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Cognitive Dysphagia and Effectively Managing The Five Systems

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Abstract

In order for a person to swallow safely and effectively, five bodily systems must work in coordination. Dysphagia occurs when there is a breakdown in one or more of these systems: neurological (NeuroDys), cognitive (CogDys), muscular (MuscDys), respiratory (RespDys), and gastrointestinal (GIDys). The present perspective discusses cognitive decline preceding or concurrently degrading mechanisms of the swallow. We put forth current evidence supporting the premise that cognitive decline can cause dysphagia, and that dysphagia can cause cognitive decline through the exploration of anatomy, physiology, cellular architecture, basic homeostatic mechanisms, metabolism, and nutrition. Additionally, this perspective distinguishes CogDys from NeuroDys and provides an overview of the remaining systems. These types of dysphagia comprise a bi-directional, constantly evolving relationship in which cognition can affect neurological function, and the reverse can be true. We propose literature-driven conclusions and further discussion into considerations for certified nursing assistants, medical physicians, speech-language pathologists, occupational therapists, & physical therapists in effective dysphagia management.

Introduction

Between 2010 and 2030, the geriatric population will dramatically increase to 70 million elderly residents (Mandawat, Mandawat, Mandawat, & Tinetti, 2012). Among these, 10–15 million Americans will live beyond the age of 85 (U.S. Census Bureau, 2012), a group afflicted by progressively deteriorating chronic diseases. Chronic diseases comprise more than 80% of the leading causes of death and debilitation among older Americans, are rarely cured, negatively impact one's quality of life, contribute to declines in cognitive and neuronal functioning, and prevent older Americans from being able to remain in their respective independent communities (Federal Interagency Forum on Aging-Related Statistics, 2012). One chronic disease, neurocognitive impairment, affects 10% of individuals over the age of 65 and 30% over the age of 90. Not surprisingly, neurocognitive decline is the most feared of the chronic diseases in American seniors (Njegovan, Man-Son-Hing, Mitchell, & Molnar, 2001).

The incident of neurocognitive impairment correlates with dysphagia (Kind, Anderson, Hind, Robbins, & Smith, 2011), a largely preventable chronic affliction, and can predict future functional disability, institutionalization, and mortality (Njegovan et al., 2001; White, O'Rourke, Ong, Cordato, & Chan, 2008). The devastating consequences of neurocognitive impairment and dysphagia are well known: neuropsychiatric disturbances, neurological atrophy, increased ventricular volume, reduced overall activity, isolation, dehydration, malnutrition, aspiration, choking, pneumonia, and death (Barer, 1989; Clave & Cichero, 2012; Gordon & Chambers, 2010; Winchester et al., 2013). Data-driven conclusions lead the community to recognize that some subsets of bodily systems work as a unit for effective functioning. Here, evidence that this phenomenon is found in deglutition will be discussed.

Effectively managing five bodily systems involved in deglutition, namely neurological, cognitive, muscular, respiratory, and gastrointestinal dysphagia, is important to the future safety of the aging community. It is a firmly held conviction among practitioners and an expectation among patients that accurate determination of diagnosis and prognosis is an important medical goal, and that functional abnormalities of the oropharyngeal swallowing should be accurately defined (Cook & Kahrilas, 1999). The knowledge presented in the current literature review perspective will provide the health care practitioner with greater understanding of the systems involved in deglutition, and when deglutition is impaired.

Within practitioners, the speech-language pathologist (SLP) is particularly adept at recognizing both the obvious and subtle characteristics of dysphagia and cognitive impairment. The SLP understands how adequate nutrition and hydration can guard against traumatic incidents (e.g., brain injury, fall/fracture, frailty, respiratory complications, weight loss, dehydration, and skin integrity issues), and how the bi-directional relationship of improving overall patient health strengthens and improves the patient's performance of activities of daily living (Rejeski & Mihalko, 2001). Thus, all final conclusions will be framed from the perspective of the interdisciplinary team with particular emphasis on the role of the SLP.

Evidence from the literature supports the bi-directional role of the Five Systems of Dysphagia (5SDys) affecting the ability to safely swallow (Bleach, 1993; Clacagno, Ruoppolo, Grasso, De Vicentiis, & Paolucci, 2002; Clave & Cichero, 2012; Cook, 2008; Cook & Kahrilas, 1999; Irwin, 2006; Janssens, 2005; Janssens, Pache, & Nicod, 1999; Kumar, 2010; Lanspa, Jones, Brown, & Dean, 2013; Njegovan, Man-Son-Hing, Mitchell, & Molnar, 2001; Njegovan et al., 2001; Nordenstedt et al., 2006; Perng et al., 2007; Rangarathnam, Kamarunas, & McCullough, 2014; Rofes et al., 2011; Smithard, 2002; Vaeizi, Hicks, Abelson, & Richter, 2003: White et al., 2008). Therefore, the present literature review seeks to go further by establishing, specifically, the bi-directional relationship between cognitive and neurological function in the healthy older population, and those suffering from a form of decline. An overview of cognition and neurological function will be provided and the co-evolving plasticity of the neurocognitive system will be discussed. The dichotomy and interaction of perceptual awareness and perceptual control will be elucidated.

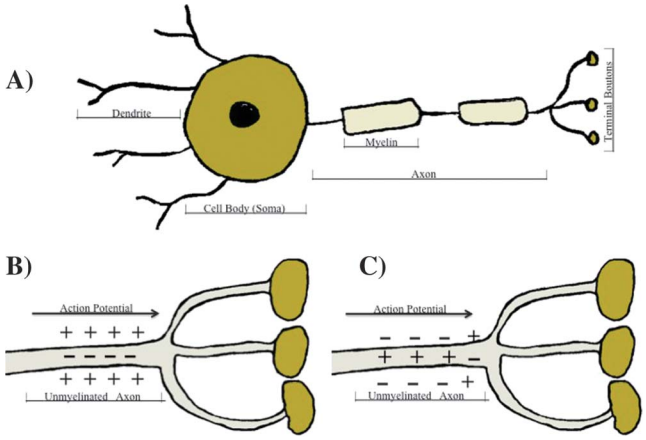
Additionally, the present literature review seeks to demonstrate the bi-directional relationship between cognitive decline and dysphagia, as well as how this bi-directional relationship is affected by and affects 5SDys. Finally, we provide literature-driven conclusions and further discussion into the considerations for the SLP, the nursing staff consisting of, but is not limited to, registered nurses, director of nursing, certified nursing assistants (NSG), the medical physician (MD), the physical therapist (PT), and the occupational therapist (OT). Each of these individuals has a critical role in patient stabilization and the importance of considering Cognitive Dysphagia Management in the older population today and in the future.

Literature-Driven Discussions of the Bi-Directional Relationship Between Cognition and Neurological Function

Background

To illustrate the complexity of the interaction between cognition and neurological function, one must begin by illustrating neurological function, which can be described in terms of biology, chemistry, and physics; then move onto cognition, which is metaphysical and intangible. The beginning of this discussion centers on the function of the neuron (See Figure 1). A neuron is a specialized cell built to generate biochemical, electrical activity that, when multiplied by 10^{10} billion neurons in the cortex alone, results in the emergence of cognition (Bhatnagar, 2013; Chalmers, 1995; Hameroff, 1994). Here, the key aspect is that a neuron never "thinks." The entire nervous system, itself, does not "think." Instead, the nervous system provides the biochemical and electrical framework through which consciousness can manifest in this reality (Bressler & Wise, 2010; Fox, Snyder, Vincent, Corbetta, Van Essen, & Raichle, 2005; Hameroff, 1994; Murphy, Nimmo-Smith, & Lawrence, 2003; Postle, 2006; Sporns, Tononi, & Kotter, 2005).

Figure 1. Anatomy and Physiology of a Neuron.



Dendrites (see A) receive neurotransmitters, become active, and activate the cell body, also known as the soma (see A). With sufficient activation, the soma alters the electrochemical gradient of the axon by forcing a massive influx of sodium and an efflux of potassium ions, resulting in an action potential (see B & C) that is carried to the terminal boutons (see B & C) via conduction in unmyelinated axons, and saltatory conduction in myelinated axons. When the action potential reaches the terminal boutons, synaptic transmission is activated and results in neurotransmitter release to the subsequent neuron's dendrites; and the process repeats in the subsequent neuron. Synaptic transmission is activated trillions of times, per second, in a single human's nervous system. A) Anatomy of a Myelinated Neuron. B) Electrochemical Gradient of an Unmyelinated Axon Prior to the Generation of an Action Potential. C) Electrochemical Gradient of an Unmyelinated Axon During the Conduction of an Action Potential towards the Terminal Boutons. Original Figure adapted from (Bhatnagar, 2013).

The neuron has some unique features, compared to any other cell in the body. For example, once a neuron is born, it will never again undergo "rebirth," meaning that unlike every other cell in the human body, neurons do not undergo mitosis. Once neurons are born, they live until they die, and no other version of that neuron will ever exist (Bhatnagar, 2013). Consequently, in an individual who is 100 years old, there are neurons in the body that are 100 years and 9 months

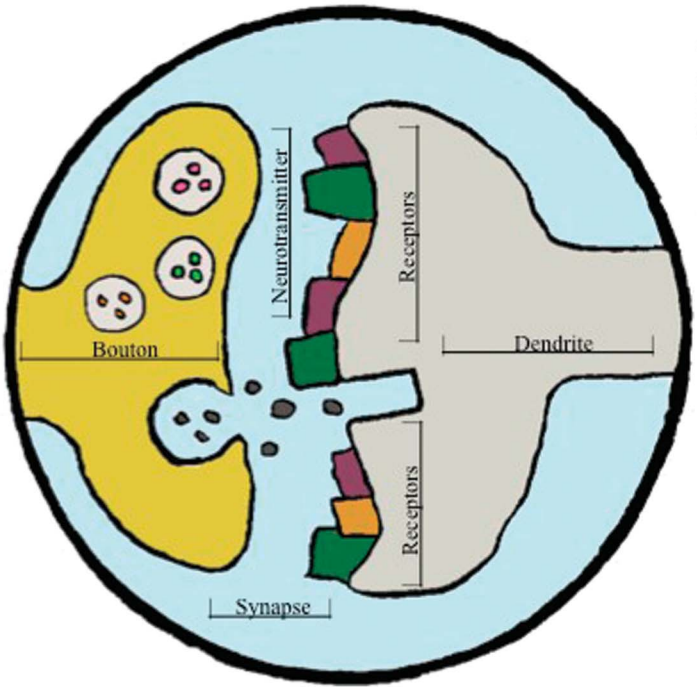
old (approximately) and will continue to function until either the neuron dies or the body dies. Another unique feature of a neuron is that the biochemical and electrical activity of that neuron flows only in one direction: from the dendrites to the cell body, then via an action potential, down an axon to the terminal boutons, triggering synaptic transmission, affecting subsequent neurons through the use of neurotransmitters (See Figures 1 & 2, respectively). This unidirectional flow of biochemical and electrical activity allows the nervous system to have some physical and functional organization, which prevents, as much as possible, a neuron from firing an action potential when it is not supposed to (Bhatnagar, 2013).

Figure 2. Synaptic Transmission and Neurotransmitters.

Acetylcholine: It is the chief neurotransmitter at the neuromuscular junction, the primary neurotransmitter involved in memory encoding/consolidation/recall, it is critical for sleep/wake cycles, and can inhibit basal ganglia activity.

Dopamine: It modulates limbic and prefrontal functions, regulates basal ganglia motor functions, and is involved in reward pathways. Cortical and limbic projections are involved in cognition, emotion and motivation.

Serotonin: It regulates arousal, emotions and pain perception. It is secreted in brainstem and limbic system, yet 95% of all serotonin is found peripherally in blood platelets, and in the GI tract. Together with norepinephrine, serotonin fluctuates sleep and wakefulness. It is also involved in the overall level of arousal and slow-wave sleep. It contributes to the pain-control system.



Glutamate: Facilitates fast synaptic transmission. It is the primary excitatory neurotransmitter.

GABA: It regulates excitability of neurons, pain perception, and inhibits basal ganglia movements.

Norepinephrine: It regulates sleep, attention and mood in conjunction with reticular projections. It is responsible for flight-fight reaction, it is thought to be involved in generating paradoxical sleep with brain wave patterns similar to wakeful state, REM, and maintaining attention and vigilance.

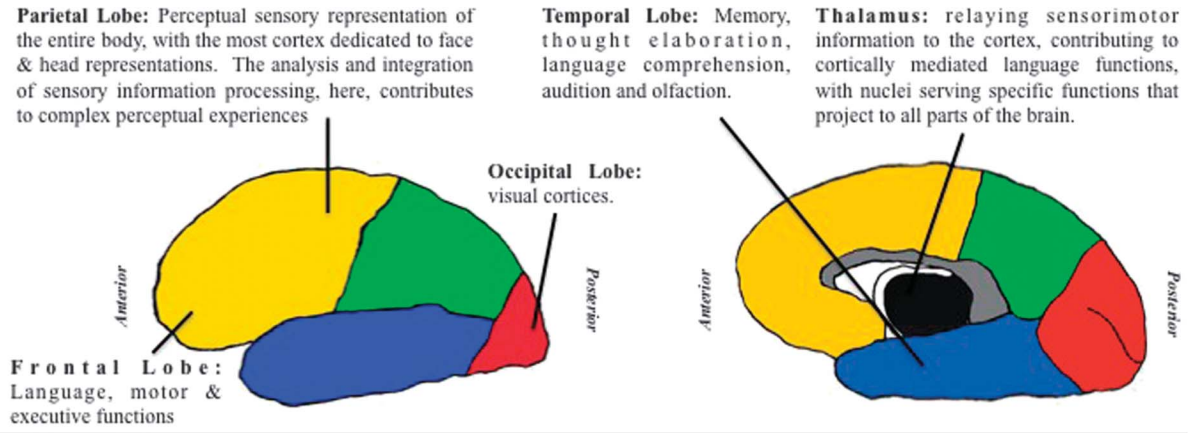
Neurotransmitters are stored in the terminal boutons, where, after activation via an action potential, neurotransmitter is release into the synapse (i.e. acetylcholine, dopamine, serotonin, glutamate, GABA and/or norepinephrine). If the neurotransmitter that is released from the presynaptic neuron comes upon a receptor of the same type on the postsynaptic neuron, the postsynaptic dendrite will be activated, possibly resulting in an action potential on the postsynaptic neuron. Original Figure adapted from (Bhatnagar, 2013).

Scaled to the level of coordinating regions of neurons, physical and functional organization is an important feature in the adaptive (e.g., plastic) relationship of cognition and neurological function. Large-scale functional networks are intimately intertwined with cognitive function, allowing for the total conscious experience to emerge through this highly organized and highly complex interaction of biochemical and electrical energy. The nervous system's physical delineations,

which facilitate its functional organization (See Figure 3), allow cognition and neurological function to be as efficient as possible. This system-level interaction provides each of us with the tools necessary to execute our daily routines, and is guided by our own motivations and purpose.

Figure 3. Divisions of the Nervous System.

A)



B)

Central Nervous System (CNS): Brain, brainstem, cerebellum, spinal cord, meninges, thalamic/sub-thalamic and limbic structures.

Peripheral Nervous System (PNS): Cranial nerves, spinal nerves, peripheral nerves, neuromuscular junction

Subdivisions:

Somatic Nervous System is under conscious control

Autonomic Nervous System (ANS) is not under conscious control. It regulates the activity of organs, such as the salivary glands, lungs, blood vessels, stomach, intestines, kidneys and bladder

Sympathetic Nervous System affects energy expenditure by activating visceral organs and glands in response to fight, flight and fright

Parasympathetic Nervous System affects energy conservation and restoration of visceral functions back to normal levels after sympathetic activations

Afferent is "bottom/up." **Efferent** is "top/down."

Corpus callosum (shown in grey): It is a fiber bundle, connecting the right and left hemispheres

Hypothalamus: Consisting of many nuclei, this communicates through neural and hormonal efferents with the brain, brainstem, and spinal cord to control the ANS by using overlapping neuronal circuitry to serve autonomic, endocrine, and regulatory functions; it controls drive and emotion, facilitates the maintenance of body temperature, blood volume, food and water intake, body mass, reproduction and the regulation of circadian rhythms

Basal Ganglia: regulates muscle tone, cognitive functional and cortical efferent processing, by slowing or inhibiting the activity of other loops and the motor cortex, thus affecting thalamo-cortical-thalamic circuits

Cognition, the psyche and the nervous system interact in order for the conscious experience to occur. It is both a top/down (efferent) and bottom/up (afferent) phenomenon, which is coevolving at any given moment. The complicated interaction of the physical and metaphysical provides the nervous system an amazing feature: adaptation. The nervous system's adaptive qualities suggest great prospect in terms of interventions and future patient outcomes, in many ways. A) The Basics of the Cortex. B) Key Concepts & Terms. Original Figure.

A neuron's physical location, physical function (Luna et al., 2001), and key biochemical and electrical features are all specialized to serve their specific purpose for an efficient use of energy, and, when operating healthily, do not require a significant level of cognitive control (Bhatnagar, 2013; Cabeza & Nyberg, 2000; Fox et al., 2005). Then, greater energy and cognitive demand is dedicated to those cognates that require more energy than others (language, executive function, cognitive integration, imagination, feelings, desires, etc.). These structural and functional divisions work separately and in conjunction with one another to activate, deactivate, or modulate one

or many different parts of the nervous system (Bressler & Wise, 2010; Fox et al., 2005; Murphy et al., 2003; Sporns, Chivalo, Kaiser, & Hilgetag, 2004).

Separating out the role of cognition in the neurocognitive relationship, cognitive processes are those that involve in coordinating biochemical and electrical activity across brain regions, hemispheres, and multiple structures (See Figure 3); yet most of this coordination is below conscious awareness. One key difference between cognition and neurological function is that cognition is highly organized, but not physically divided. Much of the interaction of cognition and neurological function can be conceptualized based on structural regional delineations; nevertheless, the root distinctions of cognitive processes span many regions of the nervous system and are not as easily divisible as their neurological counterparts (Barrett, 2009; Bhatnagar, 2013). The physical architecture may influence the metaphysical consciousness, and consciousness may affect the physical architecture (Dalley, Everitt, & Robbins, 2011; Sarter, Givens, & Bruno, 2001; Sporns et al., 2005). Thus, the relationship is bi-directional and must be perceived as such in order to truly understand both the healthy and unhealthy manifestations of this relationship.

One cognate that can illustrate several key points of neurocognitive functioning, and leave room for future discussions, is neurocognitive plasticity (Greenwood & Parasuraman, 2010; Jones et al., 2006; Mercado III, 2009). Neurocognitive plasticity refers to the ability of the cognitive and nervous systems to adapt to the introduction of new stimuli, and/or the reorganization of neurocognates already in existence (Bhatnagar, 2013). Moreover, the plastic nature of the neurocognitive relationship may provide protection against neurocognitive insults (Greenwood & Parasuraman, 2010). While the mechanisms for plasticity are not well understood, it is known that differences in brain volume across the lifespan do not correlate to intelligence, and cortical deterioration does not explain cognitive dysfunction (Bhatnagar, 2013; Greenwood & Parasuraman, 2010). The only part of the brain with neural atrophy that has been related to cognitive decline is the entorhinal cortex, an area where deterioration is among the first signs of Alzheimer's-related neuronal loss (Winchester et al., 2013).

Biophysical properties of the brain are subtle, change over time, and neuronal cell-type specific. Cognitive training can improve function even in cortical areas that have suffered serious insult, such as in dementia, ischemia, or traumatic brain injury (Acevedo & Lowenstein, 2007; Bhatnagar, 2013; Greenwood & Parasuraman, 2010; Valenzuela & Sachdev, 2009). Although some studies show linear decline in many cognitive domains from young adult to late adulthood, the amount of white matter (e.g., myelinated neurons; See Figure 1A) in the brain actually increases during that time. Furthermore, synapse loss only occurs after age 65 and is reversible (Bhatnagar, 2013). Neurocognitive plasticity can be stimulated by environmental demands and supported by biochemical, molecular, and environmental factors, which enhance brain integrity and/or promote the birth of new neurons the a process called neurogenesis (Bhatnagar, 2013; Greenwood & Parasuraman, 2010). These changes can occur over days/weeks and years/decades, resulting in larger scale neuronal network reorganization, facilitated by cognition and can promote both cognitive integrity (preserved cognitive ability) and brain integrity (preserved brain structure/function) in healthy old age (Bhatnagar, 2013; Greenwood & Parasuraman, 2010). Promoting neurocognitive plasticity involves (1) promoting novelty and exposure to environmental changes, which have been correlated with resistance to brain and cognitive aging; (2) lower carbohydrate, higher protein, and antioxidant-rich diets; (3) physical activity that promotes cardiorespiratory fitness, which increases cortical volume, increases synapses, promotes neurogenesis, clears toxic elements from the brain, improves cerebrovasculaturization, and enhances memory function. An effective approach which implements lessons from 5SysDys literature include implementation of a speech/cognitive therapy plans that recognizes the bi-directional role of cognition and neurology in overall function as describe here, combined with environmental enrichment, dietary changes, and aerobic physical activity (Ahlskog, Geda, Graff-Radford, & Petersen, 2011; Baker et al., 2010; Colcombe & Kramer, 2002; Colcombe, Kramer, McAuley, Erickson, & Scalf, 2004; Colcombe, et al., 2006; Cotman, Berchtold, & Christie, 2007; Kramer et al., 2003; Voss, Nagamatsu, Liu-Ambrose, & Kramer, 2011) can have beneficial long-term

patient outcomes. Success, here, is guided by the interdisciplinary roles of SLP, PT, RT, and OT in effective 5SDys management, particularly in the cognitive and neurological domains.

Neurological and Cognitive Control of Deglutition

Evaluating the role of neurocognitive control over deglutition begins with the brainstem (See Figure 3). Polysynaptic input from many cortical areas into corticospinal fibers modifies the reflexive swallow, depending on the characteristics of the bolus being swallowed. There is no specific cortical sidedness for deglutition control, but there is hemispheric dominance (Smithard, 2002). Deglutition involves the temporal arrangement of the oropharyngeal structures from a respiratory to a digestive pathway, the transfer of the bolus from the mouth to the esophagus, and the recuperation of the respiratory configuration (Bhatnagar, 2013). Sensory input by physiochemical properties of the bolus is required during bolus preparation to trigger and modulate deglutition (Rofes et al., 2011). Taste, pressure, temperature, nociceptive, and general somatic stimuli from the oropharynx and larynx are transported through cranial nerves to the brainstem's central pattern generator, acting as a short extension of the cortex, connecting the spinal cord to the thalamic/sub-thalamic and cortical regions (Bhatnagar, 2013). There, stimuli are integrated and organized with information from the cortex (Rofes et al., 2011), demonstrating that deglutition is a multi-regional and asymmetrical neural representation in caudal sensorimotor and lateral premotor cortex, insula, temporopolar cortex, amygdala, brainstem, and cerebellum, with widespread implications across all of the body systems involved in deglutition.

The reticular formation of the brainstem plays an important role in regulating cortically mediated functions, by modifying cortically generated functions and integrating all sensorimotor stimuli with internally generated thoughts, emotions, and cognition. This area affects the regulation of respiration and deglutition, via the pontine pneumotaxic center, and the regulation of the trigeminal nerve (CN V), facial nerve (CN VII), glossopharyngeal nerve (CN IX), vagus nerve (CN X), and hypoglossal nerve (CN XII). The pontine pneumotaxic center regulates rhythm of the medullary respiratory center, controlling the basic inspiration and expiration rhythm and the depth, with feedback coming from the level of serum carbon dioxide (Bhatnagar, 2013). The cerebellum analyzes and synthesizes sensorimortor information and provides corrective feedback to the motor cortex of the opposite hemisphere to the motor action. This is a bi-directional pathway between the neurological and motor (e.g., muscular) systems, facilitating information on pain, touch, temperature, and proprioception between the body and brain (Bhatnagar, 2013). The motor cortex, cerebellum, brainstem, cranial nerves, spinal nerves of the cervical/phrenic nerve plexus, and thoracic nerves affect the neuromuscular junctions of the face, larynx, tongue, pharynx, diaphragm, shoulder, neck, external/internal intercostal muscles, rectus abdominus, internal oblique, external oblique, and transverse abdominus (Bhatnagar, 2013). Coordinating top/down and bottom/up (See Figure 3) functioning of this system and overall perceptual awareness is central to safe deglutition.

One perspective on the effects of neurological function in dysphagia may be that it is largely a breakdown in neuromuscular output and sensory input to the CNS. However, research indicates that dysphagia in older persons is more frequently a *functional disorder* of deglutition. The neurological detection of the swallow is not necessarily a significant indicator of swallow safety; studies have shown individuals who aspirate a bolus, largely, do not have an impaired gag reflex (Bleach, 1993). Therefore, the propensity to aspirate a bolus must involve a more complicated mechanism than just the pure sensory reflex. Other evidence suggests that impaired older individuals exhibit delayed deglutition compared to healthy older individuals, leading to unsafe deglutition and aspiration in neurological patients. Impairment can be attributed to compromised sensations, decreased cortical tissue, and delays in synaptic conduction of the afferent CNS nerves. These impairments can be due to aging, neurodegenerative disorders, and/or ischemia, delirium, confusion, dementia, and the effects of medications (Clacagno et al., 2002; Lynette, Gallo, & Johnson, 2014; Rofes et al., 2011). Conclusively, one must take into account the *functional breakdown of deglutition* when making recommendations, to increase patient safety.

For example, a patient with a cough could experience reduced oxygen saturation greater than 3% and experience a change in the quality of his or her voice. The interdisciplinary team should consider this information as a clinical sign of impaired safety and piecemeal deglutition along with oropharyngeal residue as signs of impaired efficacy (Rofes et al., 2011). Oropharyngeal residue, impaired sensation, and overall reduction in nutritional status are indicative of Neurological (NeuroDys), Muscular (MuscDys), Gastrointestinal (GIDys), and Respiratory Dysphagia (RespDys). Furthermore, a breakdown in these systems may result in an overall decline in health status, which could result in the onset of cognitive impairment, resulting in Cognitive Dysphagia (CogDys).

Healthy cognitive function in deglutition involves cognitive strategies for executing neurological functions, adapting behaviors for safe deglutition under various conditions, adjusting body positions, perceiving the body as unit, and perceiving the entire eating experience as a unit. "Awareness," here, encompasses more than just being aware that the experience is taking place (Thompson & Varela, 2001). It involves being aware of one's body as a whole as well as a sum of its parts. When considering the patient's diagnostic status, clinicians should consider how the sum of the parts affects 5SDys. Effectively managing CogDys, then, includes evaluating higher-level cognates, higher-level sensory reception and perception, attention, memory, cognitive organization, problem solving/judgment, reasoning, executive function, and neuropsychiatric disturbances such as agitation, impulsivity/dis-inhibition, and apathy that may interfere with effective oral intake (Kumar, 2010; Logemann, Veis, & Colangelo, 1999). CogDys, then, can be defined as the breakdown of 5SDys, with cognitive decline preceding the breakdown of the remaining systems, or being the result of the breakdown of the remaining systems.

The Muscular, Respiratory, and Gastrointestinal Systems of Dysphagia

The Muscular System of Dysphagia

MuscDys is one of the more tangible concepts of dysphagia to comprehend and does not require a detailed explanation. MuscDys has a strong physical and mechanical component to the breakdown of the swallow; it is quantifiable and is often associated with conventional diagnoses of dysphagia in long-term care (Archem & DeVault, 2005; Cook, 2008; Rofes et al., 2011). Widespread evidence for MuscDys comes from distinct diseases of motor and muscular function. For example, all patients with degenerative diseases of the basal ganglia, cerebellum, and sub-thalamic regions experience dysphagia complications (Kumar, 2010; Rangarathnam et al., 2014). In another example, 34% of multiple sclerosis patients and 95% of patients with severe brainstem impairment experience dysphagia complications. Research noted that brainstem lesions affected the muscular control of the lips, tongue, soft palate, vocal folds, and diaphragm; potentially interfering with all stages of the swallow (Clacagno et al., 2002), a demonstration of NeuroDys, CogDys, MuscDys, and RespDys. The relationship among MuscDys, RespDys and GIDys will be elucidated in subsequent sections.

The Respiratory System of Dysphagia

Healthy respiratory function during the swallow can simply be described as the ability to hold one's breath for 1 or 2 seconds in order for the vocal cords to close, the epiglottis to retrovert to cover the trachea, and the bolus to safely pass over the protected airway and through the esophageal inlet during the pharyngeal stage of the swallow (Rofes et al., 2011; Seikel, King, & Drumright, 2010). When the process begins to break down due to trauma or disease, complications arise. According to a review of the evidence (Janssens, 2005; Janssens et al., 1999), with individuals between 25 and 75 years of age, there is a significant reduction in the compliance of the chest wall (e.g., the rib cage and upper thorax) and the diaphragm-abdomen compartment (e.g., lower thorax) for normal respiration, resulting in widespread deleterious MuscDys effects. Calcifications of the costal cartilages and chondro-sternal junctions change to the shape of the thorax, modifying chest wall mechanics. Degenerative joint disease of the dorsal spine is also a common cause of chest wall non-compliance in patients without cardiac or pulmonary disease (Seikel et al., 2010). The

coordination of the musculature and body position significantly affects the relative pressures of the respiratory tract (e.g., oral, pharyngeal, subglottal, intralobar, etc.). Age-related changes to the body can alter the relative pressures of the respiratory tract, making inspiration more difficult (Seikel et al., 2010).

The curvature of the spine and the diameter of the chest increase with age; decreasing the curvature of the diaphragm and altering its pressure/force-generating capacity for respiration, concurrent to a diaphragmatic muscular control decreasing significantly by 70 years of age. Some evidence links this reduced neuromuscular capacity to a decreased nutritional status in older persons (Janssens et al., 1999). A deficiency in these concentrations of the body's chemistry negatively impacts NeuroDys and CogDys, possibly concurrent to: (1) impaired electrolyte balance; (2) impaired pH balance; (3) reduced oxygen (O_2) utilization; (4) increased serum concentrations of carbon dioxide (CO_2); (5) a general decrease in peripheral muscle strength in older individuals; (6) a reduction in the number of cross-sectional muscle fibers and area; (7) a loss of peripheral type II motor neurons; (8) a loss of neuromuscular synapse control; and (9) in patients with CHF, a reduction in the metabolic energy storage necessary for neuromuscular control (Bhatnagar, 2013; Janssens, 2005). A breakdown in one or more of these functions can devastate 5SDys.

Respiration occurs reflexively, but can be under voluntary control to a degree. A sensor system near the carotid sinus responds to the quantity of serum O_2 and CO_2 and serum pH. When O_2 levels decline below a specific criterion level or when CO_2 or pH increase beyond a certain level, a signal mediated by glossopharyngeal nerve is relayed to the brainstem's respiratory center, thereby increasing the respiration rate. In addition, inspiration and expiration centers have a bi-directional function as excitation of the inspiration inhibits expiratory musculature, and vice versa (Bhatnagar, 2013). Taken together, there exists a clear and complex interaction of the NeuroDys, CogDys, MuscDys and RespDys.

The Gastrointestinal System of Dysphagia

Healthy gastrointestinal function in deglutition involves coordination of the digestive, muscular, and neurological systems such that movement through the alimentary canal is unidirectional, without the bolus returning to the pharyngeal cavity after having entered the esophageal cavity via the upper esophageal sphincter (UES). GIDys occurs when that gastrointestinal function moves in the opposite direction and reappears in the pharynx. This dysfunction often occurs secondary to UES impairment (Vaeizi et al., 2003). For example, in a patient on a gastrostomy tube (G-tube) source of nutrition, making the successful transition from a G-tube to oral source of nutrition requires appropriate identification of compensatory techniques that will facilitate successful unidirectional passage of the bolus through the alimentary canal (Logemann et al., 1999). Effective nutrition and neurocognitive/muscular coordination of deglutition is critical to patient safety, and each 5SDys is affected when gastric acid from the esophageal stage of the alimentary canal penetrates the pharynx and larynx. While the esophageal and gastric portions of the alimentary canal are highly adept at handling the pH of gastric acid, the pharynx and larynx are not. In fact, gastric acid can cause ulcers of the pharynx and larynx much more rapidly than the development of esophageal or gastric ulcers (Vaeizi et al., 2003).

Understanding the complexities of Gastroesophageal Reflux Disease (GERD) affecting deglutition is imperative to effective management and rehabilitation of dysphagia. There is evidence for a close causative relationship between gasteroesophageal reflux (GER) and respiratory disorders. For example, pathologic reflux has been documented in 62% of patients with severe COPD (Perng et al., 2007), and one large population-based study provides evidence that several respiratory disorders, including asthma, are linked to GER symptoms. In that study, results indicate that reflux causes respiratory complications rather than vice-versa. The study considers that bronchodialator medication might predispose GER and that half of all coughs and wheezes were associated with GER (Nordenstedt et al., 2006). One way in which the neurocognitive relationship may affect GIDys is when a breakdown in the neurocognitive relationship can cause UES dysfunction, and GERD can cause a breakdown in any of 5SDys.

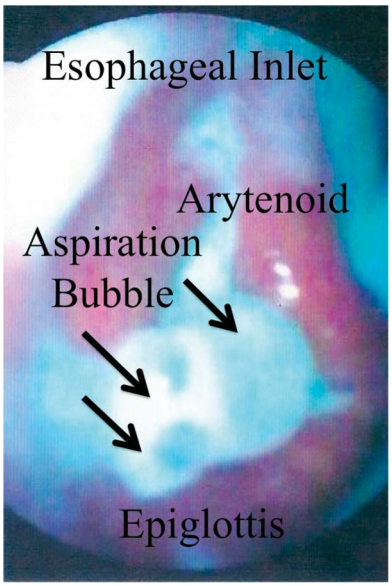
The Interdisciplinary Practitioner's Role in Effectively Managing the Five Systems of Dysphagia With an Emphasis on Cognitive Dysphagia

Repeat hospital admissions correlate with CogDys (O'Malley, Caudry, & Grabowski, 2011), are frequent, costly, preventable, and deleterious (Lanspa et al., 2013). Among all hospitalized Medicare beneficiaries, nearly 20–30% are readmitted within 1–3 months of admission, have multiple medical co-morbidities, greater length of hospital stay, and recent hospitalizations. Medicare beneficiaries are likely to be discharged to a skilled nursing facility and soon be rehospitalized with a 15–40% chance of mortality within 1 year (Lum, Studenski, Degenholtz, & Hardy, 2012). The time between hospitalizations decreases markedly as the number of previous hospitalizations increases, and the shortest time between hospitalizations occurs between the first and second hospitalizations. Thereafter, the time between hospitalizations decreases as successive hospitalizations increase (O'Malley et al., 2011).

Undiagnosed or underdiagnosed dysphagia is a major preventable contributor to repeat hospital admissions. Unfortunately, treatment for dysphagia varies greatly (Loeb, Becker, Easy, & Walker-Dilks, 2003), which is possibly related to the inconsistency of hospital discharge communications when a dysphagia patient is transferred to long-term care. For example, almost half of all patient discharge summaries omitted all of the dysphagia recommendations made within the SLP notes, and only 13% of discharged patients had summaries that included all of the SLP's dysphagia management recommendations (Kind et al., 2011). Shockingly, 60% of general food recommendations and 22% of liquid consistency recommendations other than "thin" were omitted from hospital discharge communications to long-term care (Kind et al., 2011).

Studies note a dramatic increase in safety and a reduction in penetration and/or aspiration when necessary dietary modifications concentrate patient caloric and protein intake with lower volumes of food that are consumed with greater safety (Rofes et al., 2011). An effective dysphagia management program is cost-effective and correlates with a reduction in aspiration pneumonia rates and complications (Cook & Kahrilas, 1999). NeuroDys patients who have a reduction in bolus volume and enhanced bolus viscosity significantly improved, particularly in regards to penetration and aspiration (See Figure 4). Water and thin liquids increase the prevalence of penetration and aspirations risks for these patients. Therefore, risk is incrementally reduced when an increase in the viscosity of a fluid occurs, beginning with a nectar consistency (Rofes et al., 2011).

Figure 4. Aspiration Shown via Fiberoptic Endoscopy.



A decreased neurocognitive capacity for safe deglutition (NeuroDys, CogDys) may result in impaired sensation, perception and coordination of the pharyngeal and laryngeal structures (MuscDys), resulting in penetration and/or aspiration (RespDys). Posterior pharyngeal wall (Top) and anterior near epiglottis (Bottom) noted. Arytenoid and epiglottis indicated. Image provided by Dysphagia Management Systems LLC.

Effectively managing dysphagia is a complicated task, necessitating evaluation of each of 5SDys. If an interdisciplinary health care team does not address all 5SDys, then deglutition safety has not been thoroughly addressed. NeuroDys patients suffer from complicated interactions across 5SDys, often with side effects from medications necessary to treat the impairment, injury, or disease (Kumar, 2010; Lynette et al., 2014). For example, if a patient is receiving therapy and has a diagnosis of silent aspiration, RespDys and GIDys, the therapy team will identify important indicators of overall patient risk, such as apparent wheezing during effortful or effortless physical activity, an asymmetry of facial movement, or muscle weakness that appears or increases with the onset of dehydration. A key concept, here, is that what happens during therapy will not only predict safety and overall success, but what occurs during OT and PT could counteract the positive outcome of the SLP's therapeutic interventions; as the bidirectional nature of these systems is known.

To manage CogDys, the interdisciplinary team must focus on the area of the cortex affected by neurocognitive decline, and the plasticity of the unaffected cortical region to undergo neurocognitive compensation (Smithard, 2002). Then, the team should consider whether or not a patient is capable of realizing what the risks of dysphagia mean to his or her safety, the patient's ability to learn or relearn safe deglutition compensatory techniques, and the patient's ability to make safe judgments regarding his or her own safety. The team may also take into account the effect of medications on 5SDys, particularly CogDys, because consistently reviewing which medications the patient is taking is an important aspect of understanding the patient's cognitive and neurological capacity and future potential for safe deglutition (Falsetti et al., 2009).

The role of the MD in effectively managing CogDys includes establishing patient capacity to respond to cues and facial expressions in a functional manner that is indicative of the patient's comprehension, and educating the clinical staff in recognizing the patient's neurocognitive capacity. At times, patients may report an incident to NSG as if the incident occurred in the recent

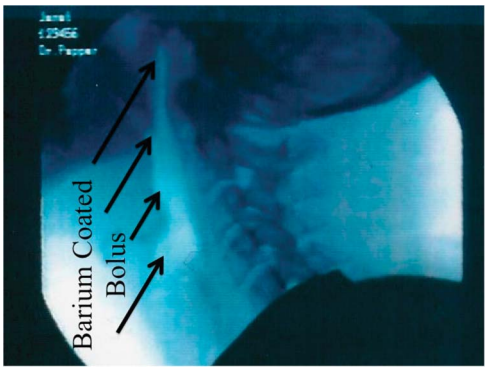
past, yet the incident possibly occurred a long time ago or not at all. The interdisciplinary team needs continued education in order to establish if a patient is expressing himself or herself and the situation accurately. The difficulty for OT and PT is that as the patient's CogDys continues, that patient's ability to use compensatory strategies and techniques may be significantly impaired. Additionally, the patient may not always have a clear understanding of therapeutic goals, interventions and/or outcomes, but may respond with seemingly normal answers. The SLP should work with OT and PT by providing a thorough cognitive evaluation and further consultation regarding the patient's ability to safely undergo and maintain therapeutic interventions.

When effectively managing MuscDys, the interdisciplinary team should ask themselves a series of questions: (1) Will the patient be able to perform the compensatory techniques repeatedly? (2) Will the patient's eventual fatigue increase his or her risk of aspiration? (3) Will the patient suffer from muscle aches and pains as a result of therapeutic exercises or techniques? (4) Will currently prescribed medications further exacerbate muscle dysfunction or increase fatigue? Asking these questions and testing the recommended compensatory strategies on one's self before making therapeutic recommending will elucidate tolerance capacity for repetitive exercises or if fatigue/ soreness could occur as a result of these recommendations. Furthermore, exploring other possible therapeutic techniques (e.g., massaging, brushing, icing, and thermal stimulation) may be helpful in rehabilitating muscular function of the swallow, independently or when combined.

The MD's role in MuscDys is to evaluate muscle fatigue, a lack of coordination of 5SDys, and weakness that can affect safe deglutition. Facial symmetry and tongue movement can be useful indicators, but sometimes the delicate balance of the oral mechanism motion remains elusive. The SLP expertly examines the patient and provides a detailed evaluation to the MD, demonstrating the importance of appropriate SLP referrals. The SLP and NSG must work together to establish the patient's ability to manage his or her MuscDys, and OT/PT should consider how the fatigue of the leg muscle affects the fatigue of the muscles of the pectoral girdle, mouth, and throat. Here, the key is to assume that fatigue is universal throughout the body.

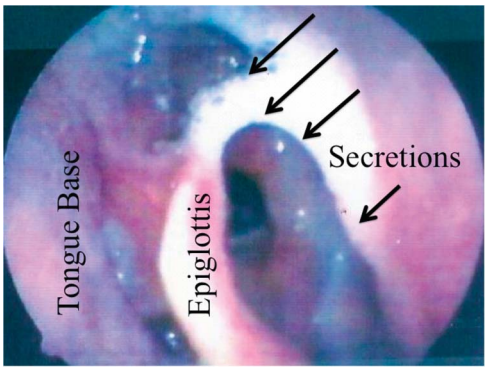
When considering the RespDys, it is important to understand the purpose of respiratory treatments on deglutition and their effects: thinning out secretions, so they may be easily expelled from the lungs. For this to happen, the patient must swallow the secretions or spit them out. Evaluating the presence of RespDys and medication-filled secretions can be facilitated by the use of instrumentation, specifically by use of the fiberoptic endoscopic evaluation of swallowing (FEES). As experienced SLPs know, FEES provides the ability to view secretions when compared to other forms of instrumentation, such as the Modified Barium Swallow (MBS; See Figure 5). For example, if a patient has a breathing treatment closely before his or her meal, residue can mix with a thickened liquid and ultimately decrease the viscosity of that thickened liquid to an unsafe consistency. The bubbling or frothy foam of the secretions can accumulate in the valleculae and pyriforms, affecting deglutition. Transit of the bolus may be affected, resulting in penetration into the laryngeal vestibule (See Figure 6), a dysphagia safety risk easily viewable via endoscopy. The evidence, here, suggests that instrumentation plays an important role in identifying 5SDys and improving the chances for effective Dysphagia Management.

Figure 5. Oral and Esophageal Stages of Deglutition Shown via MBS.



Barium coated bolus indicated. Image provided by Dysphagia Management Systems LLC.

Figure 6. Secretions in Larynx Shown via Fiberoptic Endoscopy.



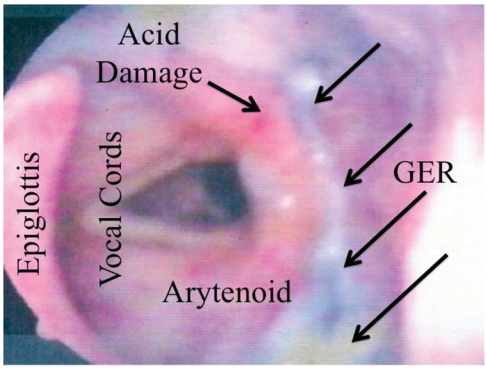
Penetration of secretions and apparent reflux from the UES only visible with FEES instrumentation (NeuroDys, CogDys, MuscDys, GIDys). Posterior pharyngeal wall (Right) and anterior region near tongue base (Left) and epiglottis indicated. Image provided by Dysphagia Management Systems LLC.

Each member of the interdisciplinary team serves an important role in the treatment of RespDys. The MD can lead the team in mitigating risk by making expert judgments. The NSG staff is involved in one of the most misunderstood risks for RespDys: medication presentation. While there are universally applied basic models of medication presentation, for our purposes, medication presentation may need to be more individualized to the patient's specific conditions. The SLP can help the MD educate the importance of medication consistency modifications for a patient who has neurological decline or loss of sensation, resulting in silent aspiration issues. The OT/PT team must consider the physical demands and flow of the patient's day in order to effectively manage RespDys to ensure that the plan of care is appropriate.

It is well known that instrumental methods for detection of GIDys are used to predict which patients who have GER and respiratory issues will respond to diet changes and anti-reflux treatment. Experienced SLPs also know that GER is difficult to detect via MBS; however, several indicators of GER are observable via FEES, and these indicators may be useful for establishing effective dysphagia management (See Figures 5 & 7, respectively). When GIDys is effectively managed, patients experience profoundly fewer re-hospitalizations for respiratory complications, less pulmonary treatment, less morbidity and mortality, less need for pulmonary medications, and less illness associated with corticosteroid therapy (Sontag, 2005). Again, effective instrumentation

and a comprehensive dysphagia evaluation may be the key to increasing patient safety and improving patient quality of life.

Figure 7. Gastroesophageal Reflux (GER) Shown via Fiberoptic Endoscopy.



Penetration of laryngeal vestibule, posteriorly, is visible (5SDys). Arytenoids, Epiglottis and Vocal Cords indicated. Image provided by Dysphagia Management Systems LLC.

The literature concludes that effective diagnosis and management of each of the five systems of dysphagia necessitates a multidisciplinary approach spanning the MD, NSG, SLP, PT, and OT departments (Rofes et al., 2011). Unfortunately, common bedside evaluations can vary widely in diagnostic accuracy, and may not achieve the desired goal. For example, the Burke's 3 oz. water swallow test does not identify 20% of aspiration in patients, and the Standard Bedside Swallow Assessment has a sensitivity of 47–68% and a specificity of only 67–87% (Rofes et al., 2011). These common bedside evaluations also involve continuous deglutition of large amounts of liquids, which may place the patient at a high risk for aspiration during the assessment. Cough, oropharyngeal residue, a fall in oxygen saturation of more than 3%, and changes in quality of voice are considered clinical signs of impaired safety and piecemeal deglutition (De Pippo, Holas, & Reding, 1994; Irwin, 2006; Rofes et al., 2011). If it is a firmly held conviction among practitioners and an expectation among patients that an accurate determination of diagnosis and prognosis is an important medical goal and that functional abnormalities of deglutition should be accurately defined (Cook & Kahrilas, 1999), then the clinical bedside evaluation in the absence of using a diagnostic tool such as FEES may not result in success of that medical goal. Research shows that the clinical bedside evaluation, alone, is not sufficient for preventing aspiration pneumonia and is not cost-effective in diagnosing dysphagia (Wilson & Howe, 2012). However, instrumentation predicts hospital readmission (Rofes et al., 2011); therefore, an effective Dysphagia Management plan should include appropriate instrumentation and coordination of the interdisciplinary team in order to evaluate all 5SDys and reduce the patient's future risk of aspiration, repeat hospitalization, and mortality. When a clinician considers 5SDys, identifying the dysfunction of each and then assessing what, if any of the systems can be rehabilitated or managed, the path to safety becomes clear.

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