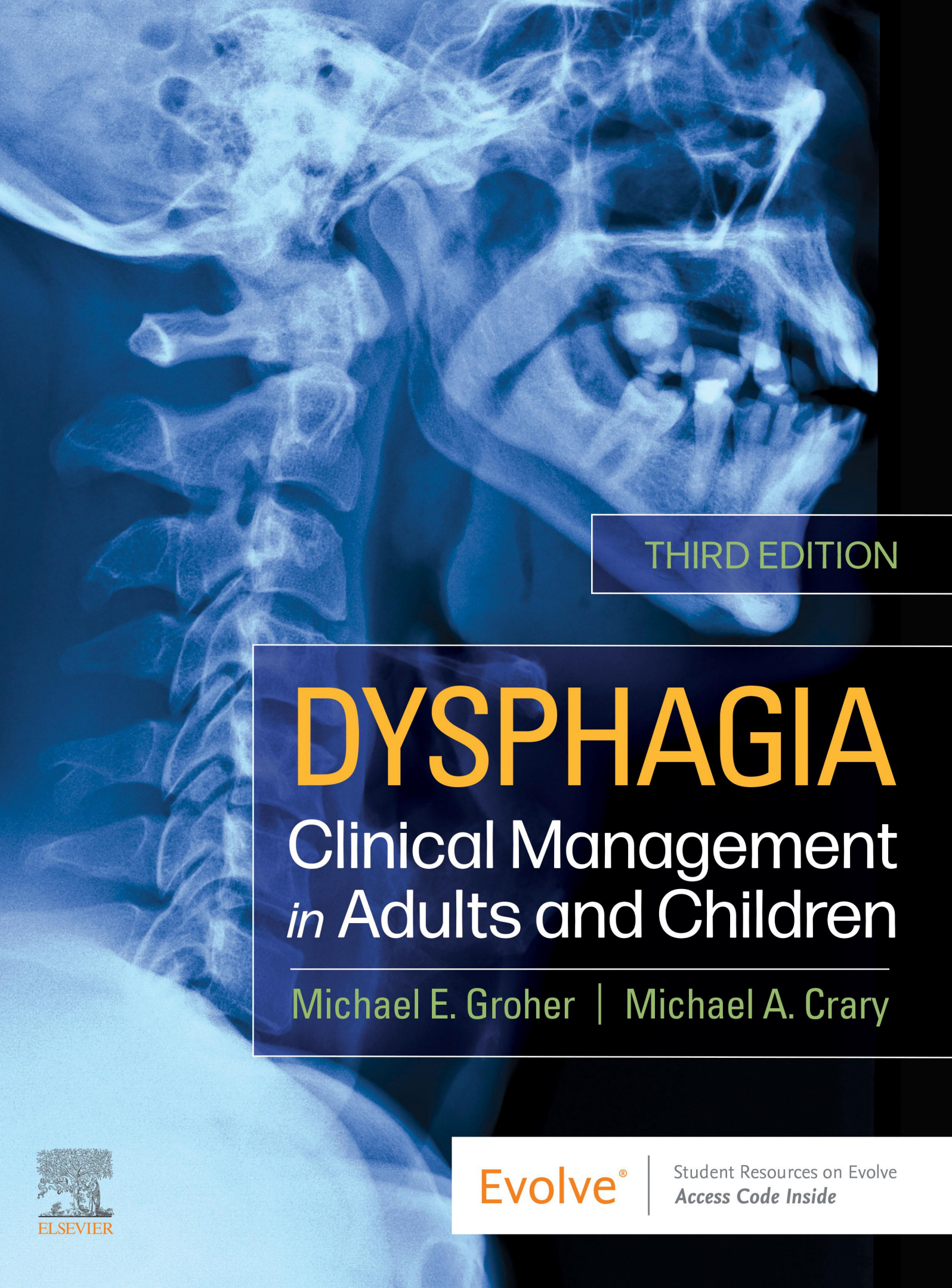

THIRD EDITION

DYSPHAGIA

Clinical Management *in* Adults and Children

Michael E. Groher | Michael A. Crary

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DYSPHAGIA

Clinical Management *in* Adults and Children

This book is dedicated to the many individuals from around the world who have influenced our own work, many of whom were pioneers in the development and support of the multidisciplinary concept of dysphagia management. The clinical and translational research that each has provided has built the foundation for a subspecialty that undoubtedly will continue to grow and benefit the patients we treat. In prior editions, we have listed them individually, but the list has grown beyond the space allotted. You know who you are!!

Clinical science can be a slow science. It requires not only dedication to time and effort, but also true commitment to the patients who will eventually benefit from these efforts. Clinical science is not without setbacks, pitfalls, and flaws. So to those who engage in clinical science in the name of helping others, we attempt to remember the words of two prominent world citizens, and we apologize if we convey their words incorrectly:

First they ignore you, then they laugh at you,
then they argue with you, then you win.
Mahatma Gandhi

It is common sense to take a method and try it.
If it fails, admit it frankly and try another.
But above all, try something.
Franklin D. Roosevelt

With best wishes to all,
M. G. & M. C.

DYSPHAGIA

Clinical Management *in* Adults and Children

Third Edition

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Preface

Welcome to the third edition of *Dysphagia: Clinical Management in Adults and Children*! As in any new field of endeavor, information accumulates rapidly and subsequently changes our perspectives. This is a good thing in the clinical sciences; we continue to add to our understanding of a problem, in this case, swallowing disorders in adults and children. First and foremost, we have updated each chapter to reflect new understandings. Access to imaging materials and critical thinking cases on Evolve has improved. Critical thinking cases that have proven so valuable in classroom discussions remain, and new ones to highlight different aspects of patient management have been added. In some instances, we have added new questions on preexisting cases.

Our focus continues to be for the clinician who wants to establish a basic and comprehensive foundation in managing infants, children, and adults with swallowing disorders. The emphasis is on the processes of providing diagnostic and treatment services for persons with dysphagia, and on the research that supports those services. Because of the comprehensive approach, some details of diagnosis and treatment will not be fully appreciated after the first reading by novice clinicians, but will be useful for journeyman clinicians. It is our opinion that the organization of this text will be an aid to the professor who is providing instruction in dysphagia management at a basic and advanced level. Aids in teaching include access to an extensive library of swallowing examinations (on the companion Evolve website); a liberal use of short, clinically based examples of a myriad of problems associated with dysphagia; critical thinking case examples (Clinical Corner boxes in the chapters); and cases that require students to analyze their own decision-making skills as they integrate historical, clinical, and imaging results using a series of prompts that probe their problem-solving skills (on the Evolve website). Unfamiliar terms have been highlighted in bold and defined in an accompanying glossary. In addition, we have tried to infuse our own biases and insights with anecdotal stories (Practice Notes in the chapters) given to us by the hundreds of patients we have treated. Amplification of these experiences is now included as ***Clinical Pearls*** that are now embedded within each chapter. These short anecdotes hopefully will provide clinicians with a better appreciation of day-to-day practices that impact patient care.

The Table of Contents has been revised. We have added an additional chapter (3), Aging and Dysphagia. The focus is on those elderly persons living in the community who may not have a typical underlying medical condition that contributes to their dysphagia. We have separated the previous Chapter 7 (Respiratory and Iatrogenic Disorders) into two separate chapters for ease of reading and teaching. Added in what is now Chapter 7 (Respiratory) is a new section detailing the etiology and consequences of aspiration pneumonia. The previous Chapter 9 (Treatment Considerations, Options, and Decisions) has been condensed into a single chapter on adult treatment, now Chapter 11. Because of the many advances in treatment approaches, this chapter has undergone extensive revision, with the addition of many key references documenting these changes.

It is our opinion that dysphagia management is best taught by illustrating approaches to problem solving. To this end, we have tried to avoid being prescriptive in favor of an emphasis on discovering available options for care and in weighing the risks and benefits of those options. Too often, prescriptive approaches in clinical care take away one's options to solve patient care problems.

The successful management of persons with dysphagia is accomplished only through the cooperation of numerous specialists (see Chapter 1). Although it is well known that a multidisciplinary approach with these patients is best, this approach also may suffer from failure to coordinate care. Often the coordination of that care is accomplished by the speech-language pathologist. In this text, we have emphasized the role of the speech-language pathologist. The roles of other disciplines are explained largely in the clinical case presentations within each chapter.

Ultimately, this is a text that highlights the problems of persons with dysphagia and how professionals might ameliorate their swallowing difficulties. It will become apparent that swallowing difficulty may be secondary to a large number of medical and sometimes nonmedical (psychogenic) disorders, and that swallowing problems are more than a physiologic change in the swallowing mechanism. The text takes the perspective that being unable to swallow normally might result in major consequences to one's medical and psychological health. Secondary medical problems such as aspiration pneumonia, undernutrition, and dehydration may predispose the patient to other complications such as

immunocompromise, mental confusion, or death. Because of this, dysphagia specialists must develop a strong background of general medical knowledge. The reader should be able to understand or be alerted to key medical concepts relating to the dysphagic circumstance within each chapter, but may have to go beyond this text for more detailed explanations of some concepts.

Being unable to ingest one's favorite foods safely, or being unable to eat normally in public, understandably will affect one's quality of life, with the potential for secondary episodes of depression, anxiety, and social withdrawal. Preparation of special diets is time consuming and in some cases, economically challenging. In short, our lifestyles frequently revolve around mealtimes. Interruptions to these normal routines are potentially devastating. Therefore treatments are geared not only to the restoration of physiologic function, but ultimately to a state of psychosocial normalcy that was disturbed as a result of a failure to swallow normally. Care of persons with dysphagia should be viewed as an attempt to rehabilitate lost function and prevent future medical complications by retaining learned rehabilitative strategies.

Managing persons with dysphagia has become a subspecialty for many healthcare professionals. For the speech-language pathologist, it is a specialty that has emerged only within the last 40 years. As clinicians have become more familiar with the issues involved in the care of dysphagic persons, clinical and basic science investigators have helped answer and ask questions that have improved the quality of that care. Many of these efforts have come together in a journal *(Dysphagia)* devoted exclusively to dysphagia; a research society that meets annually (the Dysphagia Research Society); and the largest special interest division, number 13 (Dysphagia), within the American Speech-Language-Hearing Association. There also has been a steady increase of texts with contributions from many disciplines aimed at the pathologic condition, diagnosis, and treatment of persons with swallowing disorders. It is our hope that this text will not only add to that number, but also inspire those researchers and clinicians interested in dysphagia to continue the quest to improve the lives of persons with swallowing disorders.

Contents

Part I
Foundations

CHAPTER 1
Dysphagia Unplugged

Michael E. Groher

To view additional case videos and content, please visit the evolve website.

CHAPTER OUTLINE

OBJECTIVES

1. Define *dysphagia* and its ramifications.
2. Discuss the epidemiology of dysphagia.
3. Discuss the medical and social consequences of dysphagia.
4. Provide an overview of the clinical management of dysphagia.
5. Discuss the role of persons who manage dysphagia.
6. Discuss the types of settings in which dysphagic patients might be seen and how this might affect their management.

WHAT IS DYSPHAGIA?

Dysphagia takes its name from the Greek root phagein, meaning to ingest or engulf. Combined with the prefix dys-, it connotes a disorder of or difficulty with swallowing. It is correctly pronounced with a long or short *a*. The final syllable, "ja," requires a hard pronunciation rather than the soft "dja" to avoid confusion with the communicative language disorder, *dysphasia* (see Practice Note 1-1).

Taber's Cyclopedic Medical Dictionary[1] defines five subcategories of dysphagia:

1. Constricta: narrowing of the pharynx or esophagus
2. Lusoria: esophageal compression by the right subclavian artery
3. Oropharyngeal: difficulty with propulsion from the mouth to the esophagus
4. Paralytica: paralysis of muscles of mouth, pharynx, or esophagus

PRACTICE NOTE 1-1

While acting as a consultant to a food production company, I asked them what they thought the extent of their market would be, indicating that to my knowledge we only had gross estimates of how many persons with dysphagia would benefit from specialized foods. They told me that they had been working with a firm that did an extensive analysis on this topic and had prepared a detailed report on the potential market. I asked them to send me a copy because I was interested in data that documented the **incidence** of dysphagia in the United States. Two weeks later, I received a package with a copy of the data. To my surprise, there were at least 15 pages of references. On closer inspection of the first page, I noticed that the firm they had hired had used the key word *dysphasia,* not *dysphagia*. I broke the news to them that what they had paid for was an extensive review of the literature on language disorders after neurologic injury, not swallowing disorders. What a difference a single letter can make!

5. Spastica: dysphagia from spasm of the pharynx or esophagus

In clinical practice, only *oropharyngeal dysphagia* from this list is used with any frequency. Interestingly, medical students learn that dysphagia is a swallowing problem primarily associated with disease of the esophagus. However, when used properly the term should refer to a swallowing disorder that involves any one of the three stages of swallowing: oral, pharyngeal, or esophageal. Some might extend the term to the stomach or lower gastrointestinal tract as primary disorders in these structures such as the stomach may secondarily affect other parts of the gastrointestinal tract such as the esophagus. It is not a primary medical diagnosis but rather a **symptom** of underlying disease and therefore is described most often by its clinical characteristics (**signs**). Complaints such as coughing and choking during or after a meal, food sticking, **regurgitation**, **odynophagia**, drooling, unexplained weight loss, and nutritional deficiencies all may be associated with dysphagia. Because dysphagia is a symptom of underlying disease that is not necessarily specific to the swallowing tract, it can be associated with varied diagnoses. These diagnoses are summarized in Box 1-1. Throughout this text, most of these diagnoses will receive individualized attention. See Chapter 9 for a full discussion of symptoms and signs associated with dysphagia.

Dictionary-based definitions of dysphagia imply that it is the result of a physiologic change in the muscles needed for swallowing. Physiologic change often leads to the two hallmarks of dysphagia: delay in the propulsion of a **bolus** as it transits from the mouth to the stomach or misdirection of a bolus. Misdirection can be defined as bolus material entering the upper airway or lungs, or material that enters the mouth, pharynx, or esophagus during swallowing attempts but fails to reach the stomach. In these circumstances, classification of dysphagia by either clinical or imaging examination seems warranted and straightforward. However, not all patients with physiologic abnormalities of the swallowing mechanism show obvious delay in bolus flow or misdirection of bolus flow. The question that may arise for the clinician (and often for the researcher who has selected a cohort of patients with dysphagia) is the degree of severity of physiologic changes in the swallowing musculature needed before a patient is classified as having dysphagia. For instance, physiologic changes in the swallowing musculature have been described in older persons[2]—such as reduction in tongue strength or esophageal motility—both of which may delay the delivery of food or liquid to the stomach.

BOX 1-1 SUMMARY OF CONDITIONS THAT MAY CONTRIBUTE TO DYSPHAGIA

Neurologic Diagnoses
- Stroke
- Traumatic brain injury
- Dementia
- Motor neuron disease
- **Myasthenia gravis**
- Cerebral palsy
- Guillain-Barré syndrome
- Poliomyelitis
- Infectious disorders
- **Myopathy**

Progressive Disease
- Parkinsonism
- Huntington's disease
- Progressive supranuclear palsy
- Wilson's disease
- Age-related changes

Connective Tissue/Rheumatoid Disorders
- Poly- and **dermatomyositis**
- Progressive systemic sclerosis
- Sjögren's disease
- **Scleroderma**
- Overlap syndromes

Structural Diagnoses
- Any tumor involving the alimentary tract

Iatrogenic Diagnoses
- **Radiation therapy**
- **Chemotherapy**
- **Intubation** or **tracheostomy**
- Postsurgical cervical spine fusion
- Postsurgical coronary artery bypass grafting
- Medication-related

Other or Related Diagnoses
- Severe respiratory compromise
- Psychogenic condition(s)

However, only when such changes result in perceptible changes in eating habits or associated medical complications such as **undernutrition** or **aspiration pneumonia** is a person classified as truly having dysphagia.

Clinical Pearl: When researchers describe a group of patients who are dysphagic, it may not be clear how they defined dysphagia or how they classified the severity of their subjects. Readers should be aware of this issue when interpreting the results of their study.

Because swallowing is a dynamic process, persons may not exhibit signs and symptoms of dysphagia with every swallow and every bolus type. In these cases, they may be considered to be at risk for dysphagia or, alternatively, operationally defined as dysphagic. It is also possible that the swallowing musculature is normal but the patient is not alert enough to use that musculature because of his or her decompensated medical condition. In such cases, it is assumed that attempts to swallow would result in dysphagic complications. In these cases, the patient may be classified as at risk for dysphagia. Patients may demonstrate abnormalities of behavior that interfere with the normal swallowing process; these may cause dysphagic signs and symptoms or put the patient at risk for dysphagia. Therefore, dysphagia is defined not only by abnormalities of the mechanics of the swallowing musculature, but also by the consequences of failure, or potential failure, of that musculature owing to factors not always specifically related to swallow mechanics. For this reason, the authors prefer the definition of dysphagia offered by Tanner[3]: "Dysphagia: [an] impairment of emotional, cognitive, sensory, and/or motor acts involved with transferring a substance from the mouth to stomach, resulting in failure to maintain **hydration** and nutrition, and posing a risk of choking and aspiration" (p.16). A swallowing disorder should be distinguished from a feeding disorder. A *feeding disorder* is impairment in the process of food transport outside the alimentary system. A feeding disorder usually is the result of weakness or incoordination in the hand or arm used to move the food from the plate to the mouth. In the United Kingdom and the United States, a feeding disorder, particularly in the context of infants and children, may be the same as a swallowing disorder. Persons with feeding disorders (motor transfer problems) may also be dysphagic, such as those with cerebral palsy whose neurologic disability affects both feeding (motoric transfer) and swallowing. It is not known whether a feeding disorder that might require assistance with food transport also affects the subsequent act of swallowing, perhaps by interfering with timing of swallowing events.

A swallowing disorder is also to be distinguished from an eating disorder such as **anorexia** or **bulimia** nervosa. Whereas patients with dysphagia, bulimia, and anorexia may have difficulty with poor appetite, changes in dietary selections, and problems with the oral preparation of the bolus, patients with bulimia and anorexia rarely have demonstrable changes in or complaints of swallowing difficulty.[4]

INCIDENCE AND PREVALENCE

The incidence of a disorder is the reported frequency of new occurrences of that disorder over a long time (usually at least 1 year) in relation to the population in which it occurs. The prevalence of a disorder is the number of cases in a population during a shorter, prescribed period, usually in a specific setting.

Clinical Pearl: Exact measures of the incidence and prevalence of swallowing disorders in large and various populations are impossible because of differences in accepted definitions of dysphagia, the setting in which it is measured (acute, rehabilitation, chronic), and differences in the measurement tools across studies to detect it.[5]

For instance, asking a patient if she or he has a swallowing disorder to determine the prevalence is a very different method of detection compared with the use of an imaging examination such as **videofluoroscopy**. Most demographic data that are reported relating to swallowing disorders are prevalence data. The importance of knowing the prevalence of a disorder can help guide clinicians in the detection of that disorder and therefore helps plan how resources might be devoted to that disorder. For instance, if an examiner knew that a certain abnormality was found in less than 1% of that population, the examiner may not spend time looking for that abnormality because its expected frequency of occurrence would be low. If, however, a particular abnormality was found in more than 50% of the persons with a particular disorder, the examiner would be alerted to expect the occurrence of deficits associated with that disorder. Therefore, if the data suggested that 50% of patients who have had an acute stroke could have dysphagia, and that 20% of that group might have **silent aspiration**, an examiner would expect that half of the patients with acute stroke would have swallowing impairment and about half of those are at high risk for silent aspiration. Furthermore, pneumonia develops in 37% of acute stroke patients with aspiration.[6] Knowledge of these prevalence data provides valuable assistance to medical personnel who initially screen for and manage the medical complications after acute stroke (see Chapter 4 and 9).

The American Speech-Language-Hearing Association (ASHA) estimates that 6 to 10 million Americans show some degree of dysphagia, although it is not known how these estimates were made.[7] Kuhlemeier[8] reported that the incidence of reported dysphagia in the state of Maryland rose from 3 in 1000 in 1979 to 10 in 1000, probably as a result of better reporting methods. Using these estimates, approximately 25,000 persons in Maryland in 1989 had dysphagia as either a primary or secondary diagnosis.

CLINICAL CASE EXAMPLE 1-1

The hospital's chief of staff was reviewing a request from the dysphagia team to hire an additional speech pathologist and dietitian to screen and treat patients on the hospital's new stroke and acute geriatric units. Part of the rationale for the request was based on recent published guidelines from the Centers for Medicare & Medicaid Services that screening for dysphagia on a stroke unit was prudent because of evidence that early detection may prevent associated **morbidity** and **mortality,** both of which would increase costs for the healthcare system and, by implication, the hospital. Furthermore, prevalence data from five studies were submitted indicating that at least half of the patients on the stroke unit and a similar number on the acute geriatric unit may have dysphagia. The financial officer estimated that early detection and treatment of dysphagia would result in a cost savings that far exceeded the cost of the two new employees who would be assigned to those units. After integrating the request from the dysphagia team, the evidence from the literature on prevalence, and the potential cost savings to the medical center, the chief of staff approved the request.

Prevalence by Setting

Estimates of prevalence of dysphagia vary by setting because certain age groups (older adults and premature newborns) and diagnoses (neurogenic) are more likely to demonstrate dysphagia. For instance, patients entering a **rehabilitation setting** may not have as many accompanying medical problems and dysphagia as those entering a nursing home. Conversely, infants born prematurely may have many medical problems that may secondarily result in dysphagia (see Chapter 14). In a survey of the entire population of an acute general hospital, fewer patients with dysphagia would be found in the general population compared with a survey of a special section of that hospital, such as the stroke unit.

Community

Estimates of the prevalence of dysphagia among older persons living in the community range from 16% to 22%.[9,10] One study reported on the prevalence of dysphagia in a younger cohort (14- to 30-year-olds) living in the community who had been referred for complaints of dysphagia.[11] In this selected group, 70% had demonstrable pathologic conditions that accompanied their symptoms. A systematic review of 15 studies that met criterion for review estimated that when combined, the prevalence of dysphagia in community-dwelling elderly was 15%.[12]

Acute and Chronic Geriatric Care

Of the 211 patients admitted to an acute geriatric unit in Singapore, the prevalence of dysphagia was 29% on admission and 28% at discharge.[13] In a nursing home in Maryland (chronic care), as many as 60% of residents had a combination of swallowing and feeding difficulty.[14] A similar number (53%) was found in a chronic care facility in Spain, two urban nursing homes in South Korea, and in eight nursing homes in Portugal.[15-17] One study found that when feeding and swallowing difficulty were combined, as many as 87% of the residents in a home for the aged were at risk for inadequate oral intake.[18] Follow-up data of nursing home residents with oropharyngeal dysphagia indicate a mortality rate of 45% at 1 year.[19]

Acute General Hospitals

Using the Fleming Index of Dysphagia, a tool to identify dysphagia, Layne et al.[20] found that nearly one third of their patients had a diagnosis consistent with dysphagia. These findings were nearly 18% higher than those provided by Groher and Bukatman,[21] who reported a 13% prevalence rate in similar settings. The discrepancy in prevalence was explained by the fact that patients who were dehydrated in the study by Layne et al. were classified as dysphagic, whereas this was not a marker for dysphagia used in the collection of the Groher and Bukatman data.

Acute Rehabilitation Unit

Of 307 consecutive admissions to an acute rehabilitation facility, one third of patients were dysphagic.[22] Of this group, half had dysphagia as a result of a stroke, followed by traumatic brain injury (20%), spinal cord injury and brain tumor (7%), and progressive neurologic disease (5%). On admission, the patients with the most severe dysphagia were those with traumatic brain injury, followed by stroke. The least severe dysphagia occurred in those with brain tumors.

Special Populations

Some primary medical diagnoses are more likely to precipitate dysphagic symptomatology, such as diseases that affect the central and peripheral nervous systems and disorders affecting the structures of the alimentary tract, such as cancer. An estimated 300,000 to 600,000 persons in the United States each year are affected by dysphagia from neurologic disorders alone; most cases occur after a stroke.[5] If these data are reliable, dysphagia is a common symptom after a stroke.

Stroke

Prevalence reports of dysphagia after stroke depend on when in the course of recovery the detection of a swallowing impairment was made. For instance, in acute stroke

(less than 5 days after onset) the prevalence of dysphagia may be as high as 50%, whereas 2 weeks after stroke only 10% to 28% of patients may be dysphagic. Recognizing these discrepancies, Smithard et al.[23] provided follow-up of 121 (untreated) acute stroke patients for 6 months using a clinical dysphagia examination and videofluoroscopy to detect swallowing deficits. Immediately after stroke, 51% were believed to be at risk for aspiration. After 7 days, only 27% were still considered to be at risk. At 6 months, 3% of the survivors had persistent difficulty, whereas 3% who previously were not dysphagic were now considered at risk. These results suggest that early detection is important in preventing dysphagic complications and that a significant number of patients will improve without intervention specific to their dysphagia. Similarly, comparable prevalence figures for dysphagia on admission (43% to 51%) were found by Gordon et al.[24] and Mann et al.,[25] although the latter group noted a higher prevalence of dysphagic symptoms at 6 months (50%) than other studies with prevalence rates that ranged from 3% to 9%.[23,25] Daniels et al.[26] found that 36 (65%) of 55 patients with acute stroke had dysphagia. Of these 36, more than half aspirated. Of these, two thirds did so silently, suggesting that events of aspiration could be detected only by videofluoroscopy, not the bedside examination. In a long-term follow-up, 94% of these patients returned to oral intake. Interestingly, the presence or absence of silent aspiration did not discriminate between patients who returned to successful oral feeding. After analyzing prevalence reports from two large stroke databases, Gonzalez-Fernandez et al.[27] found a significantly higher prevalence of dysphagia in Asians when compared with whites and blacks (see Clinical Corner 1-1).

CLINICAL CORNER 1-1 SEVERE DYSPHAGIA

L. G. was admitted to the hospital with a left brain stroke. On admission, he was nonresponsive and a nasogastric feeding tube was placed to provide nutrition and hydration. As his responsiveness improved, the nasogastric feeding tube was removed and he began oral feeding. As he fed himself, it was noted that he choked on most attempts and dysphagia was suspected. The clinical evaluation noted a weak tongue and poor laryngeal elevation. The imaging examination showed signs of tracheal aspiration. The diagnosis of dysphagia secondary to stroke was confirmed.

CRITICAL THINKING

1. Why might a **nasogastric tube** be placed on admission?
2. Should the nasogastric tube have been removed? Why do you think it was removed?
3. Should it be replaced, or what might the next step in care be?

Head and Neck Cancer

Surprisingly, there have been no large studies of the prevalence or incidence of swallowing disorders in unselected patients after treatment for head and neck cancer, although it is well known that dysphagia is a frequent complication. Dysphagia can result from the removal of tissue, with subsequent sensory and motor loss, and the effects of radiation therapy and chemotherapy. Before patients in their study received treatment, Pauloski et al.[28] found that 59% had symptoms consistent with dysphagia. In a large multicenter treatment trial of patients with laryngectomies who were treated with either surgery and radiation or radiation and chemotherapy, approximately 33% had some type of swallowing-related difficulty at 2-year follow-up.[29] Using a questionnaire, Maclean et al.[30] noted that 71% of their 197-person sample reported some difficulty with their swallowing. In a series of 46 patients treated by **supraglottic laryngectomy**, 60% had dysphagia after their hospital stay.[31] In 21 patients following supraglottic laryngectomy using a transoral carbon dioxide laser approach, most experienced dysphagia with aspiration after 2 weeks, but it significantly decreased at 12-month follow-up.[32] In a mixed group of 87 patients with head and neck cancer who were at least 1 year posttreatment, oropharyngeal dysphagia was present in 50.6%, mostly to solids.[33] Fifty-one percent of patients reported a decrease in their quality of life because of their swallowing disability. Evidence suggests that patients with pharyngeal tumor **resections** and those with tumors involving the tongue base are more likely to have dysphagia.[34]

Head Injury

Dysphagia is common after severe head injury. Data report that the incidence of dysphagia ranges from 4.5% (9 of 199) of consecutive admissions in an **acute care setting**[35] to an incidence of 78% (31 of 40) in a similar setting.[36] Discrepancies in reporting may be attributable to the initial severity of the injury and the method used to detect and define dysphagia. In 11 patients with severe brain injury and coma, Bremare and colleagues documented the prevalence of dysphagia after arousal using physical and endoscopic examinations. Seventy-seven percent had oral stage dysfunction, 66% with pharyngeal stage impairment, and 80% with airway protection abnormality.[37] Incidence data are available for patients who survive head injury and enter a rehabilitation setting; the incidence ranges from 27% to 30%[35,38] to 42% (218 of 524).[39] In a mixed group (type of injury and time after onset), Lazarus and Logemann[40] found that approximately half of the patients they examined with videofluoroscopy showed evidence of dysphagia. Among patients with head injuries entering a rehabilitation setting, Winstein[38] found that 27% were dysphagic on admission to rehabilitation and that only 6% were dysphagic after 5 months of rehabilitation. Of 62 consecutive patients

receiving outpatient rehabilitation, Yorkston et al.[36] reported that 13% remained dysphagic. In general, the more severe the initial injury, the higher the incidence of dysphagia. In a retrospective review of 219 patients admitted for head injury who were suspected of dysphagia, logistic regression revealed that those who were older, tracheotomized, and aphonic were more likely to enter the next level of care with a feeding tube than those who did not evidence these findings.[41] Some patients remain comatose and are unable to eat, whereas others require extensive neurosurgical procedures with prolonged intubation and mental status changes, all of which may preclude attempts at oral ingestion. However, once patients enter the rehabilitation setting, their chances of returning to oral feeding are good.

Progressive Neurologic Disease

Progressive neurologic diseases that frequently result in dysphagia include **Parkinson's disease** and its variants, **amyotrophic lateral sclerosis** (ALS), **multiple sclerosis** (MS), and myasthenia gravis; diseases of systemic rheumatic origin such as dermatomyositis, polymyositis, **rheumatoid arthritis** (RA), scleroderma, and **Sjögren's syndrome**; and variants of dementing syndromes such as Alzheimer's disease and frontotemporal disease. Systemic rheumatic disorders are far rarer than Parkinson's disease or MS but merit consideration in a discussion of dysphagia and neurologic disease. Because of the progressive nature of these disease processes, the point in disease progression at which dysphagic symptoms occur is never certain. For instance, some patients report dysphagia as the initial symptom of the disease, whereas others may never mention dysphagia. In general, however, as disease severity increases, so does dysphagia. Complications from dysphagia, particularly those that threaten pulmonary function, may lead to aspiration pneumonia and death (see Chapters 7 and 12 for a discussion of aspiration pneumonia).

Parkinson's Disease

Although dysphagia secondary to Parkinson's disease appears to be common, accurate measurements are restricted by subject selection bias and dysphagia detection methods. However, most authors agree that dysphagia occurs in at least 50% of patients with Parkinson's disease.[42-44] Pflug and colleagues studied 119 consecutive patients with early-stage Parkinson's disease using fiberoptic endoscopy. Only 5% were without some change in swallow performance that included abnormal airway protective mechanisms, increased residue, or leakage of contents into the pharynx. Most all in this group were unaware that they had any swallowing abnormality.[45] In 72 patients with Parkinson's disease of varying severity, Leopold and Kagel[46] found that as many as 82% reported swallowing difficulty. Using the Unified Parkinson's Disease Rating Scale, a scale that acquires data by self-report, Walker et al.[47] found that 32% of their patient sample complained of dysphagia. In patients with early-stage disease, Sung et al.[48] found manometric abnormalities on both liquid and more viscous bolus types with disruptions of esophageal motility during repetitive swallowing tasks. Interestingly, the esophageal abnormalities were present even before overt manifestations of dysphagia were present. That patients with Parkinson's disease may not be accurate reporters of dysphagic symptoms is well known. Kalf et al.[49] performed a meta-analysis using 12 studies to establish the prevalence of dysphagia associated with parkinsonism. One third of the patients sampled complained of dysphagia, whereas more than 80% had objective demonstrations of its presence. The prevalence of dysphagia may be higher in patients with Parkinson's disease who also have significant dementia.[50]

Amyotrophic Lateral Sclerosis

When ALS affects the **bulbar musculature**, dysphagia may be one of the first symptoms of the disease. In studies of patients with ALS at first diagnosis, 25% to 30% have evidence of bulbar symptomatology.[51,52] It can be assumed that at least one third of patients with a diagnosis of ALS will have some difficulty swallowing, particularly as the disease progresses.[53] Known characteristics of disease progression that affect the bulbar musculature result in progressively severe dysphagia symptomatology.[54]

Multiple Sclerosis

Hartelius and Svensson[55] found that more than 33% of a large series of patients with MS had either chewing or swallowing problems. Dysphagic complaints in patients receiving follow-up care in an outpatient clinic ranged between 30% and 40%.[56] Similar to those with ALS, not all patients with MS will have dysphagia unless the bulbar musculature is involved, and symptoms are more likely to appear as the disease progresses. After evaluating 143 consecutive patients with primary and secondary progressive MS, Calcagno et al.[57] confirmed dysphagic symptoms in 34%. Their study showed a positive relation between dysphagia and disease severity and between dysphagia and brainstem involvement. After surveying 309 patients with MS, DePauw et al.[58] found that 24% had chronic swallowing difficulty and another 5% admitted to transitory difficulty. As patients became more disabled according to a scale of disability measurement, the prevalence of dysphagia increased to 65%

Myasthenia Gravis

In selected populations of patients with myasthenia gravis, approximately one third will be dysphagic.[59] The prevalence of dysphagia depends largely on the extent of muscle fatigue and other medical complications such as respiratory

impairment secondary to an acute exacerbation of muscle weakness.

Clinical Pearl: Prescribed medications often can compensate for extreme muscle fatigue in patients with this diagnosis.

Muscular Dystrophy

There are no published reports of the prevalence of dysphagia in muscular dystrophy, although there are reports of swallowing dysfunction secondary to peripheral oropharyngeal and esophageal muscle weakness in those with oculopharyngeal, Duchenne, and myotonic muscular dystrophies.[60-62]

Polymyositis and Dermatomyositis

Oh et al.[63] documented the prevalence of dysphagia in those inflammatory diseases affecting muscle. Of the 783 patients studied, 62 were dysphagic. Oropharyngeal dysphagia was present in 18 with dermatomyositis, and 9 with **polymyositis**. As with other progressive neurologic conditions, with these disorders the course and response to medical therapy may differ; therefore the presence of dysphagia is variable. Because of their predilection to involve the **proximal muscle**, swallowing can be affected in these disorders. Multiple disorders of pharyngeal function following videofluoroscopic swallowing studies were noted in a small group of patients with polymyositis (6), dermatomysitis (4), and **inclusion body myositis**.[64]

Rheumatoid Arthritis

Geterude et al.[65] found that 8 of 29 patients with RA had complaints of dysphagia. In a series of 31 patients with dysphagia and RA, Ekberg et al.[66] documented pharyngeal dysfunction in 20.

Scleroderma

As many as 90% of patients with scleroderma have swallowing-related complaints.[67] Accompanying **erosive esophagitis** was found in 60% of 53 patients with scleroderma.[68] In these patients, dysphagia was always an accompanying complaint. In patients with scleroderma, dysphagic complaints are usually confined to the esophagus, although secondary effects on the oral and pharyngeal stages resulting from esophageal dysmotility should be considered.

Sjögren's Syndrome

As many as 75% of patients with Sjögren's syndrome have dysphagia.[69] As the severity of the disease increased, 64% in a sample of 101 self-reported swallowing disorders with an accompanying reduction in the quality of their life.[70] The potential of this syndrome to involve all stages of swallowing function is well known, especially in overlap syndromes such as with scleroderma.

CLINICAL CORNER 1-2 MEDICATION RISK

M. M. was admitted to the burn unit with severe burns to the head, neck, and upper torso. Because of associated pain he was heavily sedated. As his condition improved and before he was allowed to eat orally, a request for a swallowing evaluation was made because it was noticed he was not swallowing his secretions well. The evaluation of swallowing revealed normal strength of the swallowing musculature; however, he was disoriented and could not maintain his alertness level for more than 30 seconds. Because of his poor mental status and alertness level, he was not allowed to eat and was considered to be at risk for dysphagia.

CRITICAL THINKING

1. How might medications contribute to dysphagia?
2. Could poor mental status result in choking? Give some examples.
3. What cognitive functions might contribute to normal mental status for swallowing safety?

Dementia

Alagiakrishnan et al.[71] did a **systematic review** of the prevalence of dysphagia in dementia. Nineteen studies met the review criteria. Prevalence ranged from 13% to 57%, developing in the later stages of those with frontotemporal dementia and in earlier stages in those with Alzheimer's disease (see Clinical Corner 1-2).

Developmental Disability

Leslie et al.[72] discussed the need to document the true prevalence of dysphagia in those with developmental disorders to highlight the need for appropriate intervention. They could find only estimates of prevalence ranging from 36% in the community to 73% who were inpatients. After studying those patients referred for dysphagia evaluations, Chadwick and Jolliffe[73] concluded that the prevalence of those with dysphagia and concomitant mental or physical disability was 8.1%. Observations of adults with Down syndrome living in a residential facility who were eating a regular diet revealed that 56.5% were at risk for respiratory infection based on overt signs of cough during the meal.[74] Smith et al. found a similar prevalence in a younger group of hospitalized patients with Down syndrome (mean age 7.45 years). Those with significant neurologic delay or tracheostomy were more likely to be at risk for dysphagia.[75] Using videofluoroscopy as a diagnostic tool, Jackson and colleagues studied a cohort of 138 patients admitted to a teaching hospital with Down syndrome between the ages of 31 days and 18 years. Their findings showed that 65% had oral stage disability, and 56% with pharyngeal abnormality including aspiration. Of particular interest was of the 61% who aspirated, 9 of 10 did so silently.[76]

Mental Illness

Few prevalence data have been recorded on patients with mental illness who may show signs of dysphagia. Noting this omission, Aldridge and Taylor[77] completed a systematic review in an attempt to document prevalence and treatment interventions. Ten studies met the inclusion criteria documenting those with dysphagia or those who expired from choking asphyxiation. Adults with mental illness in one study were 43 times more likely to die from organic mental illness compared with the general population. Six studies revealed a range of prevalence of dysphagia from 9% to 42%. Kulkarni and colleagues noted that the side-effects of psychotropic medications produced Parkinson-like symptoms including dystonia and tardive dyskinesia. These side effects interfere with oral stage preparation that secondarily may affect pharyngeal stage function.[78] In acute and community mental health settings, the prevalence of dysphagia was 35% of those admitted, 27% in the day hospital, and 31% in long-term care.[79] None of the studies provided data on treatment intervention or outcomes (see Clinical Corner 1-3).

Phagophobia

Phagophobia, or the fear of swallowing, may be associated with psychogenic etiologic factors such as panic disorders, posttraumatic stress disorder, social phobia, or obsessive-compulsive disorders. Those with phagophobia usually describe their problem as the sensation that they are unable to swallow in the absence of any documented sensory or motor abnormality. Baijens et al.[80] reviewed 12 published studies that attempted to establish the prevalence and treatment of the disorder. Most had serious methodologic flaws with low levels of evidence that made it too difficult to establish reliable prevalence statistics.

CLINICAL CORNER 1-3 PSYCHIATRIC DIAGNOSIS

L. T. was admitted to the psychiatry unit with symptoms of acute schizophrenia. When eating, it was noted he would take excessive time to finish, with intermittent choking episodes. The speech pathologist who evaluated him for signs and symptoms of dysphagia found that the oropharyngeal swallowing musculature was intact. As she watched the patient eat, she noted a rapid feeding rate with inappropriate bite sizes. She also noted excessive talking while eating, and the choking episodes occurred during these talking periods. The patient was classified as dysphagic as a result of emotional and behavioral abnormalities.

CRITICAL THINKING

1. What other types of behavioral disorders might contribute to dysphagia?
2. Why did this patient choke while eating and talking?

Premature Infants

The incidence of infants born prematurely in the United States has increased to more than 12% of all live births and 18% of African-American births.[81] A growing concern has been the incidence of emotional and neurodevelopmental disabilities in the very low birth weight population (less than 26 weeks' gestation). Estimates indicate that as many as 90% of low-birth-weight infants may be prone to disorders of feeding.[82]

Spinal Cord Injury

In a study that evaluated the use of clinical versus imaging studies in adults with tetraplegia, Shem et al.[83] reported that 38% of the 39 patients who were enrolled had evidence of oropharyngeal dysphagia. Four subjects were diagnosed with aspiration.

Clinical Pearl: Because of positioning they may be liable to esophageal motility disorders as well as mental status changes if their injury involved the cortex in addition to the spinal cord.

CONSEQUENCES OF DYSPHAGIA

Because dysphagia frequently accompanies many medical diagnoses, it is important to appreciate its potential effect on patient care. It is well recognized that dysphagia is a symptom of disease, but it also has the potential to secondarily precipitate morbidity and mortality. As such, its influence on health can be substantial. Additionally, it can affect the patient's overall quality of life.

Medical Consequences

A potential complication of patients with oropharyngeal dysphagia is aspiration pneumonia. The treatment of aspiration pneumonia is costly, and it is associated with increased length of stay in the hospital,[84] greater disability at 3 and 6 months,[84,85] and poorer nutritional status during hospitalization.[84] One study[84] found an increased mortality risk in stroke patients for whom swallowing was considered unsafe at 6 months' follow-up, whereas another study did not find this relation at 3 months.[85] Dehydration is a frequent adjunct in those with dysphagia after stroke.[85,86] Dehydration can lead to increased mental confusion and generalized organ system failure, both of which lead to greater decompensation of swallowing.[87] Dysphagia may lead to undernutrition, which adversely affects energy levels (ability to sustain a swallow), and if severe or chronic, compromises the immune system. Compromise to the immune system potentially delays healing and increases susceptibility to infection, sepsis, and death.[87]

Psychosocial Consequences

Oral ingestion of food and liquid is a pleasurable activity for most people. Social interactions often revolve around sharing a meal. "Let's have lunch, are you free for dinner, or can we meet for an early breakfast?" Having a piece of wedding cake, being offered an hors d'oeuvre at a party, enjoying a midnight snack, and going to one's favorite restaurant are all examples of common situations that require the ability to swallow. Swallowing difficulty therefore may limit the extent to which a person might socialize, leading to major changes in a normal lifestyle (see Practice Note 1-2). Fear of overt choking episodes and the associated discomfort might contribute to social isolation and accompanying depression. Spouses and family members are equally affected because of the potential social limitations dysphagia may precipitate. Even making subtle changes in dietary preferences to compensate for dysphagia may lead to feelings of discontent. Eating may no longer be pleasurable. It becomes an activity performed only for nourishment. The need for special preparations at mealtime provides additional stress. Special dietary supplements may be costly, often posing financial burdens.

Clinical Pearl: An assessment of family burden should be an important part component of treating dysphagic patients living at home.

CLINICAL CASE EXAMPLE 1-2

A request for services was sent to the speech pathologist to evaluate a 70-year-old man for suspected dysphagia. He had lived in the nursing home for 2 years after a left brain stroke that left him with **aphasia** and poor mobility. He spent most of his day sitting in a wheelchair or in bed watching TV and was beginning to show evidence of **decubitus ulcers** on his coccyx. The nurses reported he was showing increased disinterest in his soft mechanical diet and was choking at most meals on his liquids. He rarely finished a meal. A review of his medical record revealed a consultation from the dietitian who noted that his **albumin** was 3.0 g/dL, he had lost 5% of his body weight in the past 2 weeks, and he was **hypernatremic**. Based on these parameters, the dietitian concluded that the patient was undernourished and dehydrated and wondered if his previous history of dysphagia was contributory. The patient was examined in bed. He was able to follow one-step commands and name simple objects but was not oriented to time or place. During the examination, the patient fell asleep every minute and the speech pathologist had to continually awaken him to maintain his attention and cooperation. An examination of his oral peripheral speech mechanism revealed a mild right facial weakness but otherwise was normal. Test swallows with various food items were delayed but without overt coughing. Tests with liquids revealed numerous choking episodes. Based on his physical examination and the results of his laboratory tests, it was concluded that his swallow may improve if he were properly hydrated and nourished, and that it was unlikely that hydration and nourishment could be accomplished by mouth because his alertness level was poor. Furthermore, his nutritional and hydration requirements would have to be elevated because of fluid loss from the decubitus ulcers. It also was likely that his ulcers would not heal unless his protein stores were improved. For this reason, a **nasogastric tube** was recommended with regular reevaluation of his laboratory values and mental status to make recommendations for possible return to oral feeding. It was hypothesized that because he had been eating normally before this acute change, the dysphagia was most consistent with a change in metabolic status and not related to a change in his neurologic presentation.

PRACTICE NOTE 1-2

I first met George at the New York Hospital in the outpatient clinic. He obviously was a man of means, as he told stories of extensive travel. His swallowing evaluation that day revealed it was not safe for him to eat orally because of a specific muscle weakness, and a **gastrostomy** tube was recommended. He was noticeably upset by this recommendation. Because he was only 35 years old, we suspected that this might put an end to his life as a world traveler; however, George was not convinced. After his gastrostomy was placed, to my surprise he told me he had made arrangements for a 3-week trip to Spain and Portugal. He had arranged to ship cases of formula for his tube to each hotel on his travel itinerary before his departure. When he arrived in Spain, his formula was waiting. Normally he would have dined on bouillabaisse and fresh fish with a fine Chablis. Instead, he self-administered six cans of a liquid formula per day into his gastrostomy tube and continued to enjoy the ambience of Europe. He was determined not to let his severe pharyngeal dysphagia interfere with other aspects of his life.

Clinical Management

The care of patients in whom dysphagia is suspected usually begins with a basic process of identification in an attempt to answer the question of whether dysphagia is present. This process can be the result of a simple screening, such as watching a patient eat or drink small amounts of food. Such a screening might be done after a patient has had an acute neurologic event such as a stroke. Some patients begin to eat without screening because the risk factors for dysphagia are not present. An example might be a patient who has not had any swallowing difficulty in the past, but required a feeding tube immediately after an

operation for medical purposes and who has been cleared by the physician to return to oral ingestion. As the patient returns to eating, either the medical staff or the patient notices swallowing difficulty. Outpatients may report to their general practitioner that they are having swallowing difficulty. In all these situations, a clinical evaluation of swallowing will be initiated.

Clinical Examination

The clinical evaluation should include a thorough review of the medical and psychosocial history (see Chapter 9).

Clinical Pearl: This part of the examination often is too cursory with important information not well integrated; its importance to diagnosis underestimated.

This is followed by a physical evaluation that includes a screening of mental status, an evaluation of the musculature of the head and neck, and, if appropriate, trial swallows of liquid, semisolid, and solid materials. If the clinical examination fails to adequately explain the patient's symptoms or requires more in-depth visualization of any phase of the swallowing sequence, an imaging study may be necessary. The clinical indicators for the use of imaging assessment techniques have been published by ASHA.[88]

Imaging Examination

Imaging the **aerodigestive tract** most commonly is done by barium x-ray studies, direct visualization, and measurement of pressures within the aerodigestive tract during swallowing attempts. The most common x-ray technique that assesses the oral, pharyngeal, and cervical esophageal phases of swallowing is the modified barium swallow (videofluoroscopy). ASHA provides a statement of guidelines for speech-language pathologists (SLPs) who perform this procedure.[89] A standard barium swallow (**esophagram**) may be used to evaluate the esophagus. Direct visualization of the pharyngeal, laryngeal, and esophageal compartments is done by **endoscopy**. Guidelines for the performance and interpretation of the endoscopic evaluation of swallowing by SLPs are provided by ASHA.[90] Patient preparation and positioning for each of these studies vary according to focus of the anatomic region being examined. Pressure measurements during swallowing (**manometry**) are more routinely done for clinical purposes in the esophagus than in the mouth or pharynx, although there has been an increased use in the pharynx and pharygoesophageal segment to better understand their physiology. A full discussion of these and other instrumental techniques used in the evaluation of swallowing is provided in Chapter 10.

Treatment Options

Ideally, the clinical and imaging evaluations will lead to a treatment plan.

Clinical Pearl: There is evidence that even after combining these two modalities, there may be disagreement among clinicians about what treatment should be implemented.

The goal of most treatment plans is to ensure that the patient can consume enough food and liquid to remain nourished and hydrated and that the consumption of these materials does not pose a threat to airway safety, resulting in aspiration pneumonia. If treatment is indicated, four main areas are considered: behavioral, dietary, medical, and surgical. These options may be applied as compensatory, rehabilitative, or preventive interventions (see Chapter 11)

Behavioral interventions include engaging the patient in some change in swallowing behavior. Changes may take the form of simple compensations, such as a change in posture or eating rate; in rehabilitative strategies, such as teaching a patient a new way to swallow; or in strengthening muscles. Dietary interventions might include modifications of texture, taste, or volume. Medical interventions may include a change in medication negatively affecting mental status and swallow or the placement of a nasogastric feeding tube. Surgical interventions might include mobilization of a weak vocal fold or the placement of a gastrostomy tube. Combinations of these options are common; however, the timing of each intervention is patient dependent. A full discussion of treatment planning, including options and details of rationale and use, is presented in Chapters 11 and 16.

WHO MANAGES DYSPHAGIA?

Patients who have disruptions in swallowing potentially involve many members of the medical community. Those whose dysphagia is related to the head and neck may see an otolaryngologist, dentist, SLP, or neurologist. To further define the disorder, these specialists often need the services of a radiologist. Those whose swallowing disorder may be of esophageal origin may require the services of a gastroenterologist. If the swallowing disorder is related to an acute respiratory condition, a patient may be under the care of a pulmonologist, pulmonary physical therapist, and respiratory therapist. If the swallowing disorder is related more to the process of feeding, an occupational therapist is frequently involved. If the swallowing disorder results in compromise to the nutritional system, a dietitian is consulted. While the patient is in the hospital, the nurse frequently is involved in the identification and treatment of the patient's swallowing disorder. In short, patients with swallowing disorders require the attention of many specialists who must work in

concert to achieve swallowing safety and nutritional stability. The prominence of individual roles at any given time depends on the patient presentation.

Ideally, healthcare professionals who are concerned about the patient's swallowing safety and nutritional adequacy will work together toward the mutual goal of improving the patient's swallowing performance. Coordination of effort is important if timely results are to be achieved. Some medical centers have designated swallowing teams and swallowing team leaders. In many hospitals, an SLP assumes the role of swallowing team leader. The role each specialist plays on the team varies across settings. For instance, some gastroenterologists diagnose and treat swallowing problems that involve the esophagus, but disorders of the esophagus are not their special interest. Specific interest in the swallowing-impaired patient also varies. For instance, few radiologists have a specific interest in patients who report dysphagia. The result of this variance in interest and focus is that not all swallowing disorder teams are the same, and in some cases not all potential members are represented.

Speech-Language Pathologist

SLPs have taken a leading role in the management of patients with dysphagia related to poor oral and pharyngeal swallowing mechanics. In most centers, they coordinate the swallowing team and are frequently the first professionals to perform a history and physical examination that is specific to oropharyngeal dysphagia. Based on these data, they consult other members of the dysphagia team, obtain approval from the patient's attending physician for any additional testing or referrals, and integrate the rehabilitative components of the dysphagia treatment program. Only within the past 20 years have specific practice guidelines for managing dysphagia by SLPs been developed. These include an outline of the knowledge and skills needed to treat oropharyngeal dysphagia and the need to understand the esophageal components of swallowing to make appropriate medical referrals.[91]

SLPs were evaluating and treating articulation disorders of children with cerebral palsy as early as the 1940s. Because of the decompensation of the oromotor system in children with cerebral palsy, both speech and swallowing were affected; however, treatments specific to swallowing were not a routine part of care by the SLP. Working in a medical setting studying patients with Parkinson's disease in the late 1960s, Dr. Jeri Logemann found that videofluoroscopy was ideally suited to study patients' speech and swallowing skills. Soon this technique was used to study the effects of cancer in the head and neck on swallowing performance, and in 1976 at the American Speech and Hearing Association National Convention, she presented one of the first papers by an SLP on the diagnosis and treatment of swallowing disorders after surgical procedures for cancer in the head and neck. That the paper was accepted at the convention was a monumental achievement because there was no recognized category for a paper on swallowing, and evaluating and treating patients with swallowing disorders was not within the accepted scope of practice for an SLP. This radical departure from the traditional role of the SLP raised more than a few eyebrows (see Practice Note 1-3). As Logemann was beginning her distinguished career in dysphagia management, Dr. George Larsen, also working in a medical setting with adults, began to develop treatments specific to patients with neurogenic swallowing disorders. Because so many of his patients with speech and language disorders had accompanying swallowing dysfunction, he began to search the literature for relevant treatment approaches. He discovered a literature full of descriptions of how a person swallows but no mention of how to treat the impairment. Using his background in neurology and physiology, he began to develop treatment approaches and reported them in the literature. He wrote about appropriate postures[92] and the need for some patients to bring the swallowing sequence under volitional control.[93] He was convinced that the most successful approaches would result from a team effort, and he described the use of trained feeding volunteers as part of the process.[92] The momentum to evaluate and treat swallowing disorders in children and adults grew throughout the 1980s. The momentum was sustained by the publication of two texts by SLPs summarizing empirical evidence supporting the role of the SLP and emphasizing the need for collaboration among various medical professionals.[94,95] Both texts have undergone revisions. Today, SLPs have assumed a leadership role in providing care to children and adults with oropharyngeal dysphagia. SLPs are at the forefront of providing the research and educational components that support their clinical efforts. Miller

PRACTICE NOTE 1-3

I well remember the reaction of ASHA in the 1970s and early 1980s to the acceptance of the role of the SLP in managing patients with dysphagia. It was the "new guard" versus the traditionalists. Letters to the editor flew back and forth, most arguing that this area of practice was potentially life threatening and SLPs did not have the medical background necessary to be competent. Treating patients with dysphagia labeled one as borderline heretic with threats of a breach of ethics. Today, patients with dysphagia dominate the caseloads of SLPs working in medical settings, and children with dysphagia are being managed in the public school setting. And both ASHA and the medical community have embraced the role of the SLP in these efforts.

CLINICAL CORNER 1-4 ELECTRICAL STIMULATION

Dr. Miller and I followed Dr. Larsen to a patient with occult hydrocephalus who could not initiate a swallow. Results of examination of his oral peripheral mechanism were normal, and Dr. Larsen suggested that we needed to stimulate laryngeal elevation. The following day we watched in disbelief as Dr. Larsen approached the patient with a probe tip wrapped in gauze, dipped in saline solution, and attached to a primitive facial nerve stimulator. As he applied the electric current to the thyroid notch, a swallow was initiated and the patient continued to swallow without the assistance of the stimulation. Our collective elation that "treatment" could be so easy was quickly dampened when Dr. Larsen warned it could be dangerous to use such a technique with every patient because it could trigger **laryngospasm** and death. We learned two things that day: not all treatments are for every patient and some treatments carry accompanying risk.

CRITICAL THINKING

1. Why might an electrical current facilitate swallowing?
2. Name other types of medical treatments that carry risk.
3. Find a paper that summarizes the effects of electrical stimulation on swallowing.

and Groher[96] have described a more detailed history of the involvement of the SLP in the management of swallowing disorders (see Clinical Corner 1-4).

Otolaryngologist

The otolaryngologist is skilled in the evaluation of the upper digestive tract. In particular, the use of endoscopy by otolaryngologists for direct visualization of the structures of the nasopharynx, oropharynx, pharynx, and larynx adds information relative to the structural, sensory, and motor aspects of the pharyngeal stage of swallowing. In patients with head and neck cancer who require surgery, otolaryngologists provide surgical and postsurgical management. In this regard, they must be sensitive not only to issues of cancer control, but also to the preservation of speech and swallowing functions. The otolaryngologist may be involved with the surgical placement and removal of a patient's **tracheostomy tube**. Because these tubes may interfere with normal swallowing, these specialists work with the dysphagia team to remove the tubes as soon as medically feasible.

Gastroenterologist

The gastroenterologist who participates on the swallowing disorders team usually has a special interest in the esophagus. Because primary esophageal disorders that precipitate dysphagia can have secondary effects on the pharyngeal and oral stages of swallowing, it is important to include the gastroenterologist in the evaluation of the patient who may appear to only have symptoms that relate to the oral or pharyngeal stages of swallowing (see Chapter 6). The gastroenterologist is familiar with the management of **gastroesophageal reflux disease (GERD)**, or heartburn, a symptom that may be related to dysphagia. The gastroenterologist may use special sensors that measure the amount of acid content in the alimentary tract using a test called **24-hour pH monitoring**. The gastroenterologist may use manometry, or combined impedance and manometrics, to measure esophageal motility and prescribe medications to improve esophageal motility or to control GERD. The use of esophageal endoscopy to make visual observations of the esophageal mucosa to rule out a stricture or cancer is a role of the gastroenterologist. The gastroenterologist is responsible for the nonsurgical placement of a feeding tube in the stomach called a **percutaneous endoscopic gastrostomy** tube.

Radiologist

The radiologist who may be a regular member of the swallowing disorders team often has a special interest in the gastrointestinal tract. Radiologists provide both dynamic (videofluorographic) and static (plain films) imaging of the aerodigestive tract and lung fields. Often these studies provide the diagnostic information that guides swallowing treatment. Special tests such as computed tomography performed after static images of the aerodigestive tract are done by a radiologist. The SLP frequently works in conjunction with the radiologist in performing the modified barium swallow (see Chapter 10). The interpretation of the modified barium swallow study is often done concurrently by the SLP and the radiologist.

Clinical Pearl: Not all radiologists have extensive familiarity with the modified barium swallow study and often rely on the SLP for guidance in procedure and interpretation.

Neurologist

Because the majority of patients with oropharyngeal dysphagia have swallowing impairment as a result of neurologic disease, the neurologist has an important role in the identification and subsequent management of swallowing problems. It is critical that patients with symptoms of dysphagia without a known cause be considered for evaluation by the neurologist. Some neurologic diseases that precipitate dysphagia can be treated with medication. Finding a cause is also important in providing the patient with an

explanation for the dysphagia and in providing a prognosis for future complications.

Dentist

Patients with dysphagic symptoms may be identified first by the dentist during routine dental care. Of particular interest to the dentist are any oral-stage manifestations of swallowing disorders, such as problems with chewing, bolus formation, or dental disorders such as **osteoradionecrosis** that would make swallowing painful. The dental **prosthodontist** is skilled at making appliances for the oral cavity that can facilitate swallowing in patients who have had oral structures removed because of cancer. In Japan, the dentist is often the team leader in the care of patients with dysphagia. Dental hygienists may play a role by providing oral care that limits the presence of oral pathogen formation. If colonized, such pathogens when aspirated may precipitate pneumonia and secondary lung infection.

Nurse

The nurse has 24-hour responsibility for monitoring the patient's swallowing problem. Monitoring the amount of intake and recording it in the medical record is an important role for the nurse. Not only do nurses often identify problems during eating in patients in whom dysphagia is not suspected, but they also provide the guidance necessary to help the patient with identified dysphagia use recommended swallowing strategies. Other responsibilities include administering tube feedings, maintaining good oral hygiene, and assigning volunteers to assist selected patients at mealtime.

Clinical Pearl: Because nurses are responsible for 24 hour care, they can provide important feedback about the patient's eating progress that may not get documented in the patient's medical record.

Dietitian

The dietitian assesses the patient's nutritional and hydration needs and monitors the patient's response to those needs. Because dysphagia frequently affects a patient's nutrition and hydration status, and because the result of poor nutrition and hydration affects a patient's overall medical stability, it is important to involve the dietitian in the care plan for patients with dysphagia. Because dietitians frequently monitor mealtime activities, they may be the professionals who initially detect a swallowing disorder. If specialized dysphagic diets are ordered for the patient, the dietitian may communicate with the food service to ensure that the special diet is prepared properly. If a patient is unable to eat orally, the dietitian may make a recommendation for a tube feeding. Guidelines for the amount and rate of tube feeding frequently are recommended by the dietitian. As patients return to oral feeding, the SLP and dietitian closely monitor intake. As oral feeding improves, the dietitian adjusts the amount of tube feeding to appropriate levels.[97]

Occupational Therapist

The occupational therapist is skilled in retraining the patient to self-feed. If the patient is unable to self-feed because of weakness or incoordination, the occupational therapist needs to be involved in the patient's care. Special adaptive feeding devices, such as a **plate guard** or built-up utensils for easier grasping, are ordered by the occupational therapist to assist the patient in achieving feeding independence. In some medical centers, the SLP and occupational therapist work closely with infants in the neonatal intensive care unit (NICU).

Neurodevelopmental Specialist

The NICU setting can influence the infant's brain development and organization as well as the parent-infant relationship. The neurodevelopmental specialist (NDS) is keenly aware of this relationship and will tailor the infant's care to individual needs. An NDS may be an SLP or occupational therapist who has specialized in assisting the premature infant in developmental growth by fostering supportive care during the infant's nervous system development. Neurodevelopmental care includes, but is not limited to, proper infant positioning to support neurodevelopmental tone and maturation. Often it is important to regulate the tolerance of the infant's visual, tactile, and auditory stimulation. Feeding is one of the most difficult tasks in which a premature infant can succeed. The NDS provides continued assessment regarding the timing and safety of the infant's oral feedings by breast or bottle. The NDS also monitors the infant's physiologic and behavioral responses to the environment and fosters a positive outcome.

Pulmonologist and Respiratory Therapist

Although the pulmonologist may not be a regular member of the dysphagia team, patients of pulmonologists frequently have swallowing disorders that require management by the swallowing team. Patients with respiratory disorders that require tracheostomy and **ventilatory** support (respirators) often have accompanying swallowing difficulty. Working with the respiratory therapist and pulmonologist to improve pulmonary toilet is an important step toward **decannulation**. Removing a patient's respiratory supports often is a prerequisite for improving the swallowing response.

CLINICAL CASE EXAMPLE 1-3

The SLP was called by the thoracic surgeon to the intensive care unit for a consultation. Her patient had just undergone **cardiac** bypass surgery and had respiratory complications requiring the placement of a tracheostomy tube. The patient was now medically stable and was ready to resume oral feeding. The SLP consulted with the respiratory therapist, who mentioned that the patient still required some oxygenation by facial mask for short periods during the day. After noting those times, the SLP returned when the mask was not in use because it might potentially interfere with the evaluation. On physical evaluation, the patient had reduced tongue strength and could make a weak, breathy voice only when the tracheotomy tube was occluded. She had a nasogastric tube in place for nutritional purposes. During the evaluation, the dietitian came in and told the SLP that the patient was not tolerating the feeding given by nasogastric tube and that it would be beneficial for the patient to begin to eat orally because some of those complications could be avoided. The SLP gave the patient small amounts of ice chips and water, as well as gelatin and pudding. The patient showed delayed swallowing of all materials and a weak cough on the liquids. The SLP believed that the patient might be at risk for aspiration because of pharyngeal weakness that may have involved the true vocal fold. She believed an imaging study that would allow her to observe the pharyngeal stage of swallow would be appropriate and that swallowing endoscopy would be the test of choice because it could be accomplished at the patient's bedside. She received approval for the study from the consulting physician and the test was performed the same day. Swallowing endoscopy revealed that during the coughing episodes, the patient was protecting her airway; however, there appeared to be some weakness in the left true vocal fold. She recommended that the patient start a special dysphagic diet and communicated that to the dietitian, who made the arrangements. The otolaryngologist was consulted for his opinion on whether any intervention would be appropriate for the vocal fold weakness. The SLP designed specific swallowing instructions and shared them with the patient and nursing staff. This case is a good example of how many disciplines can be involved in caring for a patient who has dysphagia.

LEVELS OF CARE

The prevalence, cause, and type of swallowing disorder that might be encountered depends in part on the setting in which the patient is seen. Correspondingly, the role of each professional may be different, or access to some medical specialties may not be available. For instance, it is rare for a gastroenterologist to have a full-time appointment in a nursing home facility or that a radiologist would be on staff in that facility. Traditionally, levels of care are divided into five categories: acute, **subacute**, rehabilitation, **skilled nursing**, and **home health**.

Acute Care Setting

In a survey of two acute care hospitals, Groher and Bukatman[21] found the prevalence of swallowing-related disorders to be 13%. The majority of these patients were found in the intensive care units and the neurology and neurosurgery units. Owing to the acute nature of their illness, patients in the acute care setting frequently have multiple medical complications, require intubation tubes connected to ventilators, have tracheostomy tubes in place, require feeding tubes for nutrition, and have frequent changes in their physical and mental status. Because their stay in the hospital may be short (2 to 5 days), their swallowing needs must be addressed rapidly. Frequently there is not sufficient time or patient cooperation because of mental status to order sophisticated laboratory tests. In this circumstance, the clinician may have to rely on the history and clinical evaluation to make a diagnosis and establish a treatment plan. If an instrumental evaluation is recommended, care must be given to scheduling.

Clinical Pearl: Developing a strong working relationship with radiology is crucial in order to get timely imaging studies when patients are in the acute stage of illness.

If the patient is able to cooperate with laboratory testing and is a candidate to proceed for further rehabilitation, his or her future care is facilitated if the acute care clinician can document the swallowing disorder with an imaging technique such as videofluoroscopy or endoscopy.

Neonatal Intensive Care Unit

Children born prematurely often must stay in the hospital for extended periods in the NICU. Specialized interventions for premature newborns such as improved systems of delivering respiratory support have resulted in higher survival rates of low birth weight infants. In the 1980s, the concept of integrated developmental care was introduced to minimize the potential for emotional and neurodevelopmental disorders after discharge. This type of care emphasizes the coordinated efforts of nurses, physicians, therapists, and other care providers toward common goals, with each discipline supporting the other. This type of care also recognizes issues of parent–child separation and the atypical environment of a hospital on the child's development.

More recently, infants admitted to the NICU are managed by "cluster care." Before the availability of cluster care, infants received medical care at any hour during the

day. However, the cluster care concept allows infants to sleep for 3 hours, after which time they are awakened for all their care, including feeding, diaper changes, and needed tests. Cluster care allows the infant to regularize his or her schedule, similar to what would occur outside the hospital environment.

Subacute Care Setting

Patients admitted to subacute care usually are not ready for a strenuous rehabilitation program. They may require additional medical monitoring but not the type of costly care of an acute admission associated with intensive care. If a swallowing treatment goal was formulated in the acute setting, the action plan to achieve that goal is implemented in the subacute unit. For instance, if the goal was to try to wean a patient from the tracheostomy tube as a way to ensure swallowing safety, the swallowing team would work toward that goal. If a patient continued to require tube feeding after leaving the acute care unit, a goal of the swallowing team in the subacute unit might be to begin restoring oral **alimentation**. Patients may stay in the subacute unit from 5 to 28 days. After this admission, they may be discharged home, to a rehabilitation facility, or to a **skilled nursing facility**.

Rehabilitation Setting

Patients who enter rehabilitation settings usually are judged to have the physical stamina needed to complete a full day of tasks oriented toward restoring lost function. In most cases, the patient will also be able to learn new information. For those with swallowing impairment, it may mean they need to learn or solidify their learning of new swallowing strategies. The role of the SLP is to teach the patient swallowing strategies (see Chapters 11 and 16). This may include special maneuvers or postures. It also may entail specialized diets. Frequently, the goal in the rehabilitation setting as it pertains to swallowing is to return the patient to a dietary level that is as near to normal as possible while ensuring swallowing safety. Swallowing safety may be defined as the maintenance of nutrition and hydration without medical complications. Not only is it considered medically unsafe for a patient to get food or fluid in the lungs, but it is also unsafe to not get sufficient nutrition and hydration to maintain normal bodily functions. For instance, lack of proper nutrition and hydration can lead to excessive fatigue, mental status changes, poor wound healing, anorexia, and a greater chance of developing infections. After a 1-month period of successful rehabilitation, the patient usually is discharged home. Those in whom medical complications develop during rehabilitation or who do not improve to a level of partial independence may be discharged to a skilled nursing facility.

Clinical Pearl: It is not uncommon that information from the acute care setting gets transferred to the rehabilitation setting. Therefore establishing good methods of communication can be crucial to the patient's recovery. On-site visitations to each other's facilities and grand round presentations help to solidify these relationships.

Skilled Nursing Facility

Patients who enter skilled nursing facilities usually have either not responded to attempts at rehabilitation, are not candidates for rehabilitation after their acute hospitalization, are too ill to be at home, or have chronic medical conditions that require monitoring in a structured environment. The prevalence of swallowing disorders in this setting has been reported to be as high as 60%.[14] The high prevalence in this setting is because the patients have multiple medical problems that predispose them to dysphagia. The majority, for instance, may have a neurologic disease that has compromised the swallowing musculature or has interfered with the cortical controls needed to complete the swallowing sequence. Their swallowing disorders are chronic. Some patients will have seen some recovery in their dysphagia, whereas others will continue to rely on tube feedings. For those who recover, it is important to help them maintain their skills. Those who must rely on tube feedings after their hospital stay will require reevaluation for the possibility of returning to oral feeding. For some, returning to oral alimentation will not be possible. Because of the potential for patients in this setting to be medically fragile, it is easy to decompensate their swallowing skills by a slight change in medical status, rather than a new, major event such as stroke. An example of this phenomenon might be a patient who is not swallowing a sufficient amount of liquids, who may then develop a urinary tract infection that results in a fever with generalized fatigue, anorexia, and a disinterest in eating. In this situation, the patient may not be ingesting enough calories to be able to sustain the strength needed to produce a safe swallow throughout the entire meal. As a consequence of fatigue, the patient is more likely to show signs of dysphagia.

Another example might be a patient who has been eating well but whose medications were changed. The unwanted side effect from the medication change could negatively affect the nervous system to create a problem with motor movement, and swallowing is secondarily affected. For example, medications that create sedative effects are capable of decompensating an already fragile swallow by slowing motor movement and interfering with the cortical controls necessary to complete an entire meal. The potential for fluctuations in metabolism in this patient population often makes it difficult to establish a single factor that precipitated the dysphagia.

It is known that patients in skilled nursing facilities usually are in older age cohorts. Not only do they endure the effects of diseases that result in dysphagia commonly found in older persons (e.g., stroke, Parkinson's disease), but they also have impairments in swallowing as a result of the aging process. Change in taste perception and in the strength and speed of the swallowing muscles are examples of these alterations. The SLP working in the skilled nursing facility is kept busy managing the large number of patients with swallowing disorders. Many patients with dysphagia are able to eat safely only if they are at the proper dietary level and only if they are following the recommended feeding strategies. Any change in baseline metabolism or any new neurologic insult may decompensate their swallowing skills so that they are at risk for developing medical complications. Many times the focus of therapeutic effort for the SLP working in the skilled nursing facility is one of prevention—attempting to keep patients as safe as possible while eating, even in the circumstance of suspected dysphagia. Such preventive efforts not only may require direct intervention with behavioral and dietary treatment strategies, but also entail monitoring of mealtime activities to ensure that patients who are at risk of aspiration are following the prescribed dysphagia treatment plan.

Often the mental or physical status of patients in the skilled nursing environment interferes with their ability to cooperate with a formal dysphagia evaluation. Clinicians must rely on a combination of the medical history and detailed observations of each meal to establish the treatment plan. If the patient is not eating orally, the clinician often must rely on the physical examination and on his or her judgment of how well the patient managed attempts at oral ingestion as part of that examination. The examination will be limited further by poor access to modified barium swallow studies or other laboratory investigations. Transportation of patients to receive these tests presents another challenge because chronically ill patients are difficult to move.

Clinical Pearl: Some regions of the country have professionals with mobile units who can provide on-site swallow imaging studies.

The chronic medical conditions of patients in skilled nursing facilities often are life threatening. For this reason, patients and their families may execute an **advance directive** (see Chapter 12). The advance directive is a statement executed by the patient or family (if they hold medical power of attorney) of their desires and wishes regarding their medical care in life-threatening situations, such as whether the patient would want to be resuscitated for cardiac arrest. Part of this directive may pertain to their wishes to sustain nutrition, especially when the support for nutrition may involve feeding tubes. Patients may elect to not be fed by a feeding tube despite the risk of aspiration and life-threatening pneumonia. In these cases, the role of the swallowing clinician is to recommend the safest mode of ingestion, making sure that the patient and family understand the potential risks.

CLINICAL CORNER 1-5 INTERDISCIPLINARY COOPERATION

An 86-year-old man who had been living in a nursing home was admitted to the hospital with a suspected right brain stroke. He was confused on admission, and the attending physician did not think it was safe for him to eat orally so a nasogastric tube was placed. At the nursing home, he was eating a modified soft diet because his teeth were in poor repair. He had a past history of GERD and **Barrett's esophagitis**. After 2 days, a swallowing evaluation was ordered before he was allowed to resume oral feeding.

CRITICAL THINKING

1. How many medical disciplines might become involved with this patient? Who and why?
2. What are the chances that he will be dysphagic based on his history? Are age and prior living setting considerations in this case? How might these facts affect the diagnosis and treatment?
3. Are there any special issues revolving around which side of the brain was injured that might relate to dysphagia?

Home Health

Patients who have left the hospital or the rehabilitation setting for home may require additional monitoring or direct treatment from therapists who perform their responsibilities in the patient's home environment. Patients who are unable to swallow should receive regular reevaluations for attempts at oral feeding unless oral feeding is contraindicated by the medical care team. Most often, the clinician responsible for managing the swallowing disorder in the home environment is ensuring that the patient is following the swallowing strategies or has improved to a point at which consideration should be given to changing the dietary level. These changes often are made in consultation with the patient and family and are based on the physical examination and observations of eating (review Clinical Corner 1-5).

TAKE HOME NOTES

1. Dysphagia is a symptom of a disease, not a primary disease. It is characterized by a delay or misdirection of something swallowed as food moves from the mouth to the stomach. It has both medical and psychosocial consequences on a patient's quality of life.

2. A *feeding disorder* usually refers to the process of food transport. An eating disorder may not be related to a swallowing disorder.
3. The prevalence of dysphagia is highest in patients with neurologic disease.
4. Patients in acute care intensive care units and those in skilled nursing facilities tend to be at highest risk for dysphagia.
5. There may not be a clear link between dysphagic symptoms and the patient's primary medical diagnosis in patients who reside in skilled nursing facilities.
6. Patients in skilled nursing facilities are medically fragile, and their swallowing response can be easily decompensated by fatigue or an acute medical condition such as an infection.
7. Aspiration of liquid and food is the consequence of those materials entering the airway below the level of the vocal folds.
8. Aspiration of liquid or food may or may not produce a lung infection known as aspiration pneumonia.
9. Respiratory impairments such as those requiring an endotracheal tube or tracheostomy tube also interfere with swallowing.
10. The SLP frequently is the coordinator of the swallowing team and therefore needs to have an understanding of each team member's perspective of the dysphagic patient. Many specialists could become involved in the care of a patient with dysphagia.
11. The evolution of the NICU has provided advanced technologies to maintain survival for infants as young as 23 weeks' gestational age. The feeding specialist in the NICU often is skilled in neurodevelopmental studies.

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CHAPTER 2
Normal Swallowing in Adults

Michael E. Groher

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CHAPTER OUTLINE

OBJECTIVES

1. Define the key anatomic structures involved in swallowing.
2. Define the groups of muscles that participate in swallowing.
3. Define the peripheral and central neurologic controls for swallowing.
4. Discuss the key physiologic components that occur when moving a bolus from the mouth to the stomach.
5. Discuss how normal swallowing is affected by bolus type and delivery.
6. Describe swallowing associated with normal aging.

Normal swallowing includes an integrated, interdependent group of complex feeding behaviors emerging from interacting cranial nerves of the brainstem and governed by neural regulatory mechanisms in the medulla, as well as in sensorimotor and limbic cortical systems.

Clinical Pearl: A thorough understanding of the complexities of the swallowing mechanism demands considerable time. However, it is important to remember that all diagnostic and treatment approaches that are discussed in this text stem from knowledge of how the system functions normally in order to grasp how abnormality is detected and what one can do to ameliorate its consequences.

Healthy individuals simultaneously perform the sequential sensory and motor patterns of mastication and swallowing with little effort and conscious awareness. For purposes of simplification, such sensory-guided discriminatory feeding and sensory-cued, stereotyped swallowing behaviors usually are divided into four stages (Practice Note 2-1): (1) the oral preparatory stage, in which food is masticated in preparation for transfer; (2) the oral stage, which entails the transfer of material from the mouth to the oropharynx; (3) the pharyngeal stage, in which material is transported away from the oropharynx, around an occluded **laryngeal vestibule**, and through a relaxed cricopharyngeus muscle into the upper esophagus; and (4) the esophageal stage, in which material is transported through the esophagus into the gastric cardia. An additional stage of swallowing that precedes the oral stage has been proposed by Leopold and Kagel,[1] who argue that visual appreciation of the bolus before its placement in the oral cavity may send a cognitive message that may help stimulate saliva during bolus preparation.

Knowledge of the anatomic and physiologic aspects of this interdependent group of voluntary and involuntary behaviors requires detailed study if the goal is to rehabilitate persons with dysphagia, which may be caused by a wide array of neurologic and structural impairments resulting from injury or disease affecting the central nervous system, cranial nerves, and muscles.

PRACTICE NOTE 2-1

A single bolus of varying texture and size can be chewed and swallowed while a person holds a conversation, and at the same time a beverage may be imbibed while various portions of the more solid food are held in the mouth. With relaxation of the pharyngeal constrictors, a sword can be passed from the pharynx through the cricopharyngeal muscle (not recommended without practice) and, with effort, a person can swallow solids while standing on his or her head!

NORMAL ANATOMY

The oral cavity extends from the lips anteriorly to the nasopharynx posteriorly, and it contains the tongue, gums, and teeth. The oral cavity is separated from the nasal cavity by the bony palate and velum (soft palate). It is composed of a highly mobile lower jaw, or mandible, consisting of a U-shaped body containing important ridges for muscle attachments. The upper jaw, or maxilla, meets the zygomatic or cheek bone and is adjoined by the L-shaped palatine bones, lying posterior to the nasal cavity. The perpendicular part of the palatines forms the back of the nasal cavity, whereas the horizontal part forms the back of the bony palate. The velum and posterior nasopharyngeal wall seal and open communication between the nasal and oral cavities during swallowing and respiratory behaviors, respectively. The nasopharynx lies above the velum, and the oropharynx lies posterior to the mouth. The pharynx extends below to the esophagus; its inferior portion is called the hypopharynx and is separated from the esophagus by the cricopharyngeal muscle (Figure 2-1). The cartilaginous larynx lies anterior to the hypopharynx at the upper end of the trachea, suspended by muscles attached to the hyoid bone. The cricoid cartilage lies above the trachea, with the thyroid cartilage above it. Both are suspended from muscles attached to the hyoid bone, which itself is suspended between the jaw, tongue, and sternum by suprahyoid and infrahyoid musculature.

The respiratory system is protected during pharyngeal swallow by occlusive muscular constriction of the laryngeal vestibule and downward displacement of the epiglottis. The true vocal cords are at the inferior margin of the **laryngeal ventricle** and are attached anteriorly at the thyroid cartilage and posteriorly at the arytenoid cartilages. The vestibular (false) vocal folds separate the ventricle and the vestibule. The epiglottis extends from the base of the tongue into the pharyngeal cavity.

The valleculae are lateral recesses at the base of the tongue on each side of the epiglottis. The piriform sinuses are lateral recesses between the larynx and the anterior hypopharyngeal wall (Figure 2-2). These recesses serve as important anatomic landmarks in the videoradiographic assessment of pharyngeal swallow. Figure 2-3 shows a lateral view of the key anatomic structures in the region of the head and neck.

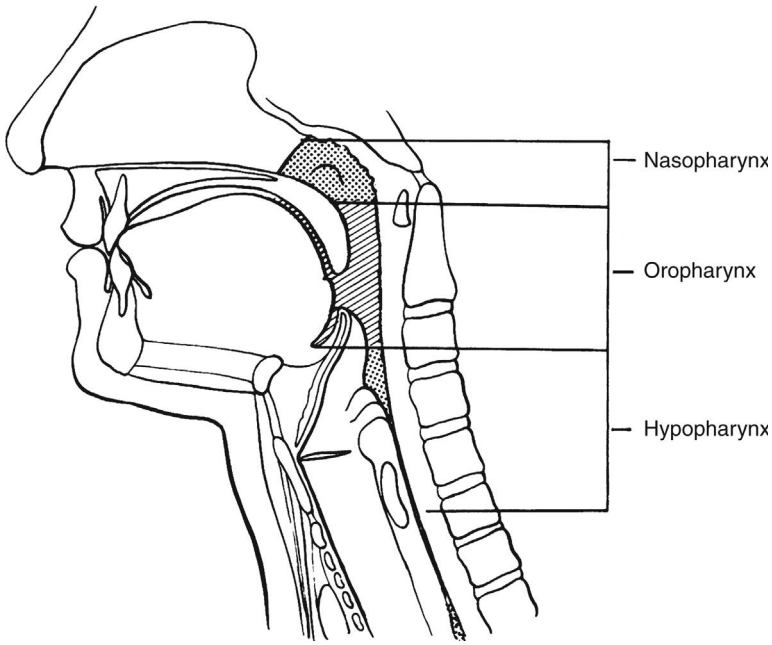

FIGURE 2-1 Lateral view of the anatomy of the head and neck with demarcations of three major regions: nasopharynx, oropharynx, and hypopharynx.

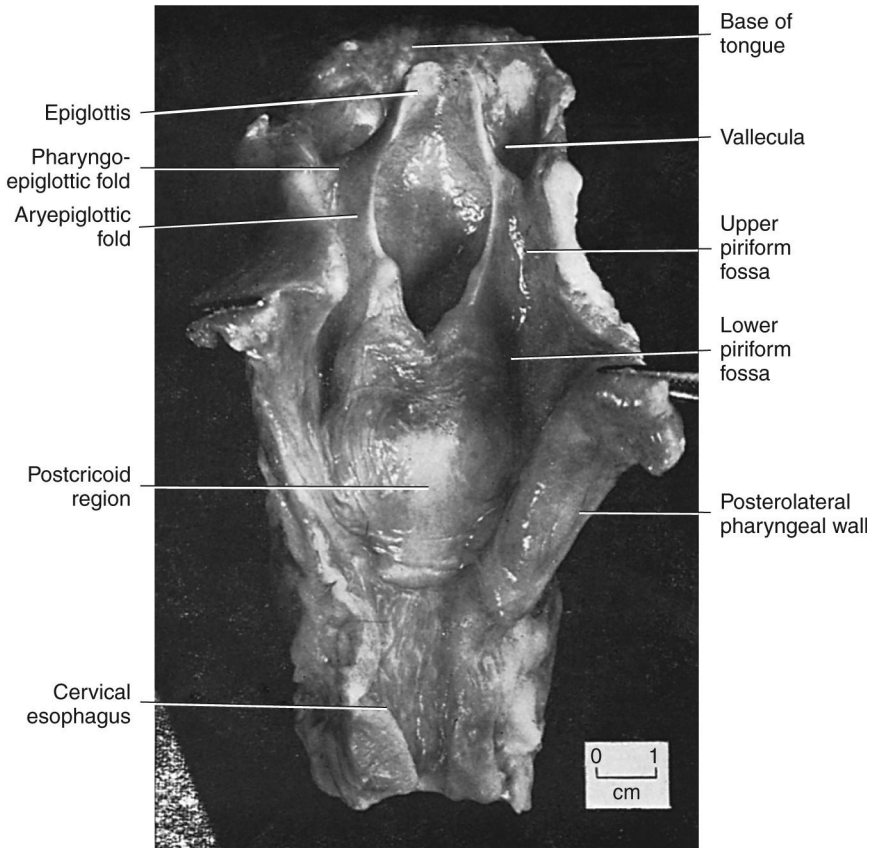

FIGURE 2-2 Anatomic specimen of the pharyngeal compartment as it surrounds the airway. The bolus flows into the **vallecular spaces** and around the epiglottis inferiorly into the piriform fossa before entering the esophagus.

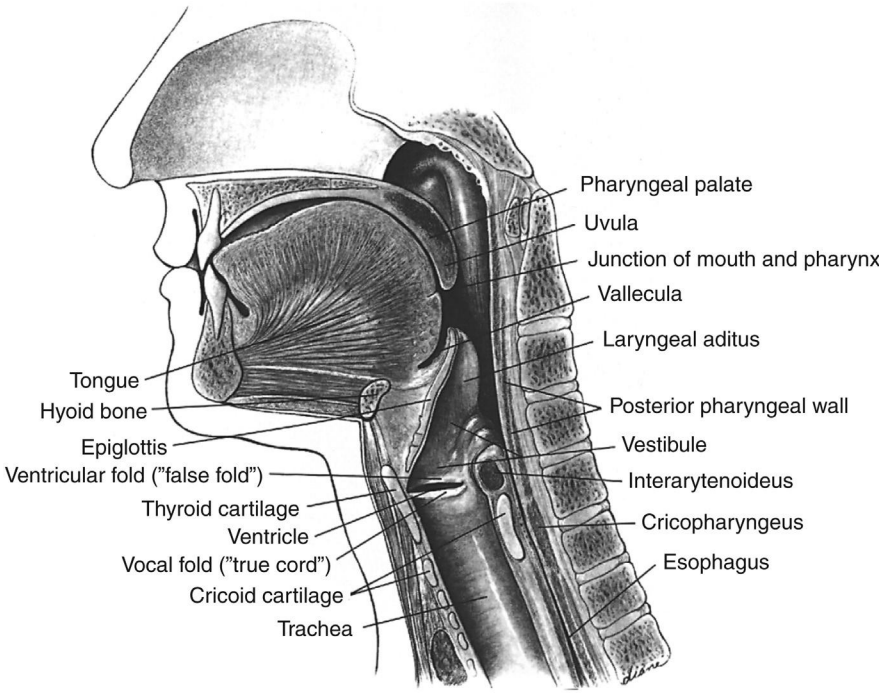

FIGURE 2-3 Lateral view of the anatomy of the head and neck pertinent to swallowing. (From Bosma JF, Donner MW, Tanaka E, et al. Anatomy of the pharynx, pertinent to swallowing. *Dysphagia*. 1986;1:24.)

Oral Preparatory Stage

The mandibular branch of the trigeminal nerve (cranial nerve [CN] V) innervates the principal muscles for chewing behaviors. The primary muscles of chewing are the masseter, temporalis, and pterygoid muscles, which attach to the sphenoid wing of the temporal bone. The masseter closes the jaw while the temporalis moves it up, forward, or backward (Table 2-1). The medial pterygoid muscles work bilaterally to elevate the mandible while they shift the jaw to the opposite side unilaterally. The lateral pterygoid muscles work together, pulling down or forward while moving the jaw or chin to the opposite side unilaterally. Both sets of pterygoid muscles cooperate to grind in mastication.

The facial nerve (CN VII) innervates lower facial muscles attached to the maxillae and mandible of the skull. These include the buccinator muscles, which compress the lips and flatten the cheeks in the movement of food across the teeth (Table 2-2). The buccinator fibers blend with those of the orbicularis oris, the sphincter of the lips.

The hypoglossal nerve (CN XII) innervates the tongue, which contains four separate intrinsic muscle masses that have different effects on the shape, contour, and function of the tongue.

Oral/Pharyngeal Stage

The pharyngeal cavity of the neck, which is suspended from the base of the skull and anchored to the top of the sternum, is formed by 26 pairs of **striated muscles** innervated by six cranial and four cervical nerves. The horseshoe-shaped hyoid bone in the neck serves as a fulcrum that provides a mechanical advantage for pharyngeal musculature associated with swallowing behaviors of the posterior tongue, pharynx, and larynx.

In the nasopharynx, five muscles adjust the position of the velum with respect to the food bolus: the palatoglossal and levator veli palatini muscles (pharyngeal plexus and accessory nerve), which elevate the soft palate and seal the nasopharynx; the tensor veli palatini (mandibular branch of the trigeminal nerve), which tenses the palate and dilates the orifice of the eustachian tube; the palatopharyngeal muscle (pharyngeal plexus and spinal accessory nerve), which depresses the soft palate, approximates the palate or pharyngeal folds, and constricts the pharynx; and the muscularis uvula (spinal accessory nerve), which shortens the soft palate (Table 2-3).

The hypoglossal (CN XII), trigeminal (CN V), and facial (CN VII) nerves innervate the suprahyoid group of muscles. The hypoglossal nerve supplies the geniohyoid, which draws the hyoid bone up and forward, depressing the jaw, and the trigeminal nerve supplies the mylohyoid, which elevates the hyoid bone and tongue and depresses the jaw (Table 2-4). The digastric muscles contain anterior and posterior bellies. The anterior belly is innervated by the mandibular branch of the trigeminal nerve (CN V) and depresses the jaw or raises the hyoid bone, whereas the posterior portion is innervated by the facial nerve (CN VII) and elevates or retracts the hyoid. The facial nerve (CN VII) innervates the stylohyoid muscle, which elevates the hyoid bone during swallowing. In addition, the hyoglossus and the genioglossus serve as laryngeal elevators, as well as extrinsic tongue muscles, and are designed to depress the tongue or help elevate the hyoid bone when the tongue is fixed. The accessory nerve (CN XI), in association with the hypoglossal (CN XII) nerve, innervates the styloglossus, which draws the tongue up and back during swallowing. The glossopharyngeal (CN IX) and accessory (CN XI) nerves also cause the palatoglossus to raise the back of the tongue and lower the velum. The styloglossus and palatoglossus raise the back of the tongue and lower the sides of the soft palate.

The vagus nerve (CN X) and the spinal accessory nerve (CN XI) innervate the muscular pharynx, whose superior, middle, and inferior constrictor muscles constitute its external circular layer and work together to transport a bolus of food toward the esophagus during swallowing. Three other muscles constitute the internal longitudinal layer of the pharynx: the palatopharyngeus, stylopharyngeus, and salpingopharyngeus. The stylopharyngeus (glossopharyngeal nerve) elevates the pharynx, and to some extent the larynx, during

TABLE 2-1 Muscles of Mastication

Muscle	Origin	Insertion	Nerve	Action
Temporalis	Temporal fossa of skull	Ramus and coronoid process of mandible	Trigeminal	Elevates or closes mandible; retracts mandible
Masseter	Zygomatic arch	Ramus of mandible	Trigeminal	Elevates or closes mandible
Medial pterygoid	Palatine bone, lateral pterygoid plate, tuberosity of maxilla	Ramus of mandible	Trigeminal	Elevates or closes mandible
Lateral pterygoid	Great wing of sphenoid and lateral pterygoid plate	Neck of condyle of mandible	Trigeminal	Depressor or opener of mandible; protrudes mandible; permits side-to-side movement of mandible

TABLE 2-2 Muscles of the Face

Muscle	Origin	Insertion	Nerve	Action
Orbicularis oris	Neighboring muscles, mostly buccinators; has many layers of tissue around the lips	Skin around lips and angles of the mouth	Facial	Closes, opens, protrudes, inverts, and twists lips
Zygomaticus minor	Zygomatic bone	Orbicularis oris in upper lip	Facial	Draws upper lip upward and outward
Levator labii superior	Below infraorbital foramen in maxilla	Orbicularis oris in upper lip	Facial	Pulls up or elevates upper lip
Levator labii superior alaeque nasi	Process of maxilla	Skin at mouth angle, orbicularis oris	Facial	Raises angle of the mouth
Zygomaticus major	Zygomatic bone	Fibers of the orbicularis oris, angle of the mouth	Facial	Draws upper lip upward; draws angle of mouth upward and backward; the smiling muscle
Levator anguli oris	Canine fossa of maxilla	Lower lip near angle of the mouth	Facial	Pulls up corners of mouth
Depressor anguli oris	Outer surface and above lower border of mandible	Skin of cheek, corner of mouth, lower border of mandible	Facial	Draws lower lip down; draws angle of mouth down and inward
Depressor labii inferior	Lower border of the mandible	Skin of lower lip, orbicularis oris	Facial	Depresses lower lip
Mentalis	Incisor fossa of mandible	Skin of chin	Facial	Pushes up lower lip; raises chin
Risorius	Platysma, fascia over the masseter skin	Angle of mouth, orbicularis oris	Facial	Draws corners or angle of mouth outward; causes dimples; gives expression of strain to face
Buccinator	Alveolar process of maxilla, buccinators ridge of mandible	Angle of mouth, orbicularis oris	Facial	Flattens cheek; holds food in contact with teeth; retracts angles of the mouth

TABLE 2-3 Muscles of the Palate

Muscle	Origin	Insertion	Nerve	Action
Levator veli palatini	Apex of temporal bone	Palatine aponeurosis of soft palate	Vagus and accessory	Raises soft palate
Tensor veli palatini	Fossa of sphenoid bone	Palatine aponeurosis of soft palate	Trigeminal	Stretches soft palate
Palatoglossus	Undersurface of soft palate	Side of tongue	Vagus and accessory	Raises back of tongue during the first stage of swallowing
Palatopharyngeus	Soft palate	Pharyngeal wall	Vagus and accessory	Shuts off nasopharynx during second stage of swallowing
Uvulae	Posterior nasal spine and palatine aponeurosis	Into uvula to form its chief bulk or content	Vagus and accessory	Shortens and raises uvula

swallowing, and the salpingopharyngeus (accessory nerve and pharyngeal plexus) draws the lateral walls of the pharynx up. The palatopharyngeus muscle draws the velum down.

The cricopharyngeal muscle is an important single muscle that lies at the transition level between the pharynx and the esophagus. Functionally, it is separate from both the pharynx and the esophagus and acts as a sphincter, relaxing during passage of the bolus from the pharynx into the esophagus. It is innervated by both pharyngeal branches of the vagus and sympathetic fibers from the middle and inferior cervical ganglia. The key muscles used in the oral and pharyngeal stages of swallowing are shown in Figure 2-4.

TABLE 2-4 Suprahyoid Muscles

Muscle	Origin	Insertion	Nerve	Action
Mylohyoid (anterior belly digastric)	Inner surface of mandible	Upper border of hyoid bone	Trigeminal	Elevates tongue and floor of mouth; depresses jaw when hyoid bone is in fixed position
Digastric (anterior belly)	Intermediate tendon by loop of fascia to hyoid bone	Lower border of mandible	Trigeminal	Raises hyoid bone if jaw is in fixed position; depresses jaw if hyoid bone is in fixed position
Geniohyoid	Mental spine of mandible	Hyoid bone	Cervical (C1 and C2) through hypoglossal	Draws hyoid bone forward; depresses mandible when hyoid bone is in fixed position
Stylohyoid	Stylohyoid process of temporal bone	Body of hyoid at greater cornu	Facial	Elevates hyoid and tongue base
Hyoglossus	Greater cornu of hyoid	Into tongue sides	Hypoglossal	Tongue depression
Genioglossus	Upper genial tubercle of mandible	Hyoid, inferior tongue, and tip of tongue	Hypoglossal	Protrusion and depression
Styloglossus	Anterior border of styloid process	Into side of tongue	Hypoglossal	Elevates up and back
Palatoglossus	Anterior surface of soft palate	Dorsum and side of tongue	Glossopharyngeal, vagus, and accessory	Narrows fauces and elevates posterior tongue

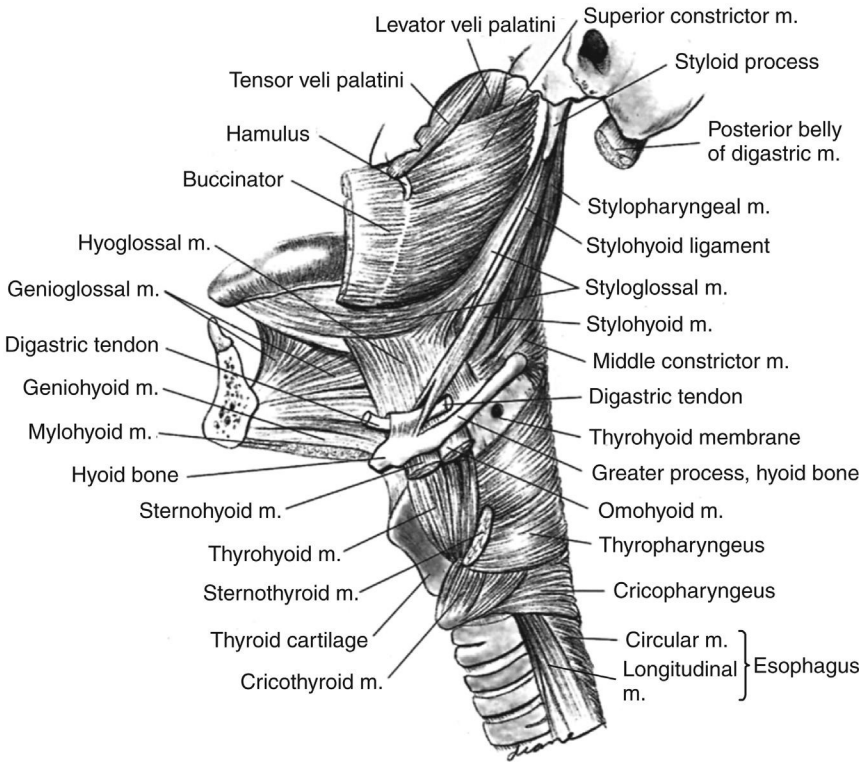

FIGURE 2-4 Lateral view of the key muscles of the head and neck used in swallowing. (From Bosma JF, Donner MW, Tanaka E, et al. Anatomy of the pharynx, pertinent to swallowing. *Dysphagia.* 1986;1:24.)

Esophageal Stage

The esophagus is a distensible tube, approximately 21 to 27 cm (10 inches) long, connecting the pharynx (at C6) and stomach (at T12). It is separated from the pharynx by the pharyngeal esophageal segment (PES) and from the stomach by the lower esophageal sphincter (LES). Under resting conditions, the esophageal **lumen** is collapsed, creating a potential space that can easily distend up to 3 cm to accommodate swallowed air, liquids, or solids. The esophagus is lined with a protective, stratified, squamous epithelium that covers an inner layer of circular fibers and an outer layer of longitudinal fibers. At its proximal end (upper fourth) the muscle is striated, whereas the distal two-thirds are composed of **smooth muscle**. The middle third, in the region of the aorta, is a combination of smooth and striated muscles. As it courses through the thorax at the level of the carina, the esophagus runs lateral and posterior to the left ventricle of the heart, creating a natural bend as it courses anteriorly toward the diaphragmatic hiatus. After passing the diaphragmatic hiatus, it connects to the body of the stomach at the level of the LES. The smooth muscle of the LES is arranged in a specialized spiral configuration as it joins the inner oblique muscle zone of the stomach. The relation of the esophagus to the heart and tracheobronchial tree, as well as its path through the diaphragmatic hiatus, is shown in Figure 2-5.

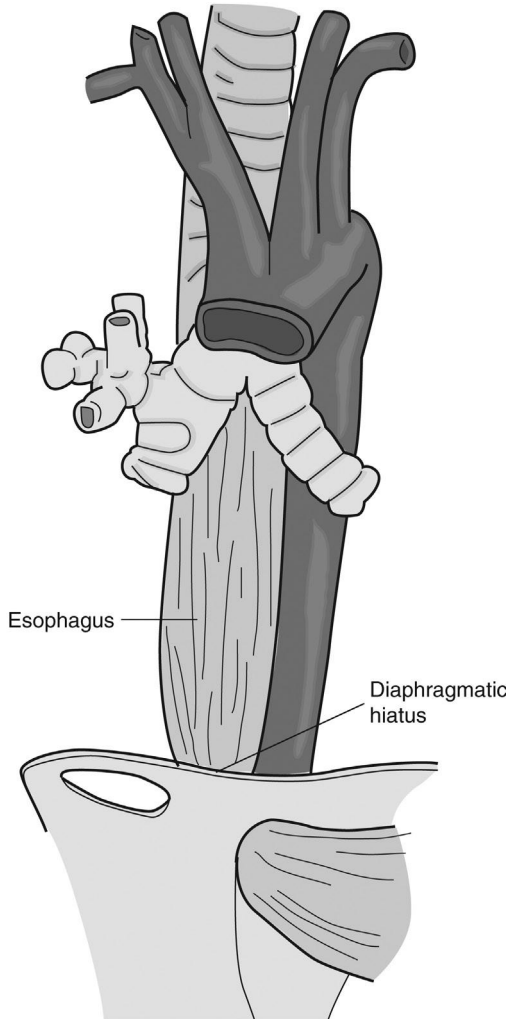

FIGURE 2-5 The esophagus courses through the chest cavity and through a hiatus in the diaphragm, ending at the level of the stomach.

NORMAL PHYSIOLOGY

Many studies have examined the normal aspects of the oropharyngeal swallow sequence. The rationale usually given for such studies is that clinicians must be able to compare normative data with patient data to determine whether an abnormality exists. Although this approach to detection has heuristic appeal, studies of the normal swallow have revealed significant variability among normal (healthy) subjects, particularly in the oral preparatory and oral stages of swallowing.[2-5] Part of this variability is attributable to subject selection, bolus type, and the tools used to measure swallow performance. Other variability seems inherent in the swallowing process. It appears that the mechanism for swallowing must be variable to accommodate the variations of bolus type and amount for successful ingestion in different circumstances of eating, such as eating while talking, in varied environments, and at various rates. The astute clinician will not ignore those aspects of normal swallow performance that have been empirically evaluated but should also be imminently cognizant of placing a person's functional swallow in the context of his or her swallowing complaint, past medical history, and the results of physical and instrumental examinations. Busy clinicians often make timing comparisons to normal values based on real-time observations with particular attention to changes in timing as it might affect actual invasion or potential threat to the airway.

Clinical Pearl: Studies that focus on timing of swallow events do not always agree; however, on most measurements, there is sufficient agreement that allows clinicians to compare normal to abnormal.

Normal swallowing performance depends on the rapid transfer of the bolus from the oral cavity to the stomach. A liquid bolus may pass through the pharynx within 2 seconds and enter the stomach in less than 5 seconds. Efficient movement is accomplished by the strength of the neuromuscular contraction exerted on the bolus and on the forces of gravity. Efficient bolus movement is accomplished when coordinated neuromuscular contractions and relaxations create zones of high pressure on the bolus and

PRACTICE NOTE 2-2

Try experiencing the effects of an open valve (the lips) and a closed nasal passage on your own swallowing performance. First swallow your saliva as usual. Then try to swallow your saliva with your lips open, noticing the differences in effort expended. Do the same thing with the nose open and then pinch the nostrils closed and swallow.

zones of negative pressure below the level of the bolus. Some parts of the swallowing chain, such as the esophagus, remain under negative pressure because of their location. Creating zones of high and low pressure is largely accomplished by the coordination and strength of the swallowing valves: lips, velum, airway closure, and the PES opening and closing. A patent nasal airway also may be important (Practice Note 2-2). The tongue provides the initial positive driving force. The tongue's posterior deflection provides the basis for laryngeal elevation by applying traction to the hyoid bone. Efficient (i.e., timely and strong) laryngeal elevation helps create a negative zone of pressure in the pharynx, particularly in the region of the PES. This allows the bolus to move rapidly, and therefore safely, from a zone of high pressure into a zone of negative pressure. Moving from a zone of high pressure into another zone of high pressure caused by a pathologic condition (e.g., muscle weakness or incoordination) inhibits bolus flow and results in **stasis** and residue that may be aspirated into the airway.

Oral Preparation

Food or liquid in the mouth stimulates taste, temperature, and pressure (touch) receptors. The primary receptors of taste are located on the tongue, on the hard and soft palates, in the pharynx, and in the supralaryngeal region. The receptors are activated by saliva. Saliva is produced by the activation of the submandibular, submaxillary (autonomic aspects of CN VII), and parotid glands (autonomic aspects of CN IX). Activation of these glands is achieved by the actions of the jaw, tongue, and hyoid bone during bolus preparation and by the inherent taste of the bolus. The primary sensory receptors on the dorsum of the tongue responsible for the perception of salt, sweet, sour, and bitter are activated by saliva. In addition to facilitating taste and bolus formation, saliva is important in the maintenance of adequate oral hygiene by controlling microorganisms, in the regulation of the acidity levels in the stomach and esophagus because of its bicarbonate composition, and in the breakdown of carbohydrates. The number of times a person swallows saliva in 1 hour can vary between 18 and 400, and it largely depends on the rate of salivary flow.[6]

Sensations of taste are carried by the chorda tympani branch of CN VII on the anterior two thirds of the tongue and through the greater petrosal branch on the hard and soft palates. Taste on the posterior third of the tongue is mediated by CN IX. Sensations of taste are sent to the nucleus tractus solitarii (NTS) in the medulla of the brainstem (see sections on neurologic controls), where they are transmitted to the sensorimotor cortex by the thalamus. Taste receptors in the region of the **laryngeal aditus** are carried to the NTS by the superior laryngeal branch of CN X. Appreciation of taste depends largely on smell. Smell sensations are carried by direct stimulation of the nasal cavity and by smell elicited by chewing, during which odors travel posteriorly into the nasopharynx. Interpretation of smell is ultimately accomplished through the thalamus to the frontal and temporal cortices by information carried by CN I. Information (memories) relating to smell may be stored in the **hippocampus**. Although it is clear that certain peripheral mechanisms are important in the elicitation of swallow, their exact role in normal and dysphagic subjects remains unclear.[7] For instance, interruptions by anesthetic injections in some of the key peripheral sensory input channels do not interfere with the motor swallow response.[7]

The coordinated action of the tongue and jaw moves a bolus laterally onto the molar table for deformation. Further deformation is accomplished by variable contacts of the tongue to the hard palate. Although the tongue may play a large role in containing the bolus in the oral cavity before swallow, evidence indicates that during solid bolus mastication, material is allowed to collect in the vallecular recesses at the tongue base before swallow initiation.[5] The ultimate role of the tongue is to manipulate, shape, hold, and then transfer the bolus into the oropharynx, signaling the onset of the oral stage of swallow as the swallowing sequence transitions into the pharyngeal stage with the passage of the bolus through the oropharyngeal port. The exact nature of the sensory cues that signal a bolus is ready for swallowing is not completely understood; however, studies have shown that the superior laryngeal nerve (SLN) branch of the vagus is important in swallow initiation[8] and in the sensory protective mechanisms of the upper airway.[9] After studying 266 normal subjects who swallowed varying types of boluses ranging from buttered bread to cake to carrots, and peanuts, Engelen et al.[10] concluded that masticatory performance when preparing a bolus is more dependent on the bolus characteristic than on oral physiology. The mechanics of bolus preparation can be appreciated with videofluoroscopy. The first images are taken as the patient faces the camera and chews a piece of cracker (Video 2-1). The undulating and varied movements of the tongue and jaw are apparent. In the lateral view, the tongue can be seen touching the hard palate as material is pushed toward the tongue base, filling the valleculae before the swallow (Video 2-2).

Oral Stage

Once the bolus is prepared, the tongue tip is elevated to occlude the anterior oral cavity at the alveolar ridge, and the bolus is held against the hard palate. The edges of the tongue dorsum contain the bolus laterally. The tongue tip and dorsum appear to work longer in containment activity than the posterior tongue after the oral stage is initiated; however, the posterior tongue is more responsible for delivering the bolus into the pharynx.[11] Before—but almost simultaneous with—the first posterior movement of the tongue, respiration ceases (see section on respiration), followed by arytenoid cartilage approximation precipitating true vocal fold adduction. Retraction of the tongue is primarily accomplished by extrinsic tongue muscles: digastricus (CN V), mylohyoid (CN V), and the geniohyoid (CN XII). The tongue base applies positive pressure to the tail of the bolus by its contact with the velum and posterior pharyngeal wall, which allows the bolus to move rapidly through the pharynx into an open PES. As the tongue propels the bolus posteriorly, the palatopharyngeal folds are pulled medially to form a slit through which the bolus can pass. The levator veli palatini muscles help elevate the velum to seal the nasopharyngeal opening. The combined action of the tongue's contact to the velum and posterior pharyngeal wall and sealing the nasopharynx contribute to the maintenance of positive pressure on the bolus as it moves toward zones of negative pressure in the hypopharynx. By the tongue's connections to the hyoid bone, and the hyoid bone's connections to the thyroid and cricoid cartilages, the larynx is pulled up and forward, resting under the tongue base that now partially covers the opening to the airway. Using 13 formalin-fixed cadaver sections, Pearson et al.[12] concluded that the geniohyoid muscle was most active in the anterior displacement of the hyoid bone, whereas the mylohyoid was most responsible for superior movement. As the larynx rises, the cartilaginous epiglottis makes its descent over the top of the airway, completing an elaborate system of airway protection that allows the bolus to be directed toward the esophagus rather than into the trachea. The extent of epiglottic descent depends on anterior hyoid displacement, tongue base retraction force, and bolus size.[13] Rapid and complete laryngeal elevation (2 to 3 cm on average) aids in creating negative pressure in the region of the hypopharynx. As the bolus enters the pharynx, it is divided by the vallecular spaces at the level of the tongue base, helping deflect it away from the airway as an additional component of airway protection.

Respiration and Swallow

Protection of the upper airway through the oropharyngeal phase of swallowing is crucial to swallowing safety. Respiration and swallowing are linked by their anatomy (common conduits of mouth and pharynx) and their neuroanatomic relations in the medulla of the brainstem. This relation is expressed functionally because respiration is inhibited by swallowing, and disorders of respiration often affect swallow safety (see Chapter 7). The period of airflow inhibition in most normal adults begins before the onset of the oral stage of swallow.[14,15] During mastication, respiratory patterns are modified from normal tidal patterns; however, respiratory pause does not occur until the bolus collects at the vallecular level.[16] A short exhalation cycle precedes the cessation of respiration. As the tail of the bolus passes through the PES, the larynx descends and respiration continues on the exhalation cycle slightly before the PES closes.[15] Exhalation is accompanied by a buildup of subglottic pressure that separates the vocal folds. This release of pressure is heard as an audible burst by using a stethoscope placed at the laryngeal level (see Chapter 9).[17] This burst of exhalation is considered a protective feature in case any swallowed material is lodged in the upper airway. This explosion of exhaled air is encouraged with the Heimlich maneuver. The pattern of exhalation-swallow-exhalation may change in normal aging[18,19] and in disease (Clinical Corner 2-1).[20] The duration of respiratory pause in normal subjects varies from 0.75 to 1.25 seconds depending on the subject's age and bolus size.[21] In general, the larger the bolus size, the longer the duration of the pause.[19] During the respiratory pause, the true vocal folds move medially but do not fully approximate.[15] It is possible that the cessation of respiration during swallowing is not physiologically tied to vocal fold movement because patients with laryngectomy show similar periods of swallow apnea compared with normal subjects.[22]

CLINICAL CORNER 2-1 HEIMLICH MANEUVER

While dining one evening, a couple noticed someone at an adjoining table suddenly jump up and complain loudly that something was sticking in his throat. He seemed quite uncomfortable and was starting to sweat. Because of the commotion, the waiter rushed over and began the Heimlich maneuver by pressing his hands around the diner's waist, forcefully pushing on his diaphragm with rapid thrusts. Unfortunately, this did not relieve his customer's symptoms and he continued to complain that something was stuck.

CRITICAL THINKING

1. Why didn't the Heimlich maneuver relieve the customer's symptoms?
2. What might have been the problem?
3. What is the key finding in this history that the Heimlich maneuver would not relieve his complaint?

Pharyngeal Stage

The pharyngeal stage begins when the bolus arrives at the level of the valleculae and ends when the PES closes.[23] When the bolus enters the pharynx, the hyoid bone continues its superior and anterior excursion toward the edge of the mandible, tilting the larynx under the retracting tongue base to protect the bolus from entering the upper airway. The false vocal folds offer further protection in conjunction with the closure of the laryngeal aditus by the aryepiglottic folds. As a result of contraction of the thyroepiglottic ligament and posterior tongue contraction, the epiglottic cartilage descends from its erect position over the laryngeal aditus. Thus many mechanisms are active in preventing the bolus from entering the upper airway. These include (1) cessation of active respiration, (2) approximation of the true and false vocal folds, (3) closure of the laryngeal aditus, (4) deflection of bolus material by the tongue base over a rising larynx, and (5) division of the bolus through the valleculae that direct the bolus around the superior aspect of the airway entrance.

As the bolus enters the pharynx, the superior, middle, and inferior constrictor muscles are activated sequentially to narrow and shorten the pharynx, contributing to peristalsis-like movements in the posterior pharyngeal wall that aid in bolus propulsion into the esophagus. The duration of pharyngeal muscle contraction is unaffected by bolus size.[24]

The forward excursion of the hyoid bone is important in applying traction forces on the PES to achieve maximum opening.[25] Before the bolus arrives in the pharynx, muscles in the region of the PES that had been closed before swallow are relaxed by parasympathetic signals carried by CN IX to the brainstem. After relaxation, the PES is pulled open during hyolaryngeal movements. As the bolus continues its descent toward the region of the PES, it remains divided as it passes lateral to the larynx into the piriform recesses of the hypopharynx, where the bolus is rejoined as it enters the esophagus. Preference for bolus flow through the pharynx has been found in healthy normal patients. Seta et al.[26] studied the preference of bolus flow in 167 normal patients. Although all patients had bolus flow in both halves of the pharynx, 58% showed no difference, 35% had left dominance, and 7% showed right dominance.[26] In addition to PES relaxation and mechanical traction, the PES is distended by the driving force of the bolus. The neurologic and biomechanical processes required for distention and closing of the PES are summarized in Figure 2-6.

As the tail of the bolus passes the region of the PES, primary esophageal peristalsis begins as the PES closes. The airway reopens and the hyoid bone returns to its resting position. These activities signal the end of the pharyngeal phase of swallow. The timing of oropharyngeal swallowing events from the beginning of vocal fold closure to the reopening of the vocal folds at the end of the swallowing sequence is depicted in Figure 2-7. (For more detail on the activity of the PES during swallowing, see Chapter 6.) The structural and biomechanical aspects of the oral and pharyngeal phases of swallowing seen in the lateral and anteroposterior planes can be appreciated in a narrated version of a videofluoroscopic examination of swallowing (Video 2-3). Video 2-4 provides a narrated version of the normal swallow as seen by endoscopy.

Esophageal Stage

Before the bolus enters the esophagus, the esophageal lumen remains closed within the chest cavity under negative pressure. Pressures generated in the closed upper esopha-

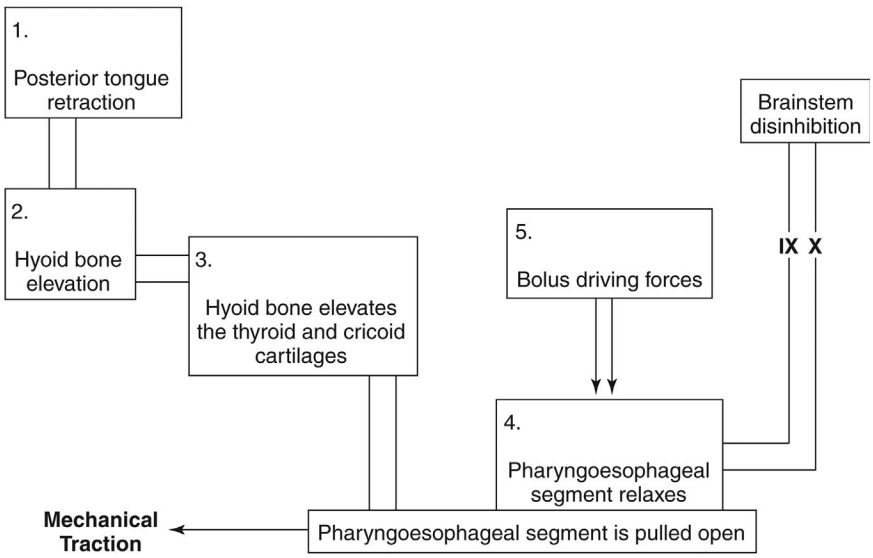

FIGURE 2-6 Schematic representation of the three mechanisms of the pharyngoesophageal segment opening. They include mechanical traction (1, 2, and 3), brainstem disinhibition (relaxation) (4), and bolus driving forces (5).

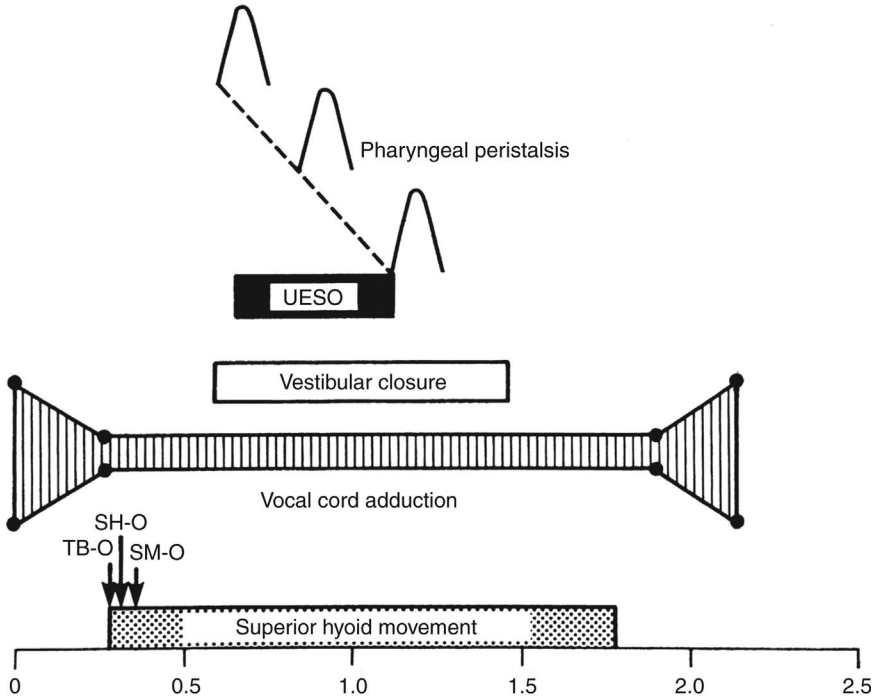

FIGURE 2-7 The relation of the time of vocal fold closure and hyoid bone elevation during a 5-mL barium swallow. Bolus transit through the pharynx and across the upper esophageal sphincter (UES) begins and ends, while the vocal folds are at maximal adduction. *SH-O,* Onset of superior hyoid movement; *SM-O,* onset of submental myoelectrical activity; *TB-O,* onset of tongue base movement; *UESO,* UES opening. (From Shaker R, Dodds WJ, Dantas RO, et al. Coordination of deglutitive glottic closure with oropharyngeal swallowing. *Gastroenterology*. 1990;98:1478.)

geal sphincter vary from 30 to 110 mm Hg, depending on patient age and the type of manometric catheter used to gather the data.[27] Esophageal swallowing tasks require an ordered pattern of function that depends on coordinated activities in three distinct zones: the proximal, striated muscle zone; the body; and the specialized smooth muscle of the distal zone. Bolus movement through these zones is characterized by an orderly, ringlike progression of contractions until the bolus enters the LES and the stomach. Liquid boluses, depending on **viscosity**, often precede this wave of contractions. The cervical portion of the esophagus works in conjunction with the hypopharynx, allowing the PES to fully relax and distend to accommodate bolus size. As the bolus enters the esophagus, a primary contraction wave (primary peristalsis) is triggered in the proximal, striated portion by vagal (CN X) efferent activity. This activity may be inhibited by multiple swallow attempts if the pharynx fails to clear its contents.[28] The motor activity in the cervical esophagus is rapid and gradually slows as it approaches the mid (level of the aortic arch) and distal esophageal regions.[29] Typically, the contraction force in the cervical esophagus is the strongest and is accompanied in time by a drop in pressure (relaxation) in the LES to allow the bolus to enter the stomach. Esophageal smooth muscle contraction (distal two-thirds) has a sequential behavior by which proximal activity successively inhibits the next most distal portion of the esophagus.[30] The bolus propagation pressures generated in the esophagus are typically measured by manometric techniques. A visual representation of primary peristalsis is presented in Figure 2-8. The radiographic representation of esophageal peristalsis is presented in Video 2-5. The patient is standing while swallowing a liquid and a semisolid bolus. The ringlike contraction waves of the esophageal lumen can be appreciated, as can the bolus entering the stomach through the LES. The primary peristaltic wave on the liquid bolus is followed by a secondary wave. It is apparent that the semisolid bolus flows at a slower pace.

Primary peristalsis is followed by secondary peristalsis. The secondary peristaltic wave follows the primary wave and is propagated by the bolus distending the esophagus. Its propagation may begin at any point in the esophageal body and often assists in primary transport of solid food boluses because the primary wave may fail to push the bolus to the level of the LES. Primary and secondary peristalsis are accompanied by longitudinal muscle contraction, resulting in shortening of the esophagus by its proximal attachments to the hypopharynx and distal attachments to the stomach (see Chapter 6 for a discussion of Zenker's **diverticulum** and esophageal shortening).

Tertiary contractions of the esophagus are random contractions that are not peristaltic (orderly) in nature and are inefficient in assisting in bolus transport. In general, they

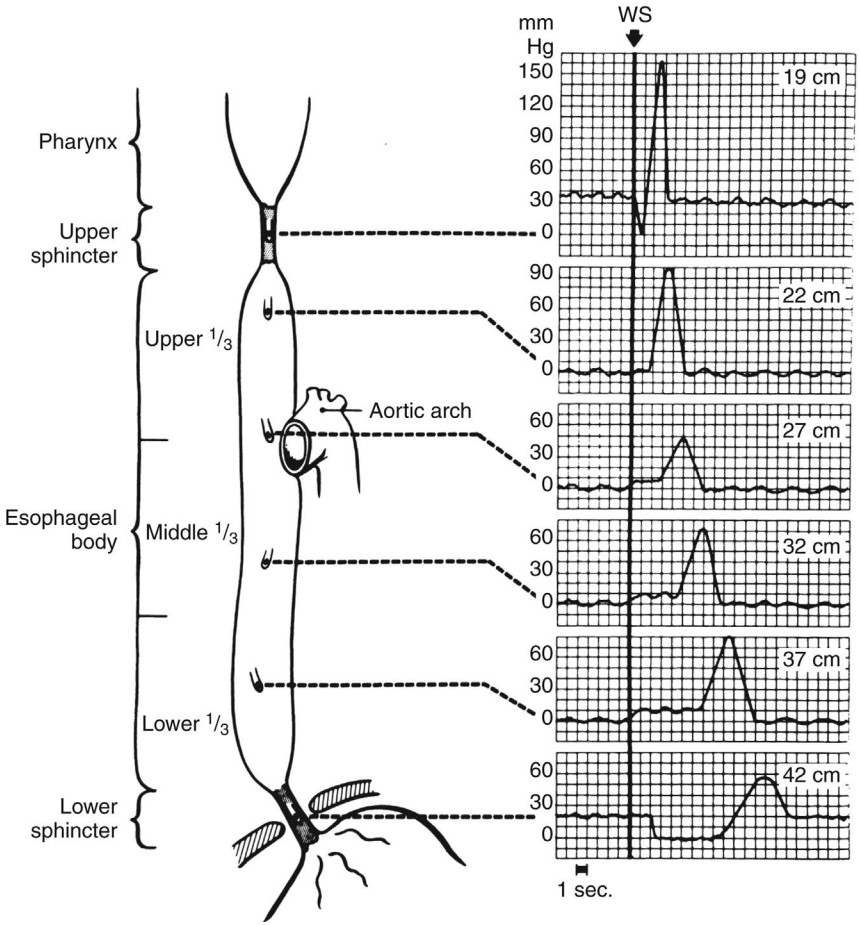

FIGURE 2-8 A manometric tracing of primary esophageal peristalsis. Pressure catheters are placed at various levels of the esophagus (19 cm from the incisors to 42 cm). Their representative measures of pressure are seen as peaks of activity on the right of the figure. Before the first pressure wave, a drop in pressure is seen from approximately 40 mm Hg (closed sphincter) to 0. This drop in pressure represents the opening and relaxation of the upper esophageal sphincter. The first primary esophageal contraction is the highest and therefore the strongest. As the bolus reaches the level of the aortic arch, the pattern of contraction is reduced because of the bending of the esophagus around the arch and the transition from striated to smooth muscle. Note that as the primary peristaltic wave begins, there is a corresponding drop in the pressure of the lower esophageal sphincter from approximately 25 mm Hg to 0 as it relaxes to await the oncoming bolus. A positive wave in the lower esophageal sphincter after this drop in pressure can be seen as a consequence of the sphincter closing.

occur independent of swallowing activity but have been reported to occur more frequently in older adults.[31] Tertiary contractions may be the result of air trapped in the esophagus, or they may result from irritation of the esophageal lumen such as from gastroesophageal reflux.

BOLUS AND DELIVERY VARIATION

Altering volume, texture, taste, and delivery method may affect the biomechanics of the normal swallow. Dietary modifications are frequently used in the treatment of patients with dysphagia (see Chapters 11 and 16 to assist in compensating for their deficits). The prescribed modifications in volume, texture (viscosity), and taste to facilitate normal swallowing are based on studies on the effects of these parameters on normal swallowing. Results from such studies are not uniform because of subject variability, measurement tools used (e.g., intramuscular and surface electromyography, ultrasound, manometry, videofluoroscopy), subject instructions (cue versus no cue),[32-34] type of bolus used and number of swallows tested,[35,36] and definitions of when specific biomechanical events begin and end. After reviewing 16 studies that investigated the temporal measurements of the normal swallow, Molfenter and Steele[35] concluded that while timing variations were apparent, they were the most stable for PES opening and the time between laryngeal closure and PES opening.[35] There are few published outcome data on the precise effects of volume, texture, and taste modification in patients with dysphagia, although these parameters are routinely modified in clinical

care. Lee et al.[37] prospectively enrolled a mixed group of 82 patients suspected of oropharyngeal dysphagia. The group was divided almost equally into a group that did not aspirate on thin or thick fluids and a group that aspirated only on thin fluids. Both groups were given 5 mL of a thin and thickened liquid. **Kinematic** analysis revealed that the thick bolus arrived earlier in the valleculae in the thin-aspirator group, resulting in longer laryngeal elevation times that delayed the opening of the PES. They concluded that changes in bolus viscosity in dysphagic patients do not affect biomechanics.

Volume and Biomechanics

Studies have shown that the normal amount of a liquid taken per swallow attempt may range from 10 to 25 mL depending on the test instructions, gender, type of cup, and body size.[38,39] Most studies that examine the effects of volume on swallowing biomechanics have studied bolus volumes that range from 1 to 20 mL. These studies have focused on the effects of volume on the movement of the hyoid bone. Movement parameters can include maximal displacement and the duration of movement, documenting total time and velocity. Some investigators have found minimal effects of hyoid displacement between small and larger boluses,[40,41] whereas others have documented larger total displacement with an incremental increase in bolus volume more prominent in men.[42,43] One study found that larger volumes had a greater effect on superior, rather than anterior hyoid, movement.[44] Other studies have not focused specifically on hyoid mechanics but rather on the biomechanical and pressure changes associated with oral and pharyngeal transit, duration of swallow apnea, and PES mechanics.

Lingual swallowing pressures with varying bolus volumes were unaffected as bolus size was increased,[45] suggesting that increased effort in oral-stage transit is not needed as the size of a liquid bolus increases. However, the tongue changed its contour to contain larger boluses before swallow onset.[36] Ekberg and Nylander[30] found no change in the speed of pharyngeal transit between small and large boluses.

A direct relation appears to exist between bolus size and the length of time the PES stays open and the onset time of relaxation. Cook et al.[46] studied 21 normal volunteers using concurrent videofluoroscopy, surface electromyography, and manometry with four different bolus sizes ranging from 2 to 20 mL. In general, as the bolus size increased the PES stayed open longer, and the onset of relaxation was closer to the onset of the anterior movement of the hyoid bone. These results suggest a possible relation between the sensory aspects of the oral stage of swallow (bolus volume) and the mechanics of the PES. These results provide further evidence of the interdependence of the stages of swallowing.

Viscosity

Studies of the effects of viscosity, taste, and bolus delivery on swallowing have focused on the changes in biomechanical effort that may be needed as these variables are changed. Measurement of swallowing effort is accomplished best with manometric techniques, allowing the investigator to document changes in swallow-generated pressures.

In general, researchers agree that swallow-generated pressures are more sensitive to changes in viscosity than are changes in volumes of the same consistency. As the consistency of the bolus becomes thicker, greater tongue pressures are needed to transport it from the oral cavity.[45] Studies have shown no differences in this effect between healthy, younger men and women.[47] The increase in generated tongue force in 62 healthy adults was highest at the point where the anterior tongue made contact with the hard palate.[48]

Pelletier and Dhanaraj[49] studied the effects of sweet, salty, sour, and bitter on swallowing pressures in 10 healthy, young subjects. Subjects were also asked to judge the palatability of each test substance from "extremely like" to "dislike." Although palatability judgments did not affect swallowing pressures, higher pressures (compared with water) were generated with the moderate-sucrose, high-salt, and high–citric acid test samples. In eight normal subjects using intramuscular electomyographic measurements, Palmer et al. concluded that a sour bolus provided increased activation of the suprahyoid musculature compared to a water bolus.[50] They concluded that the use of a sour bolus was justified as a treatment intervention, although the time of effect within a meal requires further investigation. Krival and Bates studied the swallowing pressures of 20 young women with three bolus types: carbonated, carbonation with taste, and water. Compared to water, the other two conditions showed a significant increase in swallow-related pressures.[51]

Straw drinking is a typical method to deliver a liquid bolus (Video 2-6). The patient takes multiple sips by straw. There are brief periods between each swallow when the airway opens briefly. Successful straw drinking requires adequate lip strength and intraoral pressures to draw the fluid into the oral cavity from the cup. In general, the airway must remain closed during sequential swallow attempts; therefore the biomechanical requirements may differ from single or multiple swallows from a cup. Daniels and Foundas[52] identified three distinct airway protection patterns during sequential straw drinking in 15 healthy young men, suggesting variation in how the upper airway is protected during sequential swallows using a straw with variations in the length of time the laryngeal vestibule remained closed (Clinical Corner 2-2).

Clinical Pearl: Because patients frequently drink liquid by straw, it is important to test this function when utilizing videofluorographic imaging studies.

CLINICAL CORNER 2-2 STRAW USE

A 75-year-old patient with respiratory disease was evaluated for difficulties swallowing liquids. Physical evaluation revealed that he had generalized weakness in the lips and tongue. He could take his liquids from a cup without any coughing episodes, but he had some coughing while using a straw. The patient reported that he was more comfortable using a straw and preferred it to the cup. The speech pathologist cut the straw in half, and the patient then took his liquids with the straw without any difficulty.

CRITICAL THINKING

1. Why might this patient have more difficulty using a straw than a cup?
2. Why might shortening the length of the straw improve his swallowing performance?

Younger and older normal subjects show hypopharyngeal accumulation on sequential straw swallows prior to bolus flow into the esophagus.[53]

Saitoh et al.[54] studied the effects of mastication on the normal swallow in 15 healthy, younger subjects. Boluses that required mastication usually were characterized by vallecular accumulation prior to the initiation of the swallow response because of weaker tongue-to-palate contact during mastication. Two-phase foods such as a liquid mixed with a solid may not be as easy to control in the valleculae and could put dysphagic patients at greater risk for aspiration.[52] Because viscosity often is manipulated as a treatment intervention, it is important to recognize that some ingested materials entering the oral stage requiring mastication may have their **rheologic** properties altered from the preswallow to the swallow-ready state. Hwang et al.[55] studied 20 normal subjects swallowing a cookie, banana, tofu, and cooked rice. As mastication cycles increased, mass increased and viscosity decreased only on the banana, tofu, and rice. The importance of this study is to remind clinicians that if they wish to recommend a certain food item because of its viscous, adhesive, or cohesive properties, they may need to remember that deformation of that item may provide a different rheologic profile than that associated with premasticatory measurements.

SWALLOW AND NORMAL AGING

In persons older than 65 years, some demonstrable changes in swallowing performance are attributable to age alone.

Clinical Pearl: Early studies have traditionally used 65 as a cutoff for defining olders; however, as the population lives longer, 75 and older may be more appropriate.

These changes may interact to decompensate swallowing. Some of these changes may appear as early as age 45 years.[56] These changes may be attributable to peripheral alterations in sensory perception, such as smell and taste, and decreased muscle strength secondary to changes in mass and contractility. Loss of muscle strength (force) and speed in older persons results in increased, but normal, swallow durations compared with younger cohorts.[57] Increased swallow durations were also found in healthy older adults who had more **periventricular white matter** lesions compared with healthy older adults without them.[58] Other structures involved in swallowing that may show changes in mass and contractility include the tongue, lips, jaw, velum, and lungs. Loss of elasticity in lung tissue coupled with reduced respiratory capacity and control may indirectly affect swallow because of the known interactions between breathing and swallowing. Brodsky et al.[59] found differences in respiratory patterns before and after swallows in older, healthy subjects compared with younger subjects. They speculated that this might be the result of a reduction in pharyngeal contraction pressures. Although these changes may not directly precipitate dysphagia, they may exacerbate conditions that are primary causative factors (e.g., neurologic disease). It is safe to assume that some aspects of swallowing are decompensated by normal aging and that the degree of compensation may enhance these effects in the diseased state. Robbins et al.[54] found that the ability of older persons to sustain **isometric** tasks involving the tongue may be different than in younger cohorts. These findings suggest that normal swallowing biomechanics may change under conditions of stress, such as might be imposed by hospitalization. Separating the effects of normal aging on swallowing from those in which disease is considered the primary causative factor presents a difficult clinical challenge.

Oral Stage and Aging

Tongue **hypertrophy** from fatty deposits and an increase in connective tissue results in a reduction of tongue mobility and tongue force as measured manometrically.[60] Some investigators have not found a significant difference in tongue pressure generation between normal, healthy older adults and younger cohorts,[47,61] although the time to reach maximum swallow pressures during swallowing was slower in older adults.[61] Significant differences are observed when comparing younger and older cohorts on their ability to generate maximum tongue pressures on nonswallowing tasks.[61,62] Youmans et al.[63] found that older women generated more pressure on swallows than men, and that both genders had a similar reduction of reserve strength, women greater than men. The difference between maximum isotonic pressures and the maximum pressure needed to complete a normal swallow seen in older persons, but not in younger cohorts, was discussed by Logemann et al.[62] They noted that the difference between these two measures in older persons represents a lack of pressure reserve and speculated that the difference may be important only when older persons need to rely on a pressure reserve, such as during illness. Fei et al.[64] compared 40 healthy

younger subjects younger than the age of 40 to 38 healthy persons older than 60. They confirmed that older persons did generate lower maximum isometric pressures, and that these differences affected swallow-generated pressures by bolus type that were not seen in the younger cohort. Using maximum pressure generation as a covariate when comparing the two groups, they concluded that the effect of age alone on water and saliva swallows did not account for the differences.[62]

Tanaka et al.[65] compared the frequency of swallows in a fixed time frame between healthy and semi-bedridden older and younger adults.[65] There were significant differences in swallow frequency between older and younger adults and between healthy and semi-bedridden older adults. Semi-bedridden older adults had significantly fewer swallows than age-matched older adults without disability. Because dysphagia is a more frequent occurrence in older adults, some investigators have postulated that swallow frequency measurements may be a useful tool to predict dysphagia and risk for aspiration (see Chapters 4 and 9).

Sensory changes related to aging include decrements in smell and taste,[66,67] although it is not clear whether these changes are attributable to primary loss of sensory receptors, poor oral hygiene, poor health, medications that reduce salivary flow, impaired nutritional status, or a combination of these factors.[68] Alterations in the ability to discriminate between materials with varying viscosity have been reported, although whether this is the result of primary sensory changes or a loss in the cortical representation of viscosity discrimination is not clear.[69]

Alterations in dentition necessitating the use of dentures may affect oral-stage mechanics. Ill-fitting dentures affect oral-stage preparation and may also interfere with access to the sensory receptors on the hard palate. For bolus materials that require mastication, older persons require additional time because of decreased jaw biting force.[70]

Pharyngeal Stage and Aging

Cinefluorography (in use before videofluorography) has shown that a decrease in the connective tissue in the suprahyoid musculature that supports laryngeal excursion may result in inadequate anterior laryngeal movement that secondarily reduces the opening of the PES.[71] Radiographic studies of healthy older persons show that pharyngeal constriction is normal compared with younger cohorts.[72] The restriction of PES opening is also evident on manometric studies, as evidenced by higher hypopharyngeal and intrabolus pressures in addition to increased pharyngeal contraction pressures.[58] In videofluoroscopic recordings of normal older and younger men, the older men showed significantly reduced anterior hyoid bone movement, resulting in less distention of the PES.[73] Failure of the PES to adequately distend results in shorter PES relaxation times and may explain increased higher pharyngeal contraction pressures as a compensation for shorter opening times.[74] High intrabolus pressures may be consistent with a restriction of flow through the PES and in selected older patients may explain reports of cervical dysphagia (see Chapter 6). Resting pressures within the PES are lower in older cohorts and may affect the competency of that barrier of swallowed contents that may move from the esophagus to the posterior pharynx.[75]

Videofluoroscopic swallowing studies comparing older and younger male cohorts revealed more instances of airway penetration after age 50 years.[76] Even though these threats to airway protection were evident, no subject demonstrated evidence of aspiration as a consequence of material entering the upper airway.

Studies have shown that the duration of the airway closure time in older persons is longer compared with younger cohorts.[56,77] This difference may be related to documented slower oral- and pharyngeal-stage transit times in older cohorts, resulting in a physiologic compensation to maintain airway closure and swallow safety. Changes in sensitivity in the protective reflexes in the upper airway may occur with aging. When calibrated puffs of air were delivered to the supraglottic larynx of older and younger subjects, laryngeal reflex (closure) responses were not as evident in the older subjects until the puffs of air achieved higher pressure levels.[78] Aviv et al.[78] suggested that this weaker response may indicate that the sensory mechanisms involved in upper airway protection may decompensate with normal aging.

Esophagus and Aging

In general, radiographic studies and manometrics document that esophageal motor activity decreases with age, but aging alone does not always explain dysphagic complaints. Reduction in the amplitude of esophageal contractions caused by smooth muscle thickening has been reported,[71] as well as delay in esophageal emptying and an increase in nonperistaltic contractions resulting in increased esophageal dilation and stasis[79] (review Clinical Corner 2-3).

CLINICAL CORNER 2-3 AGING ESOPHAGUS

An 82-year-old man went to his primary care physician and reported that it had become more difficult to swallow solid foods over the past few months. Six months previously, he started taking an antidepressant because he was not adjusting well to his wife's recent death. He denied choking episodes, so his doctor ordered a barium esophagram. The radiologist noted that with solid boluses, there was a mild delay of bolus flow at the level of the aortic arch and no evidence of a stricture.

CRITICAL THINKING

1. Did the patient's physician believe the swallowing problem represented new disease or normal aging?
2. Is delay at the level of the aortic arch normal? Why?

A separate chapter (Chapter 3) is devoted to other issues of aging persons and swallow performance.

NEUROLOGIC CONTROLS OF SWALLOWING

Neuroregulation of swallowing involves the activation of multiple levels of afferent and efferent pathways at different levels of the nervous system, including the cranial nerves, brainstem, cerebellum, subcortex, **limbic cortex**, and **neocortex**. Some aspects of swallowing appear to operate at a purely reflexive level, but it is more likely that swallowing does not represent a truly reflexive, brainstem-mediated response because food items are rarely swallowed the same way each time regardless of similarity in bolus type and size. As such, swallowing is believed to represent a more patterned type of neurologic response that can be influenced by control centers above the level of the brainstem. The peripheral muscles of swallowing contract sequentially but can be altered to accommodate the feeding activity. Therefore swallowing relies on both peripheral and central neurologic control systems that are activated differentially depending on the feeding circumstance. For instance, a person normally does not volitionally "think" about starting a swallowing response when eating but can "think" about swallowing when trying to swallow a pill. Although the mechanism is not totally understood, the act of swallowing potentially involves nervous system connections at multiple levels.

Peripheral and Medullary Controls

Pharyngeal swallow is initiated by sensory impulses transmitted as a result of stimulation of receptors on the fauces, tonsils, soft palate, base of the tongue, posterior pharyngeal wall, and anterior surface of the epiglottis.[80] These sensory impulses reach the NTS of the medulla primarily through the seventh, ninth, and tenth CNs. The efferent function is mediated through the ninth, tenth, eleventh, and twelfth CNs by the nucleus ambiguous (NA) (Tables 2-5 and 2-6; Figure 2-9). The highly integrated activities of swallowing depend on a combination of voluntary and involuntary control of the position of the lips, teeth, jaw, cheeks, and tongue—all mediated by multiple cranial nerves. Through innervation by the fifth CN, the masseter and pterygoid muscles provide the control of leverage, stabilization, and centering of the movable parts of the buccal cavity. Mastication depends primarily on CN V, whereas the muscles of the lips and cheeks depend on motor functions of CN VII. The extrinsic muscles of the tongue depend on the motor function of the CNs V and XII, except for the palatoglossus (elevator of the tongue root), which is innervated by CNs X and XI. All the intrinsic tongue muscles are innervated by CN XII. All the muscles of the soft palate are innervated primarily by CN X except the tensor veli palatini, which is innervated by CN V. The stylopharyngeus, a longitudinal muscle, widens the pharynx and is innervated by CN IX, whereas the palatopharyngeus is innervated primarily by CNs X and XII. The maxillary and mandibular sensory divisions of CN V are primarily involved in providing sensation pertaining to the lips, palate, teeth, inner mouth, and proprioceptive aspects of the muscles of mastication. The gag reflex and nasal regurgitation depend on the function (or dysfunction) of the glossopharyngeal and vagus nerves. Some controversy exists over the origin of the PES (cricopharyngeal) resting tone, which may not rely solely on the

TABLE 2-5 Afferent Controls Involved in Swallowing

Sensory Function	Innervation (Cranial Nerve)
General sensation, anterior two thirds of the tongue	Lingual nerve, trigeminal (V)
Taste, anterior two thirds of the tongue	Chorda tympani, facial (VII)
Taste and general sensation, posterior third of the tongue	Glossopharyngeal (IX)
Mucosa of valleculae	Internal branch of superior laryngeal nerve (vagus; X)
Primary afferent	—
Secondary afferent	Glossopharyngeal (IX)
Tonsils, pharynx, soft palate	Pharyngeal branch of vagus (X)
Pharynx, larynx, viscera	Glossopharyngeal (IX) Vagus (X)

TABLE 2-6 Efferent Controls Involved in Swallowing

Efferent/Stage	Innervation (Cranial Nerve)
Oral	
Masticatory, buccinators, floor of mouth	Trigeminal (V)
Lip sphincter	Facial (VII)
Tongue	Hypoglossal (XII)
Pharyngeal	—
Constrictors and stylopharyngeus	Glossopharyngeal (IX)
Palate, pharynx, larynx	Vagus (X)
Tongue	Hypoglossal (XII)
Esophageal	
Esophagus	Vagus (X)

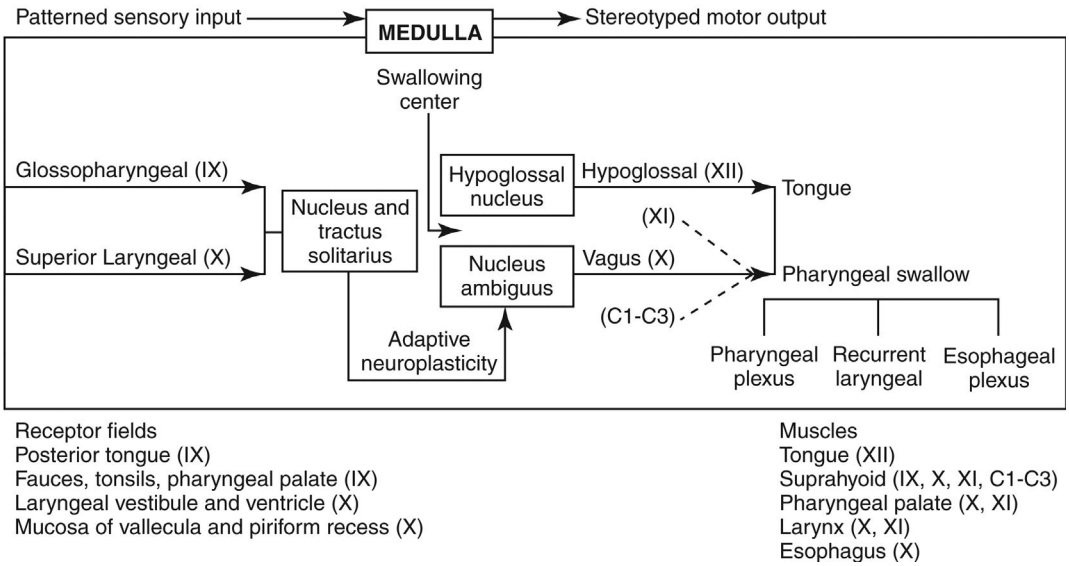

FIGURE 2-9 Conceptualization of the components of pharyngeal swallow as sensory-cued, stereotyped behaviors.

cervical sympathetic nervous system but may depend more heavily on vagal input for both contraction and relaxation.[81]

The literature refers to a swallowing center composed of key nuclei involved in afferent and efferent swallow control functions with interneuronal connections to respiratory centers in the medulla at the level of the **obex** of the fourth ventricle. This swallowing center has been defined as the dorsal NTS and ventral NA and the adjacent reticular formation.[82] In an excellent review of brainstem nuclei that are activated for swallow, Lang[83] identified medullary control centers based on swallowing stage; oral-stage activity is mediated by the trigeminal nucleus and reticular formation, the NTS receives sensory neurons for pharyngeal and esophageal function, and the NA and dorsal motor nuclei provide the motor input for the pharynx and esophagus. Based on current evidence, it is more likely that major contributions from neural activity in supramedullary structures, such as pons, mesencephalon, and limbic and cerebral cortices, also are involved in modulation of oral and pharyngeal swallowing and voluntary and involuntary behaviors.

The brainstem coordinates efferent impulse flow by way of the trigeminal, vagus, and hypoglossal cranial nerves to the muscles of the oropharynx, by way of CN X to the muscles of the hypopharynx, by way of CNs V and XII to the extrinsic muscles of the larynx, and by way of CN X to the intrinsic muscles of the larynx and esophagus. The cervical esophagus may receive two vagal efferent supplies from nerves within the neck. One comes from the recurrent laryngeal nerve (RLN) and another from the pharyngoesophageal nerve that rises proximal to the **nodose ganglion** or from an esophageal branch of the SLN. Such double innervation of the cervical esophagus in human beings has not been proved but might provide a margin of safety to prevent esophageal distention and reflux.

Sequentially timed discharges from the medulla result in movement of a bolus through successive levels of the esophageal musculature. Esophageal smooth muscle contractions have a sequential behavior by which proximal activity successively inhibits the next most distal portion of the esophagus.[84] Esophageal distention is signaled on visceral afferent nerves passing in the upper five or six thoracic sympathetic roots, presumably to the thalamus and inferior postcentral gyrus, where they may cause symptoms described as pressure, burning, gas, or aching. When such symptoms are described as pain, the referral patterns are based on sensory impulses from tissues innervated by somatic nerves that cross the corresponding spinal levels.

Motor fibers originating in the NA innervate the pharyngeal, laryngeal, and upper esophageal striated muscles. By way of the dorsal vagal nucleus, the NA also innervates the heart, lungs, and gastrointestinal tract smooth muscle.[85] Rootlets emerging from the medulla form the peripheral vagus, which exits the skull through the jugular foramen. Above the nodose ganglion, the vagus nerve sends branches to the pharyngeal plexus, which supplies the mucosa and musculature of the pharynx, larynx, and PES (Figure 2-10).[85]

The highly important branch of the vagus—the SLN—is sensory to the laryngeal mucosa and motor to the cricothyroid muscle. The vagus terminates as the RLN that loops around the aorta and returns to the larynx and hypopharynx. The RLN supplies muscles intrinsic to the larynx and is believed not to supply the cricopharyngeus, which apparently derives its innervations from the pharyngeal plexus.[86]

The neural control systems that subserve pharyngeal swallow are initiated by the action of CN afferents, but isolated central activation is not possible even though voluntary components exist. It appears that afferent impulses compe-

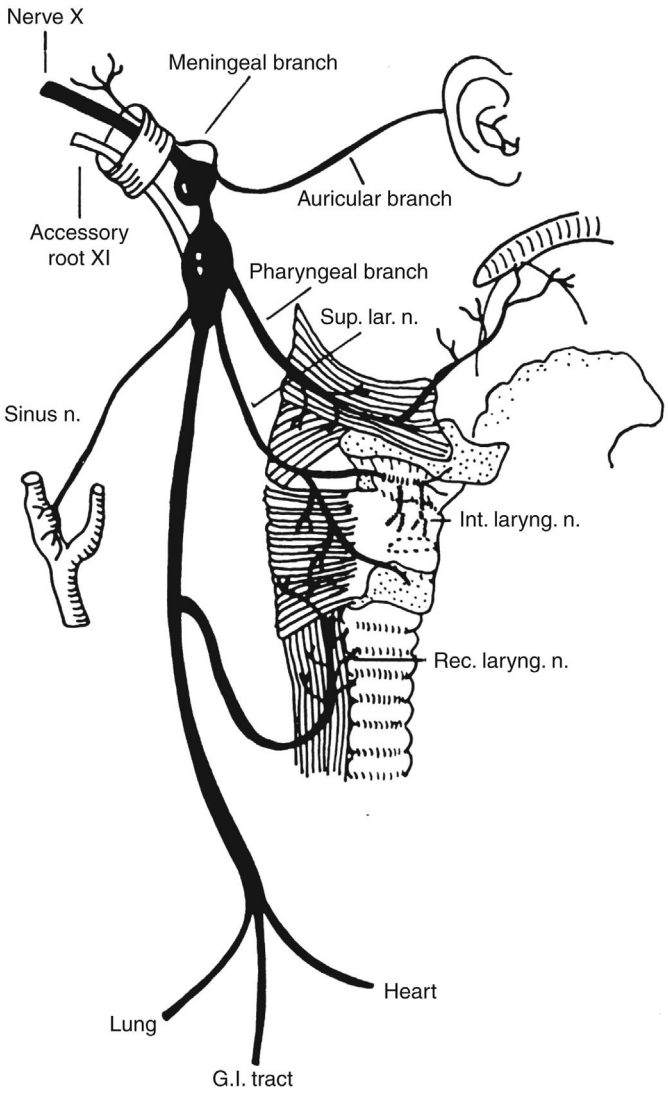

FIGURE 2-10 Schematic representation of the three peripheral branches of cranial nerve X: the pharyngeal branch to the region of the velum and pharynx; the internal and recurrent branches to the larynx; and the autonomic branch to the heart, lungs, and gastrointestinal (GI) tract. *Sup. lar.,* Superior laryngeal nerve; *Int. laryng. n.,* internal laryngeal nerve; *Rec. laryng. n.,* recurrent laryngeal nerve.

CLINICAL CASE EXAMPLE 2-1

An 86-year-old man recently had heart surgery. After surgery, he had a stroke affecting the premotor cortex of the left hemisphere. The man has a past history of depression treated with an antidepressant. He also had a history of Bell's palsy that affected CN VII in the upper and lower half of the left side of his face. He presented to the clinician with dysphagia. On examination the patient reported difficulty chewing and stated that food did not taste good. He noted considerable choking and a feeling that food was sticking in his throat. Physical examination of CN function revealed weakened right facial musculature from the stroke and weakened left facial musculature from the previous Bell's palsy. He was unable to make a tight lip seal because of bilateral CN VII nerve weakness. His tongue deviated to the right on protrusion, and range of motion was reduced (CN XII). Inspection of the oral cavity revealed moderate **xerostomia**. His voice was hoarse and breathy, although the velum rose evenly during testing of the gag reflex. His swallowing study showed poor bolus preparation, limited laryngeal elevation, pharyngeal stasis on pudding textures, and aspiration of thin liquids at the moment of swallow. It was concluded that the patient's poor bolus preparation could have been caused by multiple factors: tongue weakness, poor motor control from the involvement of a cortical motor area known to be important to bolus preparation, lack of taste appreciation from xerostomia (medication side effect), and probable involvement of the chorda tympani branch of CN VII (on the left). It was further concluded that his pharyngeal symptoms were attributable to poor laryngeal elevation caused by tongue weakness. This resulted in reduced opening of the PES, thus making it difficult for pudding to enter the esophagus, which caused the feeling that food was sticking in his throat. Liquids were aspirated because the vocal folds could not close fast enough because of the involvement of the recurrent branch of CN X that may have been damaged during the heart surgery, combined with the failure of the larynx to forcefully elevate and tilt forward because the tongue was weak. The pharyngeal branch of CNs IX and X was unaffected as evidenced by an intact gag reflex.

tent to initiate swallowing must conform to highly codified stimulus patterns that enter the NTS of the brainstem by way of its fasciculus and are relayed into the reticular formation, where connections exist to motor neurons lying in the nuclei of the fifth, seventh, and twelfth CNs and the NA.

Other brainstem motor neurons of interest in the neuroregulation of swallowing include the salivatory nuclei on either side of the genu of CNs VII and IX that provide saliva to the oral cavity and the dorsal motor nucleus of the vagus that innervates the esophageal smooth muscle (Figure 2-11).

The neuroregulatory brainstem mechanisms for pharyngeal swallow exist within the medullary reticular formation 1.5 mm from the midline on either side of the obex of the fourth ventricle. On each side of the midline is a site that communicates with the opposite side through cross-connections running behind the obex. As a result, bilateral symmetry of pharyngeal swallow is achieved. Each half of the medullary reticular formation exerts ipsilateral inhibition and excitation on appropriate motoneurons, with the exception of the inferior constrictor muscles, whose excitation is strictly contralateral.

Pharyngeal swallow involves a sequence of excitation and inhibition produced by several motor neuronal pools

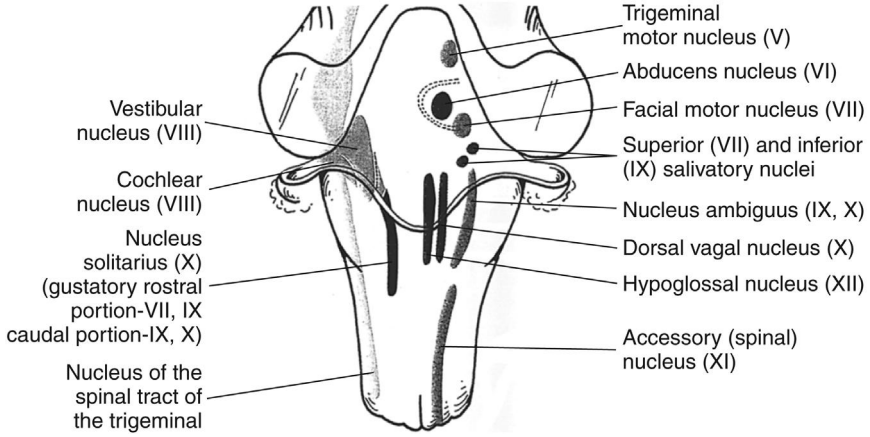

FIGURE 2-11 A view of the relations of the key brainstem nuclei involved in swallowing. Most nuclei are within close proximity in the dorsal and ventral parts of the medulla.

on each side of the brainstem.[82] Experimental unilateral destruction of the medulla eliminates swallowing in the ipsilateral musculature, except for the crossed pharyngeal constrictor muscle pathway. However, the responsiveness of the contralateral side to afferent input for the side of the lesion is still normal. For example, destruction of the left lateral medulla does not prevent right-sided swallowing if the left SLN is stimulated. This has immediate clinical relevance, especially in the case of unilateral destructive lesions to the brainstem.

The peripheral neural organization of swallowing has been largely elucidated by recording the electrical activity of involved muscles, beginning with onset of contraction in the mylohyoid and including concurrent activity in muscles innervated by CN V and those of the posterior tongue, superior constrictor, palatopharyngeus, palatoglossus, stylohyoid, and geniohyoid. These initiators constitute what has been called the leading complex.[85] Because the pharyngeal constrictor muscles form a continuous sheet of striated muscle, an overlapping "firing sequence" is observed beginning with the superior pharyngeal constrictor (the principal muscle), the middle pharyngeal constrictor, and the inferior pharyngeal constrictor, with distinct rostral (thyropharyngeus) and caudal (cricopharyngeus) components. The superior constrictor is active at the same time as the leading complex activity. A reconstruction of firing patterns leads to the conclusion that inhibition probably surrounds or brackets (in a time sense) the excitation of swallowing.[87]

The convergent supranuclear afferent systems (rostral to the brainstem) include the maxillary branch of CN V and CNs IX and X. These lead to the descending or spinal trigeminal system and the fasciculus and nucleus solitarii. The magnocellular part of the NTS receives input from the sensorimotor cortex and the ventromedial thalamus.[88] Some fibers of CNs IX and X project to the lateral cuneate nucleus (lateral portion of posterior spinal column), serving as a relay to the ventroposteromedial nucleus of the thalamus and limbic cortical system.

There are intrinsic and extrinsic neurologic controls for the esophageal components of swallowing. The extrinsic portion includes fibers that innervate the striated and smooth muscle portions of the esophagus. The striated (proximal third) portion of the esophagus is innervated by the recurrent branch of the vagus by the NA in the brainstem. Sympathetic and parasympathetic fibers leave the dorsal vagal nucleus in the brainstem, course through the NA, and innervate the smooth (distal two-thirds) muscle of the esophagus. The intrinsic portion of esophageal nervous innervation is supplied by a neural network that lies between the circular and longitudinal esophageal musculature, referred to as the mesenteric plexus.

Supranuclear Swallowing Controls

Normal oral feeding appears to involve brainstem reflex initiation by way of several types of peripheral excitation as well as a central facilitation of its limbic and cortical sensorimotor pathways. The importance of peripheral afferent stimulation cannot be underestimated because a bolus appears to be required to sustain repetitive swallowing activity. It is difficult to conceive of the act of swallowing as either purely reflexive (brainstem mediated) or purely voluntary (supranuclear mediated) because the repetitive nature of motor activity and potential differences in sensory inputs undoubtedly need to be modulated by higher cortical structures. It is conceivable that supranuclear connections to the brainstem swallowing center are necessary to continue, modify, and monitor swallowing activity when necessary as well as respond appropriately to different sensory stimuli. Conceivably, supranuclear systems are organized so that repetitive and overlearned efferent response networks (such as chewing) are maintained by a series of feedback loops that connect jaw activity to frontal motor

areas. These networks interact with interneurons that communicate with lower brainstem centers.[89] Other cortical centers appear to be reserved for modifications in swallowing activity depending on either the volitional nature of the task or changes in afferent information that may require alterations in motor performance. Kennedy and Kent[90] theorized that swallowing takes place at three different levels of nervous system organization: (1) a peripheral level that is linked to afferent bolus characteristics, (2) a subcortical level that organizes and executes learned patterns of efferent activity, and (3) a descending cortical portion that responds to any needed changes in motor activity based on perceived changes in the need to modify feeding behavior. Examples of volitional behaviors might include the need to eat faster, the need to expectorate an unwanted bolus, or perhaps the need to talk and masticate simultaneously. Investigations of these multiple pathways and centers have been conducted in human beings and animals with various laboratory techniques, including functional magnetic resonance imaging, electrical stimulation, ablation of suspected control centers, positron emission tomography, and transcranial magnetic stimulation. A complete understanding of the interrelations among centers during varying volitional and nonvolitional swallowing tasks remains speculative.

Regions of the cerebral cortex identified as active participants during swallowing are the anterior **insular cortex** with connections to the primary and **supplementary motor cortices**,[91] **orbitofrontal operculum**,[92] and the medial and superior portion of the **anterior cingulate gyrus**.[93] Interestingly, some of these areas appear to be active only for particular bolus types, such as water or a thicker liquid.[93] In animals, activation of the primary sensorimotor cortices during swallow shows both inhibitory and excitatory effects that depend on the perceived strength of the stimulus.[94] Using functional magnetic resonance imaging, Shibamoto et al.[91] found that a swallow attempt with the combination of water and a capsule activated limbic and neocortical structures as well as the cerebellum. Other studies have shown activation of multiple cortical and subcortical sites, including the basal ganglia.[95,96] From preliminary data on a small number of subjects, the right cortical hemisphere appears to be more active during volitional swallows, whereas the left is more active during reflexive activity.[97]

TAKE HOME NOTES

1. Swallowing is accomplished by a complex interaction of striated and smooth muscles whose sensory and motor components are carried by multiple cranial nerves.
2. The cranial nerves involved in swallowing send sensory information to the NTS. Motor components are organized in the NA. Together the NTS and NA compose the "swallowing center" located in the medulla of the brainstem.
3. Higher cortical control centers are capable of influencing the brainstem swallowing center.
4. The preparation and movement of a bolus during swallowing can be theoretically conceived as a series of valves that must open and close in a coordinated manner. This activity creates zones of high pressure around the bolus and zones of negative pressure below the level of the bolus. These pressure mismatches, together with gravity, create bolus flow.
5. Respiration ceases during swallowing. Protection of the airway to achieve a safe swallow is multifaceted. It is accomplished by primary airway closure at the level of the true and false vocal folds, laryngeal elevation, tongue base retraction, and epiglottic tilt.
6. The process of aging alone does not create dysphagia but may contribute to it, especially during disease-related decompensation.

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Part II
Dysphagia in Adults

Section 1
Causes and Characteristics of Dysphagia

CHAPTER 3
Aging and Dysphagia

Michael E. Groher

CHAPTER OUTLINE

OBJECTIVES

1. To provide an overview of the potential swallowing disorders in community-dwelling elders (CDE).
2. To present the prevalence of dysphagia in the CDE population.
3. To differentiate between normal aging, frailty, and sarcopenia.
4. To review the importance of detecting dysphagia in the CDE population.
5. To propose interventions as a method of preventing dysphagia and its complications in the CDE.

Recent literature has explored the possibility that as one ages, persons are more liable to develop symptoms of dysphagia that are not necessarily secondary to disease, but to changes in a generalized diminution of one's overall physical profile. Such changes may or may not impact swallow safety and are largely dependent on physical strength and speed of motor performance. Detection of those who may be at risk for developing swallowing disorders because of poor physical performance that accompanies advancing age may be important to avoid complications of undernutrition and aspiration pneumonia that potentially may shorten one's life. In a systematic review of 15 observational studies, Madhavan and colleagues concluded that there were three factors in the CDE population that increased the risk for dysphagia and its complications.[1] These include a history of clinical disease, advancing age (>70), and frailty accompanied by a reduction in activities of daily living.

The CDE population in the USA is currently estimated to be 12 million and should grow as the population of elders by 2030 is estimated to be 72 million.[2] Census bureau statistics suggest that the majority are living at home. Five

percent are living in long-term care or assisted living centers. Of those over the age of 85, the percentage climbs to 15. There is evidence in the group of elders who are living in the community that between 15% and 40% may be impacted by dysphagia that has not been fully identified.[1] Therefore, it is possible that between 11 and 29 million of elder persons, including 2 to 5 million of those in the CDE population, may be affected by unidentified dysphagia with its potential complications. In general, persons over the age of 65 in the United States are considered elderly, although this often depends on the survival statistics related to the level of healthcare provision in any given country. For instance, in Africa, where healthcare standards are lower, when one exceeds the age of 55 they are considered to be elderly.

This chapter will discuss the potential issues surrounding the group of elders who live in the community who may be at risk for dysphagic complications, and the potential need to obviate its secondary consequences such as pneumonia, undernutrition, and subsequent hospitalization.

TERMINOLOGY

Descriptions and definitions of CDE populations who may be dysphagic lack specific clarity that may make it difficult to understand the nature of their disorder due to their potential overlapping presentations.[3] As applied to a potential dysphagic CDE group, these include *successful aging*, *frailty, presbyphagia*, and *sarcopenia*.

Successful Aging

Rolfson and colleagues made the distinction between successful aging (those free of disease-related dysphagia), but who may have dysphagic complaints from normal changes in the aerodigestive tract, and those who may be considered frail with greater risk for dysphagia's complications.[3] Such changes may include loss of speed in bolus delivery due to changes in muscle strength, loss of dentition with secondary adaptations of diet with the risk of undernutrition, changes in smell and taste, and xerostomia as a consequence of medication side effects resulting in potential changes in sensory receptor activation (see Chapter 2). Failure of the muscles involved in swallow to contract rapidly and with sufficient strength may result in increased residue in each stage with subsequent spillage of contents into the airway. This may become particularly problematic as one progresses through the meal with accompanying muscle fatigue due to the effort required to finish.

Frailty

The term frailty suggests a loss of physical strength with an accompanying loss of ability to compensate for the weakness. Buchman and Bennett summarized a consensus conference that used the term *cognitive frailty* to include not only a diminution in physical status but also mental status changes including depressive syndromes, all signs short of being classified as demented.[4] Whether or not cognitive frailty is a precursor to dementia and the potential for dysphagia is unknown, but should be considered. Swallowing disorders secondary to dementing syndromes are well known (see Chapter 4). In its most severe form, frailty results in disability that may precipitate dysphagia and its complications.[5] While this progression seems logical, it does not account for those frail elderly who may not be disabled by disease-related etiologies, but are potentially at risk for dysphagia from the effects of aging alone. In short, frailty is on a continuum of severity that may or may not predict dysphagia. Presently, it is unknown what specific pattern of physical deficits might classify one as frail that predict reported or unreported symptoms of dysphagia.

As one ages, various presentations of frailty with or without dysphagia require investigation. Interestingly, the presence of dysphagia in those CDE classified as frail could be the precipitator of frailty or be a consequence of it. It is estimated that in those elders over the age of 65 that 5% will be classified as frail and 47% as pre-frail.[6] In those over the age of 85, 24% will be frail.[7] There is a lack of evidence documenting the prevalence of dysphagia in those who would be classified as frail and living in the community, although it is clear that some suffer from dysphagia. Documentation of prevalence is difficult because of underreporting and because few may seek help accepting their dysphagia as part of the normal aging process.[6,7]

Some have defined frailty when an individual has three or more of the following characteristics: weakness in hand grip, slow walking, decreased physical activity, self-reported exhaustion, and unintentional weight loss.[8] (See box 3-1) Cognitive status also may be affected, although empirical data and agreement on what constitutes cognitive status changes are needed. Muscle weakness with an accompanying loss of speed secondary to a loss of muscle mass has been

BOX 3-1 CHARACTERISTICS THAT DEFINE FRALITY

Hand grip weakness
Slower than normal walking speed
A decrease in normal physical activity
Self-reported exhaustion
Unintentional weight loss
Subtle changes in cognitive status

Attributed to changes in skeletal muscle fibers. There is evidence that as one ages, Type 2 fibers that are large and react quickly are replaced by Type 1 fibers that are small and slowly contracting.[9] These characteristics may or may not contribute to dysphagia and often are found in normal

aging. Dysphagia and its complications are more likely to appear when the person is decompensated by metabolic changes such as weight loss and undernutrition or by a hospitalization for any medically related complaint. In this circumstance, dysphagia may be transitory and disappear as the stressing agent, such as a medication that decompensated the person's health, is removed by treatment.[7]

Presbyphagia

Although not typically applied as a descriptor of dysphagia in the CDE group, Wakabayashi suggested the use of the term *presbyphagia* to describe those with normal age-related changes in the aerodigestive tract that may be associated with frailty.[10] By implication, this group may be at risk for dysphagia when there is a change in their physical or heath status. This group is to be distinguished from disease-related dysphagia in the CDE group such as from stroke or cancer versus a third group who evidence sarcopenia and accompanying dysphagia. This distinction may be important when planning treatment interventions.

Sarcopenia

The definition of what constitutes sarcopenia in older cohorts may vary dependent on one's country affiliation because of perceived differences in premorbid size (muscle mass) and accompanying strength.[10] In general, sarcopenia is a reduction of lean skeletal muscle mass with a marked loss of strength and speed of movement. It is thought to be a consequence seen most often in those who are considered to be frail, and therefore, has similar characteristics as discussed above. A related term, *dysnapenia*, has been proposed by Clark and Manini describing patients who show weakness and slowness of motor performance without loss of muscle mass.[11] The implication of this description is that slowness and weakness in performing motor tasks such as swallowing may serve as a precursor to the development of dysphagic symptomatology and therefore should be a potential marker of risk that requires monitoring. The European Consensus Group on sarcopenia divided sarcopenia into two categories: *primary sarcopenia* as a consequence of advancing age and *secondary sarcopenia* as a result of disease or undernutrition.[12] Mitchell and colleagues divided sarcopenia into three categories: presarcopenia characterized as loss of muscle mass, sarcopenia characterized by loss of strength OR physical performance, and severe sarcopenia characterized by loss of strength AND performance.[13] From these data, it is implied that those at most risk for the complications of dysphagia are those with severe and/or secondary sarcopenia. It has been suggested that dysphagia secondary to sarcopenia be considered a separate geriatric syndrome, especially in persons over the age of 70.[14] Looijaard et al. argued that measurement of a single performance on a given day may not be sufficient when classifying one with sarcopenia since measurements of motor performance on any given day can be variable.[15]

Using a screening device that included bioimpedance measures of muscle mass, hand grip strength, and gait speed calculations, Ishii and colleagues found that in 1971 Asian CDEs the prevalence of sarcopenia in those over the age of 65 was 14.2% in men and 22.1% in women.[16] The number of those with dysphagia in this cohort was not reported. Data on the number of CDE with demonstrated dysphagia who are classified with sarcopenia are lacking, partially reflecting the lack of agreement on terminology used for sarcopenia and for dysphagia. Nonetheless, there continues to be increased interest in those CDE who may either have *preclinical dysphagia* requiring preventive measures of intervention or dysphagia from frailty and/or sarcopenia that may require prevention or direct intervention.

UNDERNUTRITION

Loss of muscle mass with accompanying loss of strength and speed of motor performance may impact swallow safety, resulting in dysphagic symptomology in the CDE. For instance, loss of masticatory force, lip and tongue strength, in addition to loss of dentition, may be contributing factors.[17] Reduction of tongue pressure generation in the CDE population has been found to be associated with a low body mass index and the risk for dysphagia.[18] Diminution of tongue thickness as measured by ultrasound in the CDE has also been found to be associated with undernutrition and subsequent risk for dysphagia.[19] Loss of dentition appears to be particularly important. Sources of protein typically found in meat are too difficult to masticate and eventually become absent from dietary intake.[17] Additionally, dysphagic symptomatology may lead to negative changes in dietary preferences that in turn may impact nutritional integrity. In summary, poor nutritional status in the CDE may precipitate or complicate dysphagia.[20] As dysphagia with accompanying undernutrition worsens, immune defense systems are compromised, with resultant increased risk of aspiration pneumonia, increased hospital admissions, infection, and death. In 134 CDE over the age of 70 who were admitted to a geriatric hospital unit with pneumonia, 55% had clinical signs of dysphagia.[21] The older the patient, the more severe the pneumonia, functional status, and undernutrition.

INTERVENTION

With an increased awareness of the potential for dysphagia and its risk in the CDE comes an interest in how to intervene in an effort to avoid the complications of frailty that may result in sarcopenia, pneumonia, and death. In order to avoid these complications, there needs to be early detection of dysphagia risk by screening mechanisms, followed by

in-depth evaluation if screening is failed. Evaluations should suggest the most appropriate intervention that may include nutritional supplements, increased physical activity including strength training, aggressive oral care,[22] direct treatment and compensatory interventions targeted at the mechanism for dysphagia, or combinations of all four.

Detection

It has been suggested that closer monitoring of those CDE may be an appropriate use of resources. This is particularly important since there is a suggestion that those who may evidence dysphagia, regardless of severity, may not seek help. In this circumstance, it is easy to understand that changes in dietary habits can easily lead to loss of muscle function that further decompensates swallow, eventually leading to hospitalization for pneumonia. Hospitalization with the accompanying loss of physical activity may itself lead to a deconditioned state resulting in new dysphagic or increased dysphagic symptomatology.[23] Avoidance of this progression of events suggests early detection and a potential plan of intervention to prevent a potentially dangerous cycle of physical deterioration and its serious medical consequences.

Screening

The development of standardized screening tools that accurately identify those at risk for dysphagia in the CDE population is important because subsequent interventions will be implemented based on their results. Screening tools are designed to detect impairment that subsequently trigger further in-depth evaluation and possible intervention (see Chapter 9, screening test characteristics).

Ishii and colleagues developed a simple screening test for identifying sarcopenia.[15] Since those CDE with sarcopenia are assumed to be at increased risk for dysphagia, identification of sarcopenia is important. Sarcopenia was measured with bioimpedance measures of muscle, handgrip strength, and gait speed. The negative predictive value to detect sarcopenia using their screening tool was 97.2% for men and 93.0% for women.

Madhaven and colleagues developed a self-report screening test to identify what they termed *preclinical dysphagia* in a CDE population over the age of 60.[24] The theoretical framework used to develop the test items is presented in Figure 3-1.

The test was validated on 335 CDE who volunteered to be a part of the study. After test item reduction, 17 questions in five areas of inquiry remained: swallowing ability (coughing, food sticking, diet level), cognition, physical skills, oral health, and social support. The test was designed to be administered by any healthcare provider on any routine office visit. The test is able to differentiate between those at risk for dysphagia and those who are not. Interestingly, the two factors that predicted preclinical dysphagia were decreased communication/cognition and a reduction of physical activity. Items such as poor oral health and weight loss were not strong predictors. The authors suggested that these two variables appear to be more important in dysphagia secondary to known disease, rather than in those with symptoms in the absence of demonstrated underlying medical complications.

In a review of studies that addressed nutritional status and cognition in the elderly, Daradkeh and colleagues concluded that a thorough evaluation of one's nutritional status in the CDE is important since undernutrition often leads to

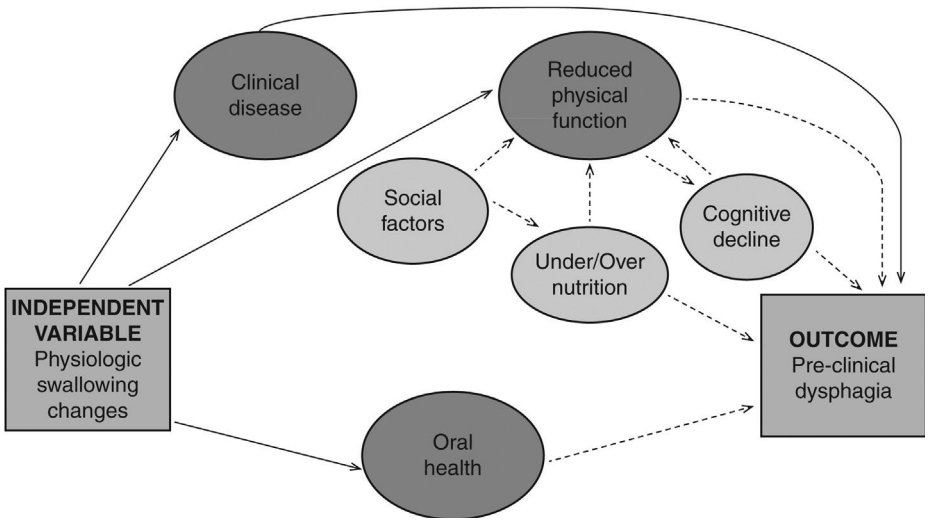

FIGURE 3-1 Proposed conceptual framework for the development of preclinical dysphagia in the CDE. Solid lines represent known causes, while dashed lines represent reported associations. (From Madhavan A, Carnaby GD, Chhabria K, et al. Preliminary development of a screening tool for pre-clinical dysphagia in community dwelling older adults. *Geriatrics*. 2018;3:90.)