**UNIVERSITY OF IOANNINA**

**SCHOOL OF HEALTH SCIENCES**

**FACULTY OF MEDICINE**

**SECTOR OF INTERNAL MEDICINE**

**2ND DEPARTMENT OF INTERNAL MEDICINE**

# Monitoring swallowing disorders in patients with ischemic stroke using speech-language scales and neuroimaging technology

**IOANNA-ELENI VIRVIDAKI**

Speech Language Pathologist

PhD Thesis

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**ΠΑΝΕΠΙΣΤΗΜΙΟ ΙΩΑΝΝΙΝΩΝ**

**ΣΧΟΛΗ ΕΠΙΣΤΗΜΩΝ ΥΓΕΙΑΣ**

**ΤΜΗΜΑ ΙΑΤΡΙΚΗΣ**

**ΠΑΘΟΛΟΓΙΚΟΣ ΤΟΜΕΑΣ**

**Β' ΠΑΘΟΛΟΓΙΚΗ ΚΛΙΝΙΚΗ**

# Καταγραφή των διαταραχών σίτισης-κατάποσης σε ασθενείς που υπέστησαν ισχαιμικό αγγειακό εγκεφαλικό επεισόδιο (ΑΕΕ) με τη χρήση λογοπαθολογικών κλιμάκων και τη βοήθεια σύγχρονων νευροαπεικονιστικών μεθόδων

**ΙΩΑΝΝΑ-ΕΛΕΝΗ ΒΙΡΒΙΔΑΚΗ**

ΠΑΘΟΛΟΓΟΣ ΛΟΓΟΥ-ΟΜΙΛΙΑΣ

ΔΙΔΑΚΤΟΡΙΚΗ ΔΙΑΤΡΙΒΗ

ΙΩΑΝΝΙΝΑ, 2020

**«Η έγκριση της διδακτορικής διατριβής από το Τμήμα Ιατρικής του Πανεπιστημίου Ιωαννίνων δεν υποδηλώνει αποδοχή των γνωμών του συγγραφέα Ν. 5343/32, άρθρο 202, παράγραφος 2 (νομική κατοχύρωση του Ιατρικού Τμήματος)».**

**Ημερομηνία αίτησης της κ. Βιρβιδάκη Ιωάννας-Ελένης:** 24-7-2013

**Ημερομηνία ορισμού Τριμελούς Συμβουλευτικής Επιτροπής**: 759α/14-2-2014

**Μέλη Τριμελούς Συμβουλευτικής Επιτροπής**:

Επιβλέπων

Μηλιώνης Χαράλαμπος, Αναπληρωτής Καθηγητής Παθολογίας του Τμήματος Ιατρικής του Πανεπιστημίου Ιωαννίνων

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Γιαννόπουλος Σωτήριος, Επίκουρος Καθηγητής Νευρολογίας του Τμήματος Ιατρικής του Πανεπιστημίου Ιωαννίνων

Νάσιος Γρηγόριος, Επίκουρος Καθηγητής Διαταραχών επικοινωνίας με αφασίες και συναφείς νευρολογικές διαταραχές του Τμήματος Λογοθεραπείας του ΤΕΙ Ηπείρου

**Ημερομηνία ορισμού θέματος**: 14-3-2014

*«Καταγραφή των διαταραχών σίτισης-κατάποσης σε ασθενείς που υπέστησαν ισχαιμικό αγγειακό εγκεφαλικό επεισόδιο (ΑΕΕ) με τη χρήση λογοπαθολογικών κλιμάκων και τη βοήθεια σύγχρονων νευροαπεικονιστικών μεθόδων»*

**ΟΡΙΣΜΟΣ ΕΠΤΑΜΕΛΟΥΣ ΕΞΕΤΑΣΤΙΚΗΣ ΕΠΙΤΡΟΠΗΣ** 910α/21-1-2020

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| **Τατσιώνη Αθηνά** | Αναπληρώτρια Καθηγήτρια Γενικής Ιατρικής του Τμήματος Ιατρικής του Πανεπιστημίου Ιωαννίνων |
| **Καλαμπόκης Γεώργιος** | Επίκουρος Καθηγητής Παθολογίας του Τμήματος Ιατρικής του Πανεπιστημίου Ιωαννίνων |
| **Κοσμίδου Μαρία** | Επίκουρη Καθηγήτρια Παθολογίας του Τμήματος Ιατρικής του Πανεπιστημίου Ιωαννίνων |
| **Μίχου Αμαλία** | Επίκουρη Καθηγήτρια Διαταραχών Επικοινωνίας του Τμήματος Λογοθεραπείας του Πανεπιστημίου Πατρών |

Έγκριση Διδακτορικής Διατριβής με βαθμό **«ΑΡΙΣΤΑ»** στις **18-2-2020**

**Ιωάννινα 26-2-2020**

**ΠΡΟΕΔΡΟΣ ΤΟΥ ΤΜΗΜΑΤΟΣ ΙΑΤΡΙΚΗΣ**

**Άννα Μπατιστάτου**

Καθηγήτρια Παθολογικής Ανατομίας

**Η Γραμματέας του Τμήματος**

**ΜΑΡΙΑ ΚΑΠΙΤΟΠΟΥΛΟΥ**

**Develop a passion for learning…**

**If you do, you will never cease to grow.**

Anthony J. D'Angelo

# PROLOGUE

This doctoral thesis was conducted in the Departments of Internal Medicine and Neurology of University Hospital of Ioannina. It is with immense gratitude that I acknowledge the guidance and mentorship of my supervisor and Professor of Internal Medicine, Dr Haralampos Milionis. Our cooperation in this research endeavor was a great honor for me. I would also like to express my sincere gratitude to Dr Grigorios Nasios, Associate Professor of Neurology at the Speech-Language Department of University of Ioannina and Dr Sotirios Giannopoulos Professor of Neurology at the University of Ioannina for their contributions and constructive criticism throughout this process.

This work would not have been possible without the eternal patience and support of my best friend and husband and our son Vassilis, who have graciously understood my passion for study and research and have allowed me to pursue my dreams to the detriment of being a wife and mother. I would also like to dedicate this work to my parents who have given me the opportunity of an education from the best institutions and support throughout my life.

I also share the credit of my work to my medical statistician Mr. George Dimakopoulos and my esteemed colleague Dr. Emilia Michou, Professor of Communication Disorders and Dysphagia who both stood out as a model of integrity and depth of knowledge in their respected fields.

I would also like to acknowledge my US colleagues, teachers and friends Amy Litwack, ASHA Board Certified Specialist in Swallowing Disorders and Dr Thomas Murry, Professor of Speech-Language Pathology in Otolaryngology for their warmth, unfailing support and willingness to offer me the benefit of their expert opinion throughout this research process. My sincere gratitude extends to my colleagues Dr. Debra Suiter, Dr. Steven Leder, Dr. Michaela Trapl, Dr. Bonnie Martin-Harris, whose work in the field of dysphagia has been truly inspirational to me throughout my clinical and academic course in the field.

Special thanks, to the physicians and other staff members of the Internal Medicine and Neurology Units for their warmth and support throughout this study, as well as my patients who are always a source of inspiration.

This thesis is only a beginning in my journey. Finally, I would like to leave the remaining space in memory of Dr. Logemann (1942-2014), a respected Professor, researcher and clinical pioneer for those of us who get aspired by the specialty field of Dysphagia. Thank you for advocating for patients and mentoring all of us, pushing us to always make sure the evidence supports what we do.

# Contents

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# *PART I*

# CHAPTER 1: INTRODUCTION TO CEREBROVASCULAR DISEASE AND STROKE

## 1.1 Definition of Cerebrovascular disease

The term cerebrovascular disease includes a variety of medical conditions that affect the blood vessels of the brain and the cerebral circulation. Arteries supplying oxygen and nutrients to the brain are often damaged or deformed in these disorders[1]. Restrictions in blood flow may occur from vessel narrowing (stenosis), clot formation (thrombosis), blockage (embolism) or blood vessel rupture (hemorrhage). The most common presentation of cerebrovascular disease is an ischemic stroke or a transient ischemic attack and sometimes a hemorrhagic stroke. For the purposes of this thesis, the pathogenesis of stroke is explained in greater length.

## 1.2 Definition and Pathogenesis of Stroke

Stroke is classically characterized as a neurological deficit caused by an acute focal injury to the central nervous system (CNS) due to a vascular cause and is a major cause of disability and death worldwide[2]. Advances in basic science, neuropathology, and modern neuroimaging have improved the understanding of ischemia, infarction, and hemorrhage in the CNS. Recent knowledge about the nature, timing and clinical recognition of stroke and its mimics in addition to imaging results have prompted updated definitions and clarifications on stroke and transient ischemic attack (TIA). The Stroke Council of the American Heart Association (AHA)/American Stroke Association (ASA) published a scientific statement in 2009 which proposed TIAs as “transient episodes of neurological dysfunction caused by focal brain, spinal cord or retinal ischemia without acute infarction”[3]. Currently, the World Health Organization defines stroke as “the interruption of blood supply to the brain, usually because a blood vessel bursts or is blocked by a clot. This cuts off the supply of oxygen and nutrients, causing damage to the brain tissue”[4]. In broader terms, therefore, stroke is an acute (emergency) neurological event in which parts of the brain undergo a sudden functional decline.

## 1.3 Types of stroke

The pathological background for stroke may either be ischemic or hemorrhagic disturbances of the cerebral blood circulation. Recent iterations by the International Classification of Diseases (ICD) system[5], classified cerebrovascular disorders chiefly as TIA, cerebral ischemic stroke, intracerebral hemorrhage (ICH) or subarachnoid hemorrhage (SAH).

### 1.3.1 Ischemic stroke (infarction)

According to the Trial of Org 10 172 in Acute Stroke Treatment (TOAST system), a stroke could be attributed to large artery atherosclerosis if the patient has clinical findings consistent with an infarction affecting the cerebral cortex or both deep and cortical structures, the brain stem, or cerebellum. Evidence for risk factors for accelerated atherosclerosis or symptomatic atherosclerotic

disease (coronary artery disease, aortic disease, and peripheral arterial disease) in other anatomic locations should further exist. Supportive brain imaging results would demonstrate a branch or large hemispheric, brain stem, or cerebellar infarction. Vascular imaging would detect either intracranial or extracranial stenosis or occlusion at the usual sites for atherosclerosis such as the origin of the internal carotid artery[6].

Conversely to these large thrombotic infarctions, some patients suffer small (<1.5 cm) deep infarcts restricted to the basal ganglia, internal capsule, thalamus, or brain stem due to a local disease of these vessels mainly related to arterial hypertension or diabetes mellitus. Traditional lacunar syndromes such as pure motor hemiparesis should not evidence large artery atherosclerosis in the clinically relevant artery on vascular imaging.

Embolic cerebral infarction is attributed to embolism of a clot in the cerebral arteries coming from other parts of the arterial system, for example, from cardiac lesions, either at the site of the valves or of the heart cardiac cavities, or due to rhythm disturbances with stasis of the blood, which allows clotting within the heart as seen in atrial fibrillation[7]. The patient should have a history of heart disease or evidence abnormal cardiac evaluation upon physical examination via electrocardiography, echocardiography. Other tests such as positive blood cultures among patients with infective endocarditis may further support a cardioembolic stroke.

In other rarer cases, typically in special populations like young adults, vascular imaging may demonstrate features consistent with a nonatherosclerotic vasculopathy such as cervical artery dissection or evidence of a prethrombotic disorder or multisystem vasculitis. This smaller group of patients would fall under the category of stroke of other determined cause according to the TOAST classification and their neurological findings may mimic either large or small artery occlusion.

Finally, some patients have no identifiable cause of stroke despite the diagnostic workup or conflicting causes are detected that could support either large or small vessel disease. There is persuasive evidence that most of these cryptogenic strokes are thromboembolic in nature[8].

### 1.3.2 Hemorrhagic stroke

Hemorrhagic subtypes of stroke, although less common than ischemic stroke and TIAs, still have a significant public health impact due to the high rates of mortality and morbidity in adults[9, 10]. ICH alone has a nearly 40% case-fatality rate at 30 days[11]. Spontaneous (non-traumatic) hemorrhages in the CNS are mainly caused by a vascular event such as arteriolar hypertensive disease[7] and result in injury to the CNS. They may occur after infarction, either spontaneously or caused by antithrombotic or thrombolytic therapy[12] and they can range in severity from minor petechial bleeding to hemorrhage causing mass effect and secondary injury[2]. As such, they are referenced with various terms in the literature as “hemorrhagic infarction,” “hemorrhagic transformation of infarction,” “ hemorrhagic conversion of infarction,” and “intracerebral hemorrhage”.

A stroke caused by intracerebral hemorrhage (ICH) is defined as “rapidly developing clinical signs of neurological dysfunction attributable to a focal collection of blood within the brain parenchyma or ventricular system that is not caused by trauma” whereas a stroke caused by subarachnoid hemorrhage (SAH) involves “rapidly developing signs of neurological dysfunction and/ or headache because of bleeding into the subarachnoid space”[2]. The presence of bleeding in the subarachnoid space (the space between the arachnoid membrane and the pia mater of the brain or spinal cord) needs to be confirmed either by brain imaging or by sampling of the cerebrospinal fluid (CSF) that occupies and circulates within the subarachnoid space. This latter subgroup of strokes, are mainly due to rupture of aneurysms at the bifurcations of large arteries at the inferior surface of the brain, arteriovenous malformation, vasculitis and cerebral amyloid angiopathy[13].

## 1.4 Epidemiology of stroke

In most developed countries, mainly the US, Canada and Australia, stroke is the third leading cause of death, after cardiac disease and cancer and a major cause of serious chronic disability in adults[14, 15]. Every year, more than **795,000** people in the United States suffer a stroke and about 610,000 of these are first or new strokes [16]. Of these strokes, approximately 87% are ischemic infarctions, 10% are primary hemorrhages, and 3% are subarachnoid hemorrhage[17]. In Europe, on an annual basis, approximately 1.1 million inhabitants suffer a stroke and ischemic stroke accounts for approximately 80% of cases[18].

Despite major improvements in primary prevention and acute treatment over the last decades, cerebrovascular accidents therefore still pose a major challenge to contemporary and future public health globally as indicated by changing demographic trends and the general projection of increased life expectancy worldwide[19]. This may also reflect an increase in stroke incidence from 1.1 million cases recorded in 2000 to more than 1.5 million in 2025[18], painting a compelling picture on the impact of stroke not only on patient and caregiver quality of life but also on health care costs.

Reliable information about the incidence of stroke globally comes from population-based stroke registries. At the beginning of the 21st century, the age-standardized incidence of stroke in Europe ranged from 95 to 290/100,000 per year, with one-month case fatality rates ranging from 13% to 35%[18]. Unique data on the incidence of stroke in Greece comes from a population-based study conducted in 1994-95 in Arcadia Province by Vemmos and colleagues[20], revealing annual incidence rates for patients aged between 45-84 years at 319.4/100,000, and for people aged >85 years at 2,661/100,000. The incidence rates were lower in women (271/100,000) than men (362/100,000). These colleagues revealed a fatality rate of 26.6% during the first 28 days after the stroke with no significant difference between the two sexes. Case fatality increased with age and was higher for intracerebral hemorrhage than for cerebral infarction.

In an effort to compare the various stroke mechanisms observed, these researchers found that the cardioembolic infarct was the most frequently noted in 23% of patients, probably due to the high

prevalence of atrial fibrillation (34%)[21], possibly attributed to insufficient primary prevention or possibly suggesting an actual increase in the prevalence of this particular risk factor. These numbers reflect higher incidence rate of stroke in Greece and implicate a poorer stroke outcome when compared to other developed countries of Western Europe and North America. Interesting data from the Eurostat Statistics Database clearly demonstrate that Greece has significantly higher mortality rates from stroke (higher by 50%) when compared to average mortality in other European countries[22].

## 1.5 Economic toll of stroke

US Statistics reveal that about 750,000 survivors of a cerebrovascular accident per year will recur and will require hospitalization, relating to an annual health care expense of $65.5 billion (Armstrong & Mosher, 2011)! In Europe, the suspected health care expenditures for stroke management exceed 38 million euros with a medium annual cost per stroke patient from 13.650€ to 34.000€ depending upon the severity of the clinical case (Brainin et al, 2000). It is surprising to find that the past few years that our country has been plagued by financial crisis, there was an increase by 17.3% in total health care costs related to hospitalization of acute stroke patient population, with numbers rising from 831.265 million euros to 1.00418 billion euros, accounting for about 10% of the total health care budget! The size of the problem in Greece appears to be vastly underestimated by both society and health care professionals in the medical world. CVD and stroke accounted for 14% of total health expenditures in 2012 to 2013, more than any major diagnostic group.

## 1.6 Functional health outcome

Beyond vital prognosis, stroke patients are also at increased risk of poor outcome within the first year of the event including re-hospitalization (33%), recurrent event (7 to 13%), dementia (7 to 23%) mild cognitive disorder (35 to 47%), depression (30 to 50%), and fatigue (35% to 92%), all of them contributing to affect health related quality of life. Given these observations, an urgent development of acute care provision, as well as resources for post-stroke therapeutic strategies, is needed.

## 1.7 Risk factors

Risk factors for stroke can be classified as modifiable and nonmodifiable. While increasing age, sex, and race/ethnicity are nonmodifiable risk factors for both ischemic and hemorrhagic stroke, arterial hypertension, smoking, diet, and physical inactivity are among some of the more commonly reported modifiable risk factors. A means of reducing the burden of stroke in the population requires both identification of modifiable risk factors and demonstration of the efficacy of stroke prevention interventions.

### 1.7.1 Nonmodifiable Stroke Risk Factors

*1.7.1.1 Age*

In general, stroke is considered a disease of ageing. Worldwide, it is recognized that strokes can and do occur at any age, but the incidence rapidly increases with age, doubling for each decade after age 55. Among adults ages 35 to 44, the incidence of stroke is 30 to 120 of 100,000 per year whereas for those ages 65 to 74, incidence is reported between 670 and 970 of 100,000 per year[23].

Emerging literature however documents a rise in the incidence and prevalence of ischemic stroke in young adults, aged 20-54 years old[24, Bejot, 2014 #617] perhaps reflecting changes in the sensitivity of diagnostic testing. In specific, results from national surveillance data showed annual increases in the prevalence of ischemic stroke hospitalizations among adults aged 14 to 44 years from 1995 to 2008[25]. Accordingly, in hemorrhagic stroke patients, the incidence is reported to increase after 45 years of age[26]. It is important to note that stroke may also occur in children mainly as a result of hemorrhage[27].

*1.7.1.2 Sex*

The relationship of sex to stroke risk depends on age. Women have as high or higher risk of stroke as men at young ages, however the relative risk at older ages is slightly higher for men[28].

Overall, women generally live longer than men and since the incidence of stroke increases with age, women typically have a higher stroke risk. This higher risk is also reflected in postmenopausal changes/hormonal changes that are associated with vascular conditions such as high blood pressure, high cholesterol and atrial fibrillation.

Follow-up data collected in 43 cohorts in 18 populations in 8 European countries surveyed for cardiovascular risk factors revealed that the risk of stroke increased by 9% per year in men and 10% per year in women[29].

*1.7.1.3 Race-ethnicity*

Literature review shows consistent ethnic disparities in stroke incidence. In general, blacks have twice a risk of stroke when compared with their white counterparts in the United States and in Europe and have higher mortality associated with stroke[30-32]. Similar findings of increased stroke incidence rates in blacks were demonstrated by a south London stroke register[33]. Lower socioeconomic status, biological differences between blacks and whites and higher prevalence of stroke risk factors in blacks contribute to these racial differences in stroke incidence and final outcome[34].

Furthermore, the risk for subarachnoid hemorrhage and intracerebral hemorrhage is substantially higher in younger black adults than white people[35]. Of further interest, racial variations have been demonstrated in the typical location of hemorrhage between American blacks and their white counterparts. Specifically, blacks are more likely to have deep or brainstem hemorrhages whereas whites are more likely to experience lobar hemorrhages[36]. The strong association between black race and first ever stroke does not remain for recurrent stroke[37].

*1.7.1.4 Genetics*

Hereditability for stroke is also known to be a nonmodifiable risk marker[38, Schulz, 2004 #627]. As is the case with other risk factors for stroke, the genetic risks of stroke vary by age, sex and race. The susceptibility for stroke increases even more when genetic conditions combine with unhealthy lifestyle choices such as tobacco use and unhealthy diet.

### 1.7.2 Modifiable Risk Factors

The well-documented modifiable risk factors are important as early identification and management of these factors can subsequently reduce the risk of stroke. Modifiable risk factors can be further divided into medical conditions and behavioral risk factors. The role of many traditional cardiovascular risk factors in causing stroke, such as arterial hypertension, diabetes mellitus, hyperlipidemia, and smoking are well established.

#### 1.7.2.1 Medical conditions

*1.7.2.1.1 Hypertension*

Hypertension is a primary risk factor for cardiovascular disease, including stroke, heart attack, heart failure and aneurysm. The higher the blood pressure, the higher the risk of stroke even among people who are not formally labelled as hypertensive. Regardless of hypertension status, blood pressure rises with increasing age, thereby increasing the lifetime risk of developing hypertension[39].

Hypertension constitutes by far the most important modifiable risk factor for stroke with a strong, direct, linear, and continuous relationship between blood pressure and stroke risk[40]. Heightened awareness and available treatment options have improved hypertension control. In addition to medication, patients with hypertension are encouraged to engage in behavioral lifestyle changes such as healthier diet and increased physical activity to reduce its negative impact.

*1.7.2.1.2 Diabetes Mellitus*

Diabetes mellitus is an independent risk factor for stroke with a 2-fold increased risk in stroke for diabetic patients, and stroke accounts for ≈20% of deaths in diabetics[39]. A steady increase in the incidence of type 2 diabetes mellitus (T2DM) related to unhealthy diet habits, obesity and reduced physical activity resulted in an exponential rise in diabetes-related cardiovascular morbidity worldwide in recent years[41]. Type 1 diabetes mellitus (T1DM) also increases the stroke risk although to a lesser degree[42]. Diabetic patients who have a stroke tend to be younger, are more likely to be black, and have a higher prevalence of other stroke risk factors[39]. The combined use of behavioral and medical interventions in diabetics reveals promising results in overall reduction of stroke risk.

*1.7.2.1.3 Hyperlipidemia*

Hyperlipidemia is defined as elevations of fasting total cholesterol concentration which may or may not be associated with elevated plasma triglyceride (TG) concentration[43]. Health care providers are concerned about hyperlipidemia because of its well documented association between elevated levels of blood lipids and the risk of cardiovascular disease, the leading cause of death in the United

States[44]. Moreover, stroke risk seems to depend on stroke subtype with a stronger association of cholesterol levels with large artery ischemic stroke than other ischemic stroke subtypes[45].

Whereas dyslipidemia is a major risk factor for coronary heart disease (CHD), its role in the pathogenesis of ischemic stroke and association with stroke risk are less clear. Overall, elevated low-density lipoprotein (LDL-C) levels appear to increase the risk of ischemic stroke as do low high-density lipoprotein (HDL-C) levels[46]. Additionally, it is reported that stroke risk for a hemorrhagic stroke increases as total cholesterol level decreases[47].

Data seems to be conflicting with regards to statin therapy. In the general patient population, the use of statins seems to consistently reduce the risk of total and ischemic stroke in patients with or without CHD[46]. However, among patients with history of previous hemorrhage, small vessel disease or cerebral amyloid angiopathy, Lauer and colleagues have demonstrated an association between statins and risk of intracerebral hemorrhage[48]. The relatively large reduction in risk of ischemic stroke and other ischemic events with statins, seems to outweigh any small increased risk of hemorrhage in most patients[39].

*1.7.2.1.4 Heart disease*

The link between heart disease and stroke is significant. People with coronary artery disease, angina or those with a history of heart attack due to atherosclerosis have significantly higher risk of having a stroke. Coronary heart disease often develops over decades as cholesterol-containing deposits (plaque) build up in the major blood vessels that supply the heart with blood, ultimately decreasing blood flow to the heart. This may be associated with chest pain (angina), shortness of breath or other coronary artery disease signs and symptoms [49]. Other heart conditions, such as heart valve defects, irregular heartbeat (including atrial fibrillation) and enlarged heart chambers, may cause blood clots that may break loose and cause a stroke.

*1.7.2.1.5 Atrial Fibrillation*

Atrial fibrillation (AF) is the most common clinically significant heart arrhythmia, with an overall prevalence of approximately 1 % in the general population[50] and an incidence which increases with each decade of life. More than one in six ischemic strokes can be traced to atrial fibrillation, whereas in people ages 80 and older, the proportion jumps to one in three[51]. The most clinically important complication from AF lies in the risk for cardiac thrombus formation and systemic embolism. Accordingly, atrial fibrillation has been shown to be a potent independent risk factor for embolic strokes[52].

Stroke risk varies greatly depending on the presence of other coexisting risk factors. Thromboprophylaxis with anticoagulants and anti-platelet agents can reduce the risk of stroke in selected patients with AF[53], but carries an increased risk of bleeding and may require lifestyle modifications such as dietary changes, and frequent monitoring if warfarin is used.

*1.7.2.1.6 History of Stroke or Transient Ischemic Attack*

A transient ischemic attack (TIA) is defined as a transient episode of neurologic dysfunction due to the focal brain, spinal cord, or retinal ischemia, without acute infarction or tissue injury[54]. It has a sudden onset and is considered a warning sign for an impending ischemic stroke. The risk is usually higher in the first 48 hours following this' mini-stroke'. Transient ischemic attacks are usually associated with a focal neurologic deficit and/or speech disturbance in a vascular territory due to underlying cerebrovascular disease.

Differentiating other potential causes which mimic TIA is important since early identification and management can potentially help in preventing a subsequent stroke. The diagnostic scheme should include but not be limited to vertigo, dizziness, seizures, headaches, bells palsy, drug withdrawal, dementia, electrolyte disorders, acute infections, syncope, and alcoholism[54].

#### 1.7.2.2 Behavioral Risk Factors

*1.7.2.2.1 Physical activity*

Physical inactivity is associated with many poor health effects, including stroke[39]. Generally, it seems that people who exercise frequently have a lower risk of stroke and stroke mortality than those who are inactive. Lee specifically showed that moderate and high levels of physical activity are associated with reduced risk of total, ischemic, and hemorrhagic strokes[55]. The relationship between physical activity and stroke may be due to the associated decrease in blood pressure, diabetes and excess body weight [56].

In his review, Mittleman and Mostofsky demonstrated that the risk of stroke acutely following moderate to vigorous exercise was significantly lower in subjects who had previously been physically active compared with those who had not[57]. Similarly, a large prospective cohort study demonstrated an inverse association between greater baseline cardiorespiratory fitness and stroke mortality in men[58], with those in the most fit group experiencing a 68% lower risk of stroke and death than those in the least fit group. This association remained after further adjustment for cigarette smoking, alcohol intake, body mass index, hypertension, diabetes mellitus, and parental history of coronary heart disease. Therefore, interventions that promote plaque stability and favourable changes in vascular wall function seem to have important implications for medical management of patients after stroke[56].

*1.7.2.2.2 Diet*

Diet influences the risk of stroke and the risk of other stroke risk factors, such as diabetes mellitus, hypertension, and dyslipidemia[39]. In specific, excess salt intake (sodium (Na+) is associated with adverse effects on the cardiovascular system such as hypertension and is significantly related to a higher risk of stroke event[59]. In contrast, habitual potassium intake is associated with lower incidence of vascular disease and over reduced stroke risk[60]. In a multicenter randomized trial in Spain, involving over 7,000 subjects, it was found that among people with high cardiovascular risk, a

Mediterranean diet supplemented with extra-virgin oil or nuts reduced the incidence of major cardiovascular events and stroke[61].

Stroke prevention programs emphasize the need for controlling body weight and specifically obesity as it is causally involved in the development of hypertension. Suk and colleagues (2003) found that abdominal obesity is an independent, potent risk factor for ischemic stroke in all race-ethnic groups and constitutes a stronger risk factor than body mass index (BMI) which is a common measure of obesity, exerting a greater effect among young people[62].

*1.7.2.2.3 Alcohol consumption*

High alcohol consumption contributes to a number of risk factors for cardiovascular disease and stroke. Heavy alcohol drinking is linked to hypertension, as well as poor blood pressure control in hypertensive patients who consume alcohol[39]. Moderate drinking has been associated with increased high-density lipoprotein cholesterol, improved insulin sensitivity and decreased levels of fibrinogen and inflammatory markers[63, Agarwal, 2002 #663]

The relationship of alcohol consumption to stroke risk has been a focus of research in several epidemiological studies concluding differing results. Its association with different stroke types has not been clearly delineated. In a large prospective cohort study involving a pool of 78,546 subjects, self-reported alcohol intake of 1 to 20 drinks/week was associated with reduced risk of any stroke. The risk increased significantly among individuals with heavy alcohol intake suggesting a J-shaped association[64]. Another meta-analysis concluded that light to moderate drinking (up to 2 drinks per day) is associated with a reduced risk of ischemic stroke and heavy drinking (more than 4 drinks per day) is related to increased risk of both ischemic and hemorrhagic stroke[65]. Further review of the literature suggests a more direct linear relationship of alcohol consumption with hemorrhagic stroke, such that consumption of even small amounts of alcohol seems to increase risk of hemorrhage[39].

*1.7.2.2.4 Cigarette smoking*

The evidence associating smoking to stroke is extremely convincing. Cigarette smoking is the single most preventable cause of mortality in society but it is still attributable to 140,000 strokes and to approximately 15% of all stroke deaths annually[66]. Smoking increases the risk of stroke in the short term by promoting thrombosis and reducing cerebral blood flow via arterial vasoconstriction[67].

Several studies conducted across various ethnicities and populations demonstrate a strong association between smoking and stroke risk, with current smokers having at least a two- to fourfold increased risk of stroke compared with lifelong nonsmokers or individuals who had quit smoking more than 10 years prior[68, 69]. These studies further suggest that environmental (second-hand) exposure to smoke is linked to increased risk of stroke even in nonsmokers. It has been reported

that the odds of a first stroke, fatal or nonfatal, were increased among nonsmokers and long-term ex-smokers exposed to second-hand smoke at home or at work compared with those not exposed [68]. In a large cohort study of over 27,000 individuals conducted in the US, it was found that environmental tobacco smoke at home of 20h or more/week (compared to those exposed to less than 1 h/week) was associated with a 1.29-fold and 1.50-fold increased risk of first ischemic stroke among men and women respectively[70]. Similar results of significant and independent association of spousal smoking with increased risk of first ischemic stroke among never-smoking women were reported by You and colleagues in a study performed in Melbourne[71]. Additionally, recent evidence suggests that patients who continue smoking after the index stroke have a nearly two-fold risk of stroke recurrence than nonsmokers and that there exists a dose–response relationship between smoking quantity and risk of a recurrent stroke[72].

### 1.7.3 Other Potential Medical Risk Factors

#### *1.7.3.1 Hypothyroidism*

Subclinical hypothyroidism is defined as elevated serum thyroid-stimulating hormone levels, outside the reference range and levels of free thyroxine ($FT_4$) values [73]. Literature review has revealed no overall effect of subclinical hypothyroidism on stroke[74]. However, a recent study found an increased risk of fatal stroke in subjects younger than 65 years and in those with higher TSH levels concentrations (7.0 to 9.9 mIU/L) [75]. These colleagues collected and analyzed individual participant data on 47 573 adults (3451 subclinical hypothyroidism) from 17 cohorts and followed up from 1972 till 2014. They detected a hazard ration (HR) of 1.57 for stroke events and a HR of 1.61 for stroke mortality for those participants with subclinical hypothyroidism compared to subjects with euthyroidism.

The mechanisms, by which subclinical hypothyroidism increase the risk for stroke event, may be explained by the increased prevalence of cardiovascular risk factors in those with subclinical hypothyroidism[75]. Specifically, thyroid hormone deficiency is related to hypertension, hypercholesterolemia and atherosclerosis[76]. Further studies reveal relationships between subclinical hypothyroidism and the risk of clinical cardiovascular outcomes such as coronary heart disease and heart failure in specific subgroups with higher TSH levels[77].

#### *1.7.3.2 Rheumatoid arthritis*

The role of inflammation in the development of cardiovascular disease has only been recognized relatively recently. Rheumatoid arthritis (RA) is a chronic, debilitating autoimmune disease that affects the joints and it is associated with cardiovascular morbidity and stroke[78]. The exact pathogenesis of cardiovascular disease, including stroke, in rheumatoid arthritis is complex and involves several intermediate factors, including dyslipidemia, elevations in serum homocysteine, impaired insulin sensitivity, and endothelial dysfunction[79].

Emerging data with over 10,000 patients with rheumatoid arthritis suggest that primarily young patients (under 65 years) had more prominent cardiovascular risk factors and higher stroke rates in comparison to age and gender matched controls[78]. In multivariate analysis, rheumatoid arthritis was shown to be independently associated with stroke risk. Similar results were reported by Dhillon and colleagues (2015) who recognized the need to carefully calculate and monitor each patient's cardiovascular risk [80].

*1.7.3.3 Chronic Obstructive Pulmonary Disease*

Worldwide, chronic obstructive pulmonary disease (COPD) and stroke are leading causes of death. Increasing evidence suggests an association between both diseases, either caused by an increased atherosclerosis risk in patients with COPD or as a consequence of shared risk factors between stroke and COPD[81]. Chronic obstructive pulmonary disease (COPD) is primarily characterized by persistent airflow limitation that is associated with an abnormal inflammatory response of the lungs to noxious particles or gases, but it is also frequently associated with systemic effects and/or comorbidities[82].

In a large prospective population-based cohort study following approximately 14,000 participants aged 45 years and older for occurrence of first-ever stroke, it was found that COPD was associated with higher risks of both ischemic and hemorrhagic stroke[81]. It was also reported that patients with COPD have an approximately 20% increased risk of stroke, which was in accordance to results from previous studies[83, 84]. Of further importance, after an acute severe exacerbation, it the risk of stroke was six-fold higher than during stable disease which led the researchers to the conclusion that acute inflammation might lead to a higher risk of stroke. In cases of severe exacerbations, smoking cessation and control of comorbid cardiovascular factors were suggested as significant preventative options.

# CHAPTER 2: INVESTIGATION OF DYSPHAGIA AND ITS ASSOCIATED COMPLICATIONS

## 2.1 The act of swallowing

Swallowing is a complex sensorimotor activity, not only peripherally but also centrally, which depends on highly organized interactions among the cerebral cortex, the brainstem swallowing center and cranial nerves V, IX, X and XII. This process has both voluntary and involuntary (reflexive) components, reflecting central regulatory pathways within swallowing centers in the cortex and brainstem respectively[85, 86].

Swallowing is essentially a reflex, which follows a set pattern initiated in the brainstem and it is a synchronous and continuous event, once triggered. The events occur in a set order, but the duration of laryngeal elevation, upper esophageal segment opening and breath holding will vary depending on the bolus characteristics such as its volume and viscosity. Stroke may affect swallowing at multiple levels due to the interruption of the feedback loop with recovery depending on the cortical recovery[87].

Swallowing has four sequential coordinated phases: the oral preparatory phase, the oral propulsive phase, the pharyngeal phase and the esophageal phase. Each of the phases of a normal swallow is described below[88]:

**Oral Preparatory Phase**: During this phase, food in the oral cavity is manipulated and masticated in preparation for swallowing. The back of the tongue controls the position of the food, preventing it from falling into the pharynx.

**Oral Propulsive Phase**: During this phase, the soft palate lifts, closing the nasopharynx and the tongue transfers the bolus of food posteriorly to the pharynx, triggering the pharyngeal swallow.

**Pharyngeal Phase:** This is the phase whereby complex and coordinated movements of the tongue and pharyngeal muscles propel the bolus from the pharynx into the esophagus. The closure of the vocal cords and the backward movement of the epiglottis prevents food or liquid from entering the trachea.

**Esophageal Phase:** During the esophageal phase of swallowing, coordinated contractions of the esophageal muscle propel the bolus through the esophagus towards the stomach.

Under normal function, the triggered pharyngeal swallow involves several cortical and subcortical pathways. The interactions of regions above the brain stem and the brain stem swallowing network are yet not fully understood, particularly in humans[85].

## 2.2 Coordination Between Swallowing and Breathing

Breathing and swallowing are physiologically linked to ensure effortless gas exchange during oronasal breathing and to prevent aspiration during swallowing[89, 90]. Breathing briefly ceases during

swallowing to protect the airway, due not only to the physical closure of the airway by soft palate elevation and epiglottic inversion, but also due to neural suppression of respiration in the brainstem[91]. In normal individuals, a highly consistent pattern of respiratory–swallow phasing has emerged for liquid swallows. These swallows usually occur during a pause in the expiratory phase of the breathing cycle[89]. The respiratory pause continues for 0.5 to 1.5 s during swallowing and respiration usually resumes with expiration[92, 93]. Swallowing during the expiratory phase perhaps aids in assuming a partially adducted true vocal fold position before the initiation of the pharyngeal swallow and later during descent of the larynx[89]. Swallowing during expiration may also precipitate the forward and upward motion of the hyoid and the larynx which is required for complete airway closure and cricopharyngeal opening in both adults and infants[93, 94]. As reported in the study conducted by Trosche and colleagues, the duration of apneic episode is relatively unchanged across boluses less than 20 ml but increases with larger boluses, a fact directly associated with increased duration of laryngeal elevation[91]. Steele and Cichero (2014)[95] further add that while swallowing large volumes of liquids (100 mL), aspiration is more common when swallow apnea is not bracketed by an exhalation–exhalation respiratory pattern.

## 2.3 Oropharyngeal Dysphagia Following A Cerebrovascular Accident (CVA)

The ultimate consequences of stroke are associated with the identification and the extent of damage to the brain cells and usually include sensory, motor and speech and language deficits. It is now well established that a quickly observed complication after a CVA is dysphagia, a term which generally is used to describe eating and swallowing difficulties. It is derived from the Greek word ‘dys’ meaning ‘with difficulty’, ‘dysfunction’ or ‘pathology’ and ‘phagia’, meaning ‘to eat’. Dysphagia refers to any disturbances affecting the safe and adequate passage of food from the mouth to the stomach such as the delay or lack of muscle coordination, the limited range of movement or the aspiration of saliva, solid and/or liquid [96, 97].

Signs and symptoms of dysphagia include: Choking on food, coughing during meals, drooling or loss of food from mouth, pocketing of food in cheeks, slow, effortful eating, difficulty when swallowing pills, avoiding food or fluids, complaining of food sticking in throat, problems swallowing[98]. Several pathophysiologic complications are associated with dysphagia. Dysfunction related to the manifestation of dysphagia post-stroke is consistently reported and complications in the pharyngeal phase of swallowing are highly prevalent.

## 2.4 Dysphagia and Lesion Topography

Empirical data reveals that lesions in specific cortical locations may be more common in patients with dysphagia or those with a risk of aspiration. Brainstem lesions are well cited as having a strong association with the presence of dysphagia[99, 100 101, 102]. In specific, brainstem lesions have been associated with pharyngeal stage impairments including decreased laryngeal elevation movement,

delay in pharyngeal triggering time as well as impaired upper esophageal opening especially evident in lateral medullary lesions[103].

Functional neuroimaging has helped us understand that in addition to known brain stem areas, volitional swallowing in humans is represented within a number of spatially and functionally distinct cortical loci which may participate differentially in the regulation of swallowing[104]. Hence, fMRI evidence highlights the possible role of the primary motor cortex in controlling swallowing [104, 105] although there seems to be a more critical component of sensory versus motor inputs in the act of deglutition. In specific, several studies have agreed on the role of the right primary sensory cortex in dysphagia[101, 106-108]and the higher occurrence of primary sensory cortex lesions in dysphagic patients has been attributed to the role of sensory inputs in controlling complex swallowing movements[100].

Martin et al[104] further revealed that stimulation of the insula was significantly lateralized to the right hemisphere for the voluntary saliva swallow, suggesting a functional hemispheric dominance of the insula for the processing of swallowing. This finding is in accordance with findings from studies that followed[86, 100, 109]. Dehaghani and his team further emphasize a significant relation between the right internal capsule and swallowing disorder. A significant association of dysphagia with lesions in the cerebellum and diencephalon was not found, however, the researchers highlight that these areas' activities be better examined by functional neuroimaging techniques in future studies.

Other observational studies have found that the presence of dysphagia correlated significantly with the vascular territory of the infarct. In their recent published study, Kim and colleagues[110] compared the patterns of post-stroke swallowing difficulties according to the vascular territories involved in the stroke of over 100 patients diagnosed with first-ever ischemic stroke. When compared, territorial anterior circulation infarcts were associated with oral phase dysfunction while territorial posterior circulation infarcts (TPI) and white matter disease resulted in complications in the pharyngeal phase of swallowing. Additionally, the incidence of penetration and aspiration was significantly increased in patients with TPI.

Similarly, Langdon and colleagues[111]followed a cohort of 88 acute stroke patients over 30 days and found that patients with lesions in the territory of anterior circulation showed a high incidence of dysphagia within one week after the stroke, representing 75% of patients with dysphagia 2 days after their stroke and 90% of those with persistent dysphagia after the first week. These findings seem to be comparable with earlier data[112, 113]which show that anterior cerebral artery infarctions were mostly implicated for dysphagia. In the study conducted by Sundar and colleagues[113], lacunar infarcts were accompanied by a lower risk of developing dysphagia (18%), possibly reflecting smaller size lesions and better collateral circulation through the circle of Willis. Upon analysis of subcortical lesions with dysphagia, brainstem strokes had the highest incidence due to lower motor involvement of bulbar swallowing mechanisms.

Finally, in 2011, Teismann et al.[99], conducted a study measuring changes in brain activity during self-paced swallowing in 37 patients in the subacute stroke recovery phase. They observed a significant bilateral reduction of cortical activation for swallowing in patients with dysphagia following hemispheric stroke. In those infarcts without dysphagia, they found bilateral activation during self-paced swallowing while in the smaller group of patients with brainstem strokes, they noted a decrease in cortical activation and a right hemispheric lateralization, which led them to conclude that the cerebral cortex is directly searching for decompensation mechanisms for the swallowing disturbances produced subcortically.

## 2.5 Dysphagia incidence and prognosis after stroke

Incidence of dysphagia varies greatly from 19% to 81% [114, 115]. The wide disparity in estimated figures is largely accounted for by the definition of 'dysphagia' utilized by researchers, the methods used to identify dysphagia, the clinical determinants considered representative of dysphagia, and the timing of the assessment post-stroke [115 2010 #158]. While observational studies suggest that in the first 24 hours post-stroke, between 30-40% of conscious individuals present with dysphagia, other researchers report findings of 50% of consecutively admitted patients assessed as being 'at risk of aspiration' due to post-stroke dysphagia[116].

In a review of studies that carefully observed the varied diagnostic methods administered to stroke survivors, Martino and colleagues found that regardless of the topographic location of the lesion, data on incidence of dysphagia varied from 37% to 45% using simple detection methods (screens), from 51% to 55% using bedside clinical examination and from 64% to 78% using instrumental testing[117]. The authors reported that although an objective test is able to identify more precisely the biomechanics of the swallowing disorder as opposed to a simple screening bedside method, it is not necessary that the resulting data effectively represent the pathophysiology of swallowing and not the effects of normal aging on the swallowing mechanism. Additionally, obtaining an instrumental swallowing evaluation is not warranted in all acute stroke cases and is not cost effective.

Dysphagia is a serious consequence of stroke because of the risk of aspiration pneumonia, malnutrition, dehydration, weight loss, airway obstruction and ultimately death [118, Mann, 2000 #268]. The severity and the characteristics of dysphagia are strictly related to the site, the type (ischemic vs. hemorrhagic) and the extent of the neurological lesion, but also related to the age and the general health conditions of the patient [119]. For many patients, dysphagia is transient and resolves spontaneously[116, 120]. Among acute stroke survivors with dysphagia, 50% return to satisfactory nutrition within 14 days from the onset of stroke, while the remaining 50% develop chronic dysphagia. The majority of patients with persistent dysphagia resort to alternative methods of nutrition such as nasogastric and gastrostomy tubes[121].

The health risk to dysphagic patients with stroke is further compounded by many physical and cognitive functions which may be impaired and affect the eating process. Decreased levels of

alertness, fatigue, inability to maintain trunk and head alignment, reduced postural stability and tone, limb and body apraxia, visual perceptual difficulties, cognitive and communication problems, as well as lack of insight and depression, can all impact the amount of food and drink stroke patients are able to consume. The coordination of the swallow mechanism affects not only the individual's level of nutrition and hydration, but is an indicator of their rehabilitative potential[122].

## 2.6 The importance of early detection and management of dysphagia

In their study, Smithard and colleagues[123]demonstrated the importance of early detection of dysphagia after a stroke. These researchers found that dysphagia recognized via the use of a bedside swallowing examination was associated with a 17% increase in the incidence rates of pulmonary inflammation when compared to patients who were not found to have any swallowing pathophysiology. In a more recent study by Writh and colleagues[124], it was reported that the risk of developing pulmonary aspiration was up to 12 times greater in stroke patients with dysphagia than in stroke patients without accompanying dysphagia.

Other studies have focused on the impact of dysphagia in the overall quality of life and found that only 45% of stroke survivors with dysphagia find pleasure in feeding while 41% of patients are experiencing anxiety or stress during meals. Interestingly enough, it was reported that more than 1/3 of patients avoid circumstances of social feeding because of their swallowing problems[125]. One fact to which all researchers unanimously conclude is that swallowing disorders are added as a negative prognostic marker in the clinical picture of a patient who suffered a CVA since they have proven to prolong hospitalization, increase the total health care costs and are associated with increased rates of premature mortality within 90 days from the onset of stroke[116, 126, 127].

Hence, early detection and management of feeding-swallowing problems can minimize not only the aforementioned associated complications of a stroke but also optimize patient outcomes, reduce the overall health care expenses[97] while striving towards the direction of maintaining any enjoyment one can still get from daily feeding. This becomes of increased importance in developing countries where data on stroke care provision are sparse and there seems to be a growing need to develop well organized stroke units and guidelines for dysphagia management[128].

## 2.7 Medical Complications of Dysphagia 'Under the Microscope'

### 2.7.1 Malnutrition and dehydration

Other studies have revealed significant relationships between dysphagia, malnutrition, and dehydration after stroke, especially in patients who receive thickened liquids or modified diets [129, 130]. This was also previously reported by Gordon and her colleagues[131] in a prospective longitudinal study whereby the condition of dehydration was defined as 'a negative fluid balance (urine output greater than fluid input), a fluid intake of less than 0 5 litres a day, a packed cell volume of 0-48 or

higher, or a urea concentration of 10 mmol/l or higher on at least one occasion'. The limited ability of patients to achieve a safe and adequate intake of food and fluids does not only pose a greater risk for malnutrition and dehydration but is also linked to an increased rate of pulmonary complications[132], longer hospital stays[133] and an increased likelihood of being discharged to long-term care facilities, which may further adversely affect patient outcome[134].

### 2.7.2 Aspiration and its adverse sequelae

In patient care, the term 'aspiration' is often used to describe the inflow of material from the oral cavity or upper gastrointestinal tract into the lungs (below the level of true vocal cords) through the larynx as shown in figure 1. True aspiration indicates tracheal contamination [135]. The term itself does not reflect the nature of the material or the specific outcome of the event[136]. The material that can be aspirated varies and includes saliva, nasopharyngeal secretions, bacteria, liquids, toxic substances, food, or gastric contents. Additional variations are noted in the outcome of an aspiration event. It can remain within the spectrum of normal physiology or result in very severe conditions such as acute respiratory distress syndrome (ARDS)[137] or aspiration pneumonia (infectious process secondary to an aspiration event) caused by macroaspiration[136]. An aspiration event however may also be silent (unwitnessed) as the patient may not elicit a cough reflex upon evaluation[138, 139]. It is, therefore, reasonable that dysphagic patients who aspirate are at increased risk of developing aspiration pneumonia.

The diagnosis of aspiration should be suspected when the stroke patient has any of the following: a subjective complaint of trouble swallowing, an abnormal chest x-ray, congested voice quality, or a delay in voluntary initiation of the swallow reflex and coughing during or after swallowing[140]. Traditionally, diagnosis is initially established through clinical assessment involving an oral motor examination followed by the introduction of one or several teaspoons of water. This process is further detailed in the following chapters.

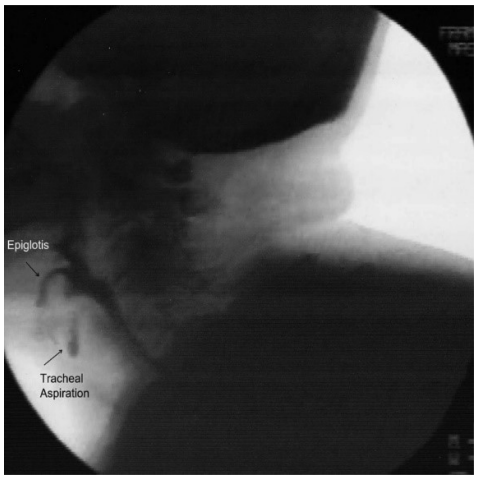



***Figure 2.1:*** Evidence of Tracheal Aspiration in Videofluoroscopy. Adapted from Aspiration Pneumonia After Stroke Intervention and Prevention by JR Armstrong and BD Mosher, 2011, *Neurohospitalist*, 1(2), p. 86.

While all stroke patients are potential aspirators, there are certain identifiable risk factors that have been recognized as greatly increasing the likelihood of aspiration[141].

These clinical risk factors for aspiration are listed in Table 1.

*2.7.2.1 Risk Factors for Aspiration Post Stroke*

**Table 2.1**

| | |
|---|---|
| Brainstem Stroke | Wet-hoarse voice quality |
| Difficulty swallowing oral secretion | Recurrent lower respiratory infections |
| Coughing/throat clearing or wet, gurgly voice quality after swallowing water | Low-grade fever or leukocytosis |
| Choking more than once while drinking 50 ml of wate | Auscultatory evidence of lower lobe congestion |
| Weak voice and cough | Immunocompromised state |

***Note.*** Table Reproduced from Dysphagia and Aspiration Following Stroke, Chapter 15, Evidence Based Review of Stroke Rehabilitation, 2018, p. 9. Retrieved from *http://www.ebrsr.com/sites/default/files/v18-SREBR-CH15-NET.pdf*

## 2.7.3 Aspiration pneumonia

Despite the propensity for recovering after acute stroke, the presence of dysphagia carries a 12-times greater risk of pulmonary complications such as aspiration pneumonia [124], an infection that increases the catabolic condition of the patient [142] and is associated with the highest attributable mortality rate of all medical complications following a CVA with over 40,000 deaths annually [14, 132, 143 2006 #469]. In the GAIN (Glycine Antagonist Neuroprotection International) study, a multicentre, multinational study of 1455 stroke subjects, 142 patients died during the first week following hospital admission. Among the fatality cases, 34 (23.9%) died from pneumonia[144]. Aspiration pneumonia is thought to occur when the lung's natural defenses are overwhelmed when excessive and/or toxic gastric contents are aspirated, leading to a localized infection or a chemical pneumonitis[141]. In her book review on how to work with dysphagia, Ruth Sanders also acknowledged the secondary effects of reduced stamina, increased likelihood of pressure sores, reduced physical recovery, reduced wound healing and increased risk of anxiety or depression[145] associated with aspiration pneumonia.

*2.7.3.1 Diagnostic criteria for aspiration pneumonia*

Clinical criteria for aspiration pneumonia vary across studies and this is believed to influence its incidence. The following table demonstrates the varied inclusion criteria reported for the diagnosis of poststroke aspiration pneumonia.

**Table 2.2 Diagnostic Criteria for Poststroke Aspiration Pneumonia**

| Study | Clinical Criteria |
|---|---|
| **Johnson et al (1993)[146]** | Either segmental consolidation or infiltrate on chest x -ray or clinical diagnosis which included an episode of respiratory difficulty with segmental moist rales on auscultation and two other symptoms including temp >100 °F, WBC >10,000 or hypoxia. |
| **DePippo et al (1994)[147]** | A positive chest x-ray or the presence of at least three of the following: temp > 100 °F, drop in PO2 > 10 torr, presence of WBC in sputum and/or positive sputum culture for pathogen. |
| **Holas et al (1994)[148]** | A positive chest x-ray or the presence of at least three of the following: temp > 100 °F, drop in PO2 > 10 torr, presence of WBC in sputum and/or positive sputum culture for pathogen. |
| **Kidd et al (1995)[149]** | Based on the production of sputum in conjunction with the development of crackles on auscultation, with or without the presence of fever or leukocytosis. |
| **Smithard et al (1996)[118]** | Presence of at least two of the following: tachypnea (> 22/min), tachycardia, aspiratory crackles, bronchial breathing or antibiotic usage. |
| **Teasell et al (1996)[150]** | Radiological evidence of consolidation, and at least one other clinical feature including granulocytosis, temp >38°C and/or shortness of breath. |
| **Carnaby et al (2006)[151]** | Diagnosed on the basis of 3 of the following indicators: temp >38°C, productive cough, abnormal respiratory exam including tachypnea, (> 22 breaths/min), tachycardia, inspiratory crackles, bronchial breathing, abnormal chest x-ray, arterial hypoxemia (PO2 < 9.3 kPa), culture of a relevant pathogen; positive chest radiography. |
| **Dziewas et al (2008)[152]** | Diagnosed on the basis of 3 of the following indicators: temp >38°C, productive cough with purulent sputum, abnormal respiratory exam including tachypnea, (> 22 breaths/min), tachycardia, inspiratory crackles, bronchial breathing, abnormal chest x-ray, arterial hypoxemia (PO2 < 9.3 kPa), culture of a relevant pathogen; positive chest radiography. |

***Note.*** Table Adapted from Dysphagia and Aspiration Following Stroke, Chapter 15, Evidence Based Review of Stroke Rehabilitation, 2018, p. 14-15. Retrieved from *http://www.ebrsr.com/sites/default/files/v18-SREBR-CH15-NET.pdf*

*2.7.3.2 Clinical Profile of Patients Viewed as 'High Risk' for Developing Poststroke Aspiration Pneumonia*

The multivariate analysis of Finlayson and colleagues[132] indicated that the older age, the presence of dysphagia, the male gender, the severity of stroke as well the premorbid level of self-care in conjunction with health comorbidities such as coronary heart disease and chronic obstructive pulmonary disease were important prognostic risk factors for developing pneumonia after stroke. Interestingly enough, severity of stroke was the factor with the most relation to aspiration pneumonia while factors such as hypertension, atrial fibrillation, cancer, progressive dementia and diabetes were not significantly associated with the development of pneumonia during the first month after the stroke. The authors added that patients with lacunar strokes were less likely to develop pneumonia than patients who suffered different types of ischemic damage. In this recent large cohort study which involved 8251 patients, the incidence of pneumonia within 30 days of ischemic stroke was 7.1% and was associated with lower life expectancy, longer hospitalization and a greater degree of disability upon discharge from the hospital[132].

Chumbler et al.[153]attempted to create and validate a simple scoring system in order to predict pneumonia after stroke based on a logistic regression model. Of the total of 1,363 patients with acute stroke, 10.5% showed new pneumonia. Researchers showed that an age over 70 years, the presence of dysphagia, the lack of consciousness related to the onset of stroke, the higher baseline score on NIHSS scale and the positive history of pneumonia are predictors of an impending pneumonia after a stroke. Similar results were reported in the study by Sellars and colleagues from the UK who used a sample of 412 patients with stroke. These researchers added dysarthria or anarthria due to global aphasia as well as a score of ≥4 in the Modified Rankin Scale in the list of high-risk factors for developing pneumonia[154]. These findings seem to be consistent with previous studies which demonstrate a closer relationship between dysarthria of speech and post stroke pneumonia[154, 155].

Although most of the aforementioned factors are not modifiable, swallowing disorders can be treated. This underlines the need for performing some form of swallowing evaluation in acute stroke population and the need to implement preventive measures for pneumonia in those with swallowing pathophysiology. The use of the NIHSS scale as a means to measure the severity of an ischemic stroke has exponentially increased in recent years. It has also been proposed as a clinical prediction tool of developing dysphagia and pneumonia risk[120]. More specifically, Okubo and his research team used the NIHSS scale for assessing patients with ischemic strokes in order to develop an algorithm for determining a patient's safer method for oral feeding until a more specialized speech-language pathology evaluation can be performed. These researchers highlighted the score of 12 in NIHSS scale as an important benchmark with an 88% sensitivity and an 85% specificity for suggesting oral feeds.

# CHAPTER 3: CLINICAL PREDICTORS OF ASPIRATION

## 3.1 Laying the Research Basis

Traditionally several studies have defined success in bedside swallowing screening as the ability of a patient to perform uninterrupted drinking without coughing or choking during or directly after swallowing[131, 147, 156] (DePippo et al, 1994, Gordon et al, 1987, Hinds & Wiles, 1998). Hinds & Wiles further highlighted the importance of recording the total time required to complete the water swallow challenge. Other studies added the element of a 'wet vocal quality' immediately after water consumption to the list of 'negative' clinical observation signs during bedside po trials, thus, placing the patient at increased risk for aspiration[157, 158]. In his review, Ramsey et al[159] found that the clinical signs with the highest predictive value for aspiration risk included a wet, gurgly voice, a weak voluntary cough, a weak reflex (involuntary) cough after swallowing, prolonged ingestion or some combination of these. The researchers pointed out that although earlier studies have linked an abnormal gag reflex and a reduced laryngopharyngeal sensation to dysphagia[158, 160, 161], these two clinical markers do not independently prognosticate aspiration in the acute stroke population[159].

These findings seem to be consistent with the results from the Terre and Mearin study, which also did not reveal any significant correlation between gag reflex and the safety of swallow[162]. In fact, it has been proven that 37% of healthy older people do not show a gag reflex on one or both sides of the mouth, yet, still retain an intact pharyngeal sensation[163]. The authors suggested that the muscles that control the gag reflex remain independent of those that control normal swallowing. Since this reflex is commonly not found in healthy people, its predictive value in determining dysphagia and/or aspiration risk is severely limited. On the other hand, pharyngeal sensation was rarely absent in the study participants which led the researchers to the hypothesis that pharyngeal sensation can serve as a better prognostic indicator of persistent swallowing difficulties.

## 3.2 Clinical markers of aspiration at pre-screening stage

In order for patients to receive oral feeds, they are required to be conscious and have some degree of intrinsic or extrinsic postural stability (trunk and head control). These pre-screening clinical determinants are identified below.

### 3.2.1 Alertness level

Common to most studies is the recognition that decreased levels of alertness may prevent sufficient oral intake and may precipitate aspiration[118, 131, 158, 164-169], as food and fluids may remain in the mouth and not move further, or they may move through the pharynx, larynx and trachea without a swallow response. This is the reason why most studies exclude unconscious patients from further analysis by explicitly stating 'lack of alertness' in their exclusion criteria at a pre-screening section of the screening tool. The Massey Bedside Swallow Screen[167] reports 100% inter-rater reliability between nurses and speech language therapists who conducted the screen where decreased alertness is essentially the only clinical indicator for the screen to be terminated if not passed. However, criteria

for inclusion in the study required all patients to be aware and able to respond to verbal and non-verbal cues. This may reflect considerable bias in the high estimation of inter-rater reliability as the researchers exclude a group of severely affected stroke patients, who may have moment by moment variations in their ability to swallow due to fluctuating levels of alertness. Hence, a bedside swallow screen should consider a continuum of alertness so that patients who are easily arousable or intermittently alert enough to be screened, are not prevented from having oral intake if their immediate condition suggests it to be appropriate.

### 3.2.2 Postural stability

Postural stability is comprised of trunk and head control and a patient has internal stability when witnessed to have voluntary control of their posture[170]. In cases where a screen needs to be conducted, this may be achievable using external supports such as pillows. The following studies suggest that postural stability is integral to oral stability, bolus manipulation and chewing, oral bolus transit and oral intake.

Studies either explicitly state postural stability as prerequisite to the swallow screen[147, 164, 165, 169, 171], or it is implied through the test requirements as a patient may be required to participate in an instrumental study such as VFFS or FEES[158, 167-169, 172]. One study requires patients to sit in a bed in, at least, a 60-degree upright posture[169]. It has been determined that patients who have no trunk or head stability are unsuitable candidates for screening for oral intake with possible effects on the safe delivery of the bolus to the mouth and oral bolus control, chewing and transit. As such, if patients with postural instability are offered oral trials as part of a swallow screening tool, they are automatically placed at a higher risk of aspirating. Any bedside swallow screening tool should therefore consider a continuum of severity that enables patients who are able to be supported in an upright posture on their own or via use of external support to undertake a swallow screen.

### 3.2.3 Wet voice quality

A wet voice quality prior to any oral intake may indicate that the patient is either unaware of or unable to initiate a swallow to remove oral secretions that have penetrated the laryngeal vestibule and are resting on the vocal cords. Therefore, administering volumes of fluids for the purpose of swallowing screening may serve to deliver more material into the larynx to be aspirated. Logemann et al[164] identified wet voice quality as a clinical marker of pharyngeal delay rather than aspiration. Other studies have associated wet voice quality with aspiration during oral trials rather than a pre-screening indicator for assessing candidacy for oral intake[118, 165-168, 173-175].

Clinically experienced swallowing specialists typically encourage patients to cough voluntarily or perform a dry swallow prior to the presentation of oral trials however, this may require more time to prepare the patient for undertaking a screen. As such, it may be beneficial for other medical practitioners to terminate the screen and refer the patient with a wet voice quality for a more detailed swallowing assessment.

### 3.2.4 Poor or absent voluntary cough

In order to assess the cough at the bedside, patients are instructed to perform a voluntary cough which relates physiologically to the voluntary adduction of the ventricular folds[118, 158, 165-167, 176]. Failure to perform a cough by closing the false vocal cords suggests that the second mechanism of airway defense during the pharyngeal stage of the swallow is compromised[177].

Coughing is usually a reflex activity associated with irritation of the vocal cords by entry of the bolus in the larynx. Sandhu[178] reports that the cough reflex is initiated by the stimulation of sensory receptors in the larynx and lower respiratory tract which subsequently send signals to the brainstem. Absence of a voluntary cough may not necessarily be indicative of inadequate closure of the vocal cords in the presence of aspiration. Patients who are unable to follow verbal instructions due to receptive aphasia or who are unable to attend to demonstration of coughing due to post stroke cognitive difficulties may be inappropriately eliminated from swallowing trials. On the other hand, the ability to perform a voluntary cough prior to any oral trials may also suggest some ability of that patient clearing any potential bolus penetrating the larynx during the actual swallow trial. As such voluntary cough documentation may be viewed as a pre-screening added precaution measure.

### 3.2.5 History of recurrent pneumonia

Pneumonia may result from infection, reflux or aspirated material. Many patients are predisposed to recurrent episodes of pneumonia due to respiratory disease, however, episodes of recurrent pneumonia may also be indicative of chronic aspiration[179]. Recurrent pneumonia is defined as two or (usually) more episodes of lower respiratory tract infection within a six-month period that are generally accompanied by fever, leucocytosis, and purulent sputum production. These episodes are separated by an asymptomatic interval of at least 1 month or clearing of the chest visible by radiograph[180]. Recurrent pneumonia is a typical clinical corollary in stroke patients who aspirate a percentage of each bolus onto the lungs[14].

Logemann [164]found that patient history of recurrent pneumonia was associated with aspiration on VFSS in combination with one of two other clinical markers: coughing and throat clearing on oral trials and/or decreased laryngeal elevation during swallowing. When two of the three variables were present, there was a reported 69% sensitivity and 73% specificity when compared to the results from the VFSS. This suggests that if patients present with a history of recurrent pneumonia, it may be advantageous to refer for an instrumental assessment of their swallow function, but it does not prevent them from participating in a screening procedure for estimating gross aspiration risk.

## 3.3 Clinical Indicators Suggestive of Physiological Dysfunction Rather Than Aspiration

The following clinical markers are not used as part of a pre-screening evaluation stage although some authors justify their use as being indicative of physiological dysfunction rather than aspiration.

### 3.3.1 Dysphonia

Dysphonia generally encompasses the auditory-perceptual symptoms of voice disorders and is characterized by altered vocal quality, pitch, loudness and vocal effort[181]. A plethora of studies identify a hoarse vocal quality as a clinical determinant of aspiration[118, 157, 158, 166, 168, 174-176, 182, 183]. Dysphonia is indicative of incomplete glottal closure, which constitutes an important mechanism of airway protection in the larynx during swallowing physiology[184]. However, there are other recognized etiologies attributed to dysphonia such as infection, vocal cord paresis or paralysis and psychiatric disorders. Therefore, although dysphonia is a clinical marker of a compromised protective mechanism of the airway, it may not be added to the list of clinical determinants of aspiration.

### 3.3.2 Abnormal gag reflex

Many studies have employed an absent gag reflex as a clinical indicator of aspiration[118, 121, 157, 158, 160, 161, 167, 176, 183]. However, it has been demonstrated that many healthy individuals actually lack a gag reflex despite uvular movement[185]. Similarly, Davies had shown that 37% of 140 individuals without swallowing difficulties did not demonstrate a gag reflex whereas pharyngeal sensation was absent in only one subject[163].

The primary role of a gag reflex involves retching which may lead to vomiting. Its presence indicates an intact sensory component of the glossopharyngeal nerve. However, awareness of the presence of penetration or aspiration of the bolus into the airway is dependent on the integrity of the superior laryngeal branch of the vagus nerve. Furthermore, although the occurrence of a gag reflex has received an 81% specificity rating, its sensitivity was only 33% when compared to VFSS[164]. The presence of a gag reflex has therefore proven to have a low predictive value in the assessment of aspiration in acute stroke.

### 3.3.3 Other

Some studies[160, 164]utilize patients' reports of swallowing difficulties as part of a dysphagia screening tool. However, such approaches may lead to unnecessary restrictions for candidacy for oral feeds since several patients present with severe language or cognitive difficulties to be alert, cognizant and capable to express themselves accurately.

## 3.4 Clinical markers of aspiration in the oral stage

### 3.4.1 Dysarthria

Dysarthria has been defined as a motor speech disorder resulting from disturbances in muscular control affecting areas of respiration, articulation, phonation, resonance and prosody[186, 187]. Physiologic characteristics include abnormal or disturbed strength, speed, range, steadiness, tone, and accuracy of muscle movements. However, because all movements of affected muscles are disturbed, disorders of feeding and swallowing often co-occur with the speech disorder.

Muscle weakness in the oral cavity may have an impact on swallow ability. Gonzalez-Fernandez and Daniels[188] note that paralysis, weakness or incoordination in the muscles of the mouth, face, and tongue associated with dysarthria, may also indicate disturbances in the oral preparatory and oral stages of swallowing. Orofacial muscle weakness can cause poor lip seal, which may cause loss of the bolus from the lips or drooling. Facial paresis after stroke due to facial nerve damage (cranial nerve VII) can cause loss of taste and sensation in the tongue and cheeks which may result in residue in the alveolar sulci, teeth and palate. Poor lingual tone in combination with a decrease in cheek tone due to facial paresis after stroke may precipitate insufficient negative pressure for bolus transit to the base of the tongue in order to trigger the swallow[189].

In patients with dysarthria, concomitant weakness in the intrinsic muscles of the tongue due to hypoglossal nerve dysfunction (cranial nerve XII) may also indicate poor formation of a cohesive bolus, delays in bolus propulsion, and premature leakage of the bolus in the pharynx[188, 190], the latter associated with increased risk of aspiration prior to the swallow[155]. In conclusion, orofacial muscle weakness has proven to adversely affect the nutritional status of a patient[118, 158, 164, 167, 174-176] as it is mainly associated with oral stage problems. However, recent data also highlights dysarthria as a predictive factor of pharyngeal stage problems associated with bolus stasis in the valleculae and aspiration risk[175, 191, 192].

## 3.5 Clinical Markers of Aspiration in the Pharyngeal Stage

In the pharyngeal stage of the swallow, bilateral or unilateral damage or incoordination of soft palate movement will allow regurgitation of bolus into the nasal cavity. The damage may further impact the pharyngeal negative pressure necessary to push the bolus through the pharynx. Logemann had earlier defined the onset of pharyngeal transit time relative to the mandible as the time when the bolus crosses the lower or anterior rim of the mandibular ramus and had reported that normal hyoid and laryngeal elevation transit time is 0.75 seconds although this can be significantly delayed post-stroke[138]. Jeri Logemann also revealed the etiopathogenesis of residue commonly observed in one or both pharyngeal spaces, namely the valleculae and the pyriform sinuses. She reported that residue in the valleculae alone may be caused by inadequate epiglottic inversion, while residue in both pharyngeal spaces may be a consequence of reduced muscle contraction in the tongue base and pharyngeal constrictor muscles, delayed or absent forward and upward hyoid movement and incoordination of the cricopharyngeal opening, increasing the risk of aspiration[138]. The quantitative analysis later reported by Hiieman and Palmer (1999) revealed that normally cricopharyngeal sphincter opens within 0.10 seconds of airway closure in response to sensory information received from the oral cavity[193]. However, cricopharyngeal relaxation and opening may be delayed, incomplete, or not well coordinated following a stroke, which may precipitate pharyngeal residue and increased risk of aspiration of material into the trachea[194]. The risk of aspiration may further increase in stroke patients due to abnormal inspiration patterns noted in this population post-swallow[195, 196].

### 3.5.1 Coughing during swallow

Cough is one of the defensive reflex systems of the respiratory tract in response to irritation. Several methodologies are reported for inducing the cough reflex at bedside. The most commonly used tussive agents include citric acid, capsaicin and ultrasonically nebulized distilled water (fog) [197]. It has been proven that women and children have a lower threshold for coughing[198, 199], while patients with chronic obstructive pulmonary disease show greater sensitivity to coughing compared to smokers who present with lower sensitivity. Evidence shows that the cough challenge cannot stand alone as a sole predictor for aspiration but when used in combination with other variables, the specificity for aspiration risk increases[200]. Miles et al found significant associations between cough reflex threshold test results and subsequent cough response to aspiration on instrumental assessment[201]. These authors stated that an inhaled concentration of 0.8 mol/L has the highest odds ration (OR) for detecting silent aspiration events.

### 3.5.2 Wet voice quality

Wet voice quality following a swallow is indicative of liquids entering the laryngeal vestibule and resting on the surface of the vocal cords. Several studies[158, 164, 169] have seen merit in including post-swallow wet voice quality in their bedside screening procedure as a clinical marker of aspiration. It is believed that a wet or gurgly voice is easily acoustically discernible at the bedside even by the non-specialized in dysphagia medical personnel.

### 3.5.3 Wet breath sounds

Similarly, acute stroke patients commonly present with wet breath which may be indicative of disordered swallowing and difficulties in secretion management. It is suggested that a clinical observation of wet breath sounds post-swallow may indicate that either saliva secretions and/or bolus intake, has entered the laryngeal vestibule and is resting on the vocal folds. However, further clinical significance cannot be attained solely based on a bedside screening procedure.

### 3.5.4 Laryngeal excursion

Forward and upward movement of the larynx facilitates more horizontal positioning of the epiglottis over the airway and relaxation of the pharyngoesophageal segment (PES) allows the bolus to move into the esophagus. Failure of the larynx to move superiorly may be evidence of incomplete closure of the laryngeal vestibule, leading to increased risk of laryngeal penetration or overt aspiration into the lungs once the airway opens for respiration to resume[158, 174, 202]. In a respective manner, failure of the larynx to move anteriorly on swallowing may suggest that the cricopharyngeal sphincter has not relaxed and opened, and that the bolus has remained in the pyriform sinuses in the pharynx[202].

### 3.5.5 Multiple swallows per bolus

Swallows are stimulated in response to presence of the bolus in the pharynx, in individuals where sensory awareness is preserved[164]. Therefore, the presence of repeated swallows may suggest that

residue has remained in the pharyngeal spaces post-swallow which may further increase the opportunity for aspiration when the laryngeal vestibule is not closed[95].

# CHAPTER 4: ASSESSMENT OF DYSPHAGIA AND ASPIRATION RISK

## 4.1 Non-Instrumental Methods for Evaluation of Dysphagia and Aspiration Risk

Worldwide clinical guidelines mandate systematic bedside swallowing screening within the first few hours of admission to the hospital after emergency management has been given and prior to offering any oral food, fluid, or medication[203-205]. Ideally, screening should take place as soon as the stroke survivor is awake and alert. Patients who pass the screening are unlikely to have significant swallowing difficulties associated with lower risk of dysphagia-related complications. On the other hand, stroke victims who fail the screening procedure are typically maintained on a 'nil per os' status until further dysphagia evaluation can be undertaken that can describe the severity of the problem and its associated parameters in detail.

## 4.2 Screening Basics

### 4.2.1 Definition and Purpose

The American Speech Language and Hearing Association (ASHA) defines a swallowing screening procedure as a "pass or fail procedure to identify individuals who require a comprehensive assessment of swallowing function or a referral for other professional and/or medical services" (ASHA). In their studies, Streiner and Eddy[206, 207] contributed to the thought process of a clinician who wishes to detect a symptom, indicating that the main objective of a screening tool is to identify early as many clinical cases as possible before the development of clinical signs that require additional management. According to Logemann, a swallowing screening protocol should be able to confirm the presence of the absence of a symptom like "aspiration risk" and the term "dysphagia assessment" should be reserved for instrumental procedures that can define the abnormal anatomy and physiology causing the swallowing problem and can record the specific characteristics of bolus transit[164, 208]. The gold standard objective methods of evaluating deglutition disorders include the fiberoptic endoscopic evaluation of swallowing (FEES) and the videofluoroscopic swallowing study (VFSS). It therefore becomes clear that the main purpose of a bedside screen is to identify as early as possible the high-risk patients so as to direct the patient more rapidly to the most appropriate care pathway.

### 4.2.2 How do we measure accuracy of screening tests?

In its simplest form, the screening test has only two outcomes: a) positive suggesting that the subject has the disease or condition or b) negative suggesting that the subject does not have the disease or condition[209]. An ideal screening test would have a positive result if and only if the subject actually has the disease and a negative result if and only if the subject did not have the disease. However, actual screening tests typically fall short of this ideal and present with inherent shortcomings. Instead, most screening tests reveal false positives and false negatives to varying degrees.

In order to determine accuracy of a screening tool, a 2 × 2 contingency table is typically used as shown below in Table 4.1. This may allow for results of the screening tool to be compared to those

of a “gold standard” or a reference test[210]. In principle, the gold standard test should reveal 100% sensitivity and 100% specificity and never make a classification error[211]. In practice however, this test may be invasive, unpleasant, too expensive or otherwise impractical to be used widely as a routine standard test and thus is more regarded as “under reasonable conditions”.

**Table 4.1 A typical 2 x 2 contingency table**

| | | Reference Standard (VFSS or FEES) | |
|---|---|---|---|
| | | Positive | Negative |
| **Screening** | Positive | a or True Positives (TP) | b or False Positives (FP) |
| | Negative | c or False Negatives (FP) | d or True Negatives (TN) |

Sensitivity = a/(a + c); Specificity = d/(b + d); PPV = a/(a + b); NPV = d/(c + d) Positive Likelihood Ratio = Sensitivity/(1 − Specificity); Negative Likelihood Ratio = (1 − Sensitivity)/Specificity

Table 4.1 shows a typical 2 × 2 table which includes:

1. The number of true positives or those individuals that have the disease and test positive.
2. The number of false positives or those individuals that do not have the disease and test positive.
3. The number of false negatives or those individuals that do have the disease and test negative.
4. The number of true negatives or those individuals that do not have the disease and test negative.

Both true and false positives and negatives are used to calculate the statistical measures of sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) which all provide information about the validity of a test. The accuracy of a screening test is generally expressed in terms of its sensitivity and specificity, but it can also be reported in terms of predictive values and false positive and negative rates as described below.

### 4.2.3 Sensitivity and Specificity Measures

Sensitivity measures a test’s ability to identify an individual with the disease as positive. Tests that are found to be highly sensitive indicate that there are few false negative results and fewer cases of disease are missed[212]. Respectively, specificity measures a test’s ability to identify an individual without the disease as negative. Tests that are found to be highly specific indicate fewer false positive results and help rule in the target condition.

### 4.2.4 Positive and Negative Predictive Values

A positive predictive value is the probability that an individual with a positive (abnormal) test finding truly has the disease of interest and represents the proportion of individuals who fail the screen and

are identified as having the target condition. On the other hand, a negative predictive value is the probability that a person with a negative (normal) test finding actually does not have the disease of interest and represents the proportion of individuals who pass the screen and are identified as not having the target condition tested.

### 4.2.5 False Positive and False Negative Rates

A false positive result suggests that a subject without the condition is misclassified as having the condition on the basis of a screening test and is calculated as: FP/(TP + FP). The ideal value for a false positive rate is 0. However, it is nearly impossible to achieve this and constitutes an inherent acceptable disadvantage when conducting a screening test in a large population. A false negative result suggests that an individual who truly has the target condition is misclassified as not having it based on the screening test. False negative rates are calculated as: FN/(TN = FN).

### 4.2.6 Likelihood Ratios

Other terms for characterizing screening tests are likelihood ratios which are defined as the probability that a given test result would be expected in a subject with a condition compared to the probability that the same result would be expected in a subject without the target condition. A positive likelihood ratio indicates how the probability of a condition shifts when the finding is present whereas a negative likelihood ratio reflects the number of times it is more likely that a negative test comes from a subject with the disease rather than a subject without the disease.

### 4.2.7 Receiver Operating Characteristic (ROC) Analysis

The diagnostic performance of a test in discriminating pathological/diseased cases from normal cases is evaluated using Receiver Operating Characteristic (ROC) curve analysis (Zweig & Campbell, 1993). In a ROC curve, the true positive rate (Sensitivity) is plotted in function of the false positive rate (100-Specificity) for different cut-off points as shown below. Each point on the ROC curve represents a sensitivity/specificity pair corresponding to a particular decision threshold (MedCalc webpage 2019). Thus a “perfect” test, a test with ideal discrimination, has a ROC curve that passes through the upper left corner (100% sensitivity, 100% specificity). Therefore, the closer the ROC curve is to the upper left corner, the higher the overall accuracy of the test. The area under the ROC curve (AUC) is a measure of how well a parameter can distinguish between two diagnostic groups (diseased/normal).

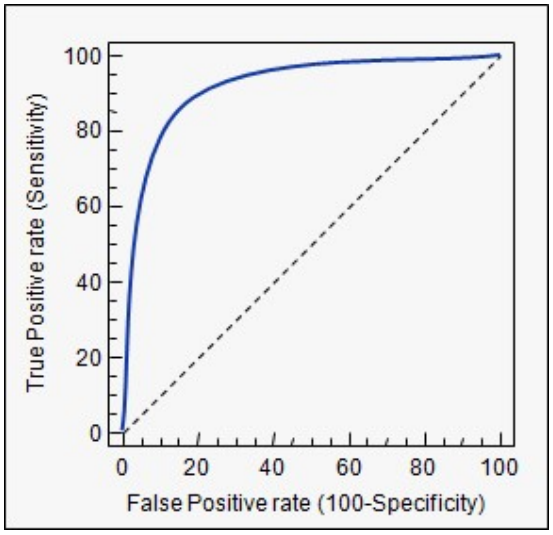



***Figure 4.1*** ROC curve illustration. Retrieved from *https://www.medcalc.org/manual/roc-curves.php*

## 4.3 Bedside Swallowing Methodologies

Stroke guidelines are stressing early dysphagia detection using standardized screening and assessment tools because of associated health benefits. Therefore, a plethora of screening methodologies have been reported in the literature and have been used in clinical practice. The discerning clinician must make an informed decision about what screen to choose and this decision seems to be multifactorial. The clinician must first consider the quality of research, ensuring adequate statistical evidence for clinical use and methodologies that are well substantiated by evidence from the literature. Other factors may include ease and time of administration and access to necessary equipment to conduct the screen.

To date, there is no formal consensus on swallowing screening. Some of the detection protocols have been accused of using small sample of subjects. Others have been criticized for their lengthy prerequisite training[213, 214]. The *3-ounce water swallow challenge* protocol[172] seems to constitute the protocol with the highest sensitivities reported, validated against the standards of FEES and VFSS, and administered to a large and heterogeneous population sample (n=3,000) compared to other screening tests.

### 4.3.1 The Water Swallow Test

As already mentioned, the water-swallowing test (WST) is frequently used in clinical practice as a functional assessment to detect aspiration and prevent pneumonia in the stroke patient population. It is a standardized test used worldwide, but the amount of water to be ingested, varies depending on the examiner. DePippo and colleagues[172] were the first to report data on the 3-ounce water swallow test via which the patient was required to demonstrate uninterrupted drinking of 90 mL of water without overt signs of aspiration. The study investigated 44 stroke rehabilitation patients and used videofluoroscopy as the objective measure for validation of its findings. Failure criteria were inability to drink continuously, cough during or up to 1 minute after completion of the screen or a wet/hoarse voice quality after the swallow. While the statistical analysis of this study was interesting,

the conclusions about aspiration of a specific percentage (greater than 10% of bolus) and thickened liquids were questioned. However, the one clinically relevant information was that with a sensitivity of 76%, the 3-ounce water challenge will potentially miss 24% of patients who aspirate.

Lim and colleagues concluded that an oxygen desaturation test combined with a 50 mL water swallow test is suitable for identifying all acute stroke patients at risk of aspiration for further evaluation and management[215]. It was emphasized again that a water swallow screen should be indicated only after careful consideration of oral motor exam findings as well as other behavioral, communicational and cognitive parameters, thus, a prudent clinician should consider the candidacy of a patient before implementing such a protocol. Similar recommendations of not overestimating the findings of a bedside water test were made by Maeshima et al[216] who reported that in cases of stroke survivors who present with both severe disability and cognitive disorders, they observed a high percentage of patients with 'silent' aspiration in subsequent videofluoroscopy.

Interesting results also came from Wu et al[217] who suggested that the speed of consumption serves as a sensitive index (85.5%) in early identification of high risk patients for the development of dysphagia, using however, a modified, larger volume of water in the test (total of 100mL). Similar findings were published in a study conducted by Osawa and colleagues[218], who evaluated 111 patients with suspected dysphagia during the second week after a stroke of diverse etiology. After administering varied volumes of water to patients who were observed both at bedside and with videofluoroscopy, these researchers concluded that the use of higher amounts of water for sequential drinking detects patients with aspiration with a higher sensitivity.

Suiter and Leder added that the performance of a patient on water swallow challenge is a predictor of a patient's ability to safely manage fluids after a stroke[168]. However, failure in the sequential water test does not indicate complete inability to manage fluids or complete inability to swallow and has often resulted in unnecessary restriction of fluids from patients' diets in approximately 50% of the cases[168, 176]. These researchers for the first time proposed the usefulness of sequential 90 mL-water swallow test in practically all medical populations regardless of age and initial admitting diagnosis.

In subsequent, more recently published studies, Leder and Suiter implemented the *Yale Swallow Protocol* in 1000 medically stable patients[219]. Failure criteria included inability to complete the task, interrupted drinking and/or coughing and choking during or immediately after the test. The researchers found that the 3-ounce water swallow challenge successfully identified all patients who could safely tolerate an oral diet 12 to 24 hours after the examination. They concluded that when the water swallow protocol is administered and interpreted by a trained clinician, it can be utilized not only as a valid method of detecting patients' aspiration risk but also as a good predictor of patients' ability to safely tolerate some form of oral diet, depending on their dentate status, without the need for further instrumental examination[220, 221].

### 4.3.2 Established Bedside Protocols With Both Non-Swallowing and Swallowing Items

In 1994, DePippo and colleagues[147] investigated again the 3-ounce water swallow test in an alternative context. Under the term, the *Burke Dysphagia Screening Test* (BDST), incorporating a screening questionnaire in conjunction with the water challenge, for years it was the instrument most commonly used to screen for dysphagia in clinical settings[222]. However, later Martino and colleagues criticized the *BDST* for lack of proper reliability measures and for its limitations to use within the rehabilitation settings and highlighted the *Toronto Bedside Swallowing Screening Tool, TOR-BSST,* as more advantageous, evaluating acute stroke population with higher sensitivity (91.3%)[214]. The measurement properties of the TOR-BSST© were established in a well-controlled study and this tool comes with a prepared education module for certification of its use.

Another popular comprehensive and non-invasive evaluation protocol was proposed by Mann and colleagues[166] who aimed to quantify the severity of neurogenic dysphagia using a simple scoring system via the *Mann Assessment of Swallowing Ability, MASA*. In 2007, Trapl and her colleagues[169] published the *Gugging Swallowing Screen-GSS* as a screening tool for evaluation of the severity of aspiration risk that could be implemented by the nursing staff and not only by trained speech language pathologists (SLPs). At that time, the GUSS was the only screening tool for dysphagia utilizing multiple consistencies for testing swallowing function and incorporated measures for patient parameters such as alertness levels and ability to manage saliva in conjunction to tolerating varied consistencies. This was considered an important factor in acute stroke-related dysphagia as patients with dysphagia are at an increased likelihood of aspirating liquids compared to semi-solids[223]. It presented with 100% sensitivity and 69% specificity for predicting aspiration risk post stroke.

In 2010, Antonios' research team[126] validated the *Modified Mann Assessment of Swallowing Ability-MMASA* as a first-time physician-administered dysphagia bedside screening tool for dysphagia following an ischemic stroke. The authors demonstrated good reliability across physician raters and ease of administration by neurologists as scoring for the MMASA could be calculated directly from findings upon initial physician examination. Therefore, it was suggested that the use of such a tool could facilitate earlier identification of post stroke dysphagia, prompting more timely and comprehensive management of a stroke patient.

In a recent systematic review of 35 bedside screening tools conducted by Schepp and colleagues[224], only four tools met the basic criteria for high sensitivity and negative predictive values when a subsequent instrumental swallow exam was performed. Two of the protocols included evaluation of mental status[126, 213], whereas the other two excluded subjects with decreased levels of consciousness[214, 225]. All protocols entailed some evaluation of oropharyngeal parameters such as dysarthria, dysphonia, and asymmetry or weakness of the face, tongue and palate. All protocols except one, included a water swallow challenge. The emergency physician screen incorporated the use of pulse oximetry in addition to clinical observations of oropharyngeal ability. None of the studies

examined measures of outcome such as pneumonia, length of hospital stay, degree of disability or death except the ICU screen which reported incidence of pneumonia at 6% in their cohort.

In conclusion, several screening tools have been proposed for examining patient's tolerance in swallowing different textures bedside. The most popular published bedside tests are compiled in Table 4.2 along with their descriptive characteristics. Each study seems to apply a different protocol of bedside trials, leading inevitably to different interpretations of clinical findings[117].Therefore, the challenge for the development of simple, high quality screening protocols that can be administered by a range of health care professionals in the acute care setting is clearly evident.

**Table 4.2 Published Bedside Tests**

| Protocol Name | Descriptive Characteristics |
|---|---|
| *Acute Stroke Dysphagia Screen*[213] | Measure of level of consciousness (Glascow Coma Scale), oral motor exam to determine the presence of dysarthria and a 3 oz water swallow test |
| *Bedside Swallow Assessment* [226] | Developed for acute stroke population. Pre-assessment questions, clinical exam, three presentations of water via teaspoon, uncontrolled volume via water in a cup. |
| *Burke Dysphagia Screening Test (BDST)* [147] | 3 oz water swallow test and clinical checklist. Developed for use within stroke rehabilitation settings. |
| *Examine Ability to Swallow (EATS)*[227, 228] | Developed for use by nurses in the acute stroke population. Uses three consistencies: semisolid, liquid and solid to assess swallowing. Contains pre-assessment criteria. |
| *Gugging Swallow Screen (GUSS)*[169] | Developed for acute stroke population. Stepwise screen that allows a graded rating with separate evaluations for non-fluid and fluid nutrition starting with non-fluid textures. |
| *Kidd Water Test*[157] | Clinical examination of tongue and facial movement, speech, sensory and perceptual function, muscle strength and pharyngeal sensation. Ability to swallow assessed by patient swallowing 50 mL of water in 5 mL allotments. |
| *Massey Bedside Swallowing Screen* [167] | Water test designed for nurse assessment in acute stroke population. Presentation of one teaspoon of water followed by cup drinking. Pre-assessment criteria. |
| *Modified Mann Assessment of Swallowing (MMASA)* [126] | Physician-administered tool for assessing dysphagia in acute stroke. Includes 12 non-swallowing items from MASA (Mann, 2002) to assess alertness, cooperation, respiration, expression, comprehension, dysarthria, saliva control, tongue movement/strength, gag, volitional cough and palate movement. |
| *Modified 30 mL Water Swallowing Test* [229] | Developed for acute stroke population. Scores six items including lip closure, tongue movement, palatal elevation, gag reflex, voice quality and motor speech function. Includes a saliva swallowing test. Swallowing is assessed |

| | |
|---|---|
| | via two presentations of water via teaspoon followed by a sequential swallowing task of 30 mL of water via cup. |
| *Standardized Swallowing Assessment (SSA)* [165] | Developed for use by nurses in the acute stroke population. Pre-swallowing check list must be passed prior to PO trials: three teaspoon sips of water followed by sequential swallowing of half cup of water. |
| *Simplified Cough Test* [200, 230] | Nurse-administered tool for identification of aspiration risk in patients with suspected stroke. |
| *Timed Water Swallowing Test* [156] | Developed for acute stroke population. Pre-assessment criteria. Initial presentation of one teaspoon of water. If isolated, patient is given 100-150 mL of water and told to drink as quickly as possible. Drinking task is timed. Residual water is measured. Number of swallows is recorded. Includes a patient questionnaire. |
| *Toronto Bedside Swallowing Screening Test (TOR-BSST©)* [214] | Developed for acute stroke population. Includes evaluation of tongue movement and ten presentations of one teaspoon of water with assessment of voice pre- and post-swallow. Requires four-hour training for certification. |
| *Rapid Aspiration Stroke Swallowing Screen (RAS)* [231] | Nurse-administered tool for identification of aspiration risk in patients with suspected stroke. Consists of nonswallowing and swallowing items. Three water swallow trials (5 mL twice, 90 mL) presented via cup or straw. |
| *Yale Swallow Protocol* [172, 208, 232] | 3 oz water presented via cup. Patient asked to drink without interruption. Inability to drink entire amount, coughing or chocking up to one min after completion or presence of post swallow wet-hoarse vocal quality indicates need for formal evaluation. Cognitive screen and oral mechanism exam. Validated with results of VFSS. |

## 4.4 Alternative diagnostic tools

### 4.4.1 Pulse Oximetry

A body of research has supported the use of pulse oximetry as a diagnostic tool to identify episodes of aspiration as they report a decline in SpO2 levels during or after the aspiration event[159, 233, 234]. Pulse oximetry involves a portable, non-invasive bedside assessment of oxygen saturation in arterial blood and thereby may serve as an indirect physiologic marker for predicting aspiration at bedside. Zaidi and colleagues found a ≈2% change in oxygen saturation levels during swallowing as indicative of aspiration if saturation is between 50% and 100%[235]. Other authors reported no association between occurrence of aspiration identified on an instrumental examination and desaturation on pulse oximetry[236-238].

### 4.4.2. Cervical Auscultation

Another method which has received a significant amount of attention for screening dysphagia is cervical auscultation. Cervical auscultation is a portable, non-invasive, well-tolerated assessment and it involves placement of a stethoscope on the lateral borders of the larynx or on the lateral borders of the tracheal wall[239]. Essentially, it provides information regarding breath sounds pre- and postswallow and translates swallow sounds. However, its accuracy in detecting physiologic events and its overall diagnostic value in swallowing are still up for debate[240]. The literature on the ability of a human observer to reliably interpret the pharyngeal sounds produced during swallowing is not dense. Moreover, in a clinical setting, the speech language pathologist evaluating a patient's swallowing function at the bedside may use such a method to listen for "notable clicks and pops" while a patient swallows"[241]. Until we are able to reduce the subjectivity inherent in nearly all clinical, human interpreted techniques, we cannot safely conclude that auscultation offers added value to the bedside clinical assessment of swallowing when compared to other imaging techniques described in the next section.

## 4.5 Instrumental Assessment Methodologies

### 4.5.1 Videofluoroscopic Examination of Swallowing (VFSS)

For years, the Videofluoroscopic Swallowing Study also termed as the Modified Barium Swallow (MBS) by Jeri Logemann, has been considered as the instrument of choice by the majority of practicing swallowing clinicians because it permits the visualization of bolus flow in relation to structural movement throughout the upper aerodigestive tract in real time[242]. VFSS also permits detection of the presence and timing of aspiration, i.e., entry of ingested material below the level of the true vocal folds into the trachea and helps to identify the physiologic and often treatable cause(s) of the aspiration[243]. Clinicians are able to observe the effects of various bolus volumes, textures and examine the efficacy of compensatory swallow maneuvers in order to inform future management.

Conduction of videofluoroscopic studies requires presence and expertise from a number of clinical staff including a radiologist/radiographer in addition to proper positioning of the patient. The patient is practically required to sit or stand in a 16-inch gap in both anterior and lateral planes and swallow barium mixed with different food or fluid consistencies. Patients must be able to demonstrate not only compliance but also a stable medical state and and relatively intact cognition in order to follow instructions. Another drawback of the study includes exposure to radiation which may limit the examination time. Lastly, the VFSS essentially offers only a snapshot of swallow ability using specific postures, head control, bolus consistency and assisted feeding by the specialist multidisciplinary team.

In 2008, Dr Martin-Harris and her team of colleagues created a standardized procedure of the videofluoroscopic assessment of swallowing impairment (MBSImp)[244] with high inter- and intrarater reliability, based on scored observations of physiologic and bolus flow measures. However, in previous years, interpretation and reporting of the study was highly subjective with different clinicians

using locally generated reporting protocols[171, 245]. Protocols vary across sites with regard to the working definition of dysphagia, bolus size and consistency, number of bolus swallows, assistance offered, cueing instructions offered and the type and number of staff present at the examination[246].

### 4.5.2 Fiberoptic Endoscopic Evaluation of Swallowing (FEES)

FEES has been proposed in recent years as a useful supplementary tool for studying swallowing[247]. It involves placement of a flexible fibreoptic endoscope at the level of the nasopharynx in order to give a clear view of the anatomy of the hypopharynx and larynx. It gives information regarding pre- and post-swallow penetration of the airway or aspiration however, no information can be obtained regarding the oral stage of swallowing. Another limitation of FEES is that it investigates the pharyngeal stage with a moment of “white-out” in the swallow due to closure of the nasopharynx by the soft palate which blocks the view and thereby aspiration during the swallow cannot be observed directly. Essentially aspiration can be observed pre-swallow and inferred post-swallow by residue in the larynx or material ejected from the trachea by coughing. Videoendoscopy also involves specialized equipment and specifically trained staff. There is the added unknown effect on swallowing of the local anesthetic spray delivered to the nostrils prior to the examination[246].

Despite the reported limitations compared to videofluoroscopy, videoendoscopy is employed as a routine method of investigation as it is a portable, safe, well-tolerated and more cost-effective assessment of swallow function at the bedside[248]. Moreover, studies report that the results obtained with videofluoroscopy and videoendoscopy correlate well in the detection of pathological aspects such as aspiration of the bolus into the airways and the presence of bolus residue in the pharynx and pharyngeal-laryngeal area[249]. In conclusion, at present, no instrumental examination can be defined as ideal for the study of swallowing, but it can be seen that, with each of these procedures, the information provided is actually complementary for the management of patients with dysphagia[250].

### 4.5.3 Other

Other instrumental procedures are available in the research setting. These include a) manometry which measures pharyngeal pressure alterations during swallowing as an adjunct to dysphagia assessments, b) scintigraphy which measures the volume of radioisotope bolus residue in the pharynx by generating a two-dimensional picture and c) ultrasound using sound waves to create an image of the oropharynx. However, these techniques are not clinically viable diagnostic examinations by a dysphagia specialist as they do not identify the nature of the patient’s deficits in order to guide effective management plans.

# *PART II*

## *SECTION I*

# CHAPTER 5: CRITICAL REVIEW OF NON-INSTRUMENTAL BEDSIDE TOOLS

## 5.1 Identification of the Unresolved Problem

The literature is dense with studies associating the presence of dysphagia and aspiration in stroke patients with increased mortality and morbidity while simultaneously highlighting the links between early recognition of dysphagia and positive changes in patient outcomes. Management dysphagia guidelines emphasize that all patients with acute stroke are kept on a 'nil by mouth' (NPO) status including medications until their swallowing safety can be established. This highlights the need for identifying the swallowing ability of all stroke patients as soon as they are awake and alert. Nowadays, most acute care settings use some kind of a swallowing screening protocol especially in developed countries. However, the current lack of a universally acceptable and validated screening protocol with high predictive value for swallowing risk has meant that various bedside methodologies are applied in practice across different clinical settings. The differences in several aspects of methodology may impact the authenticity of a test despite its reported relatively high psychometric values.

## 5.2 Formulation of Initial Research Objective

Our first main objective was to review the available bedside screening tools for assessing swallowing status and aspiration risk in acute stroke by qualitatively observing and reporting reference population study design, clinical flexibility, reliability and applicability to acute-care settings.

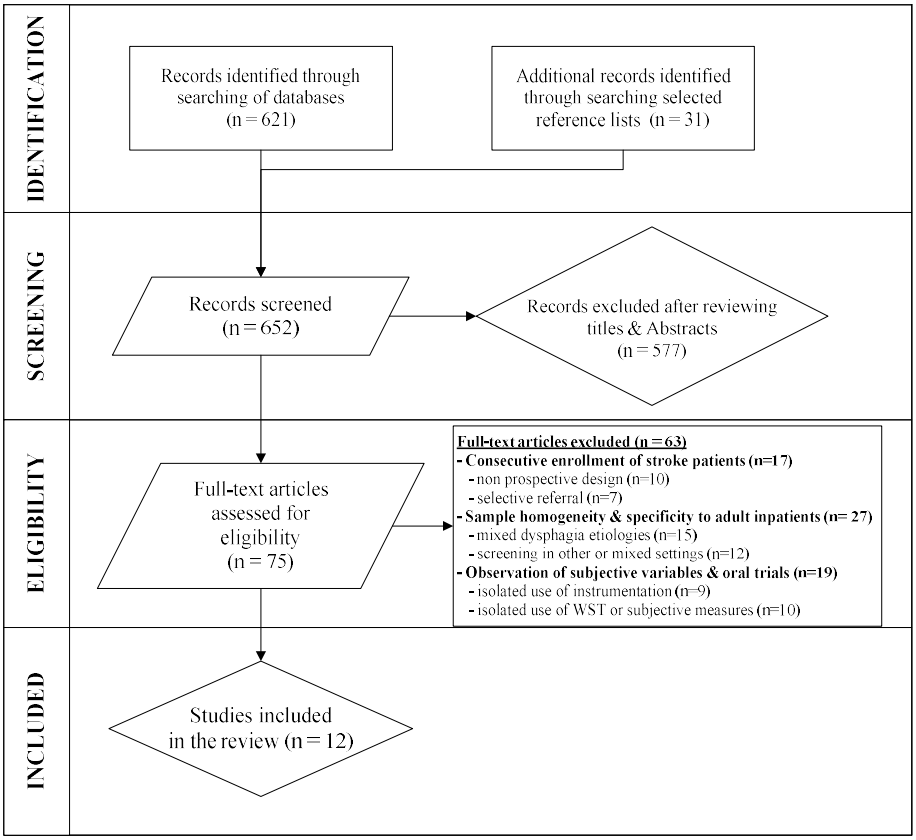
## 5.3 Methods

We performed an electronic search of the Medline (PubMed), Embase, and Cochrane databases. The search was limited to papers on humans published in English from 1991 to 2016 and for which, a full text was available. The following search terms were applied to the medical subject headings: (stroke OR cerebrovascular accident) AND (dysphagia OR swallowing OR aspiration) AND (screening OR assessment). Other potentially relevant papers were identified for full-text review from the reference lists of selected articles and from online searches of the tables of contents during the same period. Two of the authors independently completed the full article reviews to verify their inclusion, with disagreements resolved by consensus-based discussion among the review authors.

### 5.3.1 Findings of the Literature Search

Of the 652 articles retrieved, 75 original articles pertained to screening oropharyngeal swallowing impairments and aspiration risk following an acute ischemic CVA as depicted in the flowchart below (Figure 5.1). The inclusion criteria were fulfilled by only 12 articles. Information on sample size, length of training, and overall administrative burden on the clinician were noted but not required for inclusion.

***Figure 5.1 Flowchart of studies included.***




*Abbreviation:* WST, water swallowing test

## 5.3.2 Selection Criteria

Eligibility criteria included originally validated studies involving (a) the consecutive enrollment of acute-stroke patients with or without suspected dysphagia, (b) the specificity of the bedside tool for adult stroke survivors in an inpatient care unit, and (c) the combined use of subjective non-swallowing variables with subsequent food or liquid trials for estimating a patient's swallowing risk. Accordingly, papers were eliminated from full review if (a) the study did not have a prospective design, (b) the described assessment or screening protocols were administered to a heterogeneous sample of adults with confirmed or suspected dysphagia with different neurogenic etiologies, and (c) the bedside screening procedures did not entail some form of direct swallowing stimuli for determining swallowing integrity and predicting whether the patients had a high or low risk of aspiration during food and/or liquid intake.

The following exclusion criteria were applied: (a) failure to explicitly state an appraisal of swallowing status or aspiration risk after the bedside screening protocol, (b) main outcome via the bedside screening test other than the early detection of a aspiration and/or dysphagia, c) sole use of oral trials for determining aspiration risk without the use of other clinical identifiers, (d) sole use of subjective clinical indicators without subsequent bolus trials, (e) sole use of instrumental methods for detecting dysphagia and aspiration, (f) highly heterogeneous patient samples with other kinds of

neurogenic etiologies besides confirmed stroke, and (g) samples drawn exclusively from a rehabilitation setting, nursing home setting, or mixed inpatient and outpatient setting, since these were not considered patients with an acute risk of aspiration.

Articles meeting the inclusion criteria were evaluated for methodological rigor as presented in Table 5.1 1) using diagnostic study appraisal criteria from the Centre for Evidence-Based Medicine *(http://www.cebm.net/critical-appraisal/)* and the QUADAS-2 (Quality Assessment for Diagnostic Accuracy of Studies-2) tool, which are recommended by Cochrane[251]. These criteria were modified for consistency with the present study focusing on patients with stroke using clinical features associated with swallowing risk and a reference test. The criteria were rated as either 'yes' or 'no' if sufficient information was provided, or 'unclear' if the information was insufficient.

**Table 5.1: Quality Assessment Measures Utilized in Study Review**

| Study | Was selection bias minimized? | Did the study avoid inappropriate exclusions? | Was an index clinical test used? | Did all patients complete a reference standard test? | Was an appropriate protocol used? | Was there an acceptable interval between the index and reference tests? | Were the reference test results interpreted while blinded to those of the clinical test? | Were psychometric analysis data adequately reported? | Were accuracy measures adequately interpreted? | Was the description of the study protocol sufficient to allow [illegible]? | Number of criteria met |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Leigh et al. 2016[252] | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 1 | 0 | 1 | 6 |
| Edmiaston et al. 2014[253] | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 1 | 9 |
| Antonios et al. 2010[126] | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 1 | 1 | 9 |
| Turner-Lawrence et al. 2009[225] | U | 0 | 1 | 0 | U | 1 | 1 | 0 | 0 | 0 | 3 |
| Trapl et al. 2007[169] | 1 | U | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 9 |
| Ramsey et al. 2006[254] | 0 | 0 | 1 | 1 | 1 | U | 1 | 1 | 0 | 1 | 6 |
| Nishiwaki et al. 2005[229] | 0 | 1 | 1 | 1 | 1 | 1 | U | 1 | 0 | 1 | 7 |
| Leder & Espinosa 2002[176] | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 1 | 9 |
| Lim et al. 2001[215] | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 1 | 9 |
| Smith et al. 2000[255] | 1 | 0 | 1 | 1 | U | 0 | 1 | 1 | 1 | 0 | 6 |
| Daniels et al. 1998[158] | 1 | 1 | 1 | 1 | 1 | U | 1 | 1 | 0 | 1 | 8 |
| Smithard et al. 1998[256] | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 0 | 1 | 8 |

*1 indicates criteria met, 0 indicates criterion not met, U indicates unclear whether the criterion was met (assigned a final score of 0)*

## 5.4 Results

The results from the methodological appraisal of the included studies are presented in Table 5.1. None of the 12 articles were consistent with all ten quality-analysis measures. The need for informed consent and the ability to cooperate with bedside or instrumental assessment procedures resulted in a high rate of exclusion in some studies [225, 254, 255] and a bias toward patients with mild-to-moderate strokes. Five of the studies [126, 169, 176, 215, 253] conformed with nine of the methodological-rigor measures, while a study involving the hyper-acute stroke phase and emergency-room physicians [225] conformed to only three of them. The procedures in two studies[225, 255] were not described in sufficient detail or with sufficient clarity to allow their replication.

Bedside evaluation tools included different non-swallowing stimuli, such as medical history information, subjective variables [126, 176, 226, 253], oral motor measures [126, 158, 176, 226, 229, 254, 257], oxygen desaturation recordings [215, 225, 255], and scores on neurological scales such as the National Institutes of Health Stroke Severity Scale or the Glasgow Coma Scale [253]. For the purposes of this review, all of the bedside tools that were investigated in detail utilized some form of direct oral trial such as either a water swallowing test (WST) and/or a bolus swallowing test (BST) with multiple or alternative oral (per os) intake consistencies administered in varied volumes. Almost half of the included studies (42%) [126, 169, 226, 229, 252] incorporated a preliminary assessment of patient's dry (saliva) swallowing ability prior to administering swallowing trials involving boluses with other textures or specifically measured for swallowing reflex ability. One study used small aliquots of diluted radiopaque contrast agent and looked for signs of aspirated contrast in chest radiography [254].

Four of the reviewed studies involved drinking water in gradually increasing volumes ranging from 3 mL to a 90-mL sequential drinking task based on the patient's initial tolerance to smaller volumes [169, 175, 226, 252]. Each subtest was typically terminated if a patient exhibited any overt sign of swallowing difficulty, aspiration, or voice-quality compromise. Seven studies (58%) utilized a single volume of water in combination with other subjective clinical data in order to determine the swallowing integrity, aspiration, or dysphagia risk of each patient [126, 215, 225, 229, 252, 253, 255] and their eligibility to receive oral nutrition at the time of assessment. One study [176] incorporated single sips of water boluses administered via a straw, and two studies [158, 169] added swallowing of different bolus types: semisolids, solids, and liquids.

The clinical bedside screening test was completed before the diagnostic reference test in all studies. More than half of the studies (58%) used videofluoroscopy (VFSS) as the reference diagnostic test [158, 226, 229, 252-255], while 25% [169, 176, 215] used videoendoscopy (fiberoptic endoscopic evaluation of swallowing [FEES]). In the study conducted by Antonios and colleagues [126], subsequent validation was conducted by a speech language pathologist (SLP) who performed the Mann Assessment of Swallowing Ability [166] while the results from the Emergency Physician Screening tool [225] were compared with those from an unspecified standardized dysphagia assessment performed by an expert SLP. Blinding of clinical results from the health-care professional completing the diagnostic

reference test was reported for all studies except for that conducted by Leigh and colleagues [252]. The time frame between the two assessments was reported for all of the studies, but four studies used instrumental techniques with a delay of >1 day between the bedside screening and the diagnostic test [158, 229, 252, 255](ranging from a few days to several weeks post-stroke).

Across all of the included studies as listed in Table 5.2, the sensitivities of the procedures described for detecting dysphagia ranged from as low as 54.6% [252] to as high as 100% [169], while their specificities exhibited less variability, ranging from 66% [253] to 86.3% [126]. The sensitivities of the tests for identifying aspiration risk ranged from 65.2% [252] to 100% [169], and their specificities ranged from 30% [258] to 84.4% [158]. Eight studies used aspiration or the risk of aspiration as the outcome measure [158, 169, 215, 226, 229, 254, 255, 258], one study solely used dysphagia as the outcome measure [225], and the remaining studies used both aspiration and dysphagia measures [126, 252, 253].

**Table 5.2: Validity Measures of Studies Reviewed**

| Research study and protocol name (index test) | Descriptive measures and test components | Criterion standard | Main outcome | Psychometric analysis data |
|---|---|---|---|---|
| Leigh et al. 2016[252] | Check mental status and ability to open the mouth | VFSS | Dysphagia | Sensitivity = 54.6%<br>Specificity = 80.9%<br>PPV = 75.7%<br>NPV = 62.1%<br>+LR (95% CI) = 2.86% |
| *Bedside swallowing screening test* | Dry and wet swallowing tests (20-mL WST)<br><br>Descriptive three-point scale classifying the aspiration risk | | Aspiration risk | Sensitivity = 65.2%<br>Specificity = 71.4%<br>PPV = 42.9%<br>NPV = 86.2%<br>+LR (95% CI): 2.28% |
| Edmiaston et al. 2014[253] | Four screening items: mental status (Glasgow Coma Scale score <13), and presence of facial, tongue, or palatal asymmetry or weakness | VFSS | Dysphagia | Sensitivity=94%<br>Specificity=66%<br>PPV=71%<br>NPV=93% |
| *Barnes Jewish Hospital Stroke Dysphagia Screen* | Subjective signs of aspiration on 90-mL WST | | Aspiration | Sensitivity=95%<br>Specificity=50%<br>PPV=41%<br>NPV=96% |

| | | | | |
|---|---|---|---|---|
| Antonios et al. 2010[126]<br><br>*Modified MASA* | Physician-weighted screening of 12 items: alertness, cooperation, respiration, expressive dysphasia, auditory comprehension, dysarthria, saliva, tongue movement, tongue strength, gag reflex, voluntary cough, and palate movement (maximum score = 100) | Evaluation of dysphagia by SLPs using the MASA[166] | Dysphagia | Sensitivity = 92.6%<br>Specificity = 86.3%<br>PPV = 79%<br>NPV = 95% |
| | | | Aspiration | Sensitivity = 93%<br>Specificity = 53% |
| Turner-Lawrence et al. 2009[225]<br><br>*Emergency Physician Swallowing Screen* | Two-tier bedside assessment:<br>1) Voice quality, swallowing complaints, facial asymmetry and aphasia and<br>2) signs of aspiration on WST and observation of pulse oximetry desaturation (≥2%) | Formal swallowing evaluation by SLP | Dysphagia | Sensitivity=96%<br>Specificity=56%<br>+LR = 2.2 |
| Trapl et al. 2007[169]<br><br>*The Gugging Swallowing Screen* | Preliminary assessment/indirect swallowing test: vigilance, throat clearing, and SST<br><br>Subsequent direct swallowing trials with three bolus types: semisolids, liquids, and solids | FEES using Penetration-Aspiration Scale[259] | Aspiration risk (grouped according to the Penetration Aspiration Scale) | First group (n = 19)<br>Sensitivity = 100%<br>Specificity = 50%<br>PPV = 81%<br>NPV = 100%<br><br>Second group (n = 30)<br>Sensitivity = 100%<br>Specificity = 69%<br>PPV = 74%<br>NPV = 100% |
| Ramsey et al. 2006[254]<br><br>*Modified bedside swallowing test* | Oral motor function examination<br><br>Observation after three 5-mL aliquots of diluted radiopaque contrast agent<br><br>Simultaneous 10-min desaturation recordings | VFSS | Aspiration/unsafe Swallowing | Failed MBSA ± oxygen desaturation >2%<br>Sensitivity = 60%<br>Specificity = 41%<br>PPV = 28%<br>NPV = 73% |
| | | | | Failed MBSA± oxygen desaturation >5%<br>Sensitivity = 53%<br>Specificity = 67%<br>PPV = 38%<br>NPV = 79% |
| Nishiwaki et al. 2005[229] | Six oral motor items: lip closure, tongue movement, palatal elevation, | VFSS | Aspiration | Sensitivity = 72%<br>Specificity = 67%<br>(for cough/voice change in WST) |

| | | | | |
|---|---|---|---|---|
| *Modified screening tool* | gag reflex, voice quality, and motor speech function<br><br>Two swallowing screening tests: SST and 30-mL WST | | | |
| Leder & Espinosa 2002[176]<br><br>*Clinical bedside examination* | Bedside evaluation with six clinical identifiers: dysphonia, dysarthria, abnormal gag reflex, abnormal volitional cough, and voice change after swallowing<br><br>Per-os trials of single sips of water boluses via straw | FEES | Aspiration risk | Sensitivity = 86%<br>Specificity = 30%<br>PPV = 50%<br>NPV = 73% |
| Lim et al. 2001[215] | 50-mL WST (in 10-mL aliquots) and pulse oximetry recordings before and after each 10-mL WST (≥2% desaturation was clinically significant) | FEES | Aspiration | WST and oxygen desaturation test combined:<br>Sensitivity = 100%<br>Specificity = 70.8%<br>PPV = 78.8%<br>NPV = 100% |
| *Bedside aspiration test* | Monitoring for evidence of aspiration pneumonia | | Aspiration pneumonia risk | RR if evidence of aspiration on FEES = 1.24<br>(1.03 < RR < 1.49) |
| Smith et al. 2000[255]<br>*Clinical bedside examination* | Subjective evaluation of swallowing physiology at rest and on swallowing various quantities and consistencies (not clearly outlined) | VFSS | Aspiration risk | Bedside examination and oxygen desaturation≥2%<br>Sensitivity = 73%<br>Specificity = 76%<br>PPV = 55%<br>NPV = 88% |
| Daniels et al. 1998[158] | Oral motor examination<br><br>70-mL WST in small ordinal aliquots and clinical | VFSS | Aspiration risk | Stepwise logistic regression highlighted two of six predictor variables: abnormal volitional cough and cough with swallowing combined<br>Sensitivity = 69.6%<br>Specificity = 84.4% |

| | | | | |
|---|---|---|---|---|
| *Bedside swallowing examination* | swallowing trial with semisolids and solids | | | |
| Smithard et al. 1998[256] | Medical bedside assessment: consciousness level, head and trunk control, breathing pattern, lip closure, palate movement, laryngeal function, gag reflex, and voluntary cough<br><br>Signs of aspiration during WST (three 5-mL aliquots followed by60-mL challenge if passed)<br><br>Clinical judgement by an SLP | VFSS | Aspiration | Multiple logistic regression analysis revealed two independent predictors of aspiration: impairment of consciousness level and weak voluntary cough<br>Sensitivity = 75%<br>Specificity = 72%<br>PPV = 41%<br>NPV = 91% |

*Abbreviations:* **WST**, water swallowing test; **SST**, saliva swallowing test; **SLP**, speech language pathologist; **VFSS**, videofluoroscopy; **FEES**, fiberoptic endoscopic evaluation of swallowing; **MASA**, Mann Assessment of Swallowing Ability; **PPV**, positive predictive value; **NPV**, negative predictive value;**+ LR**, positive likelihood ratio; **CV**, confidence interval; **RR**, relative risk; **MBSA**, modified bedside swallowing assessment.

# *PART II*

## *SECTION II*

# CHAPTER 6: THE DEVELOPMENT OF A NOVEL SCREENING TOOL FOR POST-STROKE ASPIRATION RISK: THE FUNCTIONAL BEDSIDE ASPIRATION SCREEN (FBAS)

## 6.1 Explaining the Literature Gap

Based on the results of the critical review of non-instrumental bedside tools analyzed in Chapter 5, we concluded that there remains a strong need for an optimal clinician-friendly screening tool for the identification of aspiration risk in stroke patients. We therefore proceeded in the development of a new, clinician-friendly bedside protocol for estimating aspiration risk following stroke, intended for use by the non-specialists in dysphagia. We named this novel screening tool, the Functional Bedside Aspiration Screen (FBAS) and examined construct validity, reliability with the predictive values towards pragmatic patients' outcomes. These results are discussed in Chapter 7. The following section reveals information on the construction of the FBAS, the items included and its interpretation. As already highlighted in the previous chapter, the development of the Functional Bedside Aspiration Screen (FBAS) was based on the notion that a combination of non-swallowing and swallowing screening items would provide the highest validity for aspiration risk[260].

## 6.2 Construction of the FBAS

For the FBAS development, identification of potential screening items was performed following a group of experts' convention and discussion on recently published reviews on bedside swallow screens[224, 260-263] using content validity index methodology. Items were eliminated from the testing protocol if they were judged clinically impractical by 4 or more of the content experts in the team. The content expert team comprised of two neurologists, two internal medicine physicians, one speech-language pathologist (SLP) and one registered nurse (RN) who had >10 years of experience working full time with stroke patients in acute care settings. Our *a priori* hypothesis was that a combination of non-swallowing and swallowing screening items would provide the highest validity for aspiration risk. The final response grid and layout of the FBAS was pilot-tested with 10 newly admitted acute stroke patients. Their responses indicated high ratings for ease of administration scoring and interpretation.

In specific, we used a 4-point scale with ratings for potential items as follows: 1=not relevant/representative, 2=somewhat relevant/representative, 3=quite relevant/representative, 4=highly relevant/representative. We used the final Item-Content Validity Index (I-CVI) information to guide us in revising or deleting final items and excluded items not achieving a rating of 3 or 4 by all six content experts. For convenience, we refer to this as Scale-Content Validity Index (S-CVI/UA, universal agreement)[264].

Table 6.1 shows the relevance ratings of six experts for our 10-item scale. Five experts (all but expert 6) rated 9 out of 10 items as relevant however, the item judged not relevant differed for the five

experts. Only 5 out of 10 items received relevance ratings of 3 or 4 by all the experts (thus the S-CVI/UA=0.50).

**Table 6.1 Ratings on a 10-Item Scale by Six Experts: Items Rated 3 or 4 on a 4-Point Relevance Scale**

| | Item | Expert 1 | Expert 2 | Expert 3 | Expert 4 | Expert 5 | Expert 6 | Number in Agreement | Item-CVI† |
|---|---|---|---|---|---|---|---|---|---|
| Step 1 *Exclusionary for oral trials* | 1. alertness | x | x | x | x | x | x | 6 | 1.00 |
| | 2. positioning | x | x | x | x | x | x | 6 | 1.00 |
| | 3. saliva management | x | - | x | x | x | x | 5 | 0.83 |
| Step 2 | 4. functional language comprehension | x | x | x | x | - | x | 5 | 0.83 |
| | 5. presence of speech impairment | x | x | x | x | x | x | 6 | 1.00 |
| | 6. laryngeal response | x | x | x | x | x | x | 6 | 1.00 |
| Step 3 *Bedside oral trials* | 7. one tsp§. puree | x | x | - | x | x | x | 5 | 0.83 |
| | 8. one tb. puree | - | x | x | x | x | x | 5 | 0.83 |
| | 9. one sip of water | x | x | x | - | x | x | 5 | 0.83 |
| | 10. sequential drinking of 90cc water | x | x | x | x | x | x | 6 | 1.00 |
| | Proportion Relevant: | 0.90 | 0.90 | 0.90 | 0.90 | 0.90 | 1.00 | Mean I-CVI=0.915 S-CVI‡=0.50 Mean expert proportion =0.92 | |

†I-CVI, item-level content validity index; ‡S-CVI/UA, scale-level content validity index, universal agreement calculation method; §tsp, teaspoon; ¶ tb., tablespoon.

## 6.3 Components of the FBAS

The FBAS presented at the end of this chapter is a ten-point scale, divided into three components, which can be administered and scored in approximately 5 minutes in a step-wise fashion, as explained below. A single score of 1 denotes best performance with the maximum attainable patient score of 10.

Step 1 of the FBAS pertains to criteria for deferring protocol administration. Patients are withdrawn from direct oral trials if they show inability to maintain adequate levels of alertness, inability to sit upright in bed or chair, presence of active/congested lung sounds on auscultation and inability to initiate a saliva swallow even after dipping a sponge swab into water or using a saliva substitute to facilitate a swallowing attempt. If a patient checks “yes” for all three parameters, he/she is automatically considered eligible for continuation with oral trials (step 3 of the protocol).

Step 2 records patient-oriented clinical parameters which further serve as negative or positive predictors for aspiration risk and are scored as either severely disturbed or adequate/ ‘within normal

limits' (WNL). These include a) functional ability to comprehend language, b) presence of speech disorders known to impact oral motor diadochokinesis and c) laryngeal response as determined by the presence and the strength of a voluntary cough.

Step 3 of the FBAS consists of four sequentially performed direct swallowing subtests. Before the administration of oral trials, patients are positioned upright at 90° or as upright as possible in a bed or chair and made comfortable. The clinician initiates swallow trials with pureed consistencies in a graded volume which are followed by functional (self-) administered water swallow trials. The oral trials continue until the first subtest is failed. The total absence of the two major clinical indicators of aspiration moves the patient to the final subtest of the oral protocol, the uninterrupted drinking of 90 mL of water. This is either a self-administered 'controlled' task or the examiner provides support on the patient's cup with her hand while drinking. Failing criteria for each oral subtest include a) coughing, choking, throat clearing or b) a clear change in voice quality (wet/gurgly/hoarse voice) immediately during or after swallowing or up to one minute after the completion of the task. The examiner scores total patient performance on non-swallowing and swallowing stimuli and specific diets are prescribed based on clinical findings and patient's dentate status.

Following the initial screen for aspiration risk, all patients were monitored for possible deterioration in their functional, medical and neurological status until their hospital discharge. Performance on the FBAS leads to a binary result (aspiration risk high or low) although it is acknowledged that there may be different levels of severity of swallowing difficulty and subsequent different management needs. Patients with lower risk of aspiration (score >8) are recommended a puree diet if edentulous while dentate patients were recommended a regular diet. Patients with determined higher risk of aspiration on the FBAS maintain a nil-per-os status until further multi-disciplinary evaluation could devise appropriate management plan.

# CHAPTER 7: VALIDATION OF FBAS USE IN PATIENTS WITH ISCHEMIC STROKE

## 7.1 Materials and Methods:

### 7.1.1 Setting

All consecutive stroke patients admitted at the Departments of Internal Medicine and Neurology of a tertiary care University Hospital in North-West Greece between July 13, 2015 and January 31, 2017 (over an 18-month period) were evaluated. The study was approved by the University Hospital of Ioannina Ethics Committee and all recruited patients or their next-of-kin were informed about the study procedure and provided their written consent or ascent where appropriate. The consent form is presented in the appendices section as form A1.

### 7.1.2 Subject selection criteria

All participants aged >18 with an acute ischemic stroke confirmed by compatible computed tomography (CT) results or magnetic resonance imaging (MRI) were evaluated within 72 hours post onset. Patients with a) a tracheostomy tube in place, b) hemorrhagic stroke confirmed via neuroimaging and c) history of oropharyngeal swallowing problems preceding current symptoms were excluded. Patients with a history of multinfarction were included unless they exhibited persistent non-treated dysphagia.

### 7.1.3 Swallow Screening Measures

Aspiration risk was ascertained by the attending stroke physician using the Yale Swallow Protocol (YSP)[208] which has documented high psychometric properties in the literature[219] in addition to clinical utility indices to virtually all patients regardless of admitting diagnosis[168]. This tool, presented in the content of the appendices section as A4., has been valid for use by health-care professionals [265] of different disciplines and provides a stepwise frame for making appropriate oral diet recommendations for potential candidates without the need for further instrumental diagnostic testing. These key operating criteria made it a useful referent outcome measure tool and the authors had received prior written permission by its developers for the purposes of this research study. Within a 24-hour interval of the YSP screen, the speech-language pathologist (SLP) with >10 years of experience in working with acute brain-injured patients also implemented the novel FBAS protocol presented in chapter 6. Overall screening time for the administration of the FBAS was <10 minutes.

### 7.1.4 Other data collection: Definition of Variables

*7.1.4.1 Patient Sociodemographic and Clinical Feature Data*

Admission data for each patient were obtained through medical record review and use of a standardized questionnaire. We collected demographic data such as age, gender and patients were queried about pre-stroke use of medication and the following cardiovascular comorbidities[40, 43, 266]: arterial hypertension, diabetes mellitus, hypercholesterolemia, previous strokes/transient ischemic

attacks, coronary heart disease and atrial fibrillation. We also recorded other recognized modifiable or potential risk factors for dysphagic symptoms as emerging evidence suggests[75, 80, 82, 267] such as chronic obstructive pulmonary disease, hypothyroidism, rheumatoid arthritis, obesity, regular alcohol consumption and history of or current habitual consumption of cigarettes, pipes or cigars. The data collection form is presented at the Appendices section as form A2.

*7.1.4.2 Ischemic Stroke Classification*

All subjects included in the study underwent routine diagnostic neuroimaging procedures via CT scan or MRI for radiographic confirmation of stroke. Stroke localization was categorized as left hemispheric, right hemispheric and posterior cranial fossa infarct.

Classification of stroke etiology was completed according to the Trial of Org 10172 in Acute Stroke Treatment (TOAST)[268] as follows: 1) large-artery atherosclerosis, 2) cardioembolism, 3) small-vessel occlusion or lacunar stroke, 4) stroke of other determined etiology and 5) stroke of undetermined etiology.

Stroke specific evaluations both baseline and discharge stroke severity were quantified with the National Institute of Health Stroke Scale (NIHSS)[269].

### 7.1.5 In-Hospital and Long-Term Outcome Indicators

*7.1.5.1 Use of the National Institutes of Health Stroke Scale (NIHSS) as a measure of stroke severity and stroke outcome*

Stroke-related neurological deficits were quantified with the National Institutes of Health Stroke Scale (NIHSS) upon admission and upon discharge by the investigating lead physician or the trained SLP (proof of training is provided in the content of Supportive Documentation). The NIHSS scale has gained wide acceptance as a standard clinical assessment tool to evaluate stroke severity[270] and predict early stroke outcome[271]. Patients' performance was scored for each of the 15 items used to evaluate the effect of acute cerebral infarction on the levels of consciousness, language, neglect, visual-field loss, extraocular movement, motor strength, ataxia, dysarthria, and sensory loss. The examiner rated the patent's ability to answer questions and perform activities. Ratings for each item were scored on a 3- to 5-point scale, with 0 as normal, and there is an allowance for untestable items. Scores range from 0 to 42, with higher scores indicating greater severity. Stroke severity was stratified on the basis of NIHSS scores as follows:

0 = no impairment

1-4 = mild

5-15 = moderate

16-20 = mod-severe

21-45 = severe impairment

*7.1.5.2 Use of the Modified Ranking Scale (mRS) as a measure of functional outcome*

The modified Rankin Scale (mRS)[272, 273] was used to document patients' premorbid independence in daily activities as well as their degree of disability upon discharge from the acute care setting. According to the expanded ranking system reported by van Swieten et al (1988)[274], we thereby assigned a grade from 0-5 based on the level of independence with reference to pre-stroke activities via a guided interview process. A score of 6 denoted mortality. The ordinal outcome scale is shown below in Table 7.1 Proof of training in administration and scoring of this scale is also provided in the content of supportive documentation.

**Table 7.1 The Modified Ranking Scale**

| Ranking Grade | Description |
|---|---|
| 0 | No symptoms at all |
| 1 | 1 No significant disability despite symptoms; able to carry out all usual duties and activities |
| 2 | 2 Slight disability; unable to carry out all previous activities, but able to look after own affairs without assistance |
| 3 | Moderate disability; requiring some help, but able to walk without assistance |
| 4 | Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance |
| 5 | Severe disability; bedridden, incontinent and requiring constant nursing care and attention |
| 6 | Death* |

Ref: van Swieten et al. 1988. Table adapted from *https://www.med.unc.edu/neurology/files/2018/05/MIM-721-APRIL-03-MODIFIED-RANKIN-SCALE.pdf*

* A possible rating in the context of hospitalization and 90 days post onset denoted mortality. Patients or their family representatives were contacted via telephone 90 days post stroke onset for records of functional level using the mRS index.

*7.1.5.3 Length of Hospital Stay*

The length of stay of each stroke patient was calculated from the day of admission to the day of discharge. Specifically, hospital bed days were calculated using the admission and discharge dates by counting the days within the period of stay for each patient.

*7.1.5.4 Medical Complications*

Medical factors recognized as independent markers for poor stroke outcome as already explained in chapter 1 were recorded during hospital stay as follows: a) 'urinary tract infection', b) 'in-hospital, nosocomial respiratory infection' or c) 'other infection'[144]. Stroke-associated pneumonia was also recorded at day 90[275] and the descriptive diagnostic criteria are analyzed below.

*7.1.5.5 Pneumonia diagnostic criteria*

Pneumonia was classified by the attending physician based on chest radiographs with new focal infiltrates in addition to the presence of at least two of the following clinical features: a) symptoms of lower respiratory tract infection (body temperature>38$^{o}$ C with no other recognized cause, observations of increased respiratory secretions, new onset or worsening dyspnea or tachypnea), b) notable signs on chest auscultation (rales, inspiratory crackles or bronchial breath sounds) and c) elevated inflammatory biomarkers such as C-reactive protein (CRP) measurements. This is in close accordance with the recently proposed recommendations from the Centers for Disease Control and Prevention for the diagnosis of stroke associated pneumonia[276].

### 7.1.6 Statistical analyses

Descriptive statistics such as the median and the interquartile range (IQR) were used to summarize quantitative variables, while frequencies and proportions were used for discrete variables. The FBAS scale was expressed on both a continuous scale and a binary scale after the selection of a suitable cut-off point based on the results of the ROC curve analysis, and specifically on the score that simultaneously provided the highest sensitivity and specificity. Pearson's chi-square (Pearson $\chi^2$) tests were used to associate the FBAS outcomes with results obtained with the YSP and kappa coefficients to measure the agreement between them. Receiver operating characteristic (ROC) curves were evaluated to determine the predictive validity of the FBAS to estimate aspiration risk in ischemic stroke. Finally, in order to detect differences between groups as defined by history or other patient characteristics, Pearson $\chi^2$ tests, ANOVA or T-tests were used for independent groups depending on the type of data. In all cases the significance level was set to 0.05 and the analysis was performed with the SPSS v23.0 software.

# CHAPTER 8: RESULTS

## 8.1 The clinical characteristics of the study population upon admission and during hospitalization are summarized in Tables 8.1 and 8.2.

**Table 8.1** Patient characteristics upon admission

| Variable | Mean ± SD, N (%) |
|---|---|
| **Gender:** Male | 55 (52.9%) |
| **Marital status:** | |
| Married | 84 (80.7%) |
| Widower | 15 (14.4%) |
| **Comorbidities** | |
| Arterial hypertension | 76 (73.1%) |
| Diabetes mellitus | 29 (27,.9%) |
| Hypercholesterolemia | 38 (36.5%) |
| COPD | 8 (7.7%) |
| Atrial fibrillation | 24 (23.1%) |
| Cortical atrophy | 25 (24.0%) |
| Hypothyroidism | 8 (7.7%) |
| Rheumatoid arthritis | 4 (3.8%) |
| Hx of pacemaker insertion surgery | 6 (5.8%) |
| Extrapyramidal disorder | 3 (2.9%) |
| Obesity | 4 (3.8%) |
| History of stroke/TIA | 39 (37.5%) |
| **Smoking:** | |
| Yes | 27 (26.0%) |
| Hx of smoking | 15 (14.4%) |
| **Alcohol:** | |
| 3-5 weeks | 13 (12.5%) |
| Daily | 12 (11.5%) |
| **Medication** | |
| Antihypertensive | 68 (65.4%) |
| Hypolipidemic | 32 (30.8%) |
| Anticoagulant | 14 (13.5%) |
| Antiplatelet | 35 (33.7%) |
| Antidiabetic | 20 (19.2%) |
| Insulin | 9 (8.7%) |
| **mRS† before the index event** | |
| 0 - No symptoms | 57 (54.8%) |
| 1 - No significant disability | 33 (31.7%) |
| 2 - Slight disability | 7 (6.7%) |
| 3 - Moderate disability | 3 (2.9%) |
| 4 - Moderate severe disability | 3 (2.9%) |
| 5 - Severe disability | 1 (1.0%) |

*†mRS indicates Modified Rankin Scale*

**Table 8.2** Patient characteristics during hospitalization

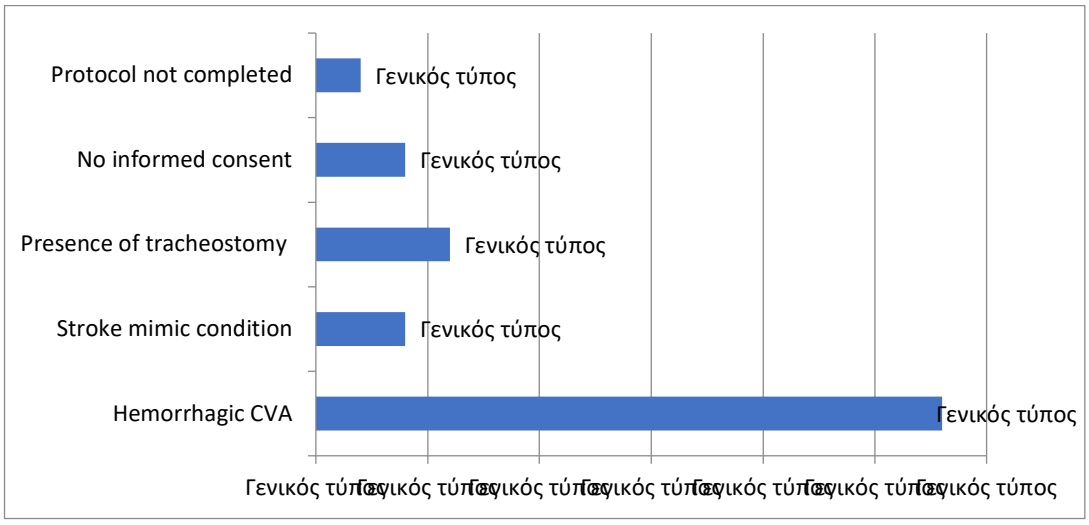
| Characteristic | Mean ± SD, N (%) |
|---|---|
| **Department** | |
| Internal medicine | 46 (44.2%) |
| Neurology | 58 (55.8%) |
| **Duration of hospitalization** | |
| <1 week | 37 (35.6%) |
| 1-2 weeks | 26 (25.0 %) |
| >2 weeks | 41 (39.4%) |
| **TOAST classification of stroke subtype** | |
| Large-artery atherosclerosis | 38 (36.5%) |
| Cardioembolism | 27(26.0%) |
| Small-vessel occlusion | 7(6.7%) |
| Stroke of other determined etiology | 3(2.9%) |
| Stroke of undetermined etiology | 29(27.9%) |
| **Lesion site** | |
| Left hemispheric | 40 (38.5%) |
| Right hemispheric | 45 (43.3%) |
| Posterior cranial fossa | 19 (18.3%) |
| **Medication** | |
| Anticoagulant | 66 (63.5%) |
| Antiplatelets | 71 (68.3%) |
| Thrombolysis | 10 (9.6%) |
| **NIHSS† entry** | |
| No impairment (0) | 3 (2.9%) |
| Mild (1-4) | 33 (31.7%) |
| Moderate (5-15) | 38 (36.5%) |
| Moderate-severe (16-20) | 10 (9.6%) |
| Severe (21-42) | 20 (19.2%) |
| **mRS‡ on discharge** | |
| 0 - No symptoms | 5 (4.8%) |
| 1- No significant disability | 20 (19.2%) |
| 2 - Slight disability | 23 (22.1%) |
| 3 - Moderate disability | 20 (19.2%) |
| 4 - Moderate severe disability | 20 (19.2%) |
| 5 - Severe disability | 13 (12.5%) |
| 6 – Death | 3 (2.9%) |
| **Complications** | |
| Urinary tract infection | 18 (17.3%) |
| Pneumonia | 26 (25.0%) |
| Other infection | 13 (12.5%) |

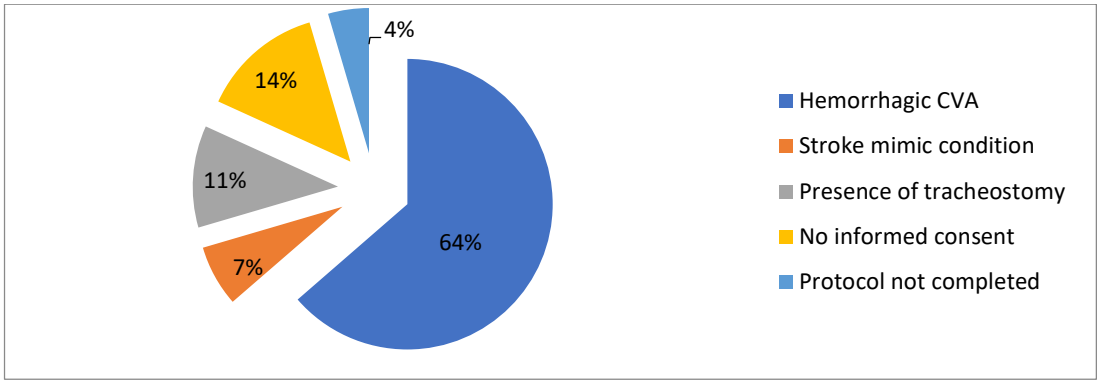
*†NIHSS, National Institutes of Health Stroke Scale*
*‡mRS, Modified Rankin Scale*

Of the total of 148 patients, 44 were excluded from our final cohort as shown in Figure 8.1. Twenty-eight patients were removed from final data analysis on the accounts of the presence of hemorrhage (subdural hematoma, intracerebral or subarachnoid hemorrhage) confirmed via neuroimaging. Similarly, 6 patients showed CT evidence of hemorrhagic transformation after cerebral infarction which manifested in a significant deterioration in clinical state and required insertion of tracheostomy tube. Another 4 patients were determined by the lead physician to have stroke mimic conditions

(typically hyponatremia or uremia) and were disqualified from participation in the study. Finally, 2 patients left before the protocol was completed and 4 patients did not provide informed consent. The reasons for removal are also graphically depicted below.

**Figure 8.1 Patients excluded**




A total of 104 acute stroke patients participated in this study (55 males and 49 females, median age 72,50, IQR 20) with confirmed brain ischemia. Of these, 40 were diagnosed with left hemisphere damage, 45 had right hemispheric damage and 19 had lesions in the posterior cranial fossa. Their most frequent comorbid conditions at initial medical work-up were arterial hypertension (73.1%), followed by hypercholesterolemia (36.5%) and previous stroke or TIA (37.5%) as presented in Table 8.2.

## 8.2 External Reliability of the FBAS with Reference to the YSP

Finally, 93 patients were administered the 90cc water challenge incorporated in both the Yale Swallow Protocol and the Functional Bedside Aspiration Screen. The remaining eleven patients met the exclusionary criteria for oral feeds. The FBAS 10-point scale was administered to all patients and

was compared with the reference standard measure YSP. The strong association found between the FBAS cut-off criterion and the YSP (Pearson $\chi^2$= 54.92, p <0.001) is shown in Table 8.3. The kappa coefficient measuring agreement between the two binary outcomes was equal to 0,76 showing "good agreement."[277].

**Table 8.3** External Reliability of the FBAS compared to the YSP

| **Count** | | | | |
|---|---|---|---|---|
| | | **FBAS** | | |
| | | Fail | Pass | Total |
| **YSP** | Fail | 40 | 8 | 48 |
| | Pass | 3 | 42 | 45 |
| **Total** | | 43 | 50 | 93 |
| Pearson $\chi^2$= 54.92, p <0.001 | | | | |

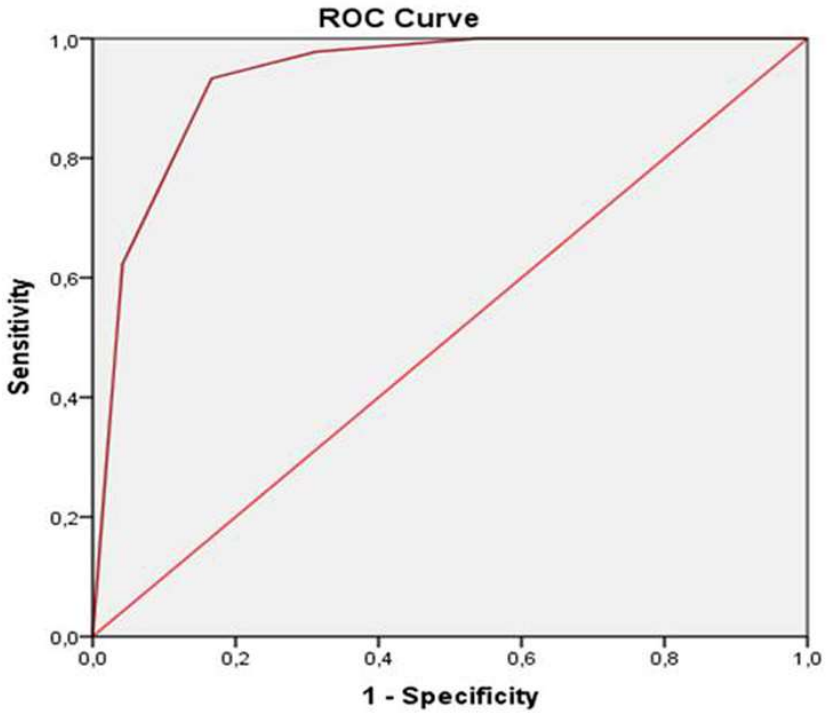
*† Functional Bedside Aspiration Screen*

*‡ Yale Swallow Protocol*

## 8.3 ROC Curve Analysis

The ROC curve depicted a discriminant ability of the FBAS test which is very close to that of the YSP (Figure 8.2). A score of ≤8 presented with 93.3% sensitivity and 83.3% specificity in deeming patient with reduced safety for oral feeds with purees and fluids while the PPV and NPV values of the test equal 84% and 93% respectively. The Area Under the Curve (AUC) equals 0,934 with a 95% CI of 0.884–0.985. A score of >8 significantly differentiated patient's tolerance for swallowing thin liquids.

**Figure 8.2** Receiver operating characteristic (ROC) curve for the ability of the Functional Bedside Aspiration Screen (FBAS) to document aspiration risk in ischemic stroke (with respect to the YSP).




**Area Under the Curve**

Test Result Variable(s): FBAS †

| Area | Std. Error[a] | Asymptotic Sig.[b] | Asymptotic 95% Confidence Interval | |
|---|---|---|---|---|
| | | | Lower Bound | Upper Bound |
| ,934 | ,026 | ,000 | ,884 | ,985 |

†FBAS, Functional Bedside Aspiration Screen

## 8.4 FBAS pass-fail outcome with respect to the lesion site

A statistically significant relationship was found also for the FBAS pass-fail outcome and the lesion site (Pearson Chi Square test = 9,762, p=0.008) as depicted in the following tables and graphs. Patients with a right lesion were more likely to fail the FBAS test (68,89%) compared to patients with a left cortical lesion (42,5%) or with a lesion in the posterior cranial fosses (31,58%). This result though is not confirmed when considering the YSP test results with similar failure percentages close to 50% (p=0,719).

**Crosstab**

| Count | | FBAS 10 | | Total |
|---|---|---|---|---|
| | | Fail | Pass | |
| **Lesion site** | Left cortical | 17 | 23 | 40 |
| | Right cortical | 31 | 14 | 45 |
| | Posterior cranial fossa | 6 | 13 | 19 |
| **Total** | | 54 | 50 | 104 |

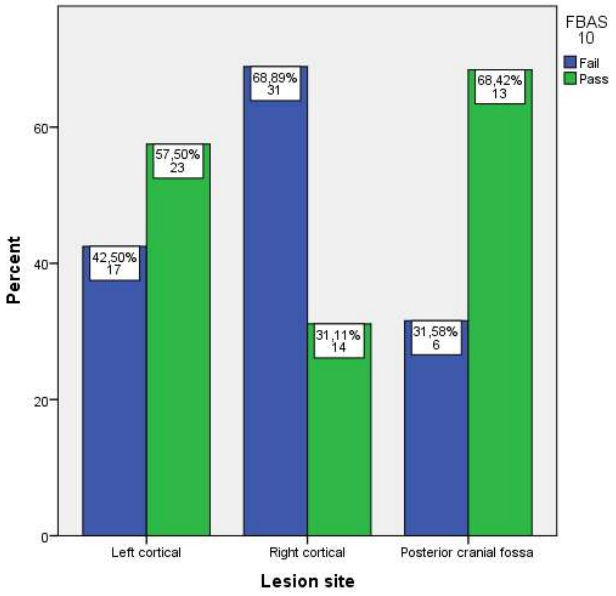



**Figure 8.3** Pass/Fail FBAS Outcome and Lesion Site

**Pass Yale * Lesion site Crosstabulation**

Count

| | | Lesion site | | | |
|---|---|---|---|---|---|
| | | Left cortical | Right cortical | Posterior cranial fossa | Total |
| Pass Yale | Όχι | 18 | 21 | 9 | 48 |
| | Ναι | 19 | 16 | 10 | 45 |
| Total | | 37 | 37 | 19 | 93 |

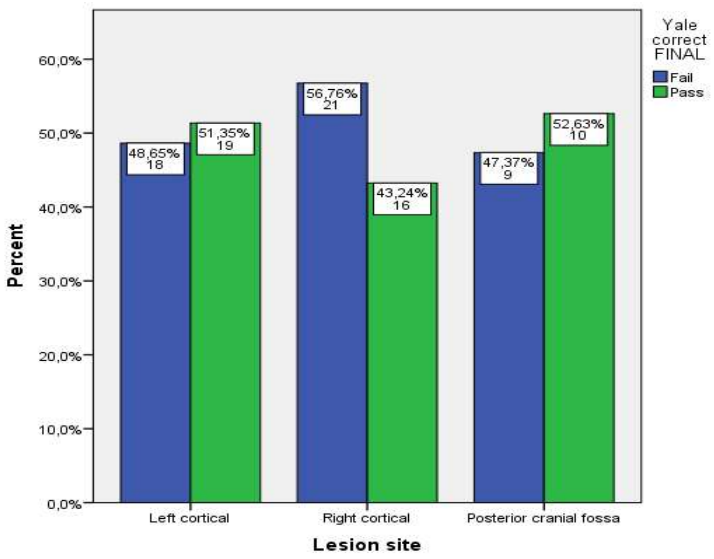



**Figure 8.4** Pass/Fail YSP Outcome and Lesion Site

## 8.5 Associations of the FBAS and YSP findings with In-Hospital and Long-Term Outcome Indicators

Apart from its "direct comparison" to the YSP test, when measured on a binary scale, the FBAS scale can provide substantial information as a scale measurement since higher scoring on this 10-point rated scale is shown to be indicative of lower risk of aspiration or generally better health outcome. Indicators of in-hospital and long-term outcome including NIHSS, mRS and duration of hospitalization are shown in Tables 8.4 and 8.5. There was an absolute accordance as far as the inference/null hypothesis is concerned in all relationships involving variables appearing on Table 4 and the two outcome variables (FBAS and YSP). Patients with better health indices were more likely to pass both the FBAS and the YSP swallowing screens. Statistically significant relationships were found in all cases except for the mRS entry measurement which reflected the patient's premorbid state. This of course could be due to the fact that the mRS entry state of the most patients (almost 85%) were characterized as non- symptomatic or with "no significant disability" rendering the statistical power of the Fisher's exact test rather inadequate to reach statistical significance.

**Table 8.4** Associations of FBAS and YSP with Outcome Indicators

| Variable | FBAS† | | YSP‡ | |
|---|---|---|---|---|
| | Fisher's Exact test | P | Fisher's Exact test | P |
| NIHSS on admission | 62.78 | <0.001 | 43.95 | <0.001 |
| NIHSS at discharge | 55.32 | <0.001 | 42.11 | <0.001 |
| mRs on admission | 7.84 | 0.111 | 2.07 | 0.836 |
| mRs at discharge | 52.16 | <0.001 | 45.71 | <0.001 |
| Nosocomial pneumonia* | 27.17 | <0.001 | 25.43 | <0.001 |
| Duration of hospitalization | 25.89 | <0.001 | 17.87 | <0.001 |
| Pneumonia within 90 days of index event | 14.39 | <0.001 | 9.02 | 0.002 |
| mRs at 90 days | 37.78 | <0.001 | 26.39 | <0.001 |

*Pearson $\chi^2$ test

† *FBAS, Functional Bedside Aspiration Screen*

‡ *YSP, Yale Swallow Protocol*

There was an inverse relationship between the performance on the FBAS and NIHSS and mRS scales as well as development of pneumonia. Lower performance on the FBAS was also significantly associated with lesion severity as reflected by higher NIHSS scores, prolonged hospital stay, greater disability or dependence after the stroke as reflected by the mRS and respiratory consequences during hospital stay and 3 months post onset (stroke-associated pneumonia within 90 days of the index event).

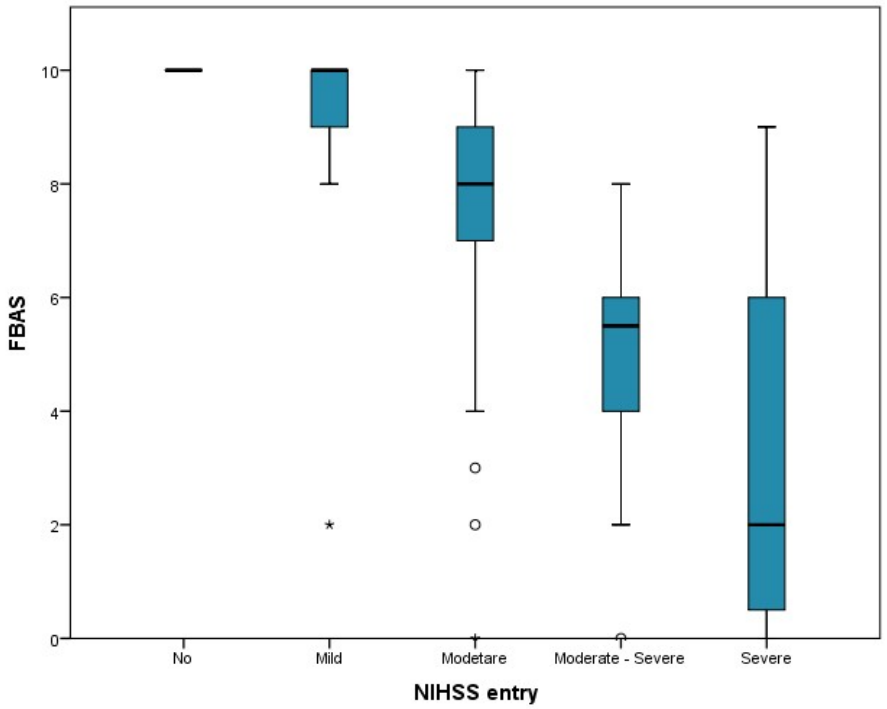



**Figure 8.5** Performance on the FBAS and initial neurological deficit

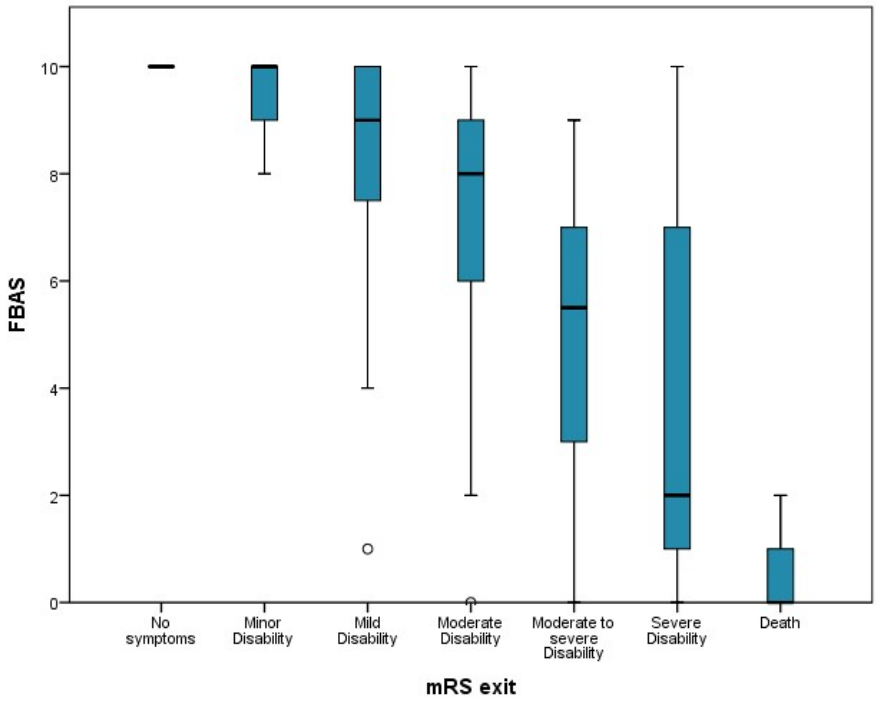



**Figure 8.6** Performance on the FBAS and dependence after stroke

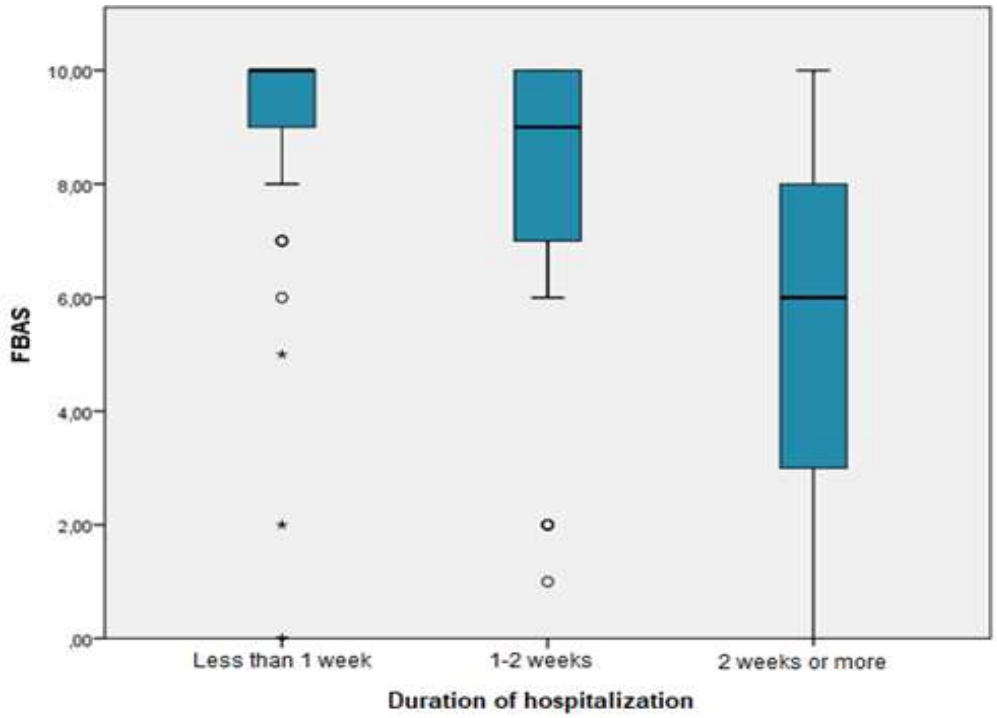



**Figure 8.7** Performance on the FBAS and length of stay

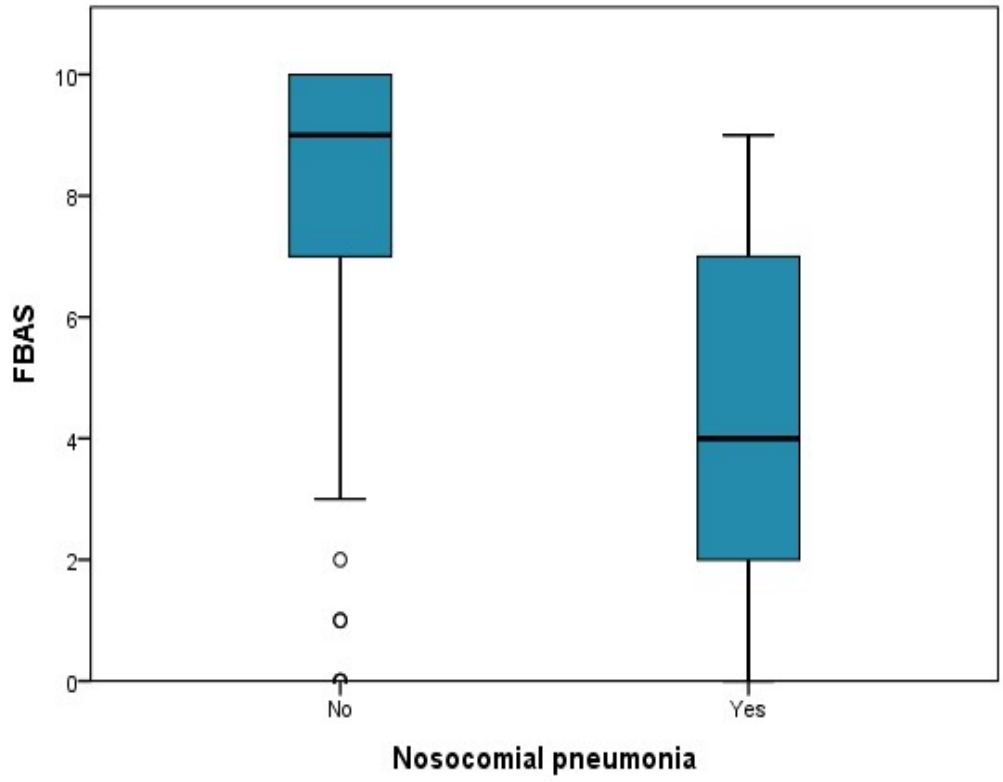



**Figure 8.8** Performance on the FBAS and in hospital pneumonia

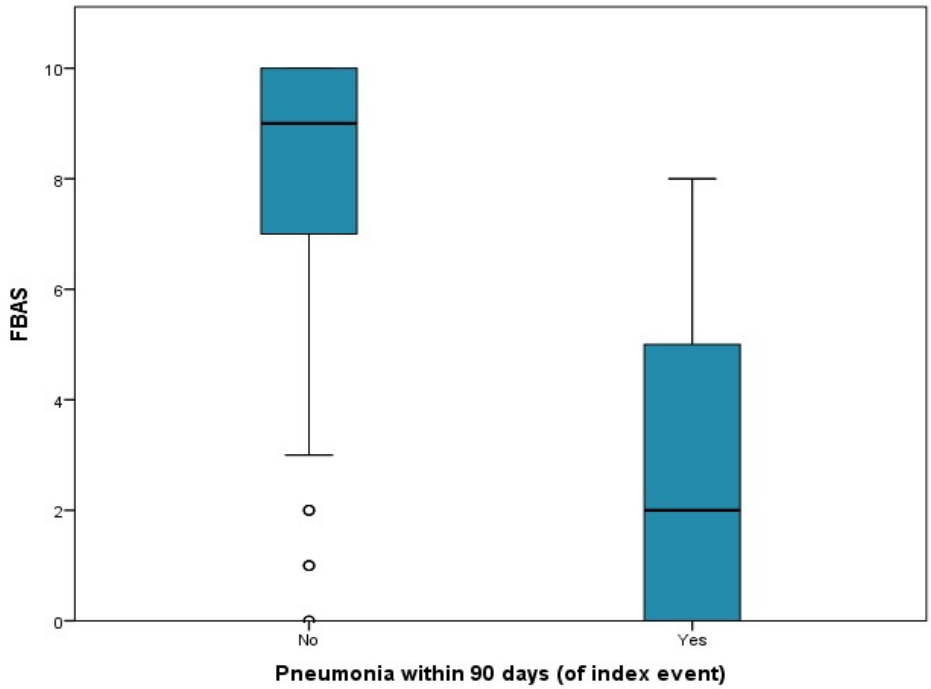



**Figure 8.9** Performance on the FBAS and pneumonia at 3 months

Of further interest, the risk of pneumonia during hospitalization for the index event and within 90 days after stroke onset was significantly higher in patients who failed either the FBAS test or the YSP test as reported in Table 8.5. It was estimated that a patient who fails the FBAS protocol is 1.82 times more likely to develop aspiration pneumonia (95% CI=1.42–2.35) and the respective risk ratio for Yale Swallow Protocol equals 1.79 times (95% CI=1.39–2.28). The relationship was also significant for the risk of pneumonia within 90 days but quite lower. Practically, a patient who fails the FBAS examination is 1.35 times more likely to develop aspiration pneumonia within the first 90 days (95% CI=1.15–1.59) and the respective risk ratio for Yale Swallow Protocol equals 1.22 times (95% CI=1.07–1.42).

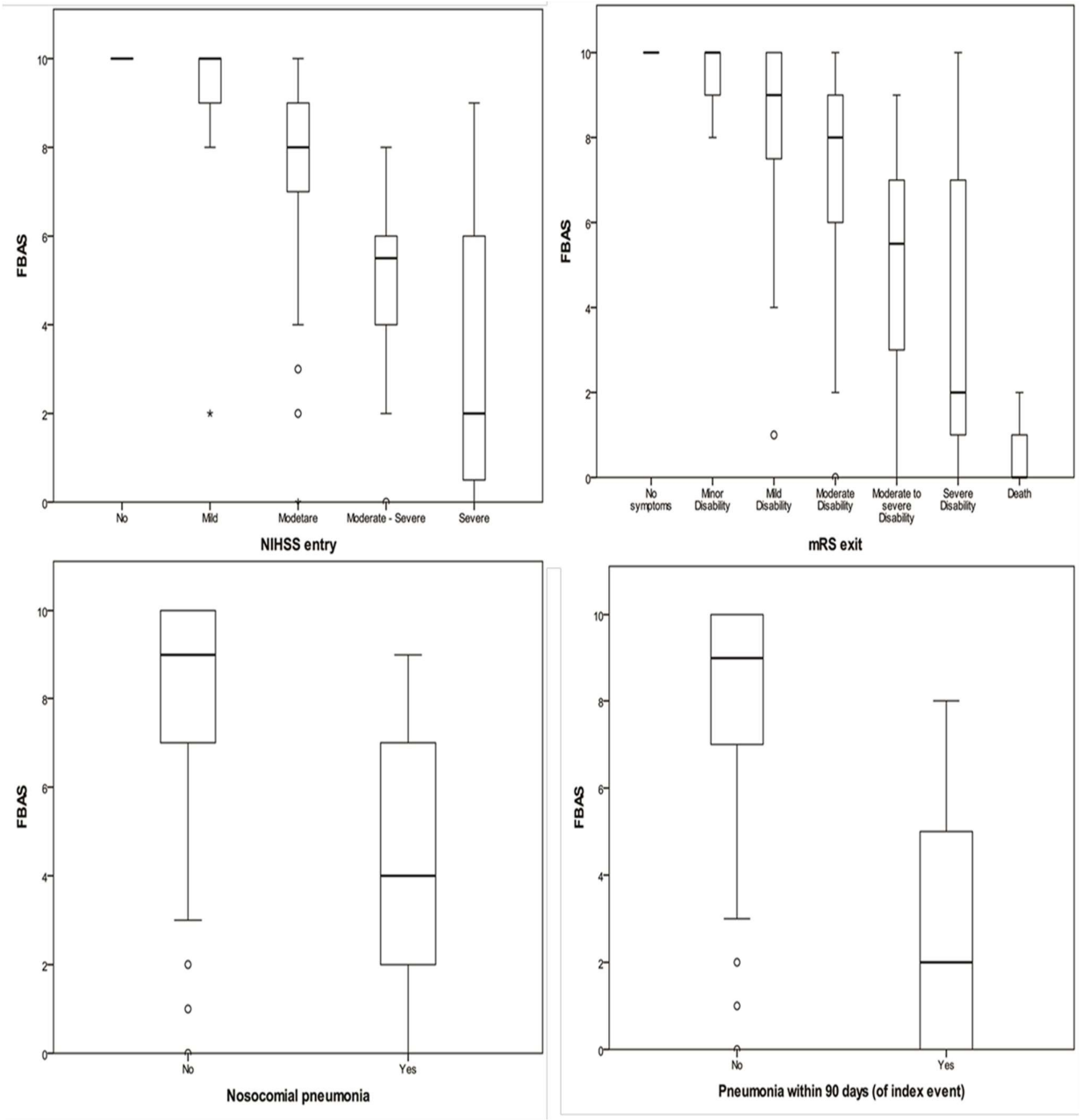
**Table 8.5** Associations of FBAS and YSP with Pneumonia Outcome

| | FBAS[+] | | | YSP[±] | | |
|---|---|---|---|---|---|---|
| **Variable** | **Fisher's Exact test** | **P** | **Risk Ratio (95% CI)** | **Fisher's Exact test** | **P** | **Risk Ratio (95% CI)** |
| Nosocomial pneumonia | 27.17* | <0.001 | 1.82 (1.43-2.32) | 25.43* | <0.001 | 1.79 (1.39-2.28) |
| Pneumonia within 90 days of index event | 14.39* | <0.001 | 1.35 (1.15-1.59) | 9.02* | 0.002 | 1.22 (1.07-1.42) |
| Pearson $\chi^2$ test | | | | | | |

[+] *FBAS, Functional Bedside Aspiration Screen ,* [±] *YSP, Yale Swallow Protocol*

Figure 8.10 below depicts comparative boxplots of the performance on the FBAS scale with measured patient outcome.

**Figure 8.10.** Comparative boxplots of the Functional Bedside Aspiration Screen (FBAS) with health outcome measures




NIHSS† entry No impairment (0), Mild (1-4), Moderate (5-15), Moderate-severe (16-20), Severe (21-42), mRS‡ exit No symptoms (0), No significant disability (1), Slight disability (2), Moderate disability (3), Moderate severe disability (4), Severe disability (5), Death (6)

# CHAPTER 9: DISCUSSION

Dysphagia with or without aspiration is highly prevalent after stroke and is associated with increased nutritional deficits and pneumonia risk. Research has shown that early recognition and management through dysphagia screening may positively alter health outcomes. Martino, Pron and Diamant (2000) found evidence suggesting that dysphagia screening leads to better health outcome as it was linked to reduced risk of developing pneumonia, reduced risk of mortality and reduced percutaneous gastrostomy (PEG) insertion rates)[96]. Accordingly, Hinchey et al (2005) revealed that pneumonia rates were 2.4% at sites implementing formal dysphagia screening protocols compared to 5.4% at sites with no formal screening protocol[222]. Joundi and colleagues recently found that failing a screen is associated with pneumonia, disability and dependence, reduced home discharge and increased mortality at one year[278]. All of these studies highlight the need to adhere to formal swallowing screening protocols in order to reduce the risk of complications in hospitalized patients. This is especially important in the stroke population given the higher incidence of swallowing-related aspiration risk within the first days after a stroke[116, 157].

Several screening and bedside assessment tools are widely available for use by the Speech-Language Pathologists[164], but fewer are available for use by other healthcare professionals who may manage these patients at an earlier time. Some tools rely on the patient case history, others on cranial nerve function or direct examination of the oral cavity, while others rely on the observation for clinical markers of aspiration during direct testing of different bolus volumes and consistencies[246]. The heterogeneity of the existing screening protocols reflects that a consensus has yet to be established.

The quality of the articles included for methodological analysis to address our first research objective, varied. Although all quality measures are important for developing a highly valid and reliable tool that can detect the swallowing status and aspiration risk following acute stroke, the exact relevance of specific quality criteria such as the short interval between assessments and the ability to replicate administration protocols may vary according to the needs of the facilities developing swallowing screening tests. Aspiration or aspiration risk was the primary outcome in the vast majority of the studies. However, a patient may present with significant dysphagia without aspiration [96] that may also lead to a reduced nutritional status and lower quality of life [222]. One study [215] also followed up on the hospital stay of the patients for evidence of aspiration pneumonia, which was not performed in any of the other studies. It appears that a screening tool needs to be able to detect both the swallowing status and aspiration risk with high sensitivity and specificity and needs to be correlated with clinical outcomes such as pneumonia. In addition, the tool should be sufficiently simple to allow it to be implemented by the hospital personnel assigned to assess and care for stroke patients.

Significant variability also existed in the components used to screen for swallowing and aspiration risk across the studies. Non-swallowing measures included medical history items such as the

presence of pneumonia, assessment of mental status, speech and language deficits, and oral motor categorical items such as unilateral jaw weakness, tongue strength, gag reflex, secretion management, and volitional cough. However, not all of these items have been demonstrated to be strong predictors of aspiration as already outlined in Chapter 3. McCullough et al. [175] examined the utility of non-swallowing bedside indicators and trial swallowing measures in detecting aspiration. Sound methodology and lengthy statistical recording for each measure were noted, but they reported low sensitivities for each measure individually, leading a clinician to the conclusion that the presence of two measures is much more meaningful than their absence. Regression analysis demonstrated that the best factors in the diagnostic model for detecting overall aspiration risk were the failure of the 90-mL water challenge (WST) and altered voice quality.

The ability to sustain adequate alertness level for a short period of time appears to be a prerequisite for direct oral trials. Furthermore, testing that a patient can actually swallow should be included in any screening tool, but the optimal method of assessment still requires investigation. Most of the studies included a WST, but the volumes administered varied over a very wide range (from 3 mL to 90 mL), and the number of trials also varied in some studies. Research has shown that silent aspiration is volume-dependent [232], and thus one concern with the bedside administration of small bolus volumes for determining aspiration risk is that the absence of overt behavioral signs such as a reflexive cough can lead to high false-negative rates. Meanwhile, there is a need to determine the optimal trade-off between assessing the swallowing ability of patients and their swallowing safety. The safety of requiring an acute-stroke patient to self-administer 90 mL of water without stopping is questionable when research has shown poor awareness of swallowing deficits in the acute phases of stroke, with most patients consuming larger volumes of water more rapidly [279].

Several of the investigators used pulse oximetry in conjunction with bedside swallowing trials to determine the presence of an aspiration risk [159, 215, 225, 255], but there are conflicting data of its usefulness [238, 254]. Lim and colleagues [215] measured oxygen desaturation 10 minutes after applying a modified WST that involved small equal aliquots. A bedside procedure that simultaneously applies a sequential drinking task and pulse oximetry measurements may provide more meaningful results while maintaining the test–retest reliability. Equally controversial findings from small subject groups were found by Smith and colleagues [255]. Although they used a combination of bedside screening and oxygen desaturation testing, they did not clearly report on their bedside assessment procedure, making it almost impossible to utilize their research in clinical practice.

Most of the studies used an instrumental reference test (VFSS or FEES) to objectively confirm the presence of dysphagia or aspiration. There is support in the literature for the need to routinely assess the swallowing function of patient in the acute phase of stroke using a diagnostic test (when this is readily available) and when the patient can sit up and cooperate with the procedure. However, few studies applied the reference standard test and the clinical index screening test within a few days of

each other, reflecting an unacceptable delay in this patient population given that the spontaneous recovery typically occurs rapidly.

The necessity for identifying post-stroke dysphagia and aspiration early in the care pathway of a patient indicates that frontline medical professionals who are the first to make contact with the patient after a CVA need to apply swallowing screening. Several studies have highlighted interdisciplinary dysphagia screening [169, 215, 225, 253], but this had limitations associated with the poorly defined screening procedures making it difficult to integrate them into clinical practice. It is clear from the existing literature that the reported statistical data can be influenced by whether patients are selectively referred due to probable dysphagia or whether they are consecutively recruited into a research study. The selection of different swallowing and non-swallowing features in the evaluation process and their perceived importance in identifying dysphagia and aspiration as well as the significant variability in the volumes and consistencies of boluses applied as direct swallowing stimuli at the bedside can further lead to discrepant assumptions.

Consistent empirical evidence is, therefore, required to achieve best practice for swallowing screens. The absence of a consensus on the best screening methodology should not be interpreted as “no screening should be performed” or that it is a “one fits all” process. Broader patient-specific and facility-specific factors should be taken into account before making any recommendation regarding oral nourishment. Based on these notions and our critical review of non-instrumental bedside tools, we developed a new, clinician-friendly bedside protocol for estimating overall aspiration risk in the acute phase of stroke intended for use by the non-specialists in dysphagia, the Functional Bedside Aspiration Screen (FBAS).

Our FBAS protocol reached high AUC values for documenting aspiration risk in patients with acute stroke with a sensitivity of 93.3% and a specificity of 83.3% in discriminating high- from low-risk patients for aspiration. We found that a score of ≤8 in our 10-point scale is a clinically significant cutoff point for reduced safety for oral feeding. In the absence of readily accessible diagnostic methodologies for evaluating swallowing, comparison of the FBAS test with the YSP revealed promising data with regards to accurate estimation of post-stroke aspiration risk. In our study, we used a larger cohort of approximately 100 prospective patients with a specific medical diagnosis yielding similarly high psychometric statistical measures. Because spontaneous recovery is generally quick in patients with stroke[117, 280], obtaining a narrow timeline between the administration of the two protocols was critical for validity.

Apart from its direct comparison to the YSP, when measured on a binary scale, the FBAS could provide substantial information as a scale measurement since higher scoring on this 10-rated scale was shown to be indicative of lower risk of aspiration or generally better health outcome. Lower performance on the FBAS was significantly associated with neurologic deficit severity as reflected in higher NIHSS entry scores, prolonged hospital stay, greater disability after the stroke (mRS indices) and with the development of in-hospital (nosocomial) pneumonia and pneumonia within 3

months post onset as depicted in Table 8.5 in the previous chapter. It was estimated that a patient who fails the FBAS protocol is 1.82 times more likely to develop nosocomial pneumonia and 1.35 times more likely to develop aspiration pneumonia within the first 90 days. Premorbid mRS was not significantly associated and this may be in part be attributed to the fact that the mRS entry state of most of our patients (almost 85%) were characterized as 'non-symptomatic' or 'with no significant disability' rendering the statistical power of the Fisher's exact test rather inadequate to reach statistical significance.

To the best of our knowledge, most existing screening tests lack data on the outcomes of pneumonia, extended hospital stay, disability or death after stroke. This attribute is considered a strength of the FBAS since it facilitates earlier implementation of effective management approaches especially for patients determined to be at risk for stroke-associated pneumonia. In addition, the FBAS protocol can be completed in <10 minutes time unlike other swallowing tests such as the MASA[166] and the TOR-BSST[214] which both require purchase and lengthy training to be administered. The brevity of completing the entire screen suggests ease of adoption in practical clinic by nonspecialized medical personnel involved in acute care admission.

With respect to its components, patient's ability to sustain adequate alertness and an upright position with some degree of head control are known prerequisites for direct oral trials[165, 260]. Fewer studies, however, have considered the relationship of patient's ability to manage own saliva after acute stroke and swallowing risk[165, 169]. Our study adds to the literature since this criterion was found to be an independent and sensitive predictor of aspiration and aspiration pneumonia[281, 282]. A preliminary investigation is also incorporated in the Gugging Swallowing Screen (GUSS)[169] according to which, the clinician scores both changes in vigilance and cough prior to the direct swallowing trial with varied consistencies. This screening tool however, does not take into consideration changes in language and speech post stroke. With FBAS administration, valuable clinical information is further collected with regards to functional language comprehension associated with a patient's ability to follow a verbal command as well as dysarthria, with the latter proven to be a strong predictive factor for both dysphagia and aspiration risk[155, 175, 231, 283].

Our protocol includes a water swallow trial (WST) to aid in recognition of aspiration risk in acute stroke as do most valid swallowing screening tests[168, 172, 175] and acknowledges as failure criteria already established sensitive indicators for aspiration[155, 166, 214, 219, 263]. A recent meta-analysis[263] revealed that when administering single sips of small and large volumes, the WST offers the best evidence for accurately discriminating patients who are aspirating.

Our constructed FBAS protocol advocates initiating bedside trials with pureed consistencies. This is in accordance with other studies[169, 284] which report that stroke patients have a significantly higher aspiration risk with liquids and that increased viscosities exert a treatment effect on the safety of deglutition. Furthermore, our protocol requires managing one tablespoon full of pureed consistency twice as a means of indirect monitoring of postswallow vallecular residue on the safety of the

swallow. The association between residue presence at the end of a swallow and penetration-aspiration on the next swallow was recently proved in a retrospective study conducted by Molfenter and Steele (2013) who found that post-swallow residue in one or both pharyngeal spaces (valleculae, pyriform sinuses) was significantly associated with impaired swallowing safety on the subsequent swallow for the same bolus. We believe that our approach helps identifying the optimal initial consistency to administer to acute stroke patients at risk for aspiration, further adding to the diagnostic capability of the FBAS.

Besides making initial diet predictions without heavily relying on objective assessment or at least a more detailed clinical swallow evaluation, the systematic application of the FBAS in acute ischemic stroke could provide prognostic information concerning the presence of pneumonia risk and outcome at 3 months. It is known that hospitals that adhere to formal screening protocols can significantly lower their rates of pneumonia[222] and that acute stroke patients who are not screened early for swallowing risk present with a higher risk of developing pulmonary complications[285-287].

Finally, accumulating recent evidence[288-291]suggests that a stroke induces an immunodepressive state increasing susceptibility for stroke-associated pneumonia (SAP). These studies prove that the pathophysiology of SAP is multifactorial and that dysphagia alone is not sufficient for its development. The PREDICT study[289] specifically reveals that SAP is the result of two independent mechanisms, aspiration of oropharyngeal secretions into the lungs and stroke-induced immunodepression syndrome (SIDS), characterized by a down-regulation of systemic cellular immune responses, which thereby lead to an increased susceptibility for pulmonary infections.

Our study has several limitations that need to be acknowledged. Although the FBAS reliably estimates aspiration risk in the very acute phase of stroke management, dysphagia remains an important risk factor for SAP and should be formally evaluated after initial stroke workup in order to make informed physiology-based treatment decisions. As such, further research with the FBAS is warranted to address both evaluation of aspiration and swallowing impairment with instrumental methodologies. Future studies will be conducted with gold standard imaging studies of swallowing such as videofluoroscopy (VFSS) or fiberoptic evaluation of swallowing (FEES). Imaging of swallowing could be reported with use of readily available scoring scales used in acute patients such as FEDSS which takes into consideration aspiration, coughing and different trial consistencies[292]. Another significant limitation of this study relates to the need for reporting inter-rater reliability measures. Concerns exist about the interpretation of all clinical features that constitute the FBAS by frontline healthcare providers working in different patient care settings where objective assessments of swallowing risk are not feasibly conducted.

## 9.1 Concluding Remarks

Dysphagia may represent difficulties with any stage of swallowing which may cause malnutrition, dehydration or aspiration. Presence of dysphagia may not necessarily cause significant health risks. However, it may increase the likelihood of aspiration which has been associated with pneumonia and poor patient outcome. Aspiration, a potential consequence of dysphagia may precipitate pneumonia, an acute and potentially life-threatening condition.

In the acute phase of stroke, nutrition and hydration needs may be managed using alternative medical interventions such as nasogastric tubes or via parenteral routes (intravenous drips or subcutaneous fluids). However, aspiration requires immediate recognition in order to reduce the risk of developing respiratory complications such as aspiration pneumonia. It is, therefore, important that any bedside swallow screening tool, developed for use in the acute stages of stroke, should focus primarily on aspiration in order to reduce the risk of developing aspiration pneumonia in post-stroke patients. Although numerous screening tools have been developed, no present screening protocol provides high specificity and sensitivity for predicting the risk of aspiration. It appears that a cluster of swallowing and non-swallowing features may achieve both high sensitivity and specificity at the bedside.

Instrumentation of swallowing function with endoscopy or fluoroscopy is considered the gold standard in the diagnosis of dysphagia and aspiration but requires specialized staff and equipment which cannot be readily performed or scheduled within a few hours of stroke onset. Although a screen is not a diagnostic tool, international guidelines dictate the importance of implementing early screens of swallowing risk before any diet recommendations are made to enhance patient quality of care, reduce the risk of developing pneumonia and yield better outcome measures.

Our newly developed, clinician-friendly, Functional Bedside Aspiration Screen (FBAS) provides high sensitivity and high negative predictive values in acute stroke ensuring that the potential for aspiration-associated complications during screening is kept to a minimum. Because of its time-efficient nature, the FBAS might easily be adopted as part of a stroke clinical support tool to prompt recognition of patients who are at risk for aspiration and pneumonia development during the acute and subacute phases of stroke onset. Further research is warranted to validate findings of the FBAS with instrumental methodologies such as fiberoptic evaluation of swallowing.

## Abstract

Stroke remains a major global health problem as one of the most common neurological disorders with a considerable socio-economic impact. Most cases of stroke represent a multifactorial disorder. Stroke is associated with high mortality rates and still constitutes the leading cause of sudden and long-term disability worldwide that affects both the survivor and caregiver.

Dysphagia is a frequent and rapidly occurring problem following a stroke. Dysphagia with subsequent aspiration has been associated with an increased risk of pulmonary complications, even death. Stroke guidelines recommend early swallow screening in order to identify all acute stroke patients at risk for aspiration. Most organized stroke units worldwide use a swallow detection tool, but consensus is not yet apparent on the best screening methodology.

The present research thesis had a dual aim: a) to critically review the literature on non-instrumental bedside swallow screening tests and b) to develop a new, clinically user-friendly and reliable screening protocol for identification of acute stroke patients at risk for aspiration. The novel protocol, the Functional Bedside Aspiration Screen (FBAS), was based on a large theoretical and empirical framework which incorporated bedside clinical indicators for aspiration.

Material and Methods: This study included a prospective cohort of patients with confirmed acute ischemic stroke. The results of the newly developed 10-point FBAS scale were compared with those of a reference test, the Yale Swallow Protocol (YSP) and health outcome indicators.

Results: A strong association was revealed between the FBAS cut-off criterion and the validated YSP. A score of ≤8 on the FBAS presented with 93.3% sensitivity and 83.3% specificity (PPV =84%, NPV=93%) in deeming a patient with reduced safety for oral intake (AUC = 0.934, CI = 0.884-0.985). An inverse relationship was found between performance on the FBAS and in-hospital and long-term outcome indicators. Patients who failed on the FBAS scale were 1.82 times more likely to develop nosocomial pneumonia (95% CI = 1.42-2.35) and 1.35 times more likely to develop pneumonia within 3 months post-onset (95% CI = 1.15-1.59).

Conclusions: The FBAS scale provides a high predictive ability for risk of aspiration in patients with acute ischemic stroke. Its stepwise approach ensures that the potential for aspiration-associated complications during screening administration is kept to a minimum. The fact that failure on the FBAS is associated with pulmonary complications enhances its diagnostic capacity compared to other popular bedside screening tools. Although no bedside screening test is a substitute for an instrumental swallow exam, the FBAS scale may be a potentially useful tool for timely prediction of aspiration risk and health outcome in acute stroke.

**Key words:** acute ischemic stroke, swallowing, screening, aspiration risk, pneumonia, health outcome

## Περίληψη

Το αγγειακό εγκεφαλικό επεισόδιο είναι μια από τις πιο συχνές σοβαρές νευρολογικές νόσους και εξελίσσεται ως μείζον πρόβλημα για τη δημόσια υγεία με εκτενείς ψυχοκοινωνικές και οικονομικές συνιστώσες. Το αγγειακό εγκεφαλικό επεισόδιο θεωρείται πολυπαραγοντική νόσος που συνδέεται με υψηλούς δείκτες θνησιμότητας και θνητότητας παγκοσμίως, ενώ παραμένει η πρώτη αιτία αιφνίδιας και μακροχρόνιας ανικανότητας ή αναπηρίας που επιβαρύνει τόσο τον ασθενή όσο και την οικογένειά του.

Η δυσφαγία είναι μια συχνή και γρήγορα εμφανιζόμενη νοσηρότητα μετά από ένα αγγειακό εγκεφαλικό επεισόδιο. Η δυσφαγία με συνεπακόλουθη εισρόφηση έχει συσχετιστεί με αυξημένο κίνδυνο πνευμονικών επιπλοκών, ακόμη και θανάτου. Οι σύγχρονες κατευθυντήριες οδηγίες αντιμετώπισης των εγκεφαλικών επεισοδίων στοχεύουν στην βελτίωση της διάγνωσης και της νοσοκομειακής περίθαλψης των ασθενών στην οξεία φάση και συνιστούν την διενέργεια έγκαιρης αξιολόγησης για τον πρώιμο εντοπισμό ασθενών ‘υψηλού κινδύνου’ για εισρόφηση. Η πλειονότητα των οργανωμένων μονάδων εγκεφαλικών επεισοδίων παγκοσμίως χρησιμοποιεί κάποιο ανιχνευτικό εργαλείο, ωστόσο δεν επικρατεί ομοφωνία στην μέθοδο ελέγχου.

Διττός στόχος της παρούσας μελέτης ήταν η κριτική επισκόπηση της βιβλιογραφίας και η ανάπτυξη ενός νέου, κλινικά εύχρηστου και αξιόπιστου πρωτοκόλλου παρά την κλίνη για την εκτίμηση του κινδύνου εισρόφησης μετά από οξεία αγγειακή προσβολή. Το τελικό προϊόν της μελέτης ήταν η παροχή θεωρητικού και εμπειρικού πλαισίου για τους υψηλής διαγνωστικής αξίας κλινικούς δείκτες εισρόφησης, οι οποίοι πρέπει να αποτελούν τον βασικό άξονα για την ανάπτυξη ενός τεκμηριωμένου εργαλείου κατάποσης παρά την κλίνη.

Υλικό και Μέθοδος: Πρόκειται για μία προοπτική μελέτη σε ασθενείς με βεβαιωμένη οξεία ισχαιμική αγγειακή προσβολή στην οποία πραγματοποιήθηκε σύγκριση των αποτελεσμάτων της νέας προτεινόμενης πρακτικής δεκάβαθμης Κλίμακας FBAS με αυτά του τυποποιημένου Πρωτοκόλλου Κατάποσης Yale (YSP) σε συνδυασμό και με δείκτες έκβασης της υγείας των ασθενών.

Αποτελέσματα: Διαπιστώθηκε ισχυρή συσχέτιση μεταξύ του κριτηρίου αποκοπής στην κλίμακα FBAS και του επικυρωμένου εργαλείου YSP. Βαθμολογία ≤8 στην κλίμακα FBAS παρουσίασε ευαισθησία 93,3% και ειδικότητα 83,3% στην εκτίμηση ασθενούς μειωμένης ασφάλειας για πρόσληψη τροφής ή υγρών από του στόματος (AUC = 0,934, CI = 0,884-0,985). Πρόσθετα ευρήματα ανέδειξαν μία αντίστροφη συσχέτιση μεταξύ των επιδόσεων στην κλίμακα FBAS και των ενδονοσοκομειακών και μακροπρόθεσμων δεικτών έκβασης. Συγκεκριμένα, ασθενείς που απέτυχαν στην κλίμακα FBAS ήταν 1,82 φορές πιο πιθανό να αναπτύξουν ενδονοσοκομειακή πνευμονική λοίμωξη (95% CI = 1,42-2,35) και 1,35 φορές πιο πιθανό να αναπτύξουν πνευμονία από εισρόφηση 3 μήνες μετά την εισβολή του εγκεφαλικού (95% CI=1.15–1.59).

Συμπεράσματα: Η κλίμακα FBAS προσφέρει υψηλή ικανότητα πρόβλεψης του κινδύνου εισρόφησης σε ασθενείς με οξύ ισχαιμικό εγκεφαλικό επεισόδιο. Η κλιμακούμενη προσέγγιση κατά την εφαρμογή

της διασφαλίζει ότι οι πιθανότητες εμφάνισης επιπλοκών σχετιζόμενες με εισρόφηση κατά τη διάρκεια της εξέτασης περιορίζεται στο ελάχιστο. Το δεδομένο ότι η αποτυχία στην κλίμακα FBAS συνδέθηκε με πνευμονική λοίμωξη, ενισχύει την διαγνωστική της δυνατότητα συγκριτικά με άλλα δημοφιλή εργαλεία παρά την κλίνη του ασθενούς. Η κλίμακα FBAS δεν υποκαθιστά τη διαγνωστική αξία ενός απεικονιστικού ελέγχου κατάποσης, αλλά μπορεί να αποτελέσει ένα δυνητικά χρήσιμο εργαλείο εκτίμησης του κινδύνου εισρόφησης και της έκβασης των ασθενών με οξύ αγγειακό εγκεφαλικό επεισόδιο.

**Λέξεις κλειδιά:** οξύ ισχαιμικό εγκεφαλικό, κατάποση, ανίχνευση, κίνδυνος εισρόφησης, πνευμονία, δείκτες έκβασης

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# APPENDICES

## Α1. Ενημερωτικό Έντυπο Συγκατάθεσης

Καλείστε να συμμετάσχετε σε μία έρευνα με θέμα *«Η καταγραφή των διαταραχών σίτισης-κατάποσης έπειτα από ισχαιμικά ΑΕΕ με την χρήση λογοπαθολογικών κλιμάκων και τη βοήθεια σύγχρονων νευροαπεικονιστικών μεθόδων»* κατά τη νοσηλεία σας στο Π.Γ.Ν. Ιωαννίνων, στα πλαίσια του ερευνητικού έργου της Παθολογικής και της Νευρολογικής Κλινικής με τη στήριξη της Ιατρικής Σχολής Ιωαννίνων. Η συμμετοχή σας σε αυτήν την έρευνα είναι εθελοντική και δεν θα υπάρξουν κυρώσεις ή απώλεια των δικαιωμάτων σας εάν αρνηθείτε να συμμετάσχετε ή εάν αποφασίσετε να αποσυρθείτε μελλοντικά ενώ έχετε ήδη δηλώσει τη συγκατάθεσή σας.

Παρακαλώ λάβετε υπόψη τα παρακάτω:

1) Στην έρευνα αυτή, θα συμμετέχουν συνολικά 200 ασθενείς που υπέστησαν πρόσφατα αγγειακό εγκεφαλικό επεισόδιο.
2) Η συμμετοχή σας σε αυτή την έρευνα περιλαμβάνει την καταγραφή δεδομένων από την εκτίμηση διαταραχών σίτισης-κατάποσης.
3) Δεν θα υπάρξει κάποιος κίνδυνος με την συμμετοχή σας στην έρευνα αυτή.
4) Δεν υπάρχει ή θα υπάρξει στο μέλλον οποιασδήποτε μορφής κέρδος, έμμεσα ή άμεσα, από την συμμετοχή σας σε αυτή την έρευνα.
5) Τα δεδομένα σας καλύπτονται από πλήρης εμπιστευτικότητα και ανωνυμία. Μελλοντικές δημοσιεύσεις των αποτελεσμάτων της παρούσας μελέτης σε έγκυρα επιστημονικά περιοδικά θα πραγματοποιηθούν υπό καθεστώς ανωνυμίας.

Σε περίπτωση που χρειάζεστε πρόσθετες διευκρινίσεις, μπορείτε να επικοινωνήσετε με τον Επιβλέποντα Αναπληρωτή Καθηγητή Παθολογίας της έρευνας αυτής, κ. Χαράλαμπο Μηλιώνη (Β' Παθολογική Κλινική ΠΓΝ Ιωαννίνων, τηλ. 2651099624).

Σας ευχαριστούμε για τη συνεργασία.

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**Ενημερώθηκα για τη μελέτη και δέχομαι να πάρω μέρος σ' αυτήν.**

*(Σε περίπτωση αδυναμίας του ασθενούς ζητείται συγκατάθεση από τον οικείο-συνοδό του)*

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| Υπογραφή Συμμετέχοντος/Νόμιμου Πληρεξουσίου Αντιπροσώπου | Ημ/νία |

## Α2. Ερωτηματολόγιο Καταγραφής Δεδομένων Ασθενή

# Ερωτηματολογίου: _____

| | | | | | |
|---|---|---|---|---|---|
| **Ονοματεπώνυμο:** | | | | | |
| **Εσωτερικός Κωδικός Ασθενή:** | | | | | |
| **Φύλο:** | ☐ 1. Άνδρας<br>☐ 2. Γυναίκα | **Ηλικία:** | ☐ 0. 18-55<br>☐ 1. 55-75<br>☐ 2. 75 + | **Οικογενειακή Κατάσταση:** | ☐ 0. Άγαμος<br>☐ 1. Έγγαμος<br>☐ 2. Ζωντοχήρος/Χήρος |

| **Στοιχεία Επικοινωνίας Οικείου Συνοδού** | | | |
|---|---|---|---|
| **Ονοματεπώνυμο:** | | **Ημερομηνία Επικοινωνίας:** | |
| **Βαθμός Συγγένειας:** | | **Τηλέφωνο:** | |

| | | | |
|---|---|---|---|
| **Κλινική Εισαγωγής:** | ☐ Παθολογική Β'<br>☐ Νευρολογική | **Ημερομηνία Εισαγωγής:** | |
| | | **Ημερομηνία Εξιτηρίου:** | |
| | | **Διάρκεια Νοσηλείας:** | ☐ ≤ 1 εβδομάδα<br>☐ 1-2 εβδομάδες<br>☐ > 2 εβδομάδες |

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| **Εντοπισμός Εμφράκτου:** | ☐ ΑΡ Ημισφαιρική Βλάβη<br>☐ ΔΕ Ημισφαιρική Βλάβη<br>☐ Οπίσθιου Κρανιακού Βόθρου |
| Απεικονιστικά Ευρήματα Αναλυτικά: | |
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| **Αγγειακό Εγκεφαλικό Επεισόδιο ΑΕΕ** | **Ισχαιμικό** | | | **Αιμορραγικό** | | |
| | ☐ 1. Αθηροθρομβωτικό<br>☐ 2. Καρδιοεμβολικό<br>☐ 3. Κενοτοπιώδες<br>☐ 4. Άλλης Καθορισμένης Αιτιολογίας<br>☐ 5. Αταξινόμητο ή άλλης άγνωστης αιτιολογίας | | | ☐ 6. Ενδοεγκεφαλικό<br>☐ 7. Υπαραχνοειδές | | |
| | **ΟΧΙ** | **ΝΑΙ** | | **ΟΧΙ** | **ΝΑΙ** | |

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| **Συννοσηρότητα** | Αρτηριακή Υπέρταση | ☐ | ☐ | ΧΑΠ | ☐ | ☐ |
| | Σακχαρώδης Διαβήτης | ☐ | ☐ | Βηματοδότης | ☐ | ☐ |
| | Δυσλιπιδαιμία | ☐ | ☐ | Ρευματοειδής Αρθρίτιδα | ☐ | ☐ |
| | Κολπική Μαρμαρυγή | ☐ | ☐ | Επιληψία | ☐ | ☐ |
| | Στεφανιαία Νόσος | ☐ | ☐ | Υποθυρεοειδισμός | ☐ | ☐ |
| | Αγγειακή Άνοια – Εγκεφ. Ατροφία | ☐ | ☐ | Καρδιακή Ανεπάρκεια | ☐ | ☐ |

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| **Ιστορικό ΑΕΕ/ΤΙΑ** | ☐ 0. ΟΧΙ<br>☐ 1. ΝΑΙ | **Κάπνισμα** | ☐ 0. ΟΧΙ<br>☐ 1. ΝΑΙ<br>☐ 2. Πρώην καπνιστής | **Αλκοόλ** | ☐ 0. ΟΧΙ<br>☐ 1. 1-2 φορές/εβδομάδα<br>☐ 2. 3-5 φορές/εβδομάδα<br>☐ 3. Καθημερινά |
| **Ιστορικό Διαταραχών Κατάποσης** | ☐ 0. ΟΧΙ<br>☐ 1. ΝΑΙ | | | | |

| | | **0. ΟΧΙ** | **1. ΝΑΙ** | **Παρατηρήσεις** |
|---|---|---|---|---|
| **Αγωγή πριν το ΑΕΕ** | Ανθυπερτασικά | ☐ | ☐ | |
| | Υπολιπιδαιμικά | ☐ | ☐ | |
| | Αντιαιμοπεταλιακά | ☐ | ☐ | |
| | Αντιπηκτικά | ☐ | ☐ | |
| | Αντιδιαβητικά | ☐ | ☐ | |
| | Ινσουλίνη | ☐ | ☐ | |
| **Αγωγή κατά τη νοσηλεία** | Αντιαιμοπεταλειακή Αγωγή | ☐ | ☐ | |
| | Αντιπηκτική Αγωγή | ☐ | ☐ | |
| | Θρομβόλυση | ☐ | ☐ | |
| **Αγωγή μετά το ΑΕΕ** | Ανθυπερτασικά | ☐ | ☐ | |
| | Υπολιπιδαιμικά | ☐ | ☐ | |
| | Αντιαιμοπεταλιακά | ☐ | ☐ | |
| | Αντιπηκτικά | ☐ | ☐ | |
| | Αντιδιαβητικά | ☐ | ☐ | |
| | Ινσουλίνη | ☐ | ☐ | |
| **Επιπλοκές κατά τη νοσηλεία** | Λοίμωξη πλην ουροποιητικού/αναπνευστικού | ☐ | ☐ | |
| | Λοίμωξη ουροποιητικού | ☐ | ☐ | |
| | Πνευμονία/Λοίμωξη Αναπνευστικού | ☐ | ☐ | |
| | Αυξημένος Δείκτης CRP | ☐ | ☐ | |

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| **Πνευμονία εντός 90 ημερών** | ☐ | ☐ | |

| **Συμπτωματολογία** | | **0. Άνευ** | **1-4. Ήπια** | **5-15. Μέτρια** | **16-20. Μέτρια-Σοβαρή** | **21-42. Σοβαρή** |
|---|---|---|---|---|---|---|
| **Κλίμακα NIHSS** | Εισαγωγής | ☐ | ☐ | ☐ | ☐ | ☐ |
| | Εξόδου | ☐ | ☐ | ☐ | ☐ | ☐ |

| | | **0. κανένα σύμπτωμα** | **1. καμία σημαντική αναπηρία** | **2. ήπια αναπηρία** | **3. μέτρια αναπηρία** | **4. μέτρια – σοβαρή αναπηρία** | **5. σοβαρή αναπηρία** | **6. απεβίωσε** |
|---|---|---|---|---|---|---|---|---|
| **mRS** | πριν το ΑΕΕ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | κατά την έξοδο | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |
| | mRS 90 | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ | ☐ |

| **Πρωτόκολλο Κατάποσης YALE** | | | | | |
|---|---|---|---|---|---|
| **Βήμα 1ο : Πρωτόκολλο** | | **Μη Εφαρμόσιμο ☐** | | **Εφαρμόσιμο ☐** | |
| | | | **0. ΟΧΙ** | **1. ΝΑΙ** | **Παρατηρήσεις** |
| **Βήμα 1α. Γνωστική Εξέταση** | Προσανατολισμός | Πρόσωπο | ☐ | ☐ | |
| | | Χώρο | ☐ | ☐ | |
| | | Χρόνο | ☐ | ☐ | |
| | Ακολουθία οδηγιών | Άνοιγμα Στόματος | ☐ | ☐ | |
| | | Προβολή Γλώσσας | ☐ | ☐ | |
| | | Χαμόγελο | ☐ | ☐ | |
| **Βήμα 1β. Στοματική Εξέταση** | | Φραγή Χειλέων | ☐ | ☐ | |
| | | Εύρος γλωσσικής κίνησης | ☐ | ☐ | |
| | | Συμμετρία Προσώπου | ☐ | ☐ | |
| **Βήμα 2ο: Διαδοχική Πόση** | Pass | | ☐ | ☐ | |
| | Fail | Βήχας/πνιγμός κατά την διάρκεια της πόσης | ☐ | ☐ | |
| | | Βήχας/πνιγμός μετά τη δοκιμασία πόσης | ☐ | ☐ | |
| | | Διακοπτόμενη πόση | ☐ | ☐ | |
| **Βήμα 3ο:** | | Εάν απαντήθηκε ‘fail’ → NPO → επανοχορήγηση σε 24 ώρες ή παραπομπή σε απεικονιστική αξ/ση | | | |

| Πρωτόκολλο FBAS | | |
|---|---|---|
| **Βήμα 1ο: Κριτήρια Αποκλεισμού για Δοκιμή Σίτισης Δια του Στόματος** | | |
| | **0. ΟΧΙ** | **1. ΝΑΙ** |
| **Εγρήγορση** | ☐ 0. Βυθιότητα / αδυναμία ή μη διατηρητέα εγρήγορση για σκοπούς της εκτίμησης (διακυμάνσεις επιπέδου εγρήγορσης, δυσκολία αφύπνισης με κίνηση ή ομιλία) | ☐ 1. Επαρκής εγρήγορση |
| **Παραμονή σε ορθή θέση** | ☐ 0. Αδυναμία υιοθέτησης κάθετης στάσης | ☐ 1. Θέση > 60ο |
| **Διαχείριση Σιέλου** | ☐ 0. Εμφανής σιελόρροια ή ανικανότητα έναρξης μίας ξηρής κατάποσης (κατάποσης σιέλου) ή καμία ορατή αυθόρμητη κατάποση ή ακουστικοί τρίζοντες ρόγχοι/υγρή, γάργαρη φώνηση | ☐ 1. Απουσία σιελόρροιας, παρατηρούμενες ακούσιες καταπόσεις σιέλου |
| | **Σύνολο: 3** | **Υποσύνολο Ασθενή:** |

| **Βήμα 2ο:** | **Καταγραφή άλλων κλινικών παραμέτρων** | |
|---|---|---|
| **Λειτουργική κατανόηση λόγου** | ☐ 0. Αδυναμία απόκρισης σε απλά παραγγέλματα (εκούσια ή κατόπιν μίμησης)<br>☐ 1. Ασθενής φαίνεται να αποκρίνεται σε καθημερινή συζήτηση | |
| **Παρουσία Νευροκινητικής Διαταραχής Ομιλίας** | ☐ 0. Ανάρθρια ή δυσκατάληπτη ομιλία<br>☐ 1. Ευκρινής ομιλία, απουσία δυσαρθρίας/δυσπραξίας | |
| **Λαρυγγική Αντίδραση** | ☐ 0. Αδυναμία εκτέλεσης εκούσιου βήχα ή παρατήρηση αδύναμου βήχα<br>☐ 1. Ικανότητα εκτέλεσης παραγωγικού/αποτελεσματικού εκούσιου βήχα | |
| | **Σύνολο: 3** | **Υποσύνολο Ασθενή:** |

*Η καταγραφή της οδοντοστοιχίας δεν συνυπολογίζεται στο τελικό σκορ του ασθενή στο FBAS αλλά αποτελεί ποιοτική παρατήρηση εάν ο ασθενής διακριθεί στην ομάδα χαμηλού κινδύνου εισρόφησης.*

| | |
|---|---|
| **Οδοντοστοιχία** | ☐ Νωδός<br>☐ Ελλιπής/Μερική Οδοντοστοιχία<br>☐ Πλήρης Οδοντοστοιχία (τεχνητή ή μη) |

| **Βήμα 3ο: Δοκιμές Σίτισης (μόνο εφόσον ο ασθενής πληροί κριτήρια εφαρμογής από το βήμα 1)** | | | | | |
|---|---|---|---|---|---|
| | | | **0. ΟΧΙ** | **1. ΝΑΙ** | **Παρατηρήσεις** |
| **Διαχείριση 5 cc κρεμώδους σύστασης** | Pass | | ☐ | ☐ | |
| | Fail | Βήχας/Πνιγμός/Καθαρισμός Λαιμού/Αλλαγή ποιότητας φωνής μετά την κατάποση | ☐ | ☐ | |
| **Διαχείριση 10 cc κρεμώδους σύστασης (2 φορές)** | Pass | | ☐ | ☐ | |
| | Fail | Βήχας/Πνιγμός/καθαρισμός Λαιμού/Αλλαγή ποιότητας φωνής μετά την κατάποση | ☐ | ☐ | |
| **Ελεγχόμενη πόση από** | Pass | | ☐ | ☐ | |

| | | | | | |
|---|---|---|---|---|---|
| **ποτήρι (μια γουλιά από ποτήρι ή με κουτάλι)** | Fail | Βήχας/πνιγμός/καθαρισμός λαιμού/υγρή ποιότητα φωνής αμέσως μετά την δοκιμασία πόσης | ☐ | ☐ | |
| **Διαδοχική πόση 90 cc νερού (από ποτήρι)** | Pass | | ☐ | ☐ | |
| | Fail | Βήχας/πνιγμός κατά την διάρκεια της πόσης/Καθαρισμός Λαιμού | ☐ | ☐ | |
| | | Βήχας/πνιγμός/καθαρισμός λαιμού/υγρή ποιότητα φωνής αμέσως μετά την δοκιμασία πόσης | ☐ | ☐ | |
| | | | | | |
| | | | **ΣΥΝΟΛΙΚΟ ΣΚΟΡ** | **/10** | |

**Εκτιμώμενος Κίνδυνος Εισρόφησης:**

| **Χαμηλός (Σκορ 9 ή 10)** | **Υψηλός (Σκορ 1-8)** |
|---|---|

Σκορ ≥ 9 έναρξη σίτισης δια του στόματος (συνεκτίμηση επιπέδου οδοντοστοιχίας).

Σκορ ≤ 8 Τίποτα Δια του Στόματος (NPO) και παραπομπή για πληρέστερη εκτίμηση παρά την κλίνη από εξειδικευμένο λογοθεραπευτή ή παραπομπή για εργαστηριακή αξιολόγηση του μηχανισμού κατάποσης.

## A3. The Functional Bedside Aspiration Screen

Instructions: Circle the most appropriate score for each field based on clinical examination and then summarize to receive a total score.

***Note:*** The swallowing part of the protocol is not administered if any NO answer to the criteria outlined in STEP 1 as the patient is automatically deemed unsafe for any direct swallow trial. Remain on a *non per os* status (NPO) and consult a specialist in dysphagia for further evaluation.

Patient Name _____Date _____Patient Room # _____

| **Functional Bedside Aspiration Screen (FBAS)** | | |
|---|---|---|
| **Step 1: Exclusion Criteria For Oral Trials** | | |
| | **No** | **Yes** |
| **Alertness** | ☐ 0. Lethargic or inability to maintain adequate level of alertness (fluctuations, difficulty awakening) | ☐ 1. Adequate alertness |
| **Positioning** | ☐ 0. Inability to sit upright in bed or chair | ☐ 1. Vertical position >60º with/without support |
| **Saliva Management** | ☐ 0. Inability to initiate a saliva swallow or notable rumbling/rales sounds coming from the sternum or wet/gurgly quality of voice | ☐ 1. Notable spontaneous saliva swallowing (patient should be able to initiate a saliva swallow- notable upward bedside movement of larynx even after clinician provocation) |
| | **Sum: 3** | **Total Patient Score: ____** |
| ***The above parameters need to be satisfied for continuation w/ bedside trials*** | | |

| **Step 2** | **Recording of other cognitive-linguistic and physical clinical parameters** | |
|---|---|---|
| **Functional Language Comprehension** | ☐ 0. Inability to follow simple commands or response to simple, familiar oral commands with cueing or after imitation<br>☐ 1. Patient appears to understand daily conversation | |
| **Presence of Speech (OM) Impairment** | ☐ 0. Anarthria or intelligible speech with difficulty<br>☐ 1. Clear speech, absence of dysarthria or apraxia of speech | |
| **Laryngeal response** | ☐ 0. Inability to perform a strong volitional cough<br>☐ 1. Strong volitional cough upon command | |
| | **Sum: 3** | **Total Patient Score: ____** |

*Before the administration of oral trials, patients are positioned upright at 90º or as upright as possible in a bed or chair and made comfortable.*

| **Step 3** | | **Bedside Oral Trials** | |
|---|---|---|---|
| ***Do not proceed to next step in the oral sequence unless patient receives a pass (score of 1)*** | | **0. NO** | **1. YES** |
| **Managing one teaspoon full of** | Pass | ☐ | ☐ |

| | | | | |
|---|---|---|---|---|
| **pureed consistency (i.e. cream)** | Fail | Coughing/choking/throat clearing and/or a change in voice quality immediately or up to 1 min after swallowing | ☐ | ☐ |
| **Managing one tablespoon full of pureed consistency twice** | Pass | | ☐ | ☐ |
| | Fail | Coughing/choking/throat clearing and/or a change in voice quality immediately or up to 1 min after swallowing | ☐ | ☐ |
| **Ingestion of one sip of water by cup (self-administered or supported by examiner)** | Pass | | ☐ | ☐ |
| | Fail | Coughing/choking/throat clearing and/or a change in voice quality immediately or up to 1 min after swallowing | ☐ | ☐ |
| **Sequential drinking of 90cc water (self-administered or supported by examiner)** | Pass | | ☐ | ☐ |
| | Fail | Coughing/choking/throat clearing and/or a change in voice quality during, immediately or up to 1 min after swallowing | ☐ | ☐ |
| | | Inability to perform uninterrupted drinking task | ☐ | ☐ |
| | | **Sum: 4** | **Total Patient Score: ____** | |

**SUM TOTAL** **/10**

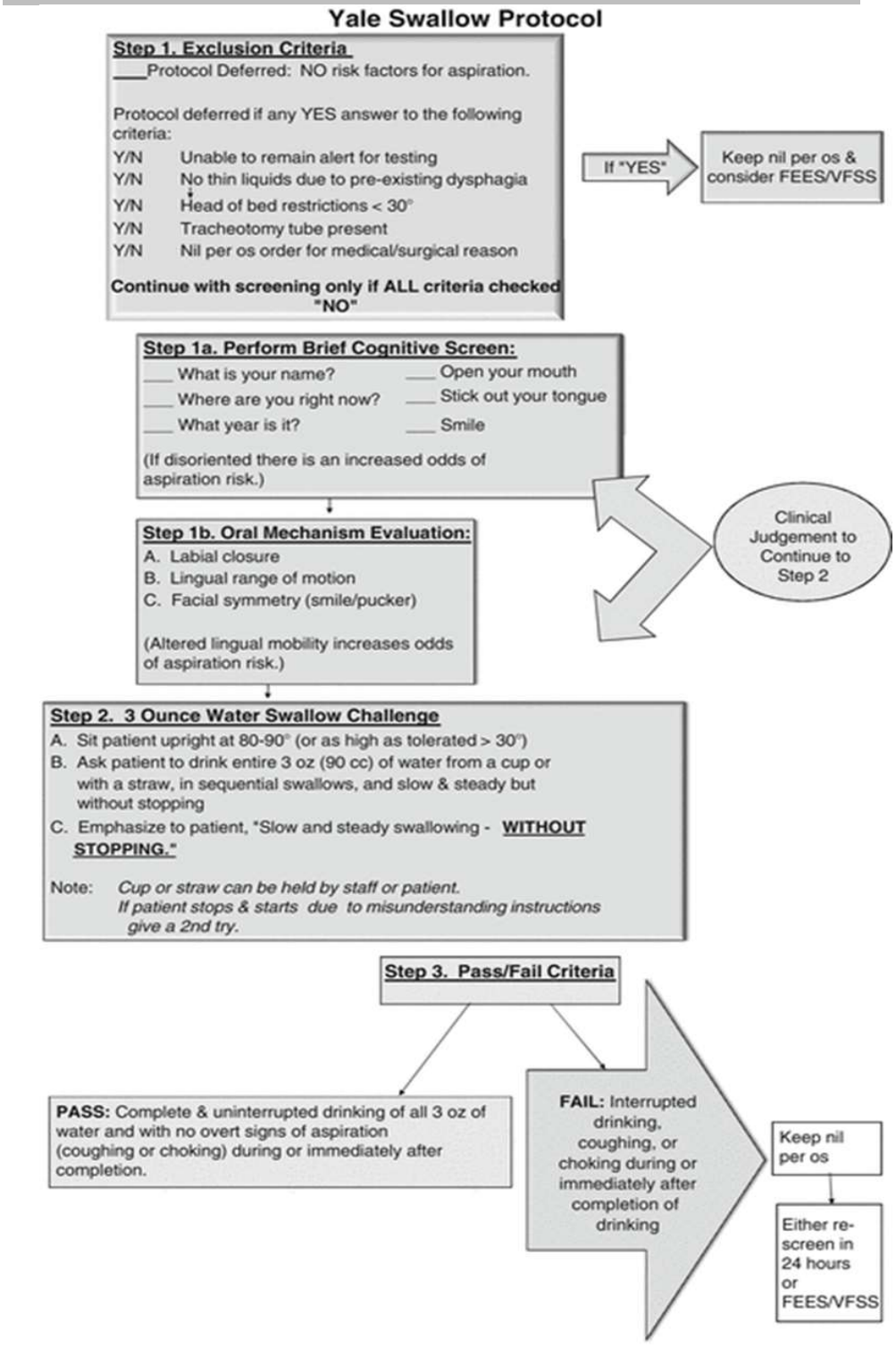
**Patients' Risk of Aspiration:**

**Low (score 9 or 10)** **High (score 1-8)**

Score ≥ ___ start oral diet and progress as tolerated considering dentate status.

Score ≤ ___ NPO and consult a specialist for a comprehensive bedside evaluation and/or instrumentation of swallow mechanics

## A4. The Yale Swallow Protocol




From *The Yale Swallow Protocol: An Evidence-Based Approach to Decision Making*, by S.B. Leder and D.M. Suiter, p. 107. DOI 10.1007/978-3-319-05113-0_13, ©Springer International Publishing Switcherland 2014. Reprinted with permission.