburn; food coming back up; odynophagia (pain during swallowing)
Diverticula	Sticking sensation/globus sensation; penetration/aspiration; coughing – wet voice

Table 2: Possible symptoms of neurological dysphagia

Screening Protocols for Aspiration Risk

It's necessary to do aspiration risk checks to avoid problems like aspiration pneumonia. Protocols often include quick ways to find out if a patient is at risk of aspiration, like the GUSS and the BAS.[47] By taking these steps, it's easy to find and fix major breathing problems earlier, which may lower their chances.[69]

Limitations of Clinical Assessments in Detecting Silent Aspiration

When patients aspirate without exhibiting obvious symptoms like coughing or choking, it is known as silent aspiration and identifying them is very difficult. The sensitivity of conventional bedside assessments to identify such occurrences is questionable.[72] The use of resource-intensive and not universally accessible advanced instrumental evaluations such as VFSS or FEES is commonly advocated for the proper diagnosis of silent aspiration.[73]

Role of Multidisciplinary Teams in Dysphagia Assessment

Stroke patients with dysphagia are best evaluated and handled by a group of professionals from different fields. So that they can give complete care, these teams usually have SLPs, chefs, nurses, and doctors on them.[47] This team effort to look at eating and diet from all sides has led to better outcomes for patients and a fuller recovery. The integration of various expertise allows for tailored interventions and continuous monitoring, adapting treatment plans as the patient's condition evolves.

Assessment Methods for Dysphagia

VFSS

The VFSS is a dynamic X-ray procedure that assesses swallowing function in real-time, allowing clinicians to visualize the oral, pharyngeal, and oesophageal phases of swallowing. During VFSS, patients swallow various consistencies of barium-infused substances, enabling the visualization of bolus transit and aspiration risks. The methodology involves positioning the patient laterally so that the swallowing process can be recorded and analysed frame by frame.

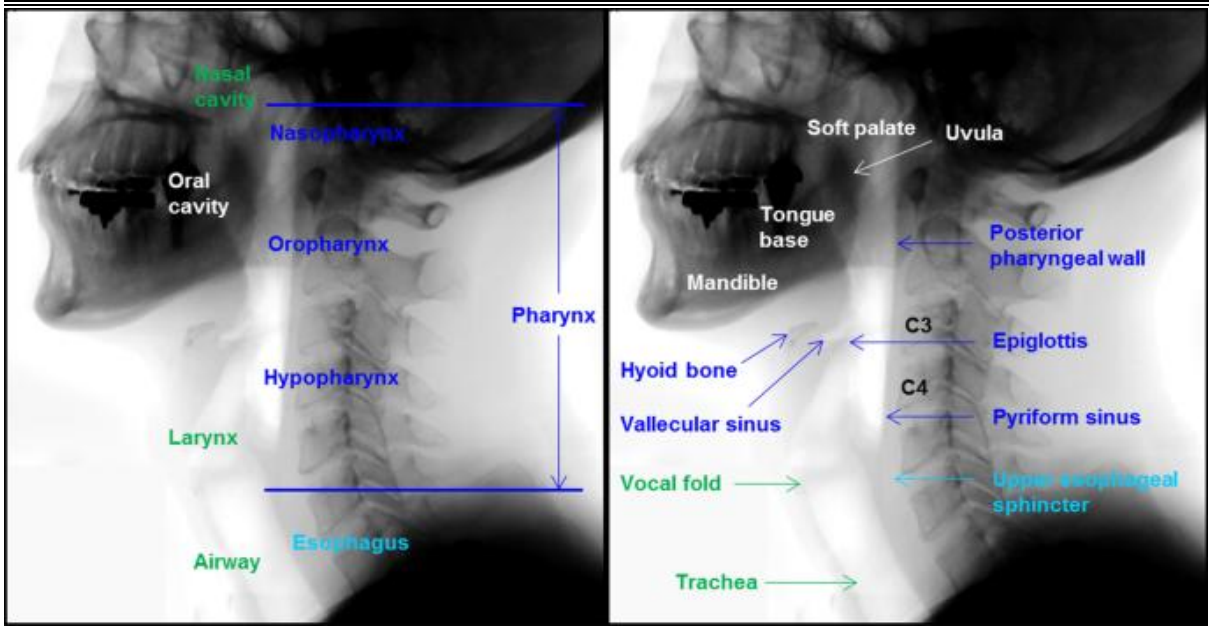


Figure 5: VFSS displaying the anatomy associated with the swallowing process[76]

This detailed examination provides insights into the coordination of structures involved in swallowing and identifies any physiological abnormalities.[74] Interpretation of VFSS requires trained clinicians to assess the timing, movement, and coordination of swallowing structures, identifying issues such as delayed swallow initiation or incomplete airway closure.[75]

Advantages and Limitations

VFSS is considered the gold standard for dysphagia assessment due to its ability to provide a comprehensive view of swallowing mechanics. It's helpful because it lets researchers see the whole eating process at once, from the mouth to the stomach. Clinicians can use this knowledge to figure out what problems are happening and change treatments as needed.[77] VFSS does have some problems, though. For example, it needs certain tools and skilled workers. Another problem is that it only gives a short "snapshot" of eating, and the radiation exposure limits how often it can be used.[78]

Radiation Exposure Considerations

Because X-rays are used in VFSS, it is important to think about how much radiation you will be exposed to. The amount of radiation must be kept as low as possible by shortening the study, even if it means the dose is often very low and below what is considered safe. Clinicians should follow the ALARA principle and find the best length and frequency of the study to keep patients safe. But the benefits of VFSS as a way to find out why someone is having trouble eating often outweigh the risks of radiation exposure.[79]

Standardized Scales and Measures

The diagnosing accuracy of VFSS is better when normal scales and measures are used. The PAS is often used to find out how much the mouth is blocked when someone is eating. It is possible to get a good idea of aspiration risk by classifying aspiration depth and response.[77] Extra standard tools, like the IDDSI, can be used to rate and categorize how well and safely people can swallow a range of food and drink textures.[80] Because of these factors, healthcare professionals can talk to each other more clearly and regularly.

FEES

When it came out in the 1980s as a way to diagnose dysphagia, the FEES changed everything. There were problems with other diagnosis methods that FEES was meant to fix by letting doctors see directly the pharyngeal stage of eating. Endoscopes that couldn't bend have been replaced by fiber optic tools that can. This makes treatments easier and more comfortable for patients.[81] FEES is now an important part of current clinical practice because it is portable, easy to use, and quick.[82]

Equipment and Technique

For FEES, a bendable camera is put in through the nose to see what is going on in the larynx and throat while the person is eating. The process lets doctors directly look at the structure and function of the eating system.

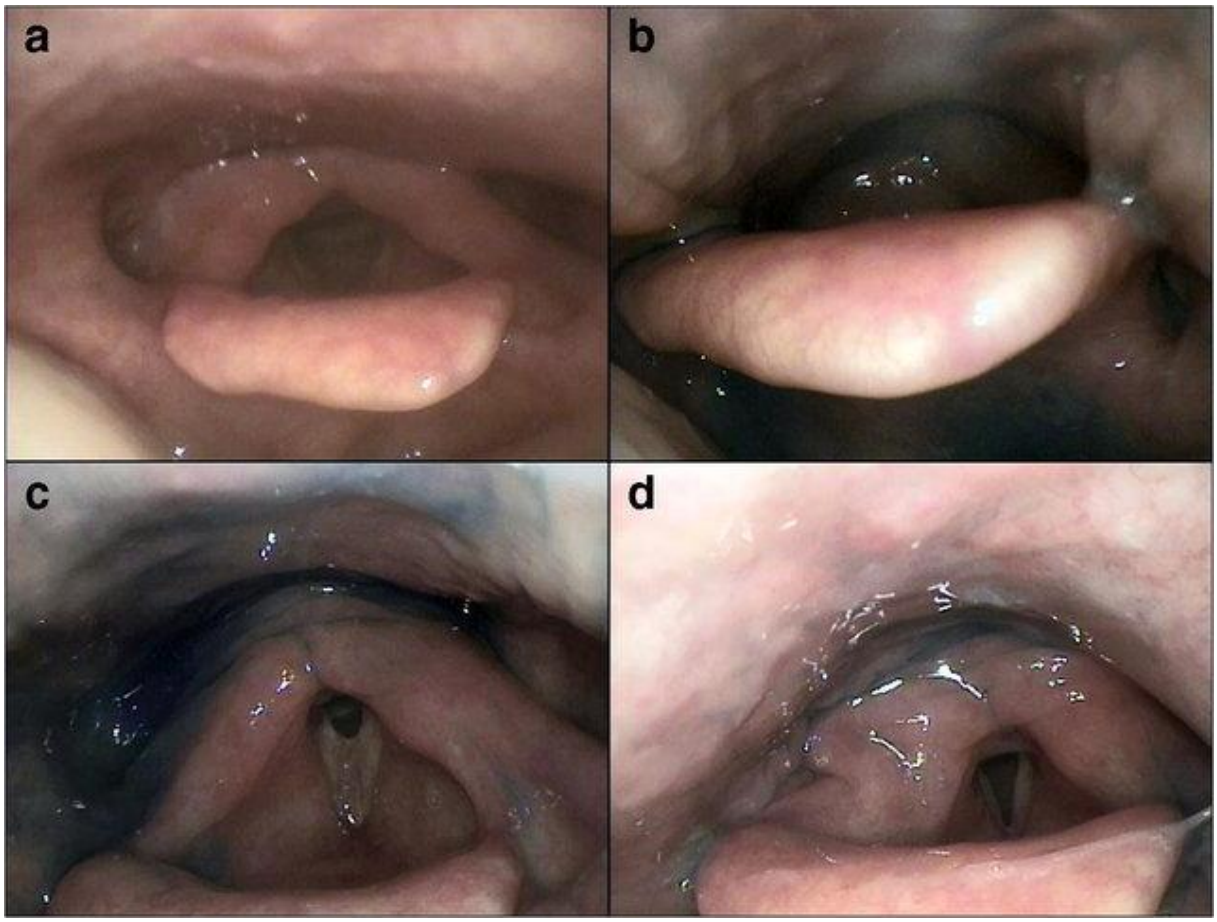


Figure 6: FEES showing four consequent phases of normal swallowing[84]

By linking the endoscope to a camera and screen, it is possible to capture and analyze pictures in real-time. When testing swallowing safety and effectiveness, patients are usually seated while a variety of liquid and food consistencies are given to them.[83] In order to detect problems like penetration, aspiration, and residue post-swallow, the method requires the interpretation of dynamic interactions by trained physicians.

Advantages and Limitations

FEES has lot of advantages, such as being portable and able to be done without radiation, which makes it a good choice for regular evaluation.[85] It shows the pharyngeal phase in great detail, which helps find aspiration and penetration that other ways may miss. But FEES is not without its drawbacks; for example, it could be uncomfortable due to its intrusive nature and there is a "whiteout" phase during swallowing when the camera cannot see anything because the pharynx closure is blocking the image.[17] In contexts without VFSS, FEES is still an effective technique, despite these limitations.

Standardized Protocols and Severity Scales

Standardized methods for FEES make sure that ratings are always correct and consistent. Doctors often use the DOSS to figure out how bad dysphagia is during FEES because it is a standard way to do it.[86] These processes cover things like where to put the patient, how consistent the food should be, and safety measures that are specific to the operation. When intensity measures are used to describe the level of speech problems, doctors can talk to each other and decide on the best course of treatment more easily.[85]

Other Methods to Assess Dysphagia

FEES is used with other tools to check for swallowing. An ultrasound can be used to look at the mouth phase of eating without hurting the person. The ultrasound shows the tongue moving and the bolus moving in real time. Manometry can be used to measure the pressure inside the stomach while eating in order to diagnose problems with oesophageal movement. By monitoring the bolus with radioactive isotopes, scintigraphy provides quantitative information on the time it takes for the bolus to go through the digestive tract and the rate at

which it is cleared.[87] When used in conjunction with FEES and VFSS, these methods allow for a more thorough evaluation of swallowing function at all developmental stages.

Comparative studies between VFSS and FESS in assessing swallowing among stroke patients

VFSS provides a comprehensive view of the oral, pharyngeal, and esophageal stages of swallowing and is hence widely regarded as the gold standard. It is especially helpful for detecting anatomical and functional problems in swallowing since it gives a complete picture of the swallowing dynamics.[78] But VFSS is not used very often since it exposes people to radiation and needs access to radioactive facilities.[83]

In contrast, FEES provides an excellent view of the laryngeal penetration as well as aspiration phases of swallowing and provides a thorough picture of the pharyngeal swallowing phase overall. There are some difficulties to overcome with VFSS, and it can be done right at the bedside, and by giving patients instant feedback.[85] The "whiteout" period that occurs during swallowing also limits FEES, which is otherwise useful, and it provides no details about the oesophageal phase.[17]

Although VFSS as well as FEES may have several values of sensitivity, research indicates that both are accurate diagnostic tools for identifying aspiration during swallowing. The mobility as well as lack of radiation exposure of FEES make it a popular choice for patients requiring ongoing monitoring or who suffer from severe dysphagia.[86] But VFSS is essential for comprehensive evaluation, especially if oesophageal involvement is thought to be involved.[78]

In general, the availability of resources and the particular clinical situation dictate which of VFSS and FEES is preferred. In terms of accessibility and real-time evaluation, FEES offers practical benefits over VFSS, which gives a larger anatomical perspective.[85]

Technical Aspects and Methodology of FEES

Equipment Specifications and Advances

Over time, FEES equipment has undergone significant development. Modern FEES systems include an adjustable eyepiece, a light source, and a computer-connected camera that allows the physician to see the patient's pharynx while they are eating.[85] The flexibility and sharpness of the endoscope have recently been improved. This has improved the image quality and increased patient comfort. As an additional method of testing sensory responses during the exam, specialized stimulators may be employed.[81]

Examination Protocols

The goal of creating FEES testing methods was to make sure that the assessment process was standardized and thoroughly evaluated. The camera is put in through the nose so that the surgeon can see the larynx and throat. Patients are asked to take foods and drinks of different textures so that the processes of eating can be studied and problems like aspiration or entry can be found.[83] The main goal is always a thorough and organized evaluation, but the steps may be slightly different depending on the patient's needs and the clinical situation..[85]

Contrast Medium Selection

Differentiation mediums, like coloured liquids, semi-solids, and solids, are used to better see the bolus and eating process during FEES. The contrast medium is chosen based on the

patient's ability to swallow and the specific medical problem that needs to be solved. Putting food colouring in the medium makes it easy to see the bolus in relation to what is around it.

Safety Considerations and Contraindications

In FEES, safety is very important because the camera is put in through the nose tube. FEES might not be a good choice for people who have recently had surgery on their noses or who have a very stuffy nose. To reduce pain and avoid problems like epistaxis, some measures are used.[83] For the safety of their patients and the smooth execution of the treatment, clinicians need extensive training on how to use the necessary equipment.[85]

Patient Preparation and Positioning

A good FEES examination requires the patient to be properly prepared and positioned. Folks should be told what kind of pain they can expect before they have surgery. They are usually sitting up straight to help their natural ways of eating. The person being examined is told to swallow a number of small items while the endoscope is carefully put into their nose.[85] Making sure the patient is ready ahead of time will help them stay calm and comfortable during the process.

Documentation Standards and Recording Systems

Documentation and recordkeeping are crucial to FEES since they provide comprehensive analysis and future reference. The examination is recorded in high-definition video, allowing students to review it subsequently. Care strategies, the patient's conduct, and any observed issues must be meticulously documented.[85] Recording systems as well as medical record systems may collaborate to facilitate the maintenance and documentation of information.

FEES Findings in Stroke Patients

Common Pathological Findings

FEES is instrumental in identifying common pathological findings in stroke patients. These include pharyngeal residue, laryngeal penetration, and aspiration, often due to impaired motor control and sensory deficits following a stroke. Such findings are critical for assessing the degree of dysphagia and planning appropriate interventions.[24]

Patterns of Aspiration

Aspiration patterns observed during FEES in stroke patients can vary. Aspiration may occur before, during, or after swallowing due to delayed swallow reflex, reduced laryngeal elevation, or inadequate airway closure. Before-swallow aspiration often results from premature spillage of the bolus into the pharynx, while during-swallow aspiration might occur due to incomplete airway protection. After-swallow aspiration is commonly linked to pharyngeal residue.[78]

Residue Patterns and Locations

Residue patterns in stroke patients typically include accumulation in the valleculae and pyriform sinuses. This can be attributed to reduced pharyngeal contraction and poor bolus clearance. FEES allows for the precise visualization of these residues, which are indicative of the swallowing inefficiency often seen in stroke patients.[88]

Laryngeal Sensation and Protective Reflexes

Laryngeal sensation and protective reflexes are often compromised in stroke patients, increasing the risk of aspiration. One type of sense cue that FEES might use to test these reactions is the presence of a bolus. Protective systems need to be treated in a certain way when they are weak, like when the cough reflex or larynx reaction is lowered.[24]

Secretion Management

It is very important to be able to control fluids when care for an individual with dysphagia. Overproduction and inadequate clearance of oropharyngeal secretions are symptoms of poor secretion control, which FEES may detect. Because aspiration of secretions may cause respiratory problems, this discovery is important.[47]

Compensatory Mechanisms

People who have had a stroke often learn to adjust for their inability to swallow. To enhance safety when eating, it may be necessary to adjust the patient's head posture or modify the size and consistency of the bolus. FEES is beneficial for physicians as it enables them to assess the efficacy of compensatory strategies and suggest appropriate modifications.[89]

PAS Findings in Stroke Cohorts

The PAS is often used to find out how much the airway has been invaded after eating. The FEES data show that people who have higher PAS scores are more likely to have entry and desire in stroke groups. This measure lets you put patients into groups based on their chance of aspiration, which lets you give them more focused care.[78]

FEES Findings Across Stroke Subtypes

varied subtypes of stroke might have varied FEES results. For example, when the primary swallowing routes are disrupted in brainstem strokes, the dysphagia that results may be more severe. Cortical strokes, on the other hand, may cause less obvious difficulties with swallowing. For proper diagnosis and treatment, it is crucial to understand these distinctions.[90]

Correlation of FEES Findings with Stroke Characteristics

Lesion Location and Swallowing Impairment Patterns

Stroke patients' patterns of swallowing difficulties are greatly affected by the site of the lesion. Researchers may examine the particulars of these issues using FEES, which is often associated with the regions of the brain that are impaired. Injury to the brainstem may result in significant dysphagia, marked by diminished pharyngeal contractions and inadequate pulmonary protection. Injury to the cortex, conversely, may postpone the initiation of swallowing..[47]

Stroke Severity (NIHSS) and Dysphagia Severity Correlation

The NIHSS is an important tool for determining the degree of severity of a stroke, and the fact that it correlates with dysphagia is intriguing. Research has shown that FEES-observed dysphagia is generally more severe in patients with higher NIHSS scores. This link shows how important it is to do thorough reviews to guide care and spot any problems before they happen.

Comparison in types of Stroke Findings

The outcomes of FEES may significantly vary between the first and later phases of a stroke. In the acute period, patients often have exacerbated dysphagia due to the direct impact of cerebral dysfunction. FEES can quickly find problems like delayed eating and the risk of aspiration during the healing phase. On the other hand, people who have had a stroke often may develop ways to compensate, and FEES may help us figure out how well these methods work and how they change over time.[43]

Predictive Value of Early FEES Assessment

Early FEES screening is very useful for predicting outcomes in stroke treatment. If eating problems are found quickly after a stroke, it may be easier to avoid aspiration asthma and other problems. Early management based on FEES data may lead to better outcomes by letting people take charge of their dysphagia and make the right changes to their treatment.[41]

Finding links between certain FEES parameters and parts of the brain

To understand how dysphagia happens in people who have had a stroke, you need to know how certain FEES factors are connected to specific brain regions. For example, a loss of larynx feeling during FEES may mean that the thalamus or sensory cortex is active, which are brain regions that handle sense information. Individualized rehab plans can better deal with the real issues with the help of this detailed map.[45]

FEES in Acute Stroke Management

Timing of First FEES Assessment After Stroke

Finding the timing of the first FEES evaluation for a stroke patient is crucial. People who recently had a stroke may show dysphagia; quick diagnosis of this issue is made possible by early screening, especially within the first 24 to 48 hours. Early FEES lowers the risk of problems such aspiration pneumonia by letting doctors rapidly evaluate the degree of eating disorders and provide suitable treatments.[41]

Impact on Nutritional Management Decisions

For people who have suffered a mild stroke, the nutritional treatment choices are much influenced by fees. Using FEES, which shows the chewing process, could help to formulate

dietary changes including changing the tastes of food and drink. While giving patients the required calorie intake, this customized method may lower their risk of choking and regurgitation.[43]

Role in Determining Feeding Strategies

People who have experienced a stroke might get great advantage from FEES as they guide best diet plans. Based on how effectively the patient can swallow and their likelihood of aspiration, it guides physicians in choosing between mouth feeding and alternative options like tube feeding. The most crucial thing is to maintain eating without compromising safety. Those with severe dysphagia particularly should pay great attention to this decision.[47]

Safety in Acute Stroke Settings

Extensive evidence has been gathered demonstrating the safety of FEES in acute stroke settings. We may do the minimally invasive procedure right at the patient's bedside if they are in a really precarious medical condition. The efficacy and safety of FEES are greatly enhanced in these settings by proper training and adherence to guidelines.[91]

Integration into Acute Stroke Care Pathways

A comprehensive approach to manage dysphagia may be assured by including FEES into acute stroke treatment plans. By making FEES a regular part of care, doctors can look at eating problems in a planned way and treat them as part of a bigger plan to handle stroke. This mix makes it possible for both better patient results and treatment continuation.[41]

Predictive Value for Aspiration Pneumonia Risk

FEES is an effective method for assessing the likelihood of aspiration pneumonia in stroke patients. Clinicians may use FEES to anticipate respiratory complications by observing occurrences such as suction and aspiration during swallowing. By using this level of foresight, we may establish methods to mitigate the danger of pneumonia, such as enhanced dining environments and improved swallowing techniques.[92]

Serial FEES Assessment in Stroke Rehabilitation

Recovery Patterns Documented Through Sequential FEES

Patients participating in rehabilitation programs post-stroke may significantly benefit by maintaining consecutive FEES recordings of their progress. Physicians may see enhancements in swallowing function over time, including a decreased risk of reflux and improved bolus clearance, via frequent patient evaluations. These patterns provide critical insights into the patient's recovery trajectory, facilitating improved treatment options.[93]

Timeframes for Reassessment

When FEES reassessments are done is very important in stroke treatment. If there is a noticeable change in swallowing abilities, it is recommended to reevaluate the patient at regular intervals, such monthly or biweekly. This method allows doctors to monitor their patients' improvement and make prompt changes to their treatment programs.[23]

Correlation with Functional Recovery Measures

Functional healing indicators and FEES data are significantly correlated in stroke patients. Enhancements seen during serial FEES are often associated with improved functional status, including the ability to do everyday activities independently and to consume food more

effectively. These connections indicate that FEES should be included into comprehensive therapeutic regimens to facilitate patient recovery.[94]

Role in Guiding Therapy Modifications

FEES is crucial for improving the treatment of stroke sufferers. The FEES system provides physicians with extensive information on swallowing, enabling the formulation of individualized treatment strategies for each patient. Based on FEES data, throat exercises or food advice may need to be changed to keep therapy successful and up to date.[45]

Long-term Outcomes Prediction

In stroke treatment, repeated FEES tests help predict how the patient will do in the long run. If clinicians can see long-term eating problems or changes, they can better plan for their patients' future needs and problems. With these prediction tools, it is much easier to plan ongoing treatment and make sure that progress continues after the first healing time.[95]

FEES-Guided Therapeutic Interventions

Dietary Modifications Based on FEES Findings

Individuals with dysphagia may significantly benefit from using FEES to modify their dietary intake. Based on the real-time picture of eating dynamics that FEES gives doctors, they may change the structure of the patient's diet by making drinks thicker or changing the way food is textured to prevent reflux. This personalized method to eating improves safety and makes sure patients get the right nutrition, which is important for their health, well-being, and life.[47]

Compensatory Strategies Validation

The FEES is very helpful for proving that compensatory methods for managing dysphagia work. It lets doctors try tools like chin tucks and head turns while eating and tells them right away if these methods lower the risk of reflux. Because of this process, clinicians can be sure that the remedial methods they pick are best for their patients.[78]

Postural Techniques Effectiveness Assessment

People who have dysphagia are often given positional techniques to help them swallow more safely and effectively. Individuals with dysphagia are often provided with positioning strategies to facilitate safer and more successful swallowing. FEES assesses the efficacy of these approaches by demonstrating changes in stomach function under various circumstances. The influence of head tilting or rotation on bolus flow and pulmonary protection may be used to optimize restorative therapies.[96]

Biofeedback Applications

People who use FEES can get biofeedback apps that tell them how well they are eating. Patients can take an active role in their treatment by seeing how their eating changes in real time and making changes on purpose. For people who are trying to get better at eating and learning new motor skills, biofeedback has been shown to be helpful.[44]

Role in NMES Therapy Guidance

FEES is crucial in the context of NMES for dysphagia. Clinicians can identify optimal electrode placements and adjust stimulation settings by observing the effects of electrical

stimulation on respiratory muscles. This tailored approach enhances the therapeutic benefits of NMES, facilitating muscle retraining and functional recovery.[43]

Integration with Exercise-Based Dysphagia Therapy

FEES may be seamlessly integrated into exercise-based dysphagia therapy, enhancing its efficacy. FEES assists physicians in formulating training strategies that address particular issues by providing comprehensive insights into the mechanics of ingestion. Comprehensive treatment tailored to the patient's individual requirements results in improved nutrition and enduring advantages.[97]

Comparison of FEES with Other Assessment Methods

FEES vs. VFSS: Concordance and Discordance

The FEES and VFSS are two prevalent methods for assessing dysphagia. The FEES and VFSS systems often concur on several aspects including the identification of aspiration and penetration events. Discrepancies may sometimes occur. For instance, because to its direct assessment of laryngeal sensation, FEES may be more effective in detecting silent aspiration compared to other techniques. Additionally, VFSS gives a fuller picture of the oral and gastric stages than FEES, which might sometimes show more eating problems. [98]

Accuracy in Detecting Aspiration

There is even more benefit to exercise-based dysphagia therapy when FEES is added to it. FEES helps therapists make problem-specific training plans by giving them detailed

information on how people eat. Patients have better eating habits and feel better for longer when their care is thorough and individualized to their specific needs.[78]

Detection of Different Pathophysiological Mechanisms

FEES is very good at finding the different pathological paths that lead to dysphagia. Because it shows the pharyngeal phase better than VFSS, it can pick up on subtle changes in muscle rhythm and time that VFSS might miss. With this in-depth look, we may be able to better understand what causes dysphagia, which in turn leads to more effective treatment methods.[99]

Clinical Decision-Making Impact Comparison:

FEES has a big effect on clinical decision-making, especially when assessments are done in real time. Because it responds right away, dietary advice and medical treatments can be changed quickly. Even though VFSS is thorough, it may need study after the fact, which could delay making a choice. Because FEES is instant, doctors can move more quickly, which leads to better results for patients.[100]

Cost-Effectiveness Analyses

Most of the time, FEES is the more cost-effective choice when compared to VFSS. Since it can be done at the table, there is no longer a need for transportation or certain tools. In situations where money is tight, FEES may be a cost-effective choice because it is easy to get. This lowers healthcare costs.[101]

Patient Preference Studies

According to opinion polls, FEES is a popular choice among patients because it is non-radiative and easy to give at the bedside. Patients often choose FEES over VFSS because they

do not have to use any tools and they do not have to be exposed to radiation. Patients with dysphagia may be more likely to stick to their treatment plans and be happy generally if they want to.[47]

Prognostic Value of FEES in Stroke Patients

Predictive Factors for Dysphagia Recovery

FEES gives us useful information about the things that can help us guess how well stroke patients will heal from dysphagia. The detailed visualization of swallowing mechanics allows clinicians to identify specific deficits and potential for recovery. Factors such as the presence of laryngeal sensation and voluntary cough during FEES are strong indicators of positive outcomes in dysphagia management. Early intervention based on these predictive factors can significantly enhance recovery trajectories.[44]

Correlation with Functional Outcomes

FEES findings correlate closely with functional outcomes in stroke rehabilitation. The ability to assess swallowing safety and efficiency through FEES helps predict the patient's overall functional recovery. Improvements observed in FEES often mirror enhancements in activities of daily living, as patients regain the ability to swallow safely and independently. This correlation underscores the importance of including FEES as part of a comprehensive stroke rehabilitation program.[43]

Risk Prediction for Pneumonia

One of the critical prognostic values of FEES is its role in predicting the risk of aspiration pneumonia. By identifying silent aspiration and other swallowing impairments, FEES allows clinicians to implement preventive measures, such as dietary modifications and swallowing

exercises, to mitigate pneumonia risk. These proactive strategies are essential in reducing pneumonia incidence, which is a common complication in stroke patients with dysphagia.[47]

Impact on Hospital Length of Stay

FEES can significantly impact hospital length of stay for stroke patients. Accurate assessment of swallowing function enables timely interventions, reducing complications such as aspiration pneumonia, which can prolong hospitalization. By optimizing swallowing management, FEES contributes to more efficient discharge planning and reduced healthcare costs, highlighting its value in acute care settings.[102]

Relationship with Long-Term Disability Measures

The relationship between FEES findings and long-term disability measures in stroke patients is noteworthy. Persistent dysphagia, as identified through FEES, can be a predictor of ongoing disability and reduced quality of life. By addressing swallowing impairments early and effectively, FEES-guided interventions can help minimize long-term disability, supporting better overall recovery and reintegration into the community.[103]

Mortality Prediction Models Incorporating FEES Data

Incorporating FEES data into models that forecast mortality may enhance their accuracy for stroke patients. Identified hazards of severe dysphagia and reflux are critical in assessing the likelihood of mortality. FEES provides extensive information about indicators that suggest improvement in dysphagia among stroke patients.[104]

Recent Advances and Future Directions in FEES

Technological Innovations in FEES Equipment

FEES gear works much better now that new technologies have been developed. High-definition images and better fiberoptic technologies have made it possible for people to see better, which has led to more accurate assessments of how eating works. Because of these improvements, patients do better because dysphagia can be diagnosed earlier and treatment choices are better thought out.[100]

Machine Learning and Automated Analysis Approaches

The use of machine learning in FEES has opened the door to new ways of automatically diagnosing swallowing problems. Machine learning systems can sort through huge amounts of data to find trends and results. This lets doctors see things that they could not see before because it would have been too expensive to have a person do the research. This technology makes it easier to diagnose dysphagia and tailor treatment plans to each person's needs by picking up on small signs of the problem.[95]

Telemedicine Applications for FEES

Telemedicine has changed the way FEES are delivered in rural and neglected areas. Specialists can now virtually do FEES exams through telemedicine. This saves patients the time and money of traveling and lets them get advice and exams in real time. This method makes patients feel better, lowers the cost of health care, and makes treatment more available to more people.[105]

Combined Modality Assessments

More and more, FEES is being used with other testing tools to get a full picture of how well someone can swallow. By combining FEES with other imaging methods, like videofluoroscopy or high-resolution manometry, it may be possible to get a fuller picture of

how the patient drinks. This unified method makes both the correctness of the evaluation and the success of the treatment better.[106]

High-Resolution Manometry with FEES

High-resolution manometry is used to measure pressure in the mouth and stomach in great detail, along with the visual information that is sent by FEES. By using both approaches together, doctors can look at both the structural and functional parts of eating. This helps them figure out the exact type of swallowing disorder and give the right treatment.[101]

Research Gaps and Opportunities

These changes, on the other hand, are not enough to fill in the big information gaps in FEES. In the future, researchers could look into how changes in technology affect clinical practice, the long-term benefits of different FEES treatments, and how to make standards for the use of mixed modes more consistent. We need to fill in these gaps if we want to learn more about eating problems and find better ways to treat them.[107]

MATERIALS AND METHODS

Study Design and Study Setting

The cross-sectional study was done at the R.L. Jalappa Hospital and Research Centre in Tamaka, Kolar, India. It is connected to Sri Devaraj Urs Medical College.

Study Period

From May 2023 to October 2024, a total of 18 months were spent on the project.

Ethics Committee Approval

Prior to implementation, the study protocol received formal approval from the Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar. The approval process included comprehensive review of participant safety measures, informed consent procedures, and data confidentiality protocols. All research activities were conducted in accordance with the Declaration of Helsinki guidelines for research involving human subjects. Written informed consent was obtained from all participants or legally authorized representatives in cases where patients demonstrated cognitive impairment affecting decisional capacity.

Inclusion Criteria

The study enrolled patients meeting the following criteria:

1. Age between 40 and 70 years
2. Clinical diagnosis of stroke confirmed by neuroimaging (CT or MRI)
3. Presence of dysphagia as determined by initial bedside swallowing assessment

4. Cognitive status adequate for participation in FEES procedure (if not initially, then upon improvement)
5. Ability to provide informed consent (personally or through legally authorized representative)

Exclusion Criteria

Patients were excluded from participation based on the following criteria:

1. History of malignancy involving the upper aerodigestive tract and/or prior treatment for such conditions
2. Previous major neck lacerations or surgical interventions affecting pharyngolaryngeal anatomy
3. Presence of spinal deformities that could independently affect swallowing biomechanics
4. Diagnosed degenerative disorders of the brain that might confound dysphagia aetiology (e.g., Parkinson's disease, Alzheimer's disease, motor neuron disease)
5. Ventilator dependency precluding safe implementation of the FEES procedure

Sample Size Estimation

The sample size was calculated using Cochran's formula: $n = Z^2 \times p \times (1-p) / d^2$, where Z represents the standard normal variate at 5% type I error (1.96), p denotes the expected proportion in the population, and d indicates the absolute error or precision. Based on findings by Zuleika et al. (2020), who documented a 62.5% prevalence of dysphagia in post-stroke patients (n=32) using FEES evaluation in an Indonesian tertiary care setting,[7] the

present study adopted p=0.625. With a precision level of 12% (d=0.12) and 95% confidence interval, the calculation yielded a minimum required sample size of 63 participants.

$n = (1.96)^2 \times 0.625 \times (1-0.625) / (0.12)^2$

$n = 3.8416 \times 0.625 \times 0.375 / 0.0144$ $n = 0.9004 / 0.0144$

$n = 62.53$ (rounded to 63 participants)

Sampling Method

Consecutive sampling was employed to recruit eligible participants from the neurology department of R.L. Jalappa Hospital. All patients admitted with stroke diagnosis during the study period were screened for eligibility according to the inclusion and exclusion criteria. Eligible patients were approached for participation after initial neurological stabilization and establishment of candidacy for FEES evaluation.

Data Collection Procedure

Patient assessment followed a standardized protocol implemented by a multidisciplinary team. Initially, comprehensive demographic and clinical data were collected, including age, gender, occupational status, comorbidities (DM, HTN, IHD, bronchial asthma), smoking status, alcohol consumption, and tobacco use. The duration of dysphagia complaints was documented as: less than 24 hours, one day to one week, one day to one month, or more than 30 days.

Neurological assessment was performed by a senior physician using the GCS to categorize severity as mild (GCS 13-15), moderate (GCS 9-12), or severe (GCS $\leq$8). All patients underwent neuroimaging (CT or MRI) to confirm stroke diagnosis and determine lesion location, which was classified according to vascular territory and neuroanatomical region

(e.g., brainstem, cerebral cortex, subcortical regions, MCA territory, ACA territory, PCA territory).

Once patients achieved adequate cognitive status for participation, FEES was performed by an experienced otorhinolaryngologist using standard equipment with a 3.2 mm flexible fiberoptic nasopharyngoscope. During the procedure, an emergency trolley remained accessible to manage potential complications. The FEES protocol evaluated multiple parameters: vocal cord/arytenoid oedema (absent, mild, moderate, severe), mucosal characteristics including dryness and ulceration, pyriform fossa pooling (absent, mild, moderate, severe), laryngeal spillover patterns (specific anatomical locations), transit delay severity (absent, mild, moderate, severe), nasal regurgitation, presence of adynamic segments, and aspiration (categorized as minor or major).

Swallowing function was assessed using standardized bolus trials (thin liquid, thick liquid, semi-solid, solid) with repeat swallow performance documented (second vs. third attempt requirement). Dysphagia severity was classified using the Dysphagia Outcome and Severity Scale (DOSS), a validated 7-point scale ranging from Level 1 (severe dysphagia) to Level 7 (normal swallowing). The DOSS has demonstrated strong inter-rater reliability (κ=0.79) and high correlation with videofluoroscopic findings in neurogenic dysphagia assessment.

Follow-up FEES evaluations were conducted at three months post-stroke to document recovery patterns, with clinical outcome categorized as improved, minimally improved, no improvement, or deceased.

Figure 7: FEES under progress

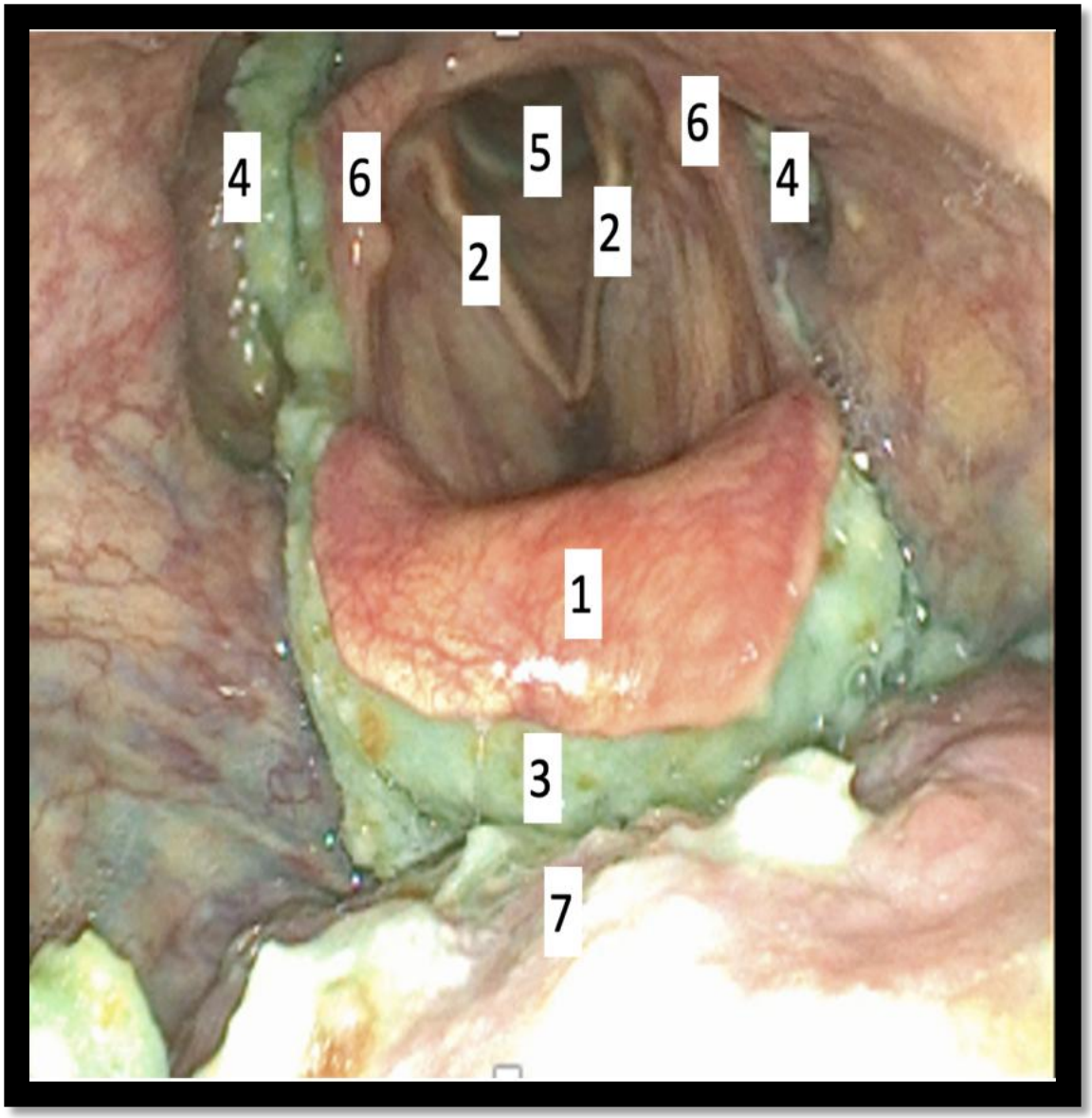


Figure 8: FEES showing the structures

1:Epiglottis

2: True Vocal Folds

3: Valeculae

4: Pyriform Fossa

5: Subglottis

6: Arytenoids

7: Base of Tongue

8. Aryepiglottic folds

9. Lat. Pharyngeal walls

Data Analysis

Statistical analysis was performed using SPSS version 26.0 (IBM Corp., Armonk, NY). Descriptive statistics were calculated for demographic and clinical variables, with continuous data presented as mean, median, mode, and standard deviation, and categorical data presented as frequencies and percentages. The primary analysis examined associations between stroke location and dysphagia parameters using Fisher's Exact Test, which was selected as the appropriate statistical method for categorical data analysis given the cell frequencies in contingency tables. This non-parametric test provided robust analysis without assuming normal distribution. For purposes of analysis, stroke locations were categorized into six primary groups: ACA territory, cerebral cortex, MCA territory, PCA territory, periventricular area, and subcortical. Dysphagia severity was classified based on DOSS scores: Level 5 (mild), Level 4 (mild-moderate), Level 3 (moderate), Level 2 (moderately severe), and Level 1 (severe). Statistical significance was established at $p<0.05$ for all analyses.

RESULTS

Table 3: Age Distribution of Post-Stroke Dysphagia Patients

Age	
Mean	61.49
Median	65.00
Mode	69
Std. Deviation	8.727

The study cohort demonstrated a mean age of 61.49 years (SD = 8.727) with a median age of 65.00 years. The modal age was 69 years, indicating a predominantly elderly population.

Table 4: Gender Distribution

Gender	Frequency	Percent
Female	20	31.7
Male	43	68.3
Total	63	100.0

Males constituted the predominant demographic group (68.3%, n=43) compared to females (31.7%, n=20).

The occupational demographics revealed a heterogeneous distribution with agricultural professions and domestic roles (housewives 22.2%, n=14) representing the largest cohorts.

Business professionals constituted 20.6% (n=13), while clerical workers, managers, drivers, attendants, and shopkeepers collectively represented approximately 23.8% of the study population.

Table 5: Duration of Dysphagia Complaints

Duration of complaints	Frequency	Percent
Less than 24 hours	28	44.4
More than 30 days	6	9.5
One day to one month	25	39.7
One day to one week	4	6.3
Total	63	100.0

Acute symptom onset (<24 hours) was documented in 44.4% (n=28) of cases, whereas 39.7% (n=25) reported symptoms persisting between one day and one month. Subacute presentations (one day to one week) were observed in 6.3% (n=4), and chronic dysphagia (>30 days) was noted in 9.5% (n=6).

Table 6: Previous History of Similar Episodes

Presence of previous past history	Frequency	Percent
No Past history	36	57.1
Presence of similar episode	27	42.9
Total	63	100.0

A majority of patients (57.1%, n=36) presented without prior history of dysphagia, while 42.9% (n=27) reported experiencing similar episodes previously.

Table 7: Alcohol Consumption Habits

Alcohol habit	Frequency	Percent
No	39	61.9
Yes	24	38.1
Total	63	100.0

Alcohol consumption was reported in 38.1% (n=24) of the study population, with 61.9% (n=39) abstaining from alcohol.

Table 8: Tobacco Chewing Habits

Tobacco chewing habit	Frequency	Percent
No	62	98.4
Yes	1	1.6
Total	63	100.0

Tobacco chewing was documented in only 1.6% (n=1) of patients, with 98.4% (n=62) reporting no such habit.

Table 9: Combined Alcohol and Smoking Habits

Both alcohol and smoking habit	Frequency	Percent
No	51	81.0
Yes	12	19.0
Total	63	100.0

Concurrent alcohol consumption and smoking was observed in 19.0% (n=12) of patients, with 81.0% (n=51) reporting absence of this combined habit.

Table 10: Smoking Habits

Smoking habit	Frequency	Percent
No	39	61.9
Yes	24	38.1
Total	63	100.0

Smoking was reported in 38.1% (n=24) of patients, identical to the proportion of alcohol consumers, while 61.9% (n=39) were non-smokers.

Figure 9: Personal habits

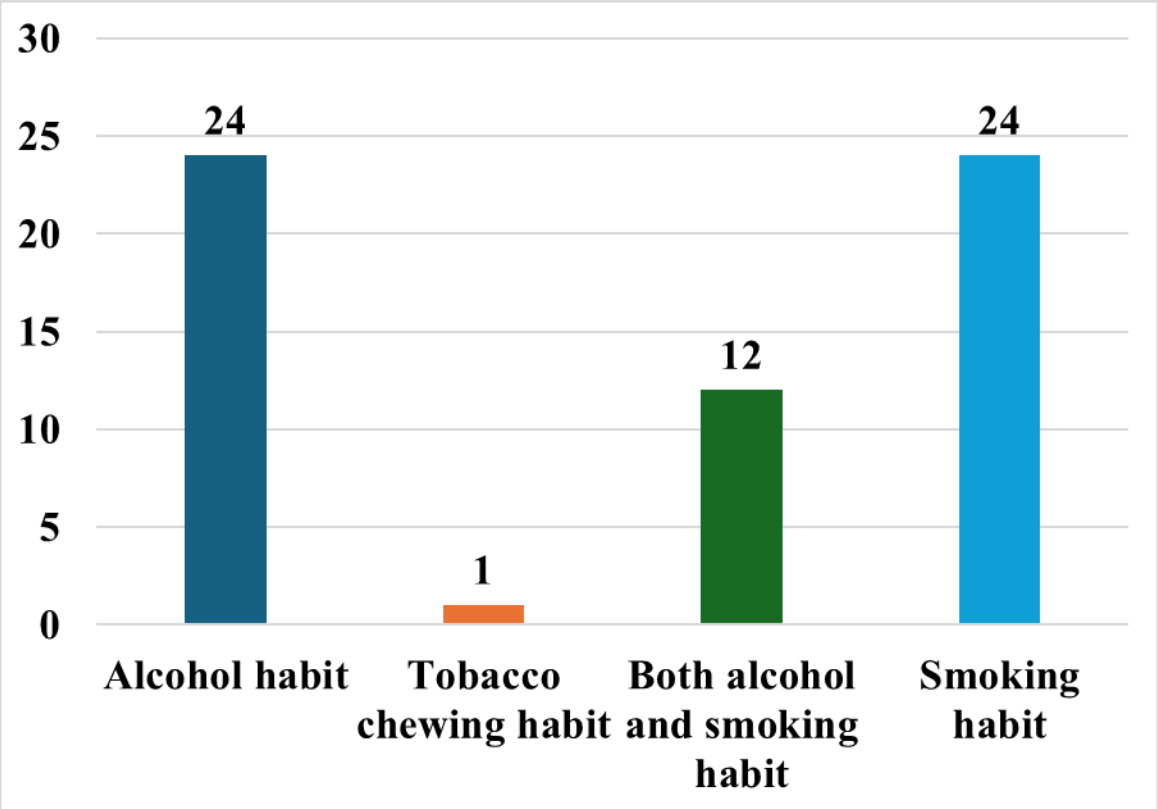


Table 11: GCS Scores at Presentation in Post-Stroke Dysphagia Patients

GCS severity category	Frequency	Percent
Severe	27	42.9
Moderate	5	8
Mild	31	49.2
Total	63	100

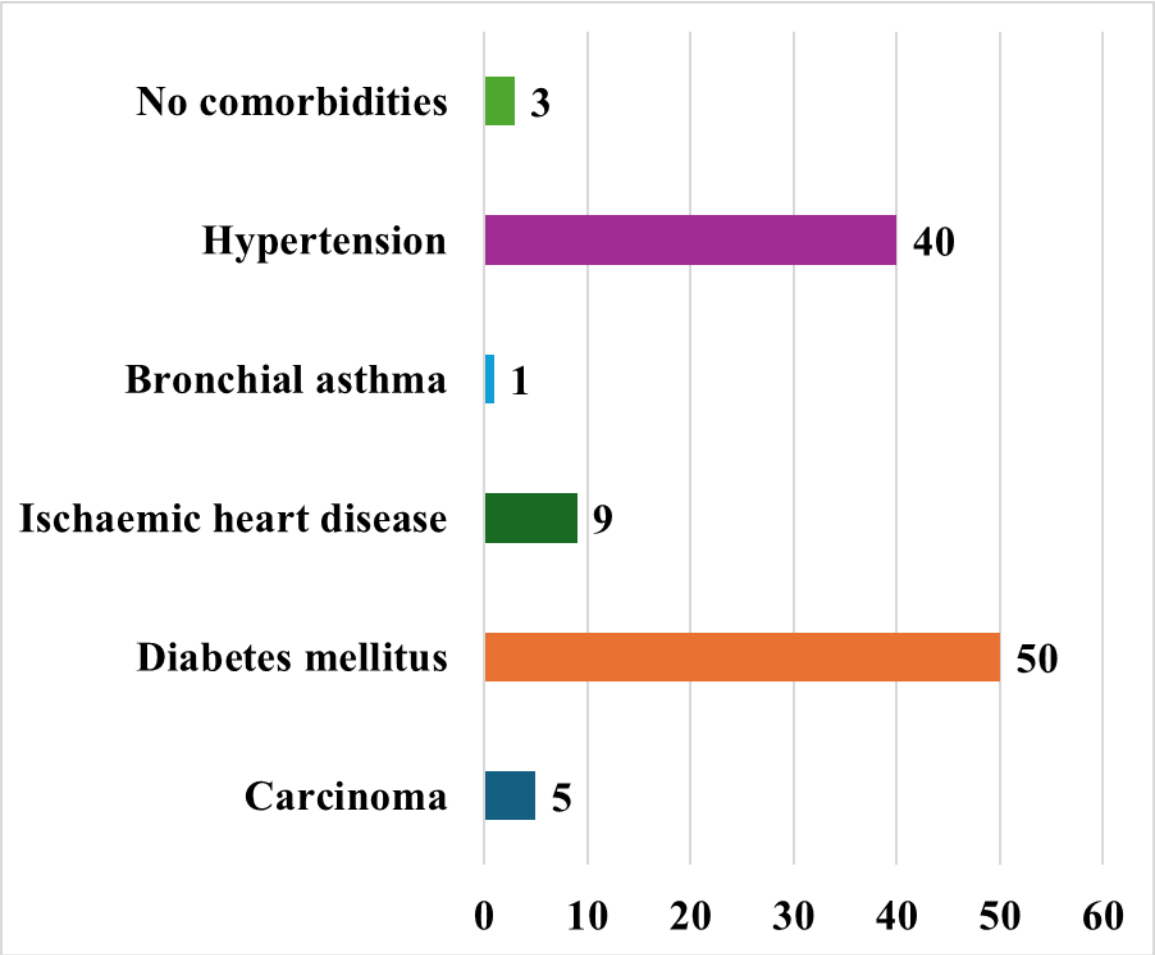
Neurological status assessment revealed that mild impairment (GCS 13-15) was observed in 49.2% of patients. Moderate impairment (GCS 9-12) was observed in approximately 8% of cases, while severe neurological compromise (GCS ≤8) was documented in 42.9% of patients.

Table 12: GCS Scores during FEES

GCS severity category	Frequency	Percent
Moderate	6	9.5
Mild	57	90.5
Total	63	100

Neurological status assessment during FEES revealed that mild impairment (GCS 13-15) was observed in 90.5% of patients. Moderate impairment (GCS 9-12) was observed in approximately 9.5% of cases.

Figure 10: Comorbidity Distribution



Type 2 DM represented the predominant comorbidity pattern (79.3%, n=50), followed by isolated HTN (63.5%, n=40). Only 4.8% (n=3) of patients were without any documented comorbidities.

Table 13: Repeat Swallow Performance in Post-Stroke Dysphagia Patients

Repeat swallow	Frequency	Percent
Present and swallow with 2nd swallow	44	69.8
Present and swallow with 3rd swallow	19	30.2
Total	63	100.0

A majority of patients (69.8%, n=44) successfully completed swallowing on the second attempt, while 30.2% (n=19) required a third attempt. This finding indicates significant but potentially recoverable neuromuscular impairment of the swallowing mechanism in the post-stroke context.

Table 14: Vocal Cord and Arytenoid Oedema

Oedema of vocal cord and arytenoids	Frequency	Percent
Absent	10	15.9
Mild	30	47.6
Moderate	10	15.9
Severe	13	20.6
Total	63	100.0

Endoscopic evaluation revealed mild oedema in 47.6% (n=30) of cases, moderate oedema in 15.9% (n=10), and severe oedema in 20.6% (n=13).

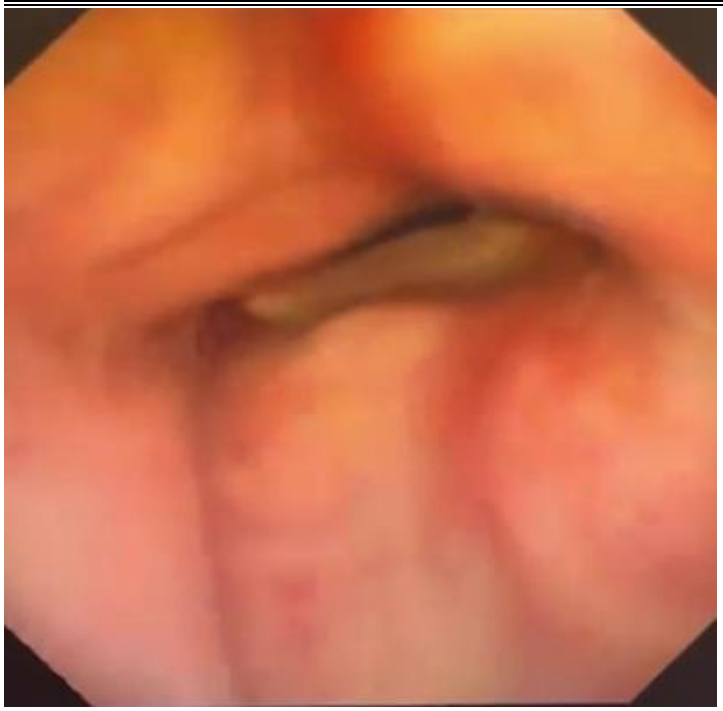


Figure 11: Tracheal Edema

Mucosal ulceration was uniformly absent (100%, n=63) across the entire cohort.

Table 15: Mucosal Dryness

Dryness of mucosa	Frequency	Percent
Absent	0	0.0
Mild	35	55.6
Moderate	21	33.3
Severe	7	11.1
Total	63	100.0

Mild mucosal dryness predominated (55.6%, n=35), with moderate dryness observed in 33.3% (n=21) and severe dryness in 11.1% (n=7).

Table 16: Pyriform Fossa Pooling

Pooling of pyriform fossa	Frequency	Percent
Absent	1	1.6
Mild	32	50.8
Moderate	22	34.9
Severe	8	12.7
Total	63	100.0

Endoscopic evaluation demonstrated mild pooling in 50.8% (n=32) of patients, moderate pooling in 34.9% (n=22), and severe pooling in 12.7% (n=8). Absence of pooling was documented in 1.6% (n=1) of cases.

Table 17: Laryngeal Spillover Patterns

Spill over into the larynx	Frequency	Percent
Absent	8	12.7
Aryepiglottic fold spill over present	41	65.1
Epiglottis-spill over present	3	4.8
Pharyngoepiglottic fold spillover present	5	7.9
Spill over in the nasopharynx	2	3.2
Spill over present just above vocal cords, vallecula	4	6.3
Total	63	100.0

Various patterns of laryngeal spillover were documented, with aryepiglottic fold being most prevalent (65.1%, n=41), followed by Pharyngoepiglottic fold spillover (7.9%, n=5). Complete absence of spillover was noted in 12.7% (n=8) of cases.

Table 18: Transit Delay Severity

Delayed transit	Frequency	Percent
Absent	9	14.3
Mild	27	42.9
Moderate	22	34.9
Severe	5	7.9
Total	63	100.0

Mild transit delay was observed in 42.9% (n=27) of patients, moderate delay in 34.9% (n=22), and severe delay in 7.9% (n=5). Normal transit was documented in 14.3% (n=9) of cases.

Table 19: Nasal Regurgitation

Nasal regurgitation	Frequency	Percent
Absent	57	90.5
Present	6	9.5
Total	63	100.0

Nasal regurgitation was largely absent (96.8%, n=61), with only 3.2% (n=2) of patients demonstrating this feature.

Table 20: Presence of Adynamic Segment

Adynamic segment		Frequency	Percent
Absent		57	90.5
Present (area of adynamic segment)	Vocal cord	3	4.8
	Pyriform sinus	2	3.1
	Arytenoid	1	1.6
Total		63	100.0

An adynamic segment was documented in only 9.5% (n=6) of patients, with 90.5% (n=57) showing no evidence of this feature. Area of adynamic segment shows 4.8% in vocal cord, 3.1% in pyriform sinus and 1 in arytenoid.

Table 21: Minor Aspiration

Minor aspiration	Frequency	Percent
Absent	27	42.9
Present	36	57.1
Total	63	100.0

Minor aspiration was observed in 57.1% (n=36) of patients, indicating a significant prevalence of this concerning clinical feature. In 42.9% (n=27) of cases, no evidence of minor aspiration was detected.

Table 22: Major Aspiration

Major aspiration	Frequency	Percent
Absent	56	88.9
Present	7	11.1
Total	63	100.0

Major aspiration was documented in 11.1% (n=7) of patients, with 88.9% (n=56) showing no evidence of this severe complication.

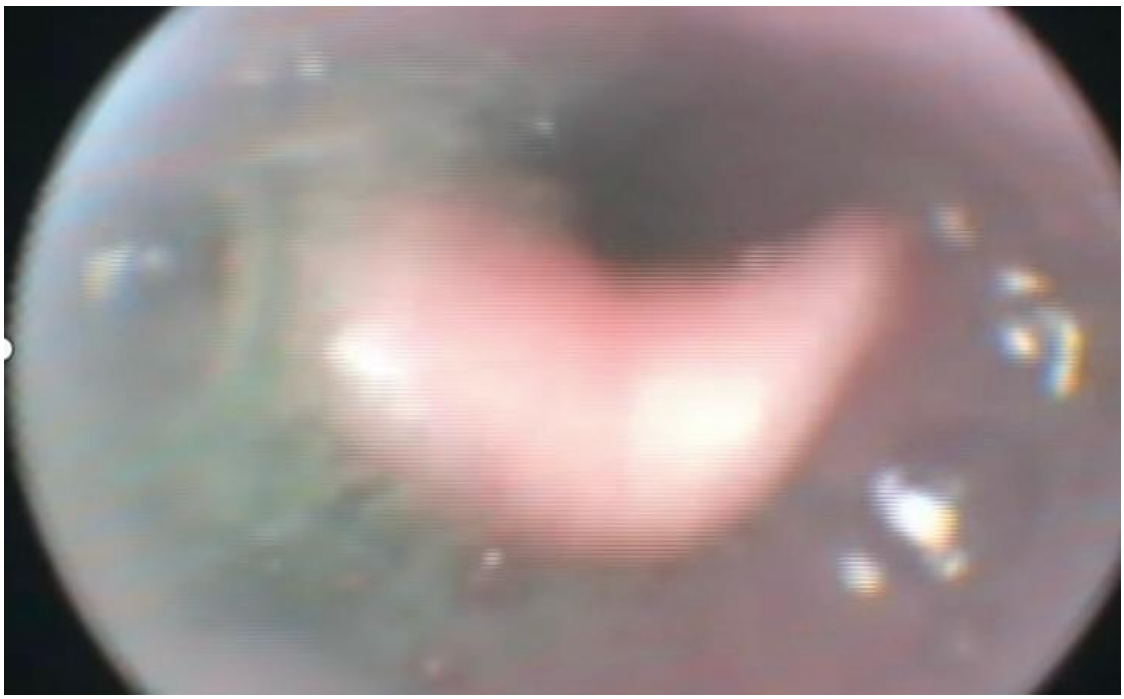


Figure 12: Major Aspiration

Table 23: Tracheostomy Status

Tracheostomy performed or not	Frequency	Percent
No	56	88.9
Yes	7	11.1
Total	63	100.0

Tracheostomy was performed in 11.1% (n=7) of patients, with 88.9% (n=56) not requiring this intervention.

Table 24: Dysphagia Severity Classification

Severity of dysphagia	Frequency	Percent
Mild dysphagia	21	33.3
Mild to moderate dysphagia	27	42.9
Moderate dysphagia	4	6.3
Profound dysphagia	2	3.2
Severe dysphagia	9	14.3
Total	63	100.0

Mild dysphagia was documented in 33.3% (n=21) of patients, with mild-to-moderate dysphagia observed in 42.9% (n=27). Moderate dysphagia was present in 6.3% (n=4), while severe dysphagia accounted for 14.3% (n=9) of cases.

Figure 13: Dysphagia Severity Classification

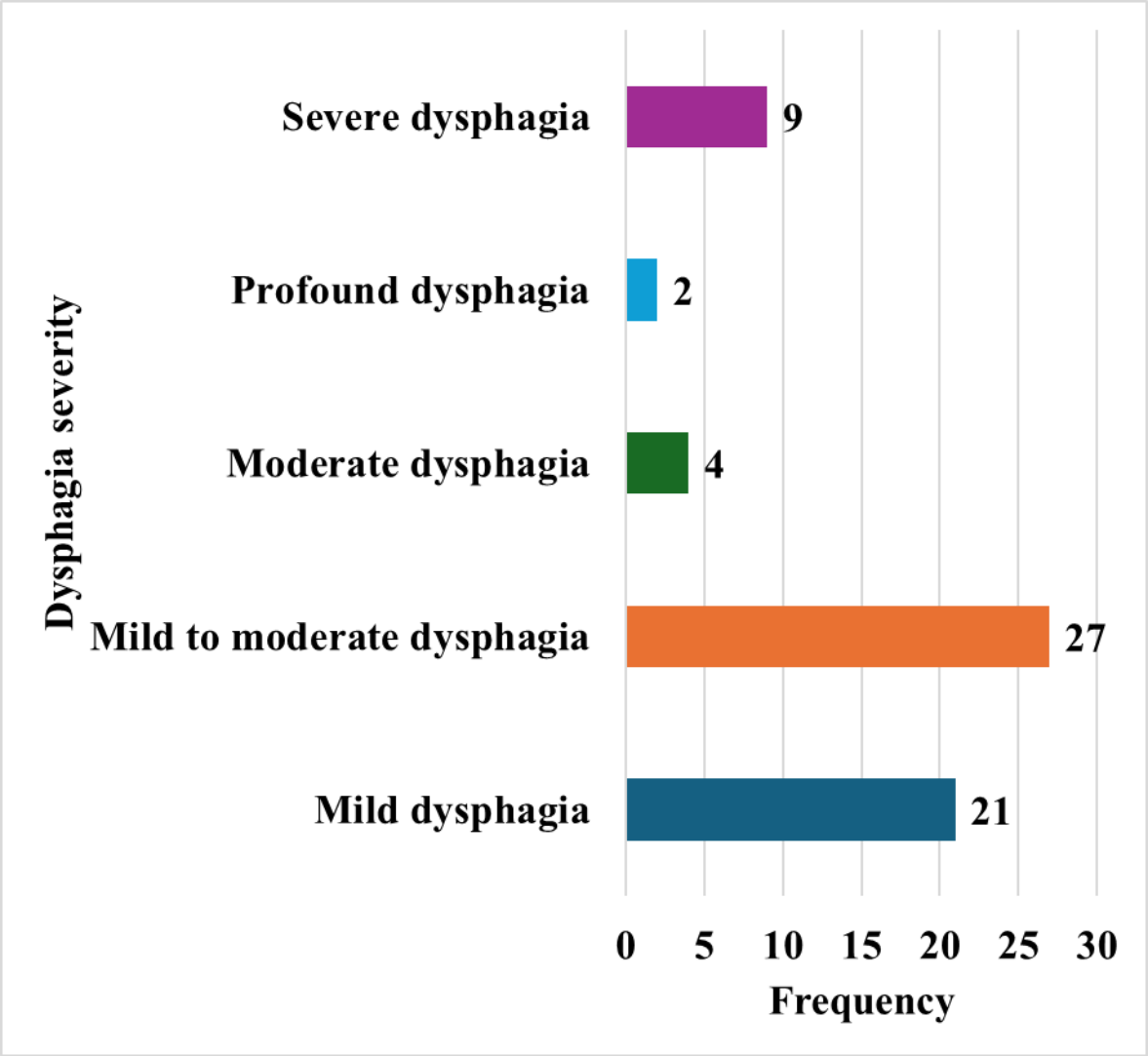


Table 25: Dysphagia Outcome and Severity Scale (DOSS) Scores

DOSS score	Frequency	Percent
1	2	3.2
2	2	3.2
3	4	6.3
4	27	42.9
5	28	44.4
Total	63	100.0

DOSS score distribution revealed Level 5 (mild dysphagia) in 44.4% (n=28) and Level 4 (mild-moderate dysphagia) in 42.9% (n=27) of patients. Level 3 (moderate dysphagia) was observed in 6.3% (n=4), while Levels 1 and 2 (severe and moderately severe dysphagia) each accounted for 3.2% (n=2) of cases.

Figure 14: Dysphagia Outcome and Severity Scale (DOSS) Scores

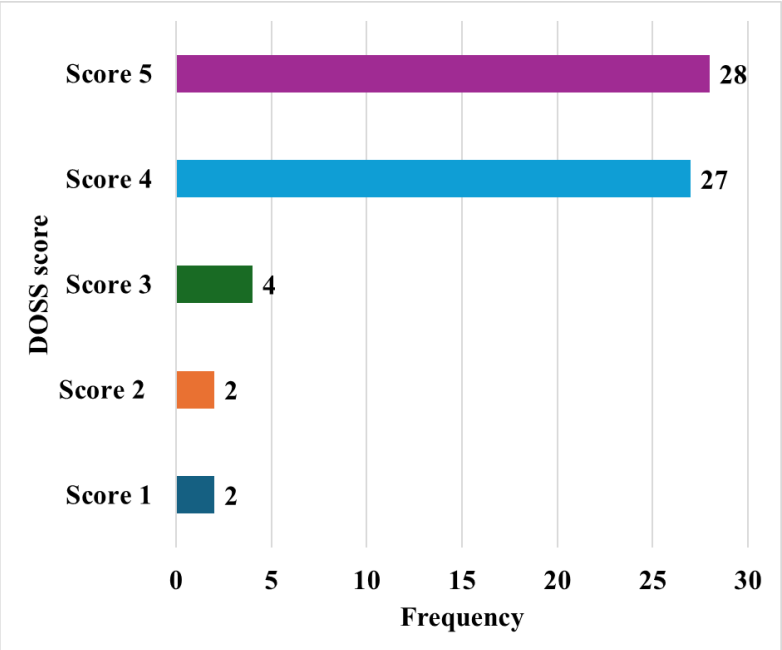


Table 26: Neuroanatomical Distribution of Stroke Lesions

Area of stroke	Frequency	Percent
Brainstem	2	3.2
Cerebral cortex	3	4.8
Cerebral cortex - Left parietal	2	3.2
Cerebral cortex - Left temporal	2	3.2
Cerebral cortex - Right parietal	2	3.2
Cortical and subcortical	3	4.8
Lateral ventricle	1	1.6
Left PCA territory	2	3.2
Left subcortical	16	25.4
MCA territory	10	15.9
MCA territory and subcortical	2	3.2
PCA territory	2	3.2
Periventricular area	3	4.8
Right ACA territory	4	6.3
Right subcortical	4	6.3
Subcortical	5	7.9
Total	63	100.0

Left subcortical strokes represented the most common lesion location (25.4%, n=16), followed by MCA territory infarcts (15.9%, n=10). Right subcortical lesions (6.3%, n=4), right ACA territory infarcts (6.3%, n=4), and subcortical strokes without lateralization specification (7.9%, n=5) were also notable. Brainstem involvement was documented in 3.2% (n=2) of cases. This neuroanatomical distribution highlights the complex neural control of swallowing and the variable impact of different lesion locations on deglutition.

Table 27: Clinical Outcomes

Follow up	Frequency	Percent
Dead	5	7.9
Improved	48	76.2
Minimal improvement	6	9.5
No improvement	4	6.4
Total	63	100.0

Clinical improvement was documented in 84.1% (n=48) of patients, with minimal improvement observed in 9.5% (n=6) and no improvement in 6.4% (n=4). Mortality was recorded in 7.9% (n=5) of cases.

Figure 15: Clinical Outcomes

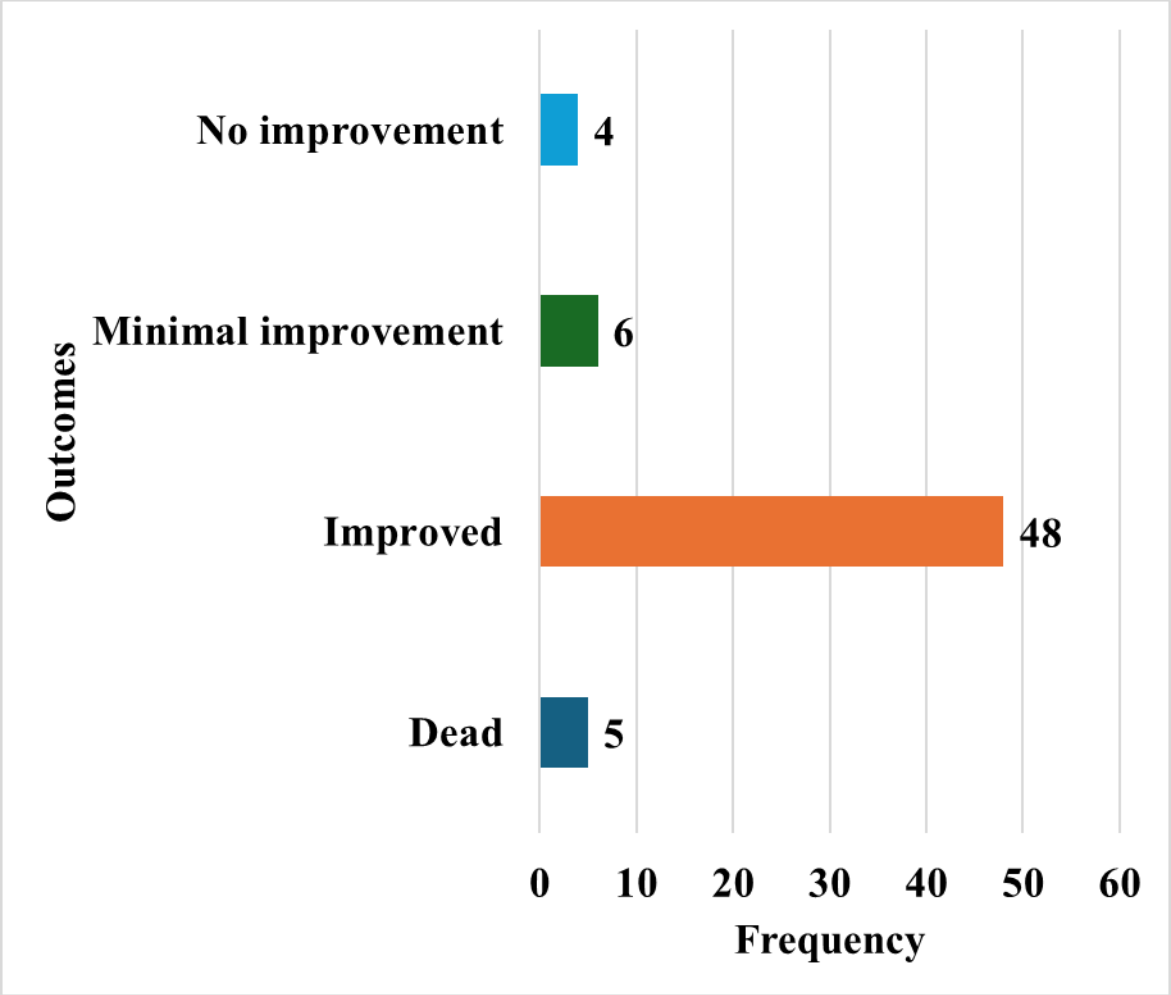


Table 28: Association Between Stroke Location and Dysphagia Severity

Area of stroke		Severity of dysphagia					Total
		Mild	Mild to moderate	Moderate	Profound	Severe	
ACA territory	n	2	4	0	0	2	8
	%	25.0%	50.0%	0.0%	0.0%	25.0%	100%
Cerebral cortex	n	2	4	3	0	0	9
	%	22.2%	44.4%	33.3%	0.0%	0.0%	100%
MCA territory	n	4	6	0	2	0	12
	%	33.3%	50.0%	0.0%	16.7%	0.0%	100%
PCA territory	n	0	0	0	0	7	7
	%	0.0%	0.0%	0.0%	0.0%	100.0%	100%
Periventricular area	n	1	2	0	0	0	3
	%	33.3%	66.7%	0.0%	0.0%	0.0%	100%
Subcortical	n	12	11	1	0	0	24
	%	50.0%	45.8%	4.2%	0.0%	0.0%	100%
Total	n	21	27	4	2	9	63
	%	33.3%	42.9%	6.3%	3.2%	14.3%	100%

Fisher's Exact Test value: 45.471, p-value = 0.007

The analysis reveals a statistically significant association between stroke location and dysphagia severity ($p = 0.007$). The most striking finding is that all PCA territory strokes (100%) manifested as severe dysphagia, representing a distinct clinical pattern compared to other lesion locations. ACA territory strokes demonstrated a heterogeneous severity profile with 25% presenting with severe dysphagia, 50% with mild-to-moderate dysphagia, and 25% with mild dysphagia. MCA territory strokes exhibited considerable clinical variability, including profound dysphagia in 16.7% of cases. Subcortical lesions demonstrated a predominance of milder presentations, with 50% classified as mild and 45.8% as mild-to-moderate dysphagia. These findings suggest substantial neuroanatomical specificity in the clinical manifestation of swallowing dysfunction.

Table 29: Association Between Stroke Location and Clinical Outcome

Area of stroke		Follow up		Total
		Improved	Poor improvement or dead	
ACA territory	n	7	1	8
	%	87.5%	12.5%	100.0%
Cerebral cortex	n	7	2	9
	%	77.8%	22.2%	100.0%
MCA territory	n	10	2	12
	%	83.3%	16.7%	100.0%
PCA territory	n	1	6	7
	%	14.3%	85.7%	100.0%
Periventricular area	n	2	1	3
	%	66.7%	33.3%	100.0%
Subcortical	n	21	3	24
	%	87.5%	12.5%	100.0%
Total	n	48	15	63
	%	76.2%	23.8%	100.0%

Fisher's Exact Test value: 2.164, p-value = 0.899

Statistical analysis did not demonstrate a significant association between stroke location and clinical outcome (p = 0.899). Improvement rates varied considerably across lesion locations, ranging from 14.3% for PCA territory strokes to 87.5% for both ACA territory and subcortical lesions. Notably, PCA territory strokes demonstrated the highest proportion of poor improvement or mortality (85.7%), despite the non-significant p-value. This finding suggests that while certain lesion locations may be associated with poorer prognosis, factors beyond neuroanatomical considerations likely exert significant influence on recovery potential in post-stroke dysphagia.

Table 30: Association Between Stroke Location and Vocal Cord/Arytenoid Oedema

Area of stroke		Oedema of vocal cord and arytenoids				Total
		Absent	Mild	Moderate	Severe	
ACA territory	n	3	2	1	2	8
	%	37.5%	25%	12.5%	25.0%	100.0%
Cerebral cortex	n	3	3	2	1	9
	%	33.3%	33.3%	22.3%	11.1%	100.0%
MCA territory	n	2	3	4	3	12
	%	16.7%	25.0%	33.3%	25%	100.0%
PCA territory	n	0	0	1	6	7
	%	0.0%	0.0%	14.3%	85.7%	100.0%
Periventricular area	n	0	1	2	0	3
	%	0.0%	33.3%	66.7%	0.0%	100.0%
Subcortical	n	2	21	0	1	24
	%	8.3%	87.5%	0.0%	4.2%	100.0%
Total	n	10	30	10	13	63
	%	15.9%	47.6%	15.9%	20.6%	100.0%

Fisher's Exact Test value: 31.26, p-value = 0.007

This table demonstrates the significant association between stroke location and laryngeal oedema severity (Fisher's Exact Test=31.26, p=0.007). PCA territory strokes exhibited the highest prevalence of severe oedema (85.7%), with no cases presenting with absent or mild oedema. This contrasts markedly with subcortical strokes, where 87.5% presented with mild oedema and only 4.2% with severe manifestations. MCA territory strokes showed considerable variability in presentation, with 25% exhibiting severe oedema. ACA territory strokes demonstrated a heterogeneous profile, with 25% presenting with severe oedema despite 37.5% showing no oedema. Overall, laryngeal oedema was present in 84.1% of the cohort, with 47.6% exhibiting mild, 15.9% moderate, and 20.6% severe presentations. These findings suggest distinct pathophysiological mechanisms affecting laryngeal vasculature and innervation according to specific vascular territories, with posterior circulation involvement strongly associated with more severe inflammatory changes.

Table 31: Association Between Stroke Location and Mucosal Dryness

Area of stroke		Dryness of mucosa				Total
		Absent	Mild	Moderate	Severe	
ACA territory	n	0	6	2	0	8
	%	0.0%	75.0%	25.0%	0.0%	100.0%
Cerebral cortex	n	0	8	1	0	9
	%	0.0%	88.9%	11.1%	0.0%	100.0%
MCA territory	n	0	6	4	2	12
	%	0.0%	50.0%	33.3%	16.7%	100.0%
PCA territory	n	0	0	2	5	7
	%	0.0%	0.0%	28.6%	71.4%	100.0%
Periventricular area	n	0	2	1	0	3
	%	0.0%	66.7%	33.3%	0.0%	100.0%
Subcortical	n	0	13	11	0	24
	%	0.0%	54.2%	45.8%	0.0%	100.0%
Total	n	0	35	21	7	63
	%	0.0%	55.6%	33.3%	11.1%	100.0%

Fisher's Exact Test value: 31.59, p-value = 0.046

Statistical analysis revealed a significant association between stroke location and mucosal dryness ($p = 0.046$). PCA territory strokes demonstrated a striking preponderance of severe mucosal dryness (71.4%), with the remaining 28.6% presenting with moderate dryness. This contrasts markedly with MCA territory strokes, where severe dryness was observed in only 16.7% of cases, and with cerebral cortex and ACA territory strokes, which predominantly exhibited mild dryness (88.9% and 75.0%, respectively). These findings suggest differential impact of specific lesion locations on autonomic control of mucosal secretion, with posterior circulation lesions potentially exerting more profound effects on this physiological parameter.

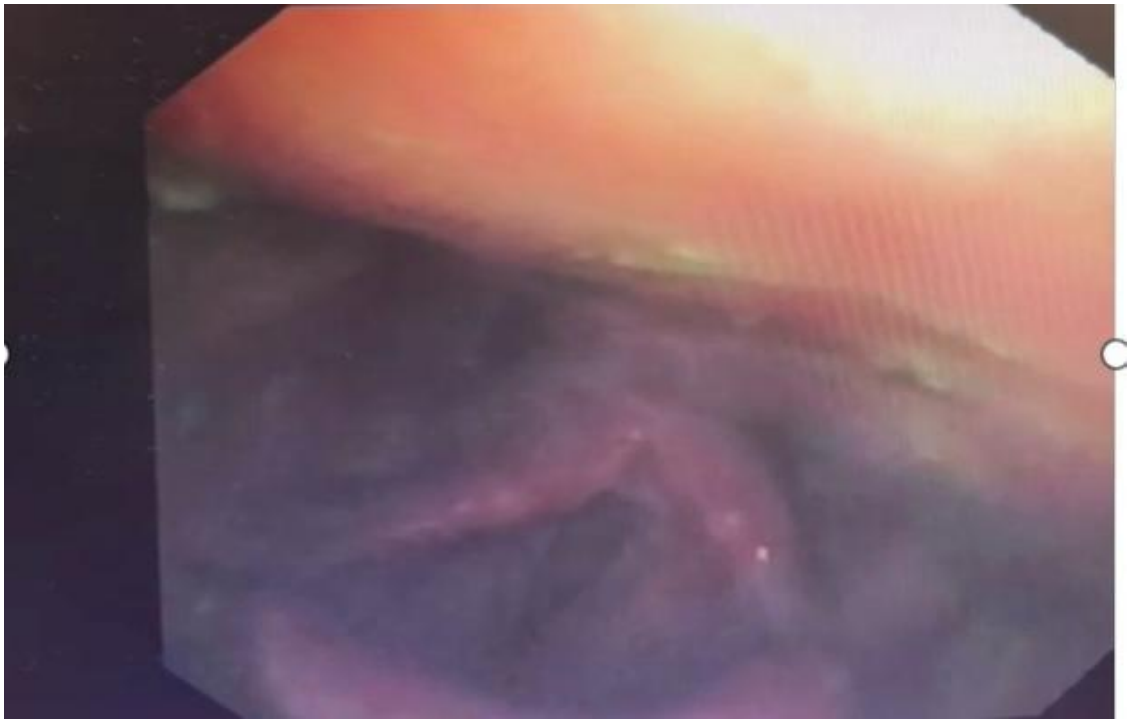


Figure 16: Bolus Residue on Pharyngeal Wall

Table 32: Association Between Stroke Location and Pyriform Fossa Pooling

Area of stroke		Pooling of pyriform fossa				Total
		Absent	Mild	Moderate	Severe	
ACA territory	n	1	3	4	0	8
	%	12.5%	37.5%	50.0%	0.0%	100.0%
Cerebral cortex	n	0	8	1	0	9
	%	0.0%	88.9%	11.1%	0.0%	100.0%
MCA territory	n	0	6	4	2	12
	%	0.0%	50.0%	33.3%	16.7%	100.0%
PCA territory	n	0	0	1	6	7
	%	0.0%	0.0%	14.3%	85.7%	100.0%
Periventricular area	n	0	2	1	0	3
	%	0.0%	66.7%	33.3%	0.0%	100.0%
Subcortical	n	0	13	11	0	24
	%	0.0%	54.2%	45.8%	0.0%	100.0%
Total	n	1	32	22	8	63
	%	1.6%	50.8%	34.9%	12.7%	100.0%

Fisher's Exact Test value: 28.05, p-value = 0.029

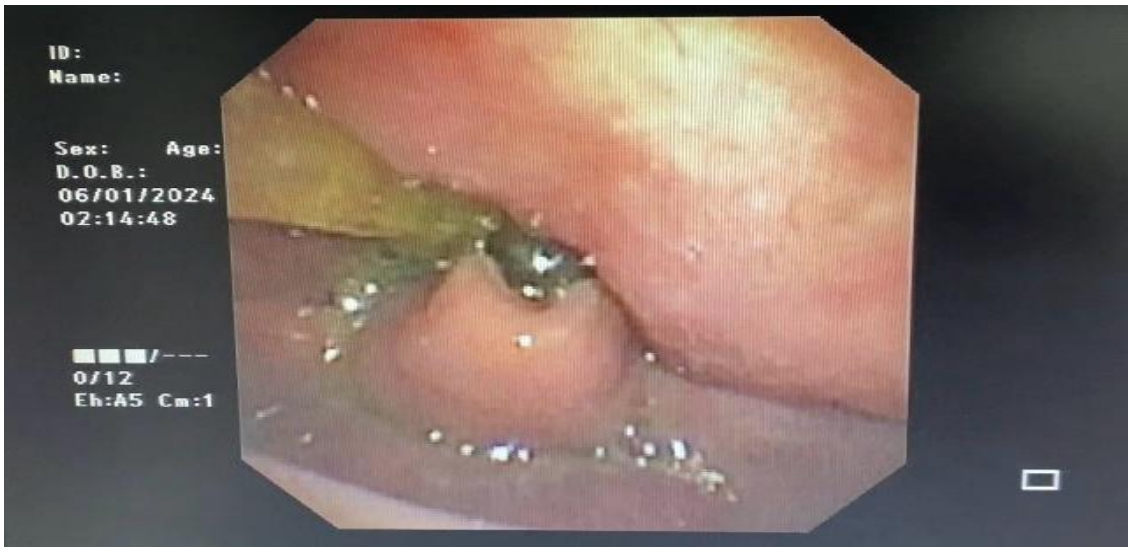


Figure 17: Pooling in Pyriform Fossa with Ryles Tube In Situ

A significant association was documented between stroke location and pyriform fossa pooling ($p = 0.029$). PCA territory strokes demonstrated a remarkably high prevalence of severe pooling (85.7%), contrasting substantially with MCA territory strokes (16.7%) and the complete absence of severe pooling in other lesion locations. ACA territory strokes showed a notable predilection for moderate pooling (50.0%), while cerebral cortex strokes predominantly exhibited mild pooling (88.9%). The marked neuroanatomical specificity in the severity distribution of this parameter suggests differential impacts of various lesion locations on pharyngeal clearance mechanisms.

Table 33: Association Between Stroke Location and Laryngeal Spillover

Area of stroke		Spill over into the larynx test		Total
		Absent	Present	
ACA territory	n	1	7	8
	%	12.5%	87.5%	100.0%
Cerebral cortex	n	1	8	9
	%	11.1%	88.9%	100.0%
MCA territory	n	2	10	12
	%	16.7%	83.3%	100.0%
PCA territory	n	0	7	7
	%	0.0%	100.0%	100.0%
Periventricular area	n	1	2	3
	%	33.3%	66.7%	100.0%
Subcortical	n	3	21	24
	%	12.5%	87.5%	100.0%
Total	n	8	55	63
	%	12.7%	87.3%	100.0%

Fisher's Exact Test value: 2.74, p-value = 0.781

No statistically significant association was observed between stroke location and the presence of laryngeal spillover (p = 0.781). Spillover was present in 87.3% of cases overall, with prevalence ranging from 66.7% in periventricular lesions to 100% in PCA territory strokes. Despite the non-significant p-value, the universal presence of spillover in PCA territory strokes may be clinically relevant. Overall, these findings suggest that laryngeal spillover represents a common pathophysiological feature across various lesion locations, potentially reflecting the generalized impact of cerebrovascular events on swallowing coordination regardless of specific neuroanatomical involvement.

Table 34: Association Between Stroke Location and Nasal Regurgitation

Area of stroke		Nasal regurgitation		Total
		Absent	Present	
ACA territory	n	8	0	8
	%	100.0%	0.0%	100.0%
Cerebral cortex	n	9	0	9
	%	100.0%	0.0%	100.0%
MCA territory	n	10	2	12
	%	83.3%	16.7%	100.0%
PCA territory	n	3	4	7
	%	100.0%	0.0%	100.0%
Periventricular area	n	3	0	3
	%	100.0%	0.0%	100.0%
Subcortical	n	24	0	24
	%	100.0%	0.0%	100.0%
Total	n	57	6	63
	%	90.5%	9.5%	100.0%

Fisher's Exact Test value: 6.15, p-value = 0.195

Statistical analysis did not demonstrate a significant association between stroke location and nasal regurgitation ($p = 0.195$). Nasal regurgitation was exclusively observed in MCA territory strokes (16.7%), with complete absence in all other lesion locations. Despite the non-significant p-value, this pattern of exclusive presentation in MCA territory strokes suggests that velopharyngeal incompetence may represent a specific pathophysiological feature of certain MCA territory lesions, potentially related to the vascular supply of velopharyngeal musculature.

Table 35: Association Between Stroke Location and Adynamic Segment

Area of stroke		Adynamic segment		Total
		Absent	Present	
ACA territory	n	8	0	8
	%	100.0%	0.0%	100.0%
Cerebral cortex	n	8	1	9
	%	88.9%	11.1%	100.0%
MCA territory	n	12	0	12
	%	100.0%	0.0%	100.0%
PCA territory	n	2	5	7
	%	100.0%	0.0%	100.0%
Periventricular area	n	3	0	3
	%	100.0%	0.0%	100.0%
Subcortical	n	24	0	24
	%	100.0%	0.0%	100.0%
Total	n	57	6	63
	%	90.5%	9.5%	100.0%

Fisher's Exact Test value: 6.65, p-value = 0.429

No statistically significant association was documented between stroke location and the presence of an adynamic segment ($p = 0.429$). An adynamic segment was exclusively observed in cerebral cortex strokes (11.1%), with complete absence in all other lesion locations. This finding, despite not reaching statistical significance, suggests that localized pharyngeal neuromuscular incompetence may represent a pathophysiological feature specific to certain cortical lesions, potentially reflecting the complex cortical representation of pharyngeal motor function.

Table 36: Dependency on Ryle's Tube Feeding Across Different Stroke Locations in Patients with Post-Stroke Dysphagia

Area of stroke		Ryles tube dependency for persistent aspiration		Total
		Yes	No	
ACA territory	n	0	8	8
	%	0.0%	100%	100.0%
Cerebral cortex	n	0	9	9
	%	0.0%	100%	100.0%
MCA territory	n	2	10	12
	%	16.7%	84.3%	100.0%
PCA territory	n	5	2	7
	%	71.4%	28.6%	100.0%
Periventricular area	n	0	3	3
	%	0.0%	100%	100.0%
Subcortical	n	0	24	24
	%	0.0%	100%	100.0%
Total	n	7	56	63
	%	11.1%	88.9%	100.0%

Fisher's Exact Test value: 17.82, p-value = 0.001

This table illustrates the relationship between anatomical location of stroke and persistent aspiration requiring continued nasogastric (Ryle's) tube feeding. PCA territory strokes demonstrated the highest proportion of tube feeding dependency (71.4%), followed by MCA territory strokes (16.7%). In contrast, no patients with ACA territory, cerebral cortex, periventricular, or subcortical strokes required tube feeding. This pattern likely reflects the differential impact of specific lesion locations on swallowing safety and efficiency, with anterior circulation cortical strokes involving swallowing centres and posterior circulation strokes affecting brainstem swallowing nuclei showing higher rates of severe dysphagia necessitating alternative feeding routes. In all, 11.1% of stroke survivors required a feeding tube. The majority of these individuals maintained regular eating and drinking habits despite varying degrees of eating disorders. These findings highlight the significance of considering the location of a neuroanatomical lesion when making clinical judgments about a patient's feeding regimen, which has substantial implications for methods of food management. Hoarse voices were observed in all seven patients who required nasogastric feeding; this indicates that vagus nerve speech and swallowing were both affected by issues with the motor pathways. Clinical evidence suggests a connection between vocal alterations and severe dysphagia necessitating alternative feeding methods, particularly in patients with MCA and PCA area injuries, which are associated with increased damage to the corticobulbar and nuclear vagal pathways.

Table 37: Prevalence of Major Aspiration According to Stroke Location in Patients with Post-Stroke Dysphagia

Area of stroke		Major aspiration		Total
		Present	Absent	
ACA territory	n	0	8	8
	%	0.0%	100%	100.0%
Cerebral cortex	n	0	9	9
	%	0.0%	100%	100.0%
MCA territory	n	1	11	12
	%	9.1%	90.9%	100.0%
PCA territory	n	6	1	7
	%	85.7%	14.3%	100.0%
Periventricular area	n	0	3	3
	%	0.0%	100%	100.0%
Subcortical	n	0	24	24
	%	0.0%	100%	100.0%
Total	n	7	56	63
	%	11.1%	88.9%	100.0%

Fisher's Exact Test value: 6.65, p-value = 0.001

This table shows the relationship between stroke location and major aspiration risk (Fisher's Exact Test=42.36, $p<0.001$). PCA territory strokes exhibited a remarkably high prevalence of major aspiration (85.7%), significantly exceeding all other stroke locations. MCA territory strokes showed the second highest prevalence (9.1%), while no cases of major aspiration were documented in ACA territory, cerebral cortex, periventricular, or subcortical strokes. The overall prevalence of major aspiration was 11.1% across the entire cohort. All 7 patients exhibiting major aspiration subsequently developed pneumonia, suggesting significant compromise of laryngopharyngeal sensation rather than just motor dysfunction. This pattern was particularly pronounced in PCA territory strokes (85.7%), where disruption of sensory pathways in the brainstem nuclei likely contributed to silent aspiration without protective reflexes, highlighting the critical distinction between sensory and motor deficits in determining respiratory complications and clinical management approaches.

Table 38: Relationship Between Tracheostomy Status and Clinical Outcome in Patients with Post-Stroke Dysphagia

Tracheostomy performed		Outcome		Total
		Improved	Poor improvement or dead	
Yes	n	1	6	7
	%	14.3%	85.7%	100.0%
No	n	47	9	56
	%	83.9%	16.1%	100.0%
Total	n	48	15	63
	%	76.2%	23.8%	100.0%

Fisher's Exact Test value: 19.74, p-value = <0.001

This table shows the outcomes after undergoing tracheostomy in post stroke dysphagia patients. Among patients who underwent tracheostomy (11.1% of the cohort), only 14.3% demonstrated clinical improvement, compared to 83.9% of those without tracheostomy. 11.1% required cuffed tracheostomy tube as they had persistent aspiration and poor improvement or mortality was documented in 85.7% due to pulmonary complications. While these findings might suggest a detrimental effect of tracheostomy, they are more likely reflect the underlying severity of neurological impairment and dysphagia that necessitated the procedure rather than a causal relationship. Tracheostomy dependency typically occurs in patients with profound neurological compromise, severe aspiration risk, or respiratory insufficiency—factors independently associated with poorer prognosis. Overall, 76.2% of the

cohort demonstrated clinical improvement, while 23.8% exhibited poor improvement or mortality, highlighting both the recovery potential and persistent risks in this patient population. Tracheostomy with cuffed tube was required in 7 patients since they had sensory deficit.

DISCUSSION

This cross-sectional study on FEES in stroke patients sheds light on how dysphagia happens after a stroke and how it is connected to different parts of the brain that are affected by the disease. It shows important changes in the severity of dysphagia, its clinical manifestations, and its anatomical relationships.

The average age of our group is 61.49 years, and 68.3% of them are men. This matches what is known about the epidemiology of stroke. Hilkens et al. (2024) did a large study (n=2,347) and found that strokes are more common in men, especially between the ages of 61 and 70, and in many different parts of the world. This result fits with what they found.[108] The data we got are also the same as those of Rangamani et al. In their NSRP in India (n=13,207), the investigators discovered that people with stroke were mostly men and had a much higher incidence rate than women in all five places they looked at. The median age of stroke patients was 59.3 years.[28]

The high rates of comorbidities in our study group, especially Type 2 diabetes (79.3%) and high blood pressure (63.5%), are in line with what is known about the risk factors for CVA. Behera et al. (2024) looked at stroke burden data from India's GBD Study (n>100,000) and found that high blood pressure and diabetes are the most important risk factors for stroke that can be changed.[26] In the same way, Atmakuru et al. (2025) did a systematic review of data-driven approaches and discovered that DM greatly raises the chance of stroke and is linked to poor functional outcomes after a stroke.[99] Not only do these metabolic conditions raise the chance of CVA, but they may also change how bad dysphagia is and how quickly patients recover.

The most common places for lesions to happen were in the left subcortical area (25.4% of cases) and the MCA region (15.9% of cases). Our study found a strong link between the site of the stroke and the severity of dysphagia (p=0.007). A study by Sun et al. (2023) looked back at FEES data from 176 people and found that subcortical strokes, especially those affecting the basal ganglia circuits, were linked to worse dysphagia than cortical strokes alone. This neuroanatomical pattern fits with what they found.[24]

Importantly, we discovered that all strokes in the PCA area (100%) caused severe dysphagia. However, the severity of strokes in the ACA region was less consistent, which may suggest that these regions are more specialized in disrupting the swallowing network. This finding goes against what has been written before. In their clinical application study of FEES in stroke (n=81), Pisegna and Murray (2018) found that infarcts in the MCA that affect the insular cortex and frontal operculum usually cause worse dysphagia than infarcts that affect other vascular areas. [12] Because dysphagia is so common in people who have had PCA area strokes in our group, we need to learn more about the possible routes by which problems with posterior circulation affect swallowing.

A lot of people who had FEES had mild vocal cord/arytenoid swelling (57.2% of the time), and there were strong links to where the stroke happened (p=0.007). The fact that severe edema was most common in PCA area strokes (85.7%), followed by MCA and ACA territory strokes (25.0%), suggests that different autonomic processes controlling the laryngeal vascular system were disrupted. The FEDSS was made by Warnecke et al. (2009), who found that laryngeal oedema was common in people who had just had a stroke, especially those whose insular brain was affected (n=153). This conclusion fits with what they saw.[22]

We discovered a strong link (p=0.046) between the site of the stroke and the dryness of the mucosa. Strokes in the PCA territory (71.4%) and MCA territory (16.7%) were linked to

serious dryness, which suggests that autonomic control over salivary secretion was disrupted in different ways. In their in-depth study, Silva-Carvalho et al. (2023) discovered that dysphagia, which is already a regular problem for people who have had a stroke, can get much worse when salivary flow is slowed down, especially in people who had cortical involvement of autonomic regions. Mucosal dryness also causes more friction during the pharyngeal phase, which can cause residue and delayed passage, as well as problems with making boluses. [109]

Pyriform fossa pooling is an important endoscopic finding that has big clinical implications. It was seen in 95.2% of our patients and was strongly linked to their brain structures (p=0.029). The level to which it was seen changed. This result is similar to what Pinho et al. (2024) found when they looked at 48 patients right after a mechanical thrombectomy and found pyriform sinus residue in 87.5% of them. This was linked to a higher risk of aspiration. [20] Our research shows that pharyngeal phase coordination is messed up most often in strokes in the PCA territory (85.7% of cases), then in strokes in the MCA territory (16.7% of cases). This means that the participation of the posterior circulation may have a bigger effect on the way the pharynx gets rid of waste.

Aspiration was found in 57.1% of our patients to be minor and 11.1% to be major, which means they were at a high risk for pulmonary problems. Leder and Espinosa (2002) compared clinical inspection and FEES in 49 people who had just had an acute stroke. They found that recorded aspiration happened in 52% of cases and silent aspiration happened in 28% of cases. [9] Elsyaad et al. (2022), for example, looked at 60 very sick acute stroke patients using FEES to find out the risk of aspiration. They found that 63.3% of cases featured aspiration, and that greater incidences were linked to lower GCS scores. [10] At the time of the FEES evaluation, 90.5% of patients had improved to mild neurological damage

(GCS 13–15). However, it is important to note that 42.9% of patients had significant neurological compromise (GCS ≤8) when they first came in, which may have led to the high aspiration rates in our study.

It is possible that this is a trait of all types of strokes because 87.3% of them had laryngeal spillover and there was no statistically significant link between the spillover and the location of the lesion (p=0.781). Ozyürek et al. (2005) also found this to be true. They looked at swallowing problems in 27 stroke patients using FEES and discovered that 78% of them had penetration, no matter where the injury was located. [19] It is important to note that all of the strokes in our group that happened in the PCA area had 100% spillover. This could mean that stopping the posterior circulation has a big effect on the defensive mechanisms in the larynx.

Aside from these underlying problems caused by the stroke, our study found very good clinical results, with 76.2% of patients getting better. There was, however, a big difference depending on where the tumor was. The p-value was not statistically significant, but the rates of improvement for PCA territory strokes were 14.3% lower than those for subcortical injuries (87.5% each) and ACA territory (87.5%). In a study by Bax et al. (2014), functional outcomes improved in 83% of cases, with little difference based on where the lesion was located, after FEES treatments led by a speech-language pathologist were used with 31 stroke patients. [21] Our study shows that the chances of fully recovering from dysphagia are much lower when the posterior circulation is involved. The smaller sample size could be one reason for the difference in how statistically significant the results are. We need to check to see if a larger sample number shows statistical significance.

69.8% of our group were able to finish swallowing on the second try, while 30.2% needed a third try. This shows that there was significant neuromuscular impairment that might be reversible. In 2006, Warnecke et al. discovered that people who had a mild stroke got better

at swallowing with each try, especially while they were still recovering. These results are backed up by their case report and general thoughts on multiple FEES exams. [8]

The DOSS score distribution in our study showed that 44.4% of patients reported a Level 5 score, 42.9% reported a Level 4 score, and 12.7% reported a more serious DOSS score. When Fransson et al. (2025) looked into the validity and dependability of DOSS when used with FEES (n=83), they found that most of the stroke patients had mild to moderate dysphagia. This intensity distribution looks a lot like what we found. When it comes to dysphagia tests, the DOSS has a close link to dysphagia and can help you see how well you are doing functionally. [86]

We saw that 11.1% of patients needed a tracheostomy for a long time. This number is mostly due to the group of patients who had serious trouble swallowing and cognitive decline. Braun et al. (2018) discovered that 13.7% of people who had an acute stroke and had trouble swallowing had a tracheostomy placed. These were mostly people whose brainstems were affected or who had large infarcts in the MCA area. As with other studies on FEES in neurological patients (n=241), this rate is about the same. People who have severe dysphagia on a regular basis may gain a lot from having a tracheostomy to protect their airways. [13]

The 11.1% of patients who needed a long-term tracheostomy were mostly those who had serious trouble swallowing and cognitive decline. Researchers Braun et al. (2018) discovered that 13.7% of people who had an acute stroke and had trouble swallowing had a tracheostomy. These were mostly people who had brainstem involvement or big MCA territory infarcts. Based on previous studies on FEES in neurological patients (n=241), this rate is typical. Chronically severely dysphagic patients may benefit greatly from tracheostomy as a way to keep their lungs safe. [13]

The 7.9% death rate in our group shows how important dysphagia is to outcomes after a stroke. Having a tracheostomy was strongly linked to bad results ($p<0.001$); 83.9% of patients who did not have tracheostomies got better, but only 14.3% of patients who did have tracheostomies did. Warnecke et al. (2009) looked at 153 patients who had been admitted with an acute stroke and found that higher FEDSS scores were directly linked to a higher chance of death within 30 days (hazard ratio = 1.9). When aspiration and severe dysphagia are present, the risk of pneumonia and death is greatly raised. [14]

Our study adds to what is known about dysphagia after a stroke by showing clear neuroanatomical links with pathologic features seen during FEES. It is a new discovery that strokes that affect the PCA area cause very bad symptoms in a number of ways. This suggests that problems with the back blood flow have a big impact on the ability to swallow. A comprehensive FEES training program for neurogenic dysphagia was set up by Dziewas et al. (2016), with a focus on the role of the anterior circulation in controlling swallowing. However, this goes against what most people think and stresses the importance of other areas also taking part. [18]

The fact that nasal vomiting was found in 9.5% of cases in our study and only in 16.7% of MCA area strokes suggests that these people may have a specific problem with how their voice and throat work together. Helliwell et al. (2023) said that velopharyngeal insufficiency was a rare but functionally important finding in stroke patients. It is especially true when the motor cortex is heavily involved in the area that controls the elevation of the soft mouth, as they pointed out in their review of the use of FEES in stroke patients. [17]

Additionally, we found that 9.5% of patients had an adynamic section, which was mostly found in the vocal cord (4.8%), pyriform sinus (3.1%), and arytenoid (1.6%). A study by Jimenez-Dominguez and Manzano-Aquiahuatl (2021) in Mexico's neurology hospitals found

that focal pharyngeal paresis is rare but diagnostically important, especially in people with discrete cortical injuries that affect how they move their pharynx. The study found that this only happens in strokes of the cerebral cortex (11.1%) and the PCA area (71.4%). [110]

Clinical Significance

The results of this study have important implications for how dysphagia is diagnosed and treated after a stroke. The strong link between the site of a stroke and certain abnormal features found by FEES makes it possible for more focused evaluation and treatment methods that are based on neuroanatomical factors.

The high rate of pyriform fossa pooling (98.4%) and minor aspiration (57.1%) in our group of patients shows how important it is to check for and treat dysphagia early in stroke patients, even if they do not have any symptoms. It also gives us more information about silent aspiration. Labeit et al. (2024) supported universal screening and instrumental evaluation in patients with positive screening results. They also did a thorough review of treatment interventions for adults who had a stroke (n>2,000 patients in total). If these signs are found early on, the risk of aspiration can be lowered through swallowing therapy, changes to the person's food, and other steps that protect the lower airway.[111]

The worst effects were seen in PCA territory strokes across a number of factors. This is a new finding that has major therapeutic implications. Patients who had a stroke and had lesions in the PCA area did not improve as much as patients who had other types of lesions (only 14.3% improved). This suggests that these patients may need more care, better monitoring, and maybe even different ways to eat sooner rather than later. After looking at FEES in 241 neurological patients, What et al. (2018) mostly talked about how important anterior circulation lesions are in managing dysphagia. This is more than just the normal focus on

MCA area strokes. [13] According to our study, people who had problems with the pharynx and larynx had the worst aspiration and needed a permanent cuffed oblique tracheostomy tube.

Even though there are some limitations because of the location of the lesion, our results showing that 76.2% of patients improved clinically give a generally positive view on the chance of recovery in post-stroke dysphagia. Trimble et al. (2023) did a study to see if it was possible to do a clinical swallowing examination and cough reflex testing on 45 people who had a hyperacute stroke. This supports their call for comprehensive rehabilitation methods. Their study shows that early intervention can improve outcomes no matter how bad the injury was at first. Our results, on the other hand, show that the location of the injury, especially if it affects the PCA region, may have a big impact on the recovery process. [73]

The strong link between dysphagia severity and certain stroke areas ($p=0.007$) gives doctors information about the prognosis that can help them make decisions and talk to patients. In our group of patients, severe dysphagia was consistently linked to strokes in the PCA area. This suggests that these patients may need more extensive treatment and have worse outcomes. For people who have had subcortical strokes, on the other hand, standard intervention methods may work better. These people tend to have less severe symptoms and a high rate of recovery (87.5% improved).

It is useful for doctors to use FEES to check for dysphagia after a stroke because it can find certain neurological problems that have effects on the brain. A study by Warnecke et al. (2009) with a sample size of 300 people showed that FEES was safe and well tolerated, even in people who had just had a stroke and had significant neurological disability. With the personal knowledge that FEES gives, intervention strategies can be better tailored to each person's needs, rather than using one-size-fits-all solutions. [22]

The aryepiglottic folds were the most common spot of laryngeal overflow (87.3%), which was seen in most of the patients. The vallecula and the pharyngoepiglottic folds were also hurt. All stroke patients in the PCA zone had clear signs of spillover, which suggests that the protective systems for the airways were more seriously harmed in this group. Even though it was not statistically significant, the fact that the link between lesion site and spillover happened so often shows how useful it is in clinical settings. As with earlier studies that looked at FEES tests in stroke patients, the fact that spillover still happens in PCA strokes suggests that the laryngeal nerves are not working as well and that the body's defense mechanisms are not working as well, which could be because the brainstem is involved. [24,47]

Repeat Swallowing Attempt

Some patients (30.2%) needed a third try to finish taking the bolus, but 69.8% were able to do it on the second try. From these results, it looks like a lot of people can make up for their trouble swallowing by keeping their neuromuscular function up. This suggests that some swallowing function has been kept, which could mean that rehabilitation is a possibility. Previous study has shown that multiple swallows can help stroke patients get better faster by increasing bolus clearance and decreasing residue. [43.]

Finally, the rather high death rate (7.9% in our study) shows how important it is to include dysphagia treatment in full stroke care plans and how much dysphagia can affect outcomes after a stroke. As Clarkson (2011) pointed out in their review of post-stroke dysphagia care, a multidisciplinary team made up of medical, nursing, and rehabilitation experts improves outcomes and lowers complications for this group of patients. [112]

CONCLUSION

Stroke is a common life threatening and often morbid condition more common in elderly and among vulnerable population who have risk factors like hypertension, atherosclerosis.

The location of cerebrovascular insult can give an expected outline of signs and symptoms. The various blood vessels supplying cranial cavity play an important role in pathophysiological and clinical features of stroke.

Dysphagia which includes aspiration is an important result and sequelae of stroke which is often ignored and not given its due importance.

The posterior circulation stroke due to posterior cerebellar artery infarction results in major dysphagia and also sensory deficit, resulting in long term aspiration which may necessitate long term cuffed tracheostomy tube.

FEES provides a dynamic assessment of swallowing and iss an easy office-based procedure and should be routinely and periodically utilized in patients with stroke. It can detect spill over and silent aspiration thereby guiding the clinician to initiate important steps and minimize morbidity.

PCA territory strokes exhibited the most severe manifestations across multiple parameters, with 100% presenting with severe dysphagia and 85.7% demonstrating severe pyriform fossa pooling, suggesting critical disruption of swallowing control mechanisms in these patients. Despite concerning pathophysiological findings, including high rates of pyriform fossa pooling (98.4%) and minor aspiration (57.1%), overall clinical improvement was documented in 76.2% of patients, though with marked variability by lesion location.

FEES demonstrated substantial clinical utility in identifying specific deficits that may inform targeted intervention strategies. The findings substantiate the value of this instrumental assessment in post-stroke dysphagia evaluation and management. The significant correlation between lesion location and specific pathophysiological features provides a foundation for more individualized prognostication and intervention planning, potentially improving functional outcomes and reducing complications in this vulnerable patient population.

STRENGTH OF THE STUDY

This study demonstrates several methodological strengths that enhance its contribution to the literature on post-stroke dysphagia. The prospective, cross-sectional design facilitated systematic documentation of FEES findings across multiple parameters, providing a comprehensive pathophysiological profile of swallowing dysfunction. The sample size (n=63) was statistically adequate based on previous prevalence data, ensuring sufficient power for the primary analyses. The standardized assessment protocol, incorporating validated measures like the DOSS, strengthens the reliability of findings. The multidisciplinary approach involving senior physicians, otorhinolaryngologists, and swallowing therapists ensured comprehensive patient evaluation and management. The integration of neuroimaging (CT/MRI) with FEES findings permitted robust neuroanatomical correlations, elucidating structure-function relationships in swallowing dysfunction. The statistical methodology appropriately employed Fisher's Exact Test for categorical analyses, accounting for the distribution characteristics of the dataset. Additionally, the inclusion of follow-up assessments at one and three months provided valuable longitudinal data on recovery patterns, distinguishing this work from purely cross-sectional analyses. The comprehensive documentation of comorbidities enabled contextualization of findings within patients' broader clinical profiles.

RECOMMENDATIONS

Future research should incorporate multicenter designs with larger, more diverse cohorts to enhance generalizability across demographic and clinical profiles. Longitudinal studies with extended follow-up periods are essential to elucidate recovery trajectories and develop prognostic models. Implementation of advanced neuroimaging techniques, including tractography and functional connectivity analysis, would provide deeper insights into structural-functional correlates of post-stroke dysphagia. Standardized quality-of-life assessments should be integrated to evaluate the broader impact of swallowing dysfunction on patients' wellbeing. Development of lesion-specific rehabilitation protocols based on the neuroanatomical correlations identified in this study represents an important clinical translation opportunity. Comparative studies of FEES versus videofluoroscopy in the same patient cohort would clarify the complementary value of these instrumental assessments. Finally, investigation of potential biomarkers (inflammatory, genetic) that might predict dysphagia severity or recovery potential could facilitate personalized intervention approaches and optimize resource allocation in rehabilitation settings.

SUMMARY

This cross-sectional study investigated the pathophysiological manifestations of dysphagia in post-stroke patients using FEES and examined their correlation with neuroanatomical lesion locations. Conducted at R.L. Jalappa Hospital from May 2023 to October 2024, the study enrolled 63 stroke patients aged 40-70 years presenting with dysphagia.

The demographic profile revealed a mean age of 61.49 years (SD=8.727) with male predominance (68.3%), consistent with established epidemiological patterns of stroke. Comorbidity analysis demonstrated high prevalence of type 2 DM (79.3%) and HTN (63.5%), reflecting the established risk factor profile for cerebrovascular disease.

Regarding symptom chronology, acute onset dysphagia (<24 hours) was documented in 44.4% of cases, while 39.7% reported symptoms persisting between one day and one month. A substantial proportion (42.9%) reported experiencing similar episodes previously, suggesting recurrent cerebrovascular events. Behavioural risk factor assessment revealed alcohol consumption and smoking in 38.1% of patients each, with concurrent use in 19.0%.

Neurological status assessment using the GCS revealed significant variability at presentation, with 49.2% demonstrating mild impairment (GCS 13-15) and 42.9% exhibiting severe neurological compromise (GCS ≤8). Notably, by the time of FEES evaluation, 90.5% had improved to mild neurological impairment, facilitating comprehensive assessment.

FEES findings demonstrated several significant pathophysiological features. Repeat swallow performance showed that 69.8% of patients successfully completed swallowing on the second attempt, while 30.2% required a third attempt, indicating significant but potentially recoverable neuromuscular impairment. Mild vocal cord/arytenoid oedema predominated

(47.6%), with moderate oedema in 15.9% and severe oedema in 20.6% of cases. Similar distributions were observed for mucosal dryness, with mild dryness in 55.6% and moderate dryness in 33.3% of patients.

Pyriform fossa pooling, a critical indicator of pharyngeal phase dysfunction, was observed in 98.4% of patients, with mild pooling in 50.8% and moderate pooling in 34.9%. Laryngeal spillover patterns were documented in 87.3% of cases, with aryepiglottic fold spillover being most prevalent (65.1%). Transit delay was observed in 85.7% of patients, predominantly mild (42.9%) or moderate (34.9%) in severity.

Aspiration risk assessment revealed minor aspiration in 57.1% of patients and major aspiration in 11.1%, highlighting the substantial risk for pulmonary complications. Tracheostomy was required in 11.1% of patients, primarily those with severe neurological compromise.

The Dysphagia Outcome and Severity Scale (DOSS) revealed Level 5 (mild dysphagia) in 44.4% and Level 4 (mild-moderate dysphagia) in 42.9% of patients. More severe categories (Levels 1-3) accounted for the remaining 12.7%, indicating a predominance of less severe swallowing dysfunction in this cohort.

Neuroanatomical analysis demonstrated that left subcortical strokes represented the most common lesion location (25.4%), followed by MCA territory infarcts (15.9%). Statistical analysis revealed significant associations between stroke location and dysphagia severity ($p=0.007$), with all PCA territory strokes manifesting as severe dysphagia, whereas subcortical strokes demonstrated predominantly mild (50.0%) or mild-to-moderate (45.8%) presentations.

Further analyses demonstrated significant associations between stroke location and specific pathophysiological features, including vocal cord/arytenoid oedema ($p=0.007$), mucosal dryness ($p=0.046$), and pyriform fossa pooling ($p=0.029$). PCA territory strokes exhibited the most severe manifestations across multiple parameters, with severe oedema in 85.7%, severe mucosal dryness in 71.4%, and severe pyriform fossa pooling in 85.7% of cases.

Clinical outcomes were variable according to lesion location, with overall improvement documented in 76.2% of patients. PCA territory strokes demonstrated markedly poorer outcomes, with only 14.3% showing improvement compared to 87.5% in both ACA territory and subcortical strokes. Mortality was recorded in 7.9% of cases, underscoring the significant impact of dysphagia on post-stroke outcomes.

These findings enhance our understanding of the neuroanatomical correlates of post-stroke dysphagia and provide a foundation for more targeted assessment and intervention strategies based on specific lesion locations and their associated pathophysiological profiles.

LIMITATION

Despite The single-center design potentially limits generalizability to broader stroke populations, particularly those in different healthcare settings or geographic regions. The age restriction (40-70 years) excludes both younger and older stroke patients, who may exhibit distinct dysphagia patterns. The cross-sectional nature of the primary analysis, despite some follow-up data, constrains causal inference regarding the relationship between lesion location and dysphagia manifestations. The exclusion of ventilator-dependent patients introduced selection bias by omitting potentially more severe cases. The study did not incorporate volumetric lesion analysis or advanced neuroimaging techniques (e.g., diffusion tensor imaging), which might have provided more precise neuroanatomical correlations. The relatively small subgroup sizes for certain lesion locations limited statistical power for some comparative analyses. The absence of pre-stroke swallowing status assessment makes it challenging to differentiate new onset from exacerbated pre-existing dysphagia. Finally, the study lacked standardized quality-of-life measures to assess the functional impact of dysphagia on patients' daily activities and psychosocial wellbeing, limiting comprehensive outcome evaluation.

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ANNEXURE

PATIENT INFORMATION SHEET

Study title: FIBEROPTIC ENDOSCOPIC EVALUATION OF SWALLOWING IN PATIENTS WITH STROKE - A CROSS SECTIONAL STUDY

Study location: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

Details:

Patients aged between 40 – 70 years, diagnosed with Stroke and having dysphagia at R.L Jalappa Hospital will be included in this study.

Patients in this study will have to undergo routine blood investigations (CBC, Coagulation profile, Serology) and a fiberoptic endoscopy will be performed for patients with stroke to assess swallowing function which has advantage of being less costly, no radiation exposure. Procedure may be associated with risk and complication such as bleeding and injury to adjacent structures which are extremely rare.

Patient will be explained about the importance of undergoing the above mentioned investigations and the procedure.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study, we will collect information from you or the person responsible for you, or both. Relevant history will be taken. This information collected will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the members of the same. There is no compulsion to agree to this study. The care you will get will not change if you do not wish to participate in this study. You will have no financial benefit by being a part of this study, nor will you incur any risk. You are required to sign/provide thumb impression only if you voluntarily agree to participate in this study.

For further information contact,

Dr. Rachana J (Post graduate)

Department of Otorhinolaryngology

SDUMC, Kolar 9845735058

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಪಾರ್ಶ್ವವಾಯು ಹೊಂದಿರುವ ರೋಗಿಗಳಲ್ಲಿ ನುಂಗುವಿಕೆಯ ಫೈಬರ್ ಆಪ್ಟಿಕ್ ಎಂಡೋಸ್ಕೋಪಿಕ್ ಮೌಲ್ಯಮಾಪನ - ಒಂದು ಅಡ್ಡ ವಿಭಾಗದ ಅಧ್ಯಯನ

ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್ ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರವು ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು,

ಟಮಕ, ಕೋಲಾರ.

ವಿವರಗಳು:

R.L ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ಪಾರ್ಶ್ವವಾಯು ರೋಗನಿರ್ಣಯ ಮತ್ತು ಡಿಸ್ಫೇಜಿಯಾ ಹೊಂದಿರುವ 40 ರಿಂದ 70 ವರ್ಷ ವಯಸ್ಸಿನ ರೋಗಿಗಳನ್ನು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲಾಗುತ್ತದೆ.

ಈ ಅಧ್ಯಯನದಲ್ಲಿ ರೋಗಿಗಳು ವಾಡಿಕೆಯ ರಕ್ತ ಪರೀಕ್ಷೆಗಳಿಗೆ ಒಳಗಾಗಬೇಕಾಗುತ್ತದೆ (CBC, ಹೆಪ್ಪುಗಟ್ಟುವಿಕೆ ಪ್ರೊಫೈಲ್, ಸೆರೋಲಾಜಿ) ಮತ್ತು ಸ್ಟ್ರೋಕ್ ಹೊಂದಿರುವ ರೋಗಿಗಳಿಗೆ ಫೈಬರ್ ಆಪ್ಟಿಕ್ ಎಂಡೋಸ್ಕೋಪಿಯನ್ನು ನುಂಗುವ ಕಾರ್ಯವನ್ನು ನಿರ್ಣಯಿಸಲು ನಡೆಸಲಾಗುತ್ತದೆ, ಇದು ಕಡಿಮೆ ವೆಚ್ಚದ ಪ್ರಯೋಜನವನ್ನು ಹೊಂದಿದೆ, ಯಾವುದೇ ವಿಕಿರಣಕ್ಕೆ ಒಡ್ಡಿಕೊಳ್ಳುವುದಿಲ್ಲ. ಕಾರ್ಯವಿಧಾನವು ಅಪಾಯ ಮತ್ತು ತೊಡಕುಗಳಂತಹ ರಕ್ತಸ್ರಾವ ಮತ್ತು ಪಕ್ಕದ ರಚನೆಗಳಿಗೆ ಗಾಯದಂತಹ ಅತ್ಯಂತ ಅಪರೂಪದ ಜೊತೆ ಸಂಬಂಧ ಹೊಂದಿರಬಹುದು.

ಮೇಲೆ ತಿಳಿಸಿದ ತನಿಖೆಗಳಿಗೆ ಒಳಗಾಗುವ ಪ್ರಾಮುಖ್ಯತೆ ಮತ್ತು ಕಾರ್ಯವಿಧಾನದ ಬಗ್ಗೆ ರೋಗಿಗೆ ವಿವರಿಸಲಾಗುವುದು.

ದಯವಿಟ್ಟು ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬದ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಸಮ್ಮತಿಸಿದರೆ, ನಾವು ನಿಮ್ಮಿಂದ ಅಥವಾ ನಿಮಗೆ ಜವಾಬ್ದಾರರಾಗಿರುವ ವ್ಯಕ್ತಿಯಿಂದ ಅಥವಾ ಇಬ್ಬರಿಂದಲೂ ಮಾಹಿತಿಯನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು

ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೈತಿಕಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಅದರ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು

ಯಾವುದೇ ಒತ್ತಾಯವಿಲ್ಲ. ನೀವು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿಯು ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದ ಭಾಗವಾಗುವುದರಿಂದ ನಿಮಗೆ ಯಾವುದೇ ಆರ್ಥಿಕ ಪ್ರಯೋಜನವಾಗುವುದಿಲ್ಲ ಅಥವಾ ನೀವು ಯಾವುದೇ ಅಪಾಯಕ್ಕೆ ಒಳಗಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಸಮ್ಮತಿಸಿದರೆ

ಮಾತ್ರ ನೀವು ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತನ್ನು ಒದಗಿಸಬೇಕಾಗುತ್ತದೆ.

ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ ಸಂಪರ್ಕಿಸಿ,

ಡಾ. ರಚನ ಜೆ (ಸ್ನಾತಕೋತ್ತರ ಪದವಿ)
ಓಟೋರಿನೋಲಾರಿಂಗೋಲಜಿ ವಿಭಾಗ
SDUMC, ಕೋಲಾರ 9845735058

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR - 563101.

INFORMED CONSENT FORM

I have been read or have been read to me and understand the purpose of the study, FIBEROPTIC ENDOSCOPIC EVALUATION OF SWALLOWING IN PATIENTS WITH STROKE - A CROSS SECTIONAL STUDY the procedure FIBEROPTIC ENDOSCOPY that will be used for swallowing assessment. The risks and benefits associated with my involvement in the study and the nature of information that will be collected and disclosed during the study have been explained.

I have the opportunity to ask my questions regarding various aspects of study and my questions are answered to my satisfaction. I understand that I remain free to withdraw from the study at any time and this will not change my future care.

I, the undersigned agree to participate in this study and authorize the collection and disclosure of my personal information for dissertation.

Signature & Name of Pt. Attendant Signature/Thumb impression & Name of patient

Relation with patient: _____

Witness: _____

Print Name of Researcher taking the consent _____

Signature of Researcher taking the consent _____

Principal Investigator's Name: Dr. RACHANA J

Mobile Number: 9845735058 Email Id: rachana.achus@gmail.com

ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನೆಯ

ಅಕಾಡೆಮಿ, ಟಮಕ, ಕೋಲಾರ - 563101.

ಮಾಹಿತಿ ನೀಡಿದ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ನಾನು ಓದಿದ್ದೇನೆ ಅಥವಾ ನನಗೆ ಓದಿದಾರೆ ಮತ್ತು ಅಧ್ಯಯನದ ಉದ್ದೇಶವನ್ನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ, ಪಾರ್ಶ್ವವಾಯು ಹೊಂದಿರುವ ರೋಗಿಗಳಲ್ಲಿ ನುಂಗುವಿಕೆಯ ಫೈಬರ್ ಆಪ್ಟಿಕ್ ಎಂಡೋಸ್ಕೋಪಿಕ್ ಮೌಲ್ಯಮಾಪನ - ಒಂದು ಅಡ್ಡ ವಿಭಾಗದ ಅಧ್ಯಯನ

ಮೌಲ್ಯಮಾಪನವನ್ನು ನುಂಗಲು ಬಳಸಲಾಗುವ FIBEROPTIC ENDOSCOPY ವಿಧಾನ. ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ

ಪಾಲ್ಗೊಳ್ಳುವಿಕೆಗೆ ಸಂಬಂಧಿಸಿದ ಅಪಾಯಗಳು ಮತ್ತು ಪ್ರಯೋಜನಗಳು ಮತ್ತು ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಸಂಗ್ರಹಿಸಿದ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸುವ ಮಾಹಿತಿಯ ಸ್ವರೂಪವನ್ನು ವಿವರಿಸಲಾಗಿದೆ.

ಅಧ್ಯಯನದ ವಿವಿಧ ಅಂಶಗಳ ಬಗ್ಗೆ ನನ್ನ ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶವಿದೆ ಮತ್ತು ನನ್ನ ಪ್ರಶ್ನೆಗಳಿಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ

ಉತ್ತರಿಸಲಾಗಿದೆ.

ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಲು ಮುಕ್ತನಾಗಿರುತ್ತೇನೆ ಮತ್ತು ಇದು ನನ್ನ ಭವಿಷ್ಯದ

ಕಾಳಜಿಯನ್ನು ಬದಲಾಯಿಸುವುದಿಲ್ಲ ಎಂದು ನಾನು ಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ನಾನು, ಕೆಳಗೆ ಸಹಿ ಮಾಡಿರುವವರು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪುತ್ತೇನೆ ಮತ್ತು ಪ್ರಬಂಧಕ್ಕಾಗಿ ನನ್ನ ವೈಯಕ್ತಿಕ

ಮಾಹಿತಿಯ ಸಂಗ್ರಹಣೆ ಮತ್ತು ಬಹಿರಂಗಪಡಿಸುವಿಕೆಯನ್ನು ಅಧಿಕೃತಗೊಳಿಸುತ್ತೇನೆ.

ಪಂ.ನ ಸಹಿ ಮತ್ತು ಹೆಸರು. ಅಟೆಂಡೆಂಟ್ ಸಹಿ/ಹೆಬ್ಬೆರಳಿನ ಗುರುತು ಮತ್ತು ರೋಗಿಯ ಹೆಸರು

ರೋಗಿಯೊಂದಿಗೆ ಸಂಬಂಧ: _____

ಸಾಕ್ಷಿ: _____

ಸಮ್ಮತಿಯನ್ನು ತೆಗೆದುಕೊಳ್ಳುವ ಸಂಶೋಧಕರ ಹೆಸರನ್ನು ಮುದ್ರಿಸಿ _____

ಒಪ್ಪಿಗೆಯನ್ನು ತೆಗೆದುಕೊಳ್ಳುವ ಸಂಶೋಧಕರ ಸಹಿ _____

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು: ಡಾ. ರಚನ ಜೆ

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 9845735058 ಇಮೇಲ್ ಐಡಿ: rachana.achus@gmail.com

PROFORMA

NAME
AGE
GENDER
ADDRESS
MOBILE NUMBER
DATE OF VISIT/ DATE OF ADMISSION
UHID NUMBER
COMPLAINTS DYSPHAGIA ASPIRATION SYMPTOMS DIFFICULTY IN PROTRUSION OF TONGUE DYSPNOEA
HISTORY OF PRESENTING ILLNESS ONSET: INSIDIOUS/SUDDEN DURATION OF SYMPTOMS PROGRESSION EXACERBATING AND RELIEVING FACTORS ASSOCIATED SYMPTOMS OCCUPATION AND HABITS OTHER COMPLAINTS
PRE EXISTING SYTEMIC ILLNESS DIABETES ☐ THYROID DISORDER ☐ TUBERCULOSIS ☐ ANAEMIA ☐ HYPERTENSION ☐ OTHERS, SPECIFY ☐
PERSONAL HISTORY HABITS- TOBACCO CHEWING- SMOKING- ALCOHOL- EXAMINATION

GENERAL PHYSICAL EXAMINATION

Vitals:

Pulse:

BP:

• RR:

Temperature :

LOCAL EXAMINATION

• Oral Cavity :

• Mouth opening: Adequate/ Trismus /Grade of Trismus (if any):

Oro-dental Hygiene: Poor/ Satisfactory

Nicotine stains: Y/ N

Site :

Retromolar Trigone

Gingivo-buccal Sulcus

Tongue

Hard palate

Floor of mouth

INDIRECT LARYNGOSCOPY:

Nose :

Ear:

SYSTEMIC EXAMINATION:

• Cardio vascular system

• Respiratory system:

Per Abdomen:

Central nervous system:

GCS

Tone:

Power of muscle:

CLINICAL DIAGNOSIS :

CHEST X-RAY:

CT SCAN:

MRI:

FIBEROPTIC ENDOSCOPY FINDINGS:

	1st MONTH	3rd MONTH
REPEAT SWALLOW		
EDEMA AND ULCERATION		
DRYNESS		
POOLING IN PYRIFORM FOSSA		
SPILL OVER LARYNX-		
DELAYED BOLUS TRANSIT-		
ASPIRATION: MINOR-		
MAJOR-		
NASAL REGURGITATION		
ANY ADYNAMIC SEGMENT -		

HOSPITALISATION:

INTERVENTION:

• OUTCOME OF INTERVENTION:

STATUS AT LAST FOLLOW UP:

IMPRESSION-

NUMBER	AGE (YEARS)	GENDER	DATE OF ADMISSION	OCCUPATION	CHIEF COMPLAINTS	DURATION	PAST HISTORY	ADDICTION	CLINICAL FINDINGS	GCS AT PRESENTATION	COMORBIDITIES	REPEAT SWALLOW	EDEMA OF VOCAL CORDS AND ARYTENOIDS	ULCERATION	DRYNESS OF MUCOSA	POOLING IN PYRIFORM FOSSA	SPILL OVER INTO LARYNX	DELAYED TRANSIT	NASAL REGURGITATION	ADYNAMIC SEGMENT	MINOR ASPIRATION	MAJOR ASPIRATION	TRACHEOSTOMY PERFORMED OR NOT	DIAGNOSIS	SEVERITY OF DYSPHAGIA-DOSS SCORE	AREA OF STROKE	FOLLOW UP	FOLLOW UP RESULTS	FOLLOW WP
1	65	MALE	18.11.2023	FARMER	C/O WEAKNESS OF LEFT UPPER LIMB & LOWER LIMB	1 DAY	H/O SIMILAR COMPLAINTS 2 MONTHS BACK	H/O CIGARETTE SMOKING SINCE 10 YEARS	LEFT PLANTAR EXTENSOR LEFT UPPER LIMB & LOWER LIMB TONE DECREASED ANGLE OF MOUTH DEVIATED TO LEFT	E, V, M_6 B/L PUPILS EQUAL & REACTIVE	K/C/O HTN SINCE 4 YEARS K/C/O TYPE 2 DIABETES MELLITUS SINCE 4 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD	ABSENT	MILD	PRESENT-MODERATE	ARYEPIGLOTTIC FOLD & ARETENOIDS SPILL OVER PRESENT	MODERATE	ABSENT	ABSENT	PRESENT	ABSENT	NO	1) ISCHAEMIC STROKE WITH LEFT HEMIPLEGIA (CVA INVOLVING POSTERIOR CIRCULATION WITH LEFT UMN TYPE PALSY) 2) CHRONIC INFARCT IN PONTOMEDULLARY JUNCTION3)HYPERTENSION 4) TYPE 2 DIABETES MELLITUS	MODERATE DYSPHAGIA-SCORE-3	BRAINSTEM	SWALLOW THERAPY GIVEN-EFFORTFUL SWALLOW,AFTER 3 MONTHS	REPEAT SWALLOW-PRESENT, EDEMA-RESOLVED,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-5-MILD DYSPHAGIA	IMPROVED
					C/O DECREASED RESPONSIVENESS	1 DAY																							
2	53	MALE	16.11.2023	FARMER	C/O DECREASED MOVEMENT OF LEFT UPPER LIMB & LOWER LIMB	10 DAYS	H/O SIMILAR COMPLAINTS 2 YEARS BACK ON RIGHT SIDE, SLOWLY IMPROVED	H/O CIGARETTE SMOKING SINCE 20 YEARS STOPPED 6 MONTHS BACK H/O ALCOHOL CONSUMPTION FOR 20 YEARS	LEFT UPPER LIMB AND LOWER LIMB TONE DECREASED, B/L PLANTAR REFLEX FLEXION	E, V, M_6 B/L PUPILS EQUAL AND REACTIVE	K/C/O HTN SINCE 5 MONTHS K/C/O TYPE 2 DIABETES MELLITUS SINCE 5 MONTHS	PRESENT AND CAN SWALLOW WITH 3rd SWALLOW	MILD	ABSENT	MILD	PRESENT-MILD	ARYEPIGLOTTIC FOLD SPILL OVER(+)	MILD	ABSENT	ABSENT	ABSENT	ABSENT	NO	1) LEFT HEMIPLEGIA 2) RECURRENT STROKE 3) HTN 4) TYPE 2 DIABETES MELLITUS	MODERATE DYSPHAGIA-SCORE-3	MCA TERRITORY	SWALLOW THERAPY GIVEN-EFFORTFUL SWALLOW,AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-ABSENT,MINOR ASPIRATION-ABSENT, DOSS SCORE-5-MILD DYSPHAGIA	IMPROVED
					C/O DIFFICULTY TO SPEAK	10 DAYS																							
3	70	FEMALE	15.12.2023	HOUSEWIFE	C/O NUMBNESS OF RIGHT UPPER LIMB & LOWER LIMB	1 DAY	H/O SIMILAR COMPLAINTS 6 MONTHS BACK	NO ADDICTIONS	PLANTAR RIGHT LOWER LIMB EXTENSOR, RIGHT SEMIFLEXED AND PRONATED TONE RIGHT - DECREASED LEFT - NORMAL	$E_4V_4M_6$ B/L PUPILS EQUAL AND REACTIVE	NO OTHER KNOWN COMORBIDITTIES	PRESENT AND CAN SWALLOW WITH 3rd SWALLOW	MODERATE	ABSENT	MODERATE	PRESENT-MODERATE	ARYEPIGLOTTIC FOLD & ARETENOIDS SPILL OVER PRESENT	MODERATE	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.RECURRENT CVA WITH RIGHT HEMIPARESIS	MILD TO MODERATE DYSPHAGIA-SCORE 4	RIGHT SUBCORTICAL	SWALLOW THERAPY GIVEN-EFFORTFUL SWALLOW,AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-5 MILD DYSPHAGIA	IMPROVED
					C/O BURNING SENSATION OF RIGHT UPPER LIMB & LOWER LIMB																								
4	53	MALE	21.12.2023	FARMER	C/O WEAKNESS OF RIGHT UPPER LIMB & LOWER LIMB	5 DAYS	H/O SIMILAR COMPLAINTS IN 5 MONTHS BACK	H/O CIGARETTE SMOKING AND ALCOHOL CONSUMPTION SINCE 10 YEARS	LEFT PLANTAR - FLEXOR RIGHT WITHDRAWAL TONE- RIGHT UPPER LIMB AND LOWER LIMB DECREASED	$E_4V_4M_6$, B/L PUPILS EQUAL AND REACTIVE TO LIGHT	K/CO TYPE 2 DM SINCE 6 MONTHS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD	ABSENT	MILD	MILD-PRESENT	ARYEPIGLOTTIC FOLD SPILL OVER(+)	PRESENT	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.CVA WITH RIGHT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS 3.NEWLY DIAGNOSED HTN	MODERATE DYSPHAGIA-SCORE-3	LEFT SUBCORTICAL	SWALLOW THERAPY GIVEN, FOLLOW UP AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-ABSENT,SPILLOVER-ABSENT, DELAYED TRANSIT ABSENT,MINOR ASPIRATION-ABSENT, DOSS SCORE-5 MILD DYSPHAGIA	IMPROVED
5	69	MALE	03.01.2024	MANAGER IN ORPHANAGE TRUST	C/O WEAKNESS OF LEFT UPPER LIMB & LOWER LIMB	2 DAYS	NO SIMILAR COMPLAINTS IN PAST	NO ILL HABITS	TONE AND POWER NORMAL	$E_4V_4M_6$	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 15 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD	ABSENT	MILD	MILD	ABSENT	MILD	ABSENT	ABSENT	ABSENT	ABSENT	NO	1. LEFT HEMIPARESIS 2.TYPE 2 DM 3.CEREBELLAR STROKE 4.HTN	MILD DYSPHAGIA-DOSS SCORE 5	LEFT SUBCORTICAL	SWALLOW THERAPY GIVEN-EFFORTFUL SWALLOW, FOLLOW UP AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-ABSENT,SPILLOVER-ABSENT, DELAYED TRANSIT ABSENT,MINOR ASPIRATION-ABSENT, DOSS SCORE-6 WITHIN FUNCTIONAL LIMITS	IMPROVED
6	66	MALE	05.01.2024	AGRICULTURIST	C/O DECREASED MOVEMENT OF LEFT UPPER LIMB & LOWER LIMB	1 DAY	NO SIMILAR COMPLAINTS IN PAST	NO ADDICTIONS	LEFT UPPER LIMB AND LOWER LIMB TONE DECREASED, PLANTAR REFLEX-RIGHT FLEXOR AND LEFT-EXTENSOR,LOSS OF WRINKLES PRESENT,EYE CLOSURE-WEAK, LOSS OF NASOLABIAL FOLD,DEVIATION OF ANGLE OF MOUTH TO RIGHT	E2 V1 M6 B/L PUPILS EQUAL AND REACTIVE	K/C/O HTN SINCE 5 YEARS K/C/O ATRIAL FIBBRILATION SINCE 15 DAYS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD	ABSENT	MILD	POOLING IN PYRIFORM FOSSA	ARYEPIGLOTTIC FOLD & ARETENOIDS SPILL OVER PRESENT	MILD	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH LEFT HEMIPARESIS 2.7LOWER MOTOR NEURON 7th CRANIAL NERVE PALSY 3.RIGHT MCA TERRITORY 4.CARDIOEMBOLIC STROKE 5.ATRIAL FIBRILLATION WITH CVR 6.ASPIRATION PNEUMONIA 7.HTN	MILD DYSPHAGIA-DOSS SCORE 3	MCA TERRITORY	PATIENT IS DEAD		DEAD
					C/O DEVIATION OF ANGLE OF MOUTH TO RIGHT	1 DAY																							

NUMBER	AGE (YEARS)	GENDER	DATE OF ADMISSION	OCCUPATION	CHIEF COMPLAINTS	DURATION	PAST HISTORY	ADDICTION	CLINICAL FINDINGS	GCS AT PRESENTATION	COMORBIDITIES	REPEAT SWALLOW	EDEMA OF VOCAL CORDS AND ARYTENOIDS	ULCERATION	DRYNESS OF MUCOSA	POOLING IN PYRIFORM FOSSA	SPILL OVER INTO LARYNX	DELAYED TRANSIT	NASAL REGURGITATION	ADYNAMIC SEGMENT	MINOR ASPIRATION	MAJOR ASPIRATION	TRACHEOSTOMY PERFORMED OR NOT	DIAGNOSIS	SEVERITY OF DYSPHAGIA-DOSS SCORE	AREA OF STROKE	FOLLOW UP	FOLLOW UP RESULTS	FOLLOW WP
7	65	MALE	25.01.2024	FARMER	C/O WEAKNESS OF LEFT UPPER LIMB & LOWER LIMB	3 DAYS	NO SIMILAR COMPLAINTS IN PAST	NO ADDICTIONS	LEFT UPPER LIMB AND LOWER LIMB TONE DECREASED, LEFT PLANTAR REFLEX-EXTENSOR	E4V4 M6 B/L PUPILS EQUAL AND REACTIVE	K/C/O HTN SINCE 1 AND HALF YEARS K/C/O BRONCHIAL ASTHMA SINCE 6 MONTHS	PRESENT AND CAN SWALLOW WITH 2ad SWALLOW	MODERATE	ABSENT	DRYNESS OF MUCOSA-MODERATE	PRESENT-MODERATE	ARYEPIGLOTTIC FOLD SPILLOVER(+)	MODERATE	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH LEFT HEMIPARESIS 2.BRONCHIAL ASTHMA 3.HTN	MILD TO MODERATE DYSPHAGIA-SCORE 4	CEREBRAL CORTEX-RIGHT PARIETAL	SWALLOW THERAPY GIVEN-SUPRAGLOTTIC SWALLOW	REPEAT SWALLOW 2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-6 MILD DYSPHAGIA	IMPROVED
					C/O DIFFICULTY TO SPEAK	3 DAYS																							
8	70	FEMALE	02.02.2024	HOUSEWIFE	C/O WEAKNESS OF RIGHT UPPER LIMB & LOWER LIMB	1 DAY	NO SIMILAR COMPLAINTS IN PAST	NO ADDICTIONS	LEFT UPPER LIMB AND LOWER LIMB TONE DECREASED, PLANTAR REFLEX-RIGHT FLEXOR AND LEFT-EXTENSOR	E1 V1 M6 B/L PUPILS EQUAL & REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS SINCE 6 MONTHS, K/C/O CARCINOMA ENDOMETRIUM SINCE 5 YEARS S/P BRACHYTHERAPY AND CHEMOTHERAPY	PRESENT AND CAN SWALLOW WITH 2ad SWALLOW	MODERATE	ABSENT	MODERATE	PRESENT-MODERATE	ARYEPIGLOTTIC FOLD SPILLOVER(+)	MODERATE	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.LEFT HEMIPLEGIA WITH UPPER MOTOR NEURON FACIAL PALSY SECONDARY TO CEREBELLAR STROKE 2.CARCINOMA ENDOMETRIUM 3.TYPE 2 DIABETES MELLITUS	MILD TO MODERATE DYSPHAGIA-SCORE 4	LEFT SUBCORTICAL	SWALLOW THERAPY GIVEN	REPEAT SWALLOW 2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-5 MILD DYSPHAGIA	IMPROVED
					C/O DEVIATION OF ANGLE OF MOUTH TO RIGHT	1 DAY																							
9	69	MALE	13.02.2024	BUSSINESSMAN	C/O DECREASED RESPONSIVENESS	1 DAY	H/O SIMILAR COMPLAINTS 1 AND HALF YEARS BACK AND 1 EPISODE 5 DAYS BACK	H/O CIGARETTE SMOKING SINCE 20 YEARS	RIGHT UPPER LIMB AND LOWER LIMB TONE DECREASED, RIGHT PLANTAR-EXTENSOR	E2V2M4, B/L PUPILS SUGGISHLY REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 10 YEARS	PRESENT AND CAN SWALLOW WITH 2ad SWALLOW	MODERATE	ABSENT	MODERATE	PRESENT-MODERATE	ARYEPIGLOTTIC FOLD SPILLOVER(+)	MODERATE	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.CVA WITH RIGHT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS 3.HTN 4.SIADH	MILD TO MODERATE DYSPHAGIA-SCORE 4	LEFT SUBCORTICAL	SWALLOW THERAPY GIVEN-AFTER 3 MONTHS	REPEAT SWALLOW 2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-5 MILD DYSPHAGIA	IMPROVED
10	51	MALE	03.01.2024	CLERK	C/O DIFFICULTY TO SPEAK ,C/O DIFFICULTY TO SWALLOW	5 YEARS,1 DAY	NO SIMILAR COMPLAINTS IN PAST	H/O ALCOHOL CONSUMPTION SINCE 10 YEARS	TONE AND POWER NORMAL, DEVIATION OF ANGLE OF MOUTH TO RIGHT	E4V1M6, B/L PUPIL EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 10 YEARS, K/C/O ISHEMIC HEART DISEASE S/P PTCA 7 YEARS BACK	REPEAT SWALLOW-3RD SWALLOW RYLES TUBE-INSITU	MILD	ABSENT	MILD	PRESENT-MODERATE	PHARYNGOEPIGLOTTIC FOLD SPILLOVER PRESENT	MODERATE	ABSENT	ABSENT	PRESENT FOR LIQUIDS	PRESENT FOR LIQUIDS	YES	1.7PSEUDOBULBAR PALSY, 2.TYPE 2 DIABETIS MELLITUDS, 3.HTN, 4.ISHEMIC HEART DISEASE S/P PTCA	SEVERE DYSPHAGIA-SCORE 2	LEFT SUBCORTICAL	SWALLOW THERAPY GIVEN-SUPRAGLOTTIC SWALLOW	REPEAT SWALLOW 2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MODERATE,SPILLOVER-PRESENT, DELAYED TRANSIT-MODERATE,MINOR ASPIRATION-PRESENT,MAJOR ASPIRATION-PRESENT DOSS SCORE-2 SEVERE DYSPHAGIA, TRACHEOSTOMY TUBE-INSITU	SAME
11	41	MALE	9.02.2024	SHOPKEEPER	C/O DECREASED RESPONSIVENESS	1 DAY	NO SIMILAR COMPLAINTS IN PAST	H/O ALCOHOL CONSUMPTION SINCE 10 YEARS	B/L PLANTARS-MUTE	E1VTM1, B/L PUPILS REACTIVE TO LIGHT	K/C/O TYPE 2 DIABETES MELLITUS SINCE 1 YEAR, RECENTLY DIAGNOSED MYOCARDIAL INFARCTION	REPEAT SWALLOW 2nd SWALLOW PRESENT	MILD	ABSENT	MILD	MILD-POOLING IN PYRIFORM FOSSA	SPILL OVER PRESENT JUST ABOVE VOCAL CORDS,VALECULLA	PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	1.ACUTE EMBOLIC HEMORRAGIC STROKE WITH HAEMORRAGIC TRANSFORMATION, 2. ACUTE PULMONARY EDEMA SECONDARY TO HEARTV FAILURE, 3. TYPE 2 DIABETES MELLITUS	MILD DYSPHAGIA-DOSS SCORE 5	SUBCORTICAL	PATIENT IS DEAD-MYOCARDIAL INFARCTION		DEAD
12	68	MALE	04.03.2024	AGRICULTURIST	C/O DECREASED MOVEMENT OF BILATERAL LOWER LIMB	3 MONTHS	NO SIMILAR COMPLAINTS IN PAST	H/O ALCOHOL CONSUMPTION SINCE 10 YEARS	BILATERAL LOWER LIMB TONE-DECREASED, LEFT PLANTAR-FLEXOR	E2V1M6 , B/L PUPILS EQUAL AND REACTIVE TO LIGHT	K/C/O HTN AND TYPE 2 DM SINCE 5 MONTHS	REPEAT SWALLOW PRESENT-3rd SWALLOW	EDEMA OF VOCAL CORDS AND ARYTENOIDS-SEVERE	ABSENT	PRESENT-SEVERE	POOLING IN PYRIFORM FOSSA-SEVERE	SPILL OVER IN THE NASOPHARYNX	PRESENT-SEVERE	PRESENT	ABSENT	LOW GRADE ASPIRATION PRESENT	PRESENT	YES	1.POSTERIOR CIRCULATION STROKE	PROFOUND DYSPHAGIA-SCORE 1	LATERAL VENTRICLE	SWALLOW THERAPY GIVEN-SUPRAGLOTTIC SWALLOW , AFTER 3 MONTHS	REPEAT SWALLOW 3rd SWALLOW PRESENT , EDEMA-MODERATE,DRYNESS-MODERATE,POOLING IN PYRIFORM FOSSA-MODERATE,SPILLOVER-PRESENT, DELAYED TRANSIT-MODERATE,NASAL REGURGITATION-PRESENT,MINOR ASPIRATION-PRESENT,MAJOR ASPIRATION-PRESENT DOSS SCORE-2 SEVERE DYSPHAGIA, TRACHEOSTOMY TUBE-INSITU	MINIMAL IMPROVEMENT
					C/O DIFFICULTY TO SPEAK	3 MONTHS																							

NUMBER	AGE (YEARS)	GENDER	DATE OF ADMISSION	OCCUPATION	CHIEF COMPLAINTS	DURATION	PAST HISTORY	ADDICTION	CLINICAL FINDINGS	GCS AT PRESENTATION	COMORBIDITIES	REPEAT SWALLOW	EDEMA OF VOCAL CORDS AND ARYTENOIDS	ULCERATION	DRYNESS OF MUCOSA	POOLING IN PYRIFORM FOSSA	SPILL OVER INTO LARYNX	DELAYED TRANSIT	NASAL REGURGITATION	ADYNAMIC SEGMENT	MINOR ASPIRATION	MAJOR ASPIRATION	TRACHEOSTOMY PERFORMED OR NOT	DIAGNOSIS	SEVERITY OF DYSPHAGIA-DOSS SCORE	AREA OF STROKE	FOLLOW UP	FOLLOW UP RESULTS	FOLLOW WP
13	63	MALE	20.05.2024	CLERK	C/O DECREASED RESPONSIVENESS	6 DAYS	H/O SIMILAR COMPLAINTS 2 YEARS BACK(RIGHT HEMIPARESIS), H/O SIMILAR COMPLAINT 15 DAYS BACK(LEFT HEMIPARESIS)	H/O ALCOHOL CONSUMPTION SINCE 10 YEARS	BILATERAL LOWER LIMB TONE AND POWER-DECREASED	E2V2M4, B/L PUPILS SUGGISHLY REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 2 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MODERATE	ABSENT	MODERATE	PRESENT-MODERATE	ARYEPIGLOTTIC FOLD SPILL OVER PRESENT	MODERATE	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.RECURRENT CVA WITH RIGHT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS 3.HYPERTENSION	MILD TO MODERATE DYSPHAGIA-SCORE 4	LEFT SUBCORTICAL	SWALLOW THERAPY GIVEN-AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-5 MILD DYSPHAGIA	IMPROVED
					C/O DECREASED MOVEMENT OF BILATERAL LOWER LIMB	1 MONTH																							
14	52	MALE	27.03.2024	BUSSINESSMAN	C/O GIDDINESS	1 WEEK	NO SIMILAR COMPLAINTS IN PAST	H/O CIGARETTE SMOKING SINCE 2 YEARS	TONE-LEFT SIDE FLACCIDITY PRESENT, LEFT UPPER LIMB AND LOWER LIMB POWER DECREASED	E4V4 M6 B/L PUPILS EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 10 YEARS, K/C/O DEEP VEIN THROMBOSIS SINCE 1 YEAR	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MODERATE	ABSENT	MODERATE	PRESENT-MODERATE	ARYEPIGLOTTIC FOLD SPILL OVER PRESENT	MODERATE	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH LEFT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS 3. HYPERTENSION 4.DEEP VEIN THROMBOSIS	MILD TO MODERATE DYSPHAGIA-SCORE 4	RIGHT SUBCORTICAL	SWALLOW THERAPY GIVEN-AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-5 MILD DYSPHAGIA	IMPROVED
					C/O SWAYING WHILE WALKING	1 WEEK																							
15	62	MALE	05.03.2024	BUSSINESSMAN	C/O SLURRING OF SPEECH	1 DAY	NO SIMILAR COMPLAINTS IN PAST	NO ILL HABITS	LEFT UPPER LIMB AND LOWER LIMB TONE AND POWER DECREASED, ANGLE OF MOUTH DEVAITION TO RIGHT, LOSS OF NASOLABIAL FOLD ON LEFT, EYE CLOSURE AND WRINKLING OF FOREHEAD NORMAL ON BOTH SIDES	E2V2M6 B/L PUPILS EQUAL AND REACTIVE TO LIGHT	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 10 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD	ABSENT	MILD	PRESENT	ARYEPIGLOTTIC FOLD & ARETENOIDS SPILL OVER PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH LEFT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS 3.HTN	SEVERE DYSPHAGIA-DOSS SCORE 2	PCA TERRITORY	SWALLOW THERAPY GIVEN-EFFORTFUL SWALLOW, FOLLOW UP AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-2 SEVERE DYSPHAGIA	NO IMPROVEMENT
					C/O DEVIATION OF ANGLE OF MOUTH TO RIGHT	1 DAY																							
					C/O WEAKNESS OF LEFT UPPER LIMB	1 DAY																							
16	65	MALE	02.04.2024	AGRICULTURIST	C/O DECREASED RESPONSIVENESS	1 DAY	NO SIMILAR COMPLAINTS IN PAST	H/O ALCOHOL CONSUMPTION SINCE 15 YEARS	B/L PLANTARS-MUTE, TONE-B/L UPPER LIMB AND LOWER LIMB NORMAL, POWER- COULDNOT BE ASSESSED AT PRESENTATION	E1V1M1, B/L PUPILS REACTIVE TO LIGHT	K/C/O HTN SINCE 5 YEARS K/C/O TYPE 2 DM SINCE 3 YEARS	PRESENT AND CAN SWALLOW WITH 3rd SWALLOW	MILD	ABSENT	MILD	MILD	SPILL OVER PRESENT JUST ABOVE VOCAL CORDS,VALECULLA	MILD	ABSENT	ABSENT	ABSENT	ABSENT	YES(12/4/2024)	1.ACUTE ISCHEMIC STROKE WITH RIGHT HEMIPARESIS COMMUNICATING HYDROCEPHALUS 2.TYPE 2 DIABETES MELLITUS 3.HTN	MILD DYSPHAGIA-DOSS SCORE 5	CEREBRAL CORTEX-LEFT PARIETAL LOBE	PATIENT IS DEAD		DEAD
17	45	MALE	05.04.2024	BUSSINESSMAN	C/O SLURRING OF SPPECH	1 DAY	NO SIMILAR COMPLAINTS IN PAST	H/O CIGARETTE SMOKING AND ALCOHOL CONSUMPTION SINCE 10 YEARS	TONE AND POWER NORMAL, PLANTAR-B/L FLEXOR	E4V5M6, B/L PUPILS EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 4 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD	ABSENT	MILD	PRESENT	ARYEPIGLOTTIC FOLD SPILL OVER PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH LEFT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS 3.HTN 4.CORONARY ARTERY DISEASE	MILD TO MODERATE DYSPHAGIA-SCORE 4	MCA TERRITORY	SWALLOW THERAPY GIVEN-EFFORTFUL SWALLOW, FOLLOW UP AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-5 MILD DYSPHAGIA	IMPROVED
					C/O WEAKNESS OF LEFT UPPER LIMB & LOWER LIMB	1 DAY																							

NUMBER	AGE (YEARS)	GENDER	DATE OF ADMISSION	OCCUPATION	CHIEF COMPLAINTS	DURATION	PAST HISTORY	ADDICTION	CLINICAL FINDINGS	GCS AT PRESENTATION	COMORBIDITIES	REPEAT SWALLOW	EDEMA OF VOCAL CORDS AND ARYTENOIDS	ULCERATION	DRYNESS OF MUCOSA	POOLING IN PYRIFORM FOSSA	SPILL OVER INTO LARYNX	DELAYED TRANSIT	NASAL REGURGITATION	ADYNAMIC SEGMENT	MINOR ASPIRATION	MAJOR ASPIRATION	TRACHEOSTOMY PERFORMED OR NOT	DIAGNOSIS	SEVERITY OF DYSPHAGIA-DOSS SCORE	AREA OF STROKE	FOLLOW UP	FOLLOW UP RESULTS	FOLLOW WP
18	65	MALE	02.04.2024	ATTENDER	C/O WEAKNESS OF RIGHT UPPER LIMB & LOWER LIMB	3 DAYS	NO SIMILAR COMPLAINTS IN PAST	H/O CIGARETTE SMOKING AND ALCOHOL CONSUMPTION SINCE 14 YEARS	LEFT UPPER LIMB AND LOWER LIMB TONE DECREASED, PLANTAR REFLEX- RIGHT FLEXOR AND LEFT- EXTENSOR	E2V1M6 , B/L PUPILS EQUAL AND REACTIVE TO LIGHT	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 5 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD	ABSENT	MILD	PRESENT	ARYEPIGLOTTIC FOLD SPILL OVER(+)	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH RIGHT HEMIPARESIS WITH LEFT CRURAL HEMIPLEGIA 2.TYPE 2 DIABETES MELLITUS 3.HTN	MILD TO MODERATE DYSPHAGIA-SCORE 4	CEREBRAL CORTEX-LEFT TEMPORAL LOBE	SWALLOW THERAPY GIVEN-EFFORTFUL SWALLOW, FOLLOW UP AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-5 MILD DYSPHAGIA	IMPROVED
19	46	MALE	29.04.2024	DRIVER	C/O WEAKNESS OF LEFT UPPER LIMB & LOWER LIMB	2 DAYS	NO SIMILAR COMPLAINTS IN PAST	H/O ALCOHOL CONSUMPTION SINCE 5 YEARS	TONE AND POWER NORMAL	E4V4 M6 B/L PUPILS EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 5 YEARS	REPEAT SWALLOW 2nd SWALLOW PRESENT	MILD	ABSENT	MILD	PRESENT	ABSENT	MILD	ABSENT	ABSENT	ABSENT	ABSENT	NO	1.LEFT HEMIPLEGIA 2.TYPE 2 DIABETES MELLITUS 3.HYPERTENSION 4.CEREBELLAR STROKE	MILD DYSPHAGIA-DOSS SCORE 5	CORTICAL AND SUBCORTICAL	SWALLOW THERAPY GIVEN	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-ABSENT,SPILLOVER-ABSENT, DELAYED TRANSIT ABSENT,MINOR ASPIRATION-ABSENT, DOSS SCORE-6 WITHIN FUNCTIONAL LIMITS	IMPROVED
20	52	MALE	26.04.2024	BUSSINESSMAN	C/O DECREASED RESPONSIVENESS	1 DAY	H/O SIMILAR COMPLAINTS 4 MONTHS BACK	H/O ALCOHOL CONSUMPTION SINCE 5 YEARS	LEFT PLANTAR-MUTE, RIGHT-FLEXOR, LEFT LOWER LIMB TONE AND POWER DECRAESED	E4V1M5, B/L PUPIL EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS SINCE 5 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD	ABSENT	MILD	PRESENT	ABSENT	MILD	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.CVA WITH LEFT LEFT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS 3.HYPERTENSION	MILD DYSPHAGIA-DOSS SCORE 5	RIGHT MCA INFARCT	SWALLOW THERAPY GIVEN-SUPRAGLOTIC SWALLOW AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-ABSENT,SPILLOVER-ABSENT, DELAYED TRANSIT ABSENT,MINOR ASPIRATION-ABSENT, DOSS SCORE-6 WITHIN FUNCTIONAL LIMITS	IMPROVED
					C/O WEAKNESS OF LEFT UPPER LIMB & LOWER LIMB	2 DAYS																								
21	69	MALE	20.04.2024	MANAGER IN ORPHANAGE TRUST	C/O WEAKNESS OF LEFT LOWER LIMB	10 DAYS	H/O SIMILAR COMPLAINTS 1 YEAR BACK	H/O CIGARETTE SMOKING SINCE 12 YEARS	LEFT PLANTAR-MUTE,RIGHT PALNTAR-EXTENSOR	E3V3M4, B/L PUPILS EQUAL AND REACTIVE TO LIGHT	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 15 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MODERATE	ABSENT	MODERATE	PRESENT	ARYEPIGLOTTIC FOLD & ARETENOIDS SPILL OVER PRESENT	MODERATE	ABSENT	PRESENT	ABSENT	ABSENT	NO	1) ISCHAEMIC STROKE WITH LEFT HEMIPLEGIA (CVA INVOLVING POSTERIOR CIRCULATION WITH LEFT UMN TYPE PALSY) 2) CHRONIC INFARCT IN PONTOMEDULLARY JUNCTION@HYPERTENSION 4) TYPE 2 DIABETES MELLITUS	MILD TO MODERATE DYSPHAGIA-SCORE 4	MCA TERRITORY AND SUBCORTICAL	SWALLOW THERAPY GIVEN-EFFORTFUL SWALLOW, FOLLOW UP AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-6 WITHIN FUNCTIONAL LIMITS	IMPROVED
					C/O DECREASED RESPONSIVENESS	3 DAYS																								
22	58	FEMALE	05.04.2024	HOUSEWIFE	C/O NUMBNESS OF RIGHT UPPER LIMB & LOWER LIMB	1 DAY	H/O SIMILAR COMPLAINTS 2 YEARS BACK	NO ILL HABITS	PLANTAR-EXTENSOR, TONE AND POWER DECREASED ON RIGHT SIDE	E4V4M6, B/L PUPILS EQUAL AND REACTIVE TO LIGHT	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 15 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	ABSENT	ABSENT	ABSENT	ABSENT	ARYEPIGLOTTIC FOLD & ARETENOIDS SPILL OVER PRESENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	CVA WITH RIGHT HEMIPARESIS	MILD DYSPHAGIA-DOSS SCORE 5	SUBCORTICAL	SWALLOW THERAPY GIVEN	NO REPEAT SWALLOW AND NO SPILL OVER,DOSS SCORE 7-NORMAL	NORMAL
					C/O BURNING SENSATION OF RIGHT UPPER LIMB & LOWER LIMB	1 DAY																								
23	51	MALE	15.04.2024	FARMER	C/O WEAKNESS OF LEFT UPPER LIMB & LOWER LIMB	3 DAYS	NO SIMILAR COMPLAINTS IN PAST	H/O CIGARETTE SMOKING AND ALCOHOL CONSUMPTION SINCE 15 YEARS	LEFT UPPER LIMB AND LOWER LIMB TONE AND POWER DECREASED, ANGLE OF MOUTH DEVAITION TO RIGHT	E2V2M3 B/L PUPILS EQUAL AND REACTIVE TO LIGHT	K/C/O TYPE 2 DIABETES MELLITUS SINCE 2 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD	ABSENT	MILD	MILD	ABSENT	MILD	ABSENT	ABSENT	ABSENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH LEFT HEMIPARESIS 2.TYPE 2 DIABETES MILLITUS 3.HTN	MILD DYSPHAGIA-DOSS SCORE 5	MCA TERRITORY	SWALLOW THERAPY GIVEN-SUPRAGLOTTIC SWALLOW,	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-resolved,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-ABSENT,SPILLOVER-ABSENT, DELAYED TRANSIT NONE,MINOR ASPIRATION-ABSENT, DOSS SCORE-6 WITHIN FUNCTIONAL LIMITS	IMPROVED
					C/O SLURRING OF SPPECH	3 DAYS																								

NUMBER	AGE (YEARS)	GENDER	DATE OF ADMISSION	OCCUPATION	CHIEF COMPLAINTS	DURATION	PAST HISTORY	ADDICTION	CLINICAL FINDINGS	GCS AT PRESENTATION	COMORBIDITIES	REPEAT SWALLOW	EDEMA OF VOCAL CORDS AND ARYTENOIDS	ULCERATION	DRYNESS OF MUCOSA	POOLING IN PYRIFORM FOSSA	SPILL OVER INTO LARYNX	DELAYED TRANSIT	NASAL REGURGITATION	ADYNAMIC SEGMENT	MINOR ASPIRATION	MAJOR ASPIRATION	TRACHEOSTOMY PERFORMED OR NOT	DIAGNOSIS	SEVERITY OF DYSPHAGIA-DOSS SCORE	AREA OF STROKE	FOLLOW UP	FOLLOW UP RESULTS	FOLLOW WP
24	67	FEMALE	19.05.2024	HOUSEWIFE	C/O WEAKNESS OF LEFT UPPER LIMB & LOWER LIMB	2 DAYS	NO SIMILAR COMPLAINTS IN PAST	NO ILL HABITS	LEFT UPPER LIMB AND LOWER LIMB TONE AND POWER DECREASED, RIGHT PLANTAR-FLEXOR, LEFT-EXTENSOR	E4V4 M6 B/L PUPILS EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS SINCE 3 YEARS, IHD SINCE 2 YEARS	PRESENT AND CAN SWALLOW WITH 3rd SWALLOW	MODERATE	ABSENT	MODERATE	PRESENT-MODERATE	ARYEPIGLOTTIC FOLD & ARETENOIDS SPILL OVER PRESENT	MODERATE	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.CVA WITH LEFT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS 3.HYPERTENSION	MILD TO MODERATE DYSPHAGIA-SCORE 4	RIGHT ACA TERRITORY	SWALLOW THERAPY GIVEN- EFFORTFUL SWALLOW, FOLLOW UP AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-6 WITHIN FUNCTIONAL LIMITS	IMPROVED
25	67	MALE	06.05.2024	FARMER	C/O WEAKNESS OF RIGHT UPPER LIMB & LOWER LIMB	1 DAY	H/O SIMILAR COMPLAINTS 2 MONTHS BACK	NO ILL HABITS	BILATERAL TONE AND POWER POWER NORMAL, SENSATION OF RIGHT LOWER LIMB REDUCED, RIGHT SIDE SWAYING PRESENT	E4V5M6, B/L PUPILS EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS SINCE 5 YEARS	REPEAT SWALLOW 3rd SWALLOW PRESENT	SEVERE	PRESENT	PRESENT-SEVERE	PRESENT	ARYEPIGLOTTIC FOLD & ARETENOIDS SPILL OVER PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	YES	1.CVA WITH RIGHT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS	SEVERE DYSPHAGIA-DOSS SCORE 2	LEFT PCA TERRITORY	SWALLOW THERAPY GIVEN	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-PRESENT,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-PRESENTT,SPILLOVER-PRESENT, DELAYED TRANSIT-MODERATE,MINOR ASPIRATION-PRESENT, DOSS SCORE-2	NO IMPROVEMENT
26	66	MALE	09.05.2024	SHOPKEEPER	C/O WEAKNESS OF LEFT UPPER LIMB & LOWER LIMB	2 DAYS	NO SIMILAR COMPLAINTS IN PAST	NO ILL HABITS	TONE AND POWER NORMAL	E4V4 M6 B/L PUPILS EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS SINCE 10 YEARS	REPEAT SWALLOW 2nd SWALLOW PRESENT	MILD	ABSENT	MILD	MILD	ABSENT	MILD	ABSENT	ABSENT	ABSENT	ABSENT	NO	1.CVA WITH RIGHT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS	MILD DYSPHAGIA-DOSS SCORE 5	CORTICAL AND SUBCORTICAL	SWALLOW THERAPY GIVEN	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-ABSENT,SPILLOVER-ABSENT, DELAYED TRANSIT-ABSENT,MINOR ASPIRATION-ABSENT, DOSS SCORE-7 NORMAL	IMPROVED
27	63	MALE	25.05.2024	FARMER	C/O WEAKNESS OF LEFT LOWER LIMB	1 WEEK	H/O SIMILAR COMPLAINTS 6 YEARS BACK	H/O CIGARETTE SMOKING AND ALCOHOL CONSUMPTION SINCE 40 YEARS	BILATERAL UPPER LIMB AND LOWER LIMB TONE-NORMAL, LEFT LOWER LIMB POWER-REDUCED	E4V5M6, B/L PUPILS EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 2 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MODERATE	ABSENT	MODERATE	PRESENT, STASIS IN VALECULLA-MODERATE	ARYEPIGLOTTIC FOLD & ARETENOIDS SPILL OVER PRESENT,CYST IN THE RIGHT ARYEPIGLOTTIC FOLD	MODERATE	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.CVA 2.TYPE 2 DM 3.HYPERTENSION	MILD TO MODERATE DYSPHAGIA-SCORE 4	RIGHT ACA TERRITORY	SWALLOW THERAPY-EFFORTFUL SWALLOW, AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-6 WITHIN FUNCTIONAL LIMITS	MINIMAL IMPROVEMENT
					C/O SWAYING WHILE WALKING TO RIGHT	1 WEEK																							

NUMBER	AGE (YEARS)	GENDER	DATE OF ADMISSION	OCCUPATION	CHIEF COMPLAINTS	DURATION	PAST HISTORY	ADDICTION	CLINICAL FINDINGS	GCS AT PRESENTATION	COMORBIDITIES	REPEAT SWALLOW	EDEMA OF VOCAL CORDS AND ARYTENOIDS	ULCERATION	DRYNESS OF MUCOSA	POOLING IN PYRIFORM FOSSA	SPILL OVER INTO LARYNX	DELAYED TRANSIT	NASAL REGURGITATION	ADYNAMIC SEGMENT	MINOR ASPIRATION	MAJOR ASPIRATION	TRACHEOSTOMY PERFORMED OR NOT	DIAGNOSIS	SEVERITY OF DYSPHAGIA-DOSS SCORE	AREA OF STROKE	F0LLOW UP	FOLLOW UP RESULTS	FOLLOW WP
28	68	FEMALE	12.05.2024	HOUSEWIFE	C/O WEAKNESS OF B/L LOWER LIMBS	15 DAYS	H/O SIMILAR COMPLAINTS 5 YEARS BACK	NO ILL HABITS	TONE AND POWER NORMAL, PLANTAR-B/L FLEXOR	E4V5M6, B/L PUPILS EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS SINCE 5YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW WITH RYLES TUBE INSITU	MODERATE, LEFT VOCAL CORD MOBILITY REDUCED	ABSENT	MODERATE	POOLING IN VALECULLA-MODERATE	PHARYNGOEPIGLOTTIC FOLD SPILLOVER PRESENT	MODERATE	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.CVA WITH LEFT HEMIPARESIS 2.TYPE 2 DM	MILD TO MODERATE DYSPHAGIA-SCORE 4	RIGHT ACA TERRITORY	SWALLOW THERAPY-EFFORTFUL SWALLOW, AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-6 WITHIN FUNCTIONAL LIMITS	IMPROVED
29	66	FEMALE	21.05.2024	HOUSEWIFE	C/O WEAKNESS OF B/L LOWER LIMBS	1 MONTH	H/O SIMILAR COMPLAINTS 2 MONTHS BACK	NO ILL HABITS	LEFT UPPER LIMB TONE AND POWER-NORMAL; RIGHT UPPER LIMB POWER-REDUCED	E4V5M6, B/L PUPILS EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS SINCE 8 YEARS, K/C/O BRONCHIAL-ASTHMA SINCE 2 YEARS	PRESENT AND CAN SWALLOW WITH 3rd SWALLOW WITH RYLES TUBE INSITU	MILD,B/L VOCAL CORDS MOBILE, DECREASED PHARYNGEAL MOVEMENT	ABSENT	MILD	PRESENT, STASIS IN VALECULLA	ARYEPIGLOTTIC FOLD & VALECULLA SPILL OVER PRESENT	MILD	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.POSTERIOR CIRCULATION STROKE 2.TYPE 2 DM 3.BRONCHIAL ASTHMA	MILD TO MODERATE DYSPHAGIA-SCORE 4	SUBCORTICAL	SWALLOW THERAPY-EFFORTFUL SWALLOW, AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-6 WITHIN FUNCTIONAL LIMITS	IMPROVED
30	59	FEMALE	08.05.2024	HOUSEWIFE	C/O WEAKNESS OF RIGHT UPPER LIMB & LOWER LIMB	1 DAY	NO SIMILAR COMPLAINTS IN PAST	NO ILL HABITS	RIGHT UPPER LIMB AND LOWER LIMB TONE AND POWER DECREASED	E3V3M5, BILATERAL PUPILS EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS SINCE 20 YEARS	PRESENT AND CAN SWALLOW WITH 3rd SWALLOW WITH RYLES TUBE INSITU	MILD,B/L VOCAL CORDS MOBILE	ABSENT	MILD	PRESENT, STASIS IN VALECULLA-MILD	ARYEPIGLOTTIC FOLD & ARETENOIDS SPILL OVER PRESENT	MILD	ABSENT	ABSENT	ABSENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH RIGHT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS	MILD DYSPHAGIA-DOSS SCORE 5	PRIVENTRICULAR AREA	SWALLOW THERAPY GIVEN-SUPERSUPRAGLOTTIC SWALLOW, AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-ABSENT,SPILLOVER-ABSENT, DELAYED TRANSIT-ABSENT,MINOR ASPIRATION-ABSENT, DOSS SCORE-7 NORMAL	IMPROVED
					C/O DECREASED RESPONSIVENESS	1 DAY																							
31	64	MALE	24.05.2024	AGRICULTURIST	C/O WEAKNESS OF RIGHT UPPER LIMB & LOWER LIMB ASSOCIATED WITH APHASIA	1 DAY	H/O SIMILAR COMPLAINTS 2 YEARS BACK	NO ILL HABITS	RIGHT UPPER LIMB AND LOWER LIMB TONE AND POWER DECREASED	E4V5M6, B/L PUPILS EQUAL AND REACTIVE	K/C/O METASTATIC ADENOCARCINOMA STOMACH	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD,B/L VOCAL CORDS MOBILE	ABSENT	MILD	MILD-POOLING IN PYRIFORM FOSSA	EPIGLOTTIS-SPILL OVER PRESENT	MILD	ABSENT	ABSENT	ABSENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH RIGHT HEMIPARESIS AND RIGHT UMN VII NERVE PALSY WITH APHASIA 2.METASTATIC ADENOCARCINOMA OF STOMACH	MILD DYSPHAGIA-DOSS SCORE 5	CEREBRAL COTTEX	SWALLOW THERAPY GIVEN-EFFORTFUL SWALLOW, FOLLOW UP AFTER 3 MONTHS, ADVISED SEMISOLID DIET	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-ABSENT,SPILLOVER-ABSENT, DELAYED TRANSIT-ABSENT,MINOR ASPIRATION-ABSENT, DOSS SCORE-7 NORMAL	IMPROVED

NUMBER	AGE (YEARS)	GENDER	DATE OF ADMISSION	OCCUPATION	CHIEF COMPLAINTS	DURATION	PAST HISTORY	ADDICTION	CLINICAL FINDINGS	GCS AT PRESENTATION	COMORBIDITIES	REPEAT SWALLOW	EDEMA OF VOCAL CORDS AND ARYTENOIDS	ULCERATION	DRYNESS OF MUCOSA	POOLING IN PYRIFORM FOSSA	SPILL OVER INTO LARYNX	DELAYED TRANSIT	NASAL REGURGITATION	ADYNAMIC SEGMENT	MINOR ASPIRATION	MAJOR ASPIRATION	TRACHEOSTOMY PERFORMED OR NOT	DIAGNOSIS	SEVERITY OF DYSPHAGIA-DOSS SCORE	AREA OF STROKE	FOLLOW UP	FOLLOW UP RESULTS	FOLLOW WP
32	74	FEMALE	14.05.2024	HOUSEWIFE	C/O WEAKNESS OF RIGHT UPPER LIMB & LOWER LIMB	1 MONTH	NO SIMILAR COMPLAINTS IN PAST	H/O TOBACCO CHEWING FOR 30 YEARS	RIGHT UPPER LIMB AND LOWER LIMB TONE AND POWER DECREASED	E4V4M6, B/L PUPILS EQUAL AND REACTIVE TO LIGHT	K/C/O HTN SINCE 15 YEARS ,NEWLY DIAGNOSED C/O CARCINOMA RIGHT LOWER GBS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD-B/L VOCAL CORDS MOBILE	ABSENT	MILD	PRESENT-POOLING IN VALECULLA	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH RIGHT HEMIPARESIS 2.HTN 3.NEWLY DIAGNOSED CASE OF CARCINOMA RIGHT LOWER GBS	MILD TO MODERATE DYSPHAGIA-SCORE 4	CEREBRAL CORTEX	SWALLOW THERAPY GIVEN-SUPERSUPRAGLOTTIC SWALLOW, AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-5 MILD DYSPHAGIA	IMPROVED
33	69	FEMALE	6.07.2024	HOUSEWIFE	C/O WEAKNESS OF RIGHT UPPER LIMB & LOWER LIMB	1 MONTH	NO SIMILAR COMPLAINTS IN PAST	NO ILL HABITS	RIGHT UPPER LIMB AND LOWER LIMB TONE AND POWER DECREASED	E4V4M6, B/L PUPILS EQUAL AND REACTIVE TO LIGHT	K/C/O TYPE 2 DIABETES MELLITUS SINCE 20 YEARS	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MILD-B/L VOCAL CORDS MOBILE	ABSENT	MILD	PRESENT, STASIS IN VALECULLA	EPIGLOTTIS-SPILL OVER PRESENT	MILD	ABSENT	ABSENT	ABSENT	ABSENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH RIGHT HJEMIPARESIS	MILD DYSPHAGIA-DOSS SCORE 5	CEREBRAL CORTEX	SWALLOW THERAPY GIVEN-EFFORTFUL SWALLOW, FOLLOW UP AFTER 3 MONTHS, ADVISED SEMISOLID DIET	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-RESOLVED,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-ABSENT,SPILLOVER-ABSENT, DELAYED TRANSIT ABSENT,MINOR ASPIRATION-ABSENT, DOSS SCORE-7 NORMAL	NORMAL
34	69	FEMALE	20.05.2024	HOUSEWIFE	C/O DECREASED RESPONSIVENESS	1 DAY	NO SIMILAR COMPLAINTS IN PAST	NO ILL HABITS	HYPERTONIA OF UPPER LIMB AND LOWER LIMB	E4V1M3, NECKL RIGIDITY PRESENT, SPEECH-APHASIA	K/C/O HTN SINCE 5 YEARS	PRESENT AND CAN SWALLOW WITH 3rd SWALLOW WITH RYLES TUBE INSITU	MILD-B/L VOCAL CORDS MOBILE	ABSENT	MILD	PRESENT, STASIS IN VALECULLA	ARYEPIGLOTTIC FOLD & VALECULLA SPILL OVER PRESENT	MILD	ABSENT	ABSENT	LOW GRADE ASPIRATION PRESENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH LEFT HEMIPARESIS 2.HTN	MILD DYSPHAGIA-DOSS SCORE 5	PERIVENTRICULAR AREA			DEAD
35	72	MALE	2.06.2024	BUSSINESSMAN	C/O WEAKNESS OF RIGHT UPPER LIMB & LOWER LIMB	1 DAY	NO SIMILAR COMPLAINTS IN PAST	H/O CIGARETTE SMOKING AND ALCOHOL CONSUMPTION SINCE 20 YEARS	RIGHT UPPER LIMB AND LOWER LIMB TONE AND POWER DECREASED	E4V4M6, B/L PUPILS EQUAL AND REACTIVE TO LIGHT	K/C/O HTN SINCE 15 YEARS,IHD SINCE 1 YEAR	PRESENT AND CAN SWALLOW WITH 2nd SWALLOW	MOFDERATE-B/L VOCAL CORDS MOBILE	ABSENT	MODERATE	POOLING IN VALECULLA-MODERATE	PHARYNGOEPIGLOTTIC FOLD SPILLOVER PRESENT	MODERATE	ABSENT	ABSENT	PRESENT	ABSENT	NO	1.ACUTE ISCHEMIC STROKE WITH RIGHT HEMIPARESIS 2.IHD 3.HTN	MILD TO MODERATE DYSPHAGIA-SCORE 4	SUBCORTICAL	SWALLOW THERAPY GIVEN-SUPRAGLOTIC SWALLOW AFTER 3 MONTHS	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-MILD,DRYNESS-MILD,POOLING IN PYRIFORM FOSSA-MILD,SPILLOVER-ABSENT, DELAYED TRANSIT-MILD,MINOR ASPIRATION-ABSENT, DOSS SCORE-6 WITHIN FUNCTIONAL LIMITS	IMPROVED
36	79	FEMALE	15.03.2024	HOUSEWIFE	C/O GIDDINESS	1 WEEK	NO SIMILAR COMPLAINTS IN PAST	NO ILL HABITS	TONE-LEFT SIDE FLACCIDITY PRESENT, LEFT UPPER LIMB AND LOWER LIMB POWER DECREASED	E4V4 M6 B/L PUPILS EQUAL AND REACTIVE	K/C/O TYPE 2 DIABETES MELLITUS & HYPERTENSION SINCE 10 YEARS	REPEAT SWALLOW 3rd SWALLOW PRESENT	SEVERE	PRESENT	PRESENT-SEVERE	PRESENT	ARYEPIGLOTTIC FOLD & ARETENOIDS SPILL OVER PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	PRESENT	YES	1.CVA WITH RIGHT HEMIPARESIS 2.TYPE 2 DIABETES MELLITUS	SEVERE DYSPHAGIA-DOSS SCORE 2	LEFT PCA TERRITORY	SWALLOW THERAPY GIVEN	REPEAT SWALLOW-2nd SWALLOW PRESENT , EDEMA-PRESENT,DRYNESS-RESOLVED,POOLING IN PYRIFORM FOSSA-PRESENT,SPILLOVER-PRESENT, DELAYED TRANSIT-MODERATE,MINOR ASPIRATION-PRESENT, DOSS SCORE-2	NO IMPROVEMENT
					C/O SWAYING WHILE WALKING	1 WEEK																							