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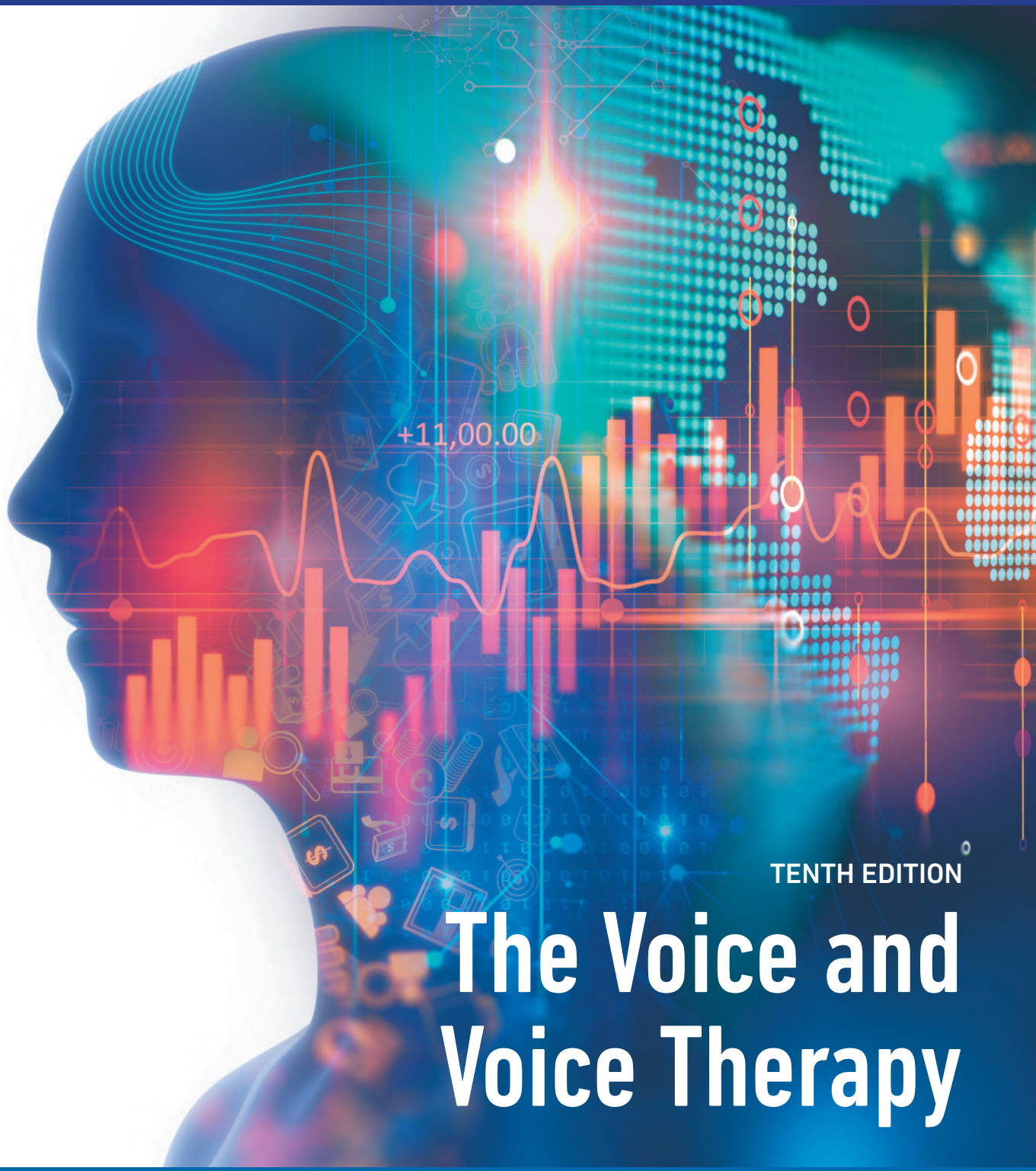

TENTH EDITION

The Voice and Voice Therapy

Daniel R. Boone | Stephen C. McFarlane
Shelley L. Von Berg | Richard I. Zraick

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Tenth Edition

The Voice and Voice Therapy

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Cover Design: Pearson CSC
Cover Art: WhiteMocca/Shutterstock
Editorial Production and Composition Services: Pearson CSC
Full-Service Project Management: Pearson CSC, Rowena Saycon and Sindhuja Vadlamani
Printer/Binder: LSC Communications, Inc.
Cover Printer: Phoenix Color/Hagerstown
Text Font: 9.5/13 Palatino LT Pro

Library of Congress Cataloging-in-Publication Data

Names: Boone, Daniel R., author. | McFarlane, Stephen C., author. | Von Berg, Shelley L., author. | Zraick, Richard I., author.
Title: The voice and voice therapy/Daniel R. Boone, Stephen C. McFarlane, Shelley L. Von Berg, Richard I. Zraick.
Description: Tenth edition. | Boston : Pearson, [2020] |Includes bibliographical references and index.
Identifiers: LCCN 2018060183 | ISBN 9780134894485 | ISBN 0134894480
Subjects: | MESH: Voice Disorders--diagnosis | Voice Disorders--etiology | Voice Disorders--therapy | Speech, Alaryngeal
Classification: LCC RF540 | NLM WV 500 | DDC 616.85/56--dc23 LC record available at https://lccn.loc.gov/2018060183

1 19

Pearson

ISBN-13: 978-0-13-489448-5
ISBN-10: 0-13-489448-0

From Dan to his sister, Barbara Boone Brueggemann, who typed the raw manuscripts of the first two editions of this textbook.

From Stephen to his wife, Patty, and his family.

From Shelley to her mother, Sarah Von Berg, an extraordinary speech pathologist and mentor.

From Richard to his wife, Amanda, and his twin daughters, Kaitlyn and Brooklyn, whose voices fill his heart with joy.

In Memoriam

DANIEL R. BOONE was born in Chicago, IL on October 30, 1927 and died suddenly on October 27th, 2018, three days shy of his 91st birthday. On the week of his passing, Dan was the keynote speaker at a voice and speech conference in Dallas. He had just published a new book featuring vignettes covering more than 60 years of aphasia, speech, and voice practice. Dan was on top of his game until the very end.

Dan graduated with a BA in speech-language pathology (with a minor in psychology) from the University of Redlands in 1951. From 1951–1953, he worked as a speech-language pathologist at the Long Beach VA Hospital with veterans of either WW II or the Korean War. He subsequently received both an MA (1954) and a PhD (1958) from Western Reserve University in Cleveland.

Dan had a series of academic appointments: Assistant Professor at Western Reserve University, 1960–1963; Associate Professor, University of Kansas Medical Center, 1963–1966; Professor, University of Denver, 1966–1973; Professor, University of Arizona, 1973–1988 and Professor Emeritus, 1988–present. For the first 20 years of his professional career, Dan was active clinically and in research with various neurogenic disorders. With the success of his voice text, *The Voice and Voice Therapy*, first published in 1971, he devoted the last 40-plus years of his career to voice disorders and treatment. As a writer, Dan authored over 100 professional articles and 18 books. In both Kansas and Colorado, he was elected president of that state's speech and hearing association. He served both as Vice President and President of the American Speech, Language, and Hearing Association (ASHA), receiving both Fellowship and Honors from that organization.

In the prologue to this edition, Dan presents a historical personal overview of the field of voice disorders and their treatments, as well as a rich history of the evolution of this textbook. We trust you will find it as interesting as we do.

It was our privilege to know Dan as a mentor, colleague, and dear friend. May his legacy be a lasting one.

Stephen, Shelley, and Richard

About the Authors

DANIEL R. BOONE celebrated his 60th year as a speech-language pathologist with the publishing of this tenth edition of *The Voice and Voice Therapy*. Dr. Boone held professorships over the years at Case Western Reserve University, University of Kansas Medical Center, University of Denver, and the University of Arizona (where he was a professor emeritus). Dr. Boone was a former president of the American Speech-Language-Hearing Association and held both a Fellowship and the Honors of that organization. He was the author of over 100 publications and was well known nationally and internationally for his many workshop presentations. Dr. Boone served as consultant to the Parkinson Voice Project in Dallas, TX, from 2011 until his death. Dr. Boone was perhaps best known for his love of his students and turning them on to the excitement of clinical voice practice.

STEPHEN C. McFARLANE is a professor emeritus at the School of Medicine at the University of Nevada, Reno. He was awarded ASHA Fellowship in 1982 and ASHA Honors in 1999. He received both his B.S. and M.S. degrees from Portland State University and his Ph.D. degree from the University of Washington. Dr. McFarlane has a long history of research interests in the area of voice disorders. Study of the outcomes from voice therapy and the development of new treatment techniques is of particular interest. His scholarly work has been published in dozens of books and journals, among them *Seminars in Speech and Language*; *American Journal of Speech Language Pathology*; *Phonoscope*; and *Current Opinion in Otolaryngology & Head and Neck Surgery*.

SHELLEY L. VON BERG teaches, practices, and researches in the areas of voice, dysphagia, and motor speech disorders in adults and children in the Department of Communication Sciences and Disorders at California State University, Chico, where she holds the rank of Professor. She earned her M.S. and Ph.D. degrees from the School of Medicine at the University of Nevada, Reno. She has presented on the assessment and intervention of neurogenic speech-language disorders nationally and abroad. She also teaches abroad on occasion. Dr. Von Berg has been published in the ASHA *Leader* Series; *Unmasking Voice Disorders*; *Language, Speech, and Hearing Services in Schools*; *Current Opinion in Otolaryngology & Head and Neck Surgery*; *Cleft Palate–Craniofacial Journal*; and *AAC Journal.*

RICHARD I. ZRAICK holds the rank of Professor and is the Director of the School of Communication Sciences and Disorders at the University of Central Florida. He was awarded ASHA Fellowship in 2014. He earned his doctorate at Arizona State University. Dr. Zraick is a clinician and teacher-scholar with over 30 years of experience in clinical practice and academia. His scholarship focuses on voice disorders, speech and voice perception, interprofessional education, healthcare simulation, and health communication. He regularly speaks about these topics at state, regional, national, and international scientific and professional conventions.

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Preface

New to This Edition

From the first publication of *The Voice and Voice Therapy* in 1971, the authors have attempted to maintain the book's relevance to students and voice clinicians alike. Each edition has incorporated the most current scientific evidence from a variety of disciplines that supports the behavioral approaches to voice assessment and intervention that are core to the text. In this tenth edition, videos demonstrating voice assessment and Voice Facilitation Approaches appear liberally throughout the chapters, accompanied by clinically relevant sidebars that guide the reader through a series of thought-provoking exercises illustrating the principles of voice anatomy and mechanics. The tenth edition also incorporates hundreds of Self-Check quizzes that map onto the chapters' learning outcomes. Each quiz is followed by feedback for all correct and incorrect answers. These pedagogical additions, along with the end-of-chapter Praxis Questions, help support the book's use in the classroom and clinic. The culmination of these elements ensures that students and their instructors, or clinicians and their colleagues, have the most current resources they need for voice and voice related disorders in a single package. Some chapter-by-chapter highlights include:

- Chapter 1, "An Introduction to Voice Disorders and Their Management," features current data on the incidence and prevalence of voice disorders in the general population and in specific populations. There are also expanded discussions of the classification of voice disorders and of the various approaches to managing the person with dysphonia.
- Chapter 2, "Normal Voice: Anatomy and Physiology Throughout the Lifespan," has been expanded considerably. It features a more comprehensive description of the anatomy and physiology of normal voice production. The detailed, colored anatomical illustrations and accompanying captions make it easy for the reader to "put it all together." This chapter can stand alone, thus eliminating the need for students, instructors, or clinicians to refer to outside source material.
- Chapter 3, "Functional Voice Disorders," presents practical approaches to identifying and managing behaviorally based voice disorders across the age spectrum. The chapter includes expanded discussions of excessive laryngeal muscle tension and the benign laryngeal pathology that may develop as a result, as well as voice disorders with a psycho-emotional basis or overlay. Special attention is given to the emotional and/or behavioral issues that might result in children with functional voice disorders. We also review evidence-based practice (EBP) studies supporting the value of our Voice Facilitating Approaches in treating persons with functional or psychogenic dysphonia.
- Chapter 4, "Organic Voice Disorders," presents practical approaches to identifying and managing organic voice disorders. The chapter includes significant updates on the risks for, and assessment and treatment of, gastroesophageal reflux diseases and laryngopharyngeal reflux. Endocrine changes and juvenile recurrent respiratory papilloma have also been expanded. We present current literature on the medical management of all organic disorders and on the role of the voice clinician in evaluation and therapy.

- Chapter 5, "Neurogenic Voice Disorders," presents the latest research in the behavioral, pharmacological, and surgical management of neurogenic voice disorders. Emphasis is placed on interventions for unilateral vocal fold paralysis and adductor spasmodic dysphonia. We also review numerous evidence-based practice (EBP) studies supporting the value of our Voice Facilitating Approaches in treating the respiration, phonation, and resonance subsystems in persons with dysarthria. Behavioral approaches that increase the respiratory support and intelligibility in Parkinson's disease are described and supported by strong clinical research.
- Chapter 6, "Evaluation of the Voice," has been updated extensively. It features the latest approaches to the auditory-perceptual evaluation of the voice and to assessment of voice-related quality of life. Multiple case studies illustrate both instrumental and noninstrumental assessment of the voice across medical and educational settings. These case studies also provide a framework for report writing and special considerations for voice populations across the lifespan. New figures illustrate instrumental approaches to identifying and quantifying voice and resonance disorders. Over a dozen new tables present the student and clinician with normative data across the lifespan for a variety of acoustic, aerodynamic, and related voice measures. This chapter can stand alone, thus eliminating the need for students, instructors, or clinicians to refer to outside source material.
- Chapter 7, "Voice Facilitating Approaches," continues to be the bedrock of this textbook. We have retained our core set of 25 Voice Facilitating Approaches and present the latest evidence-based practice (EBP) studies supporting their value in treating persons with dysphonia. Many of the cases illustrating the approaches have been updated to reflect the types of patients seen in current clinical practice, including applications for audiovisual feedback in therapy. We also discuss current literature on patient compliance and barriers to treatment.
- Chapter 8, "Therapy for Special Patient Populations," features expanded discussions of the identification and management of children, adolescents, and older adults with dysphonia. In particular, we discuss in greater detail the professional voice user, notably educators, and the management of dysphonia in this increasing population of patients. We discuss in more detail the management of dysphonia in children and adults with hearing impairment and in those with a variety of respiratory-based conditions. The section on voice, speech and communication style adaptations for those pursuing gender identity change has also been expanded.
- Chapter 9, "Management and Therapy Following Laryngeal Cancer," features detailed discussion of the medical management of patients with laryngeal cancer and the role of the voice clinician in evaluation and therapy. The illustrations and photographs that accompany the chapter are powerful learning tools for the student and clinician and can also serve as effective teaching tools for the patient. Communication options post laryngectomy are discussed in detail, including the artificial larynx, esophageal speech, and tracheoesophageal speech.
- Chapter 10, "Resonance Disorders," features both the instrumental and noninstrumental assessment of persons with disorders of nasal or oral resonance. Hypernasality, hyponasality, and assimilative nasality are discussed in depth, along with medical and behavioral approaches to each disorder. We have expanded the chapter's discussion of the team management of persons with cleft palate speech. Application of our Voice Facilitating Approaches to treatment of resonance disorders is illustrated.

Close to 1,200 references to other studies are included throughout the text. Cardinal literature from the past 40 years of voice science and care is included, as well as the most current literature from a variety of disciplines. Greater than half the references are new in this edition, with the majority representing advances in our field from the year 2012 to the present.

All new pedagogical elements supporting the use of the book for teaching include the following:

- The Learning Objectives at the beginning of each chapter have been expanded.
- Self-check multiple-choice quizzes are embedded in each chapter, complete with answer feedback for all correct and incorrect answers.
- Clinical Sidebars reinforce clinical application of material.
- Clinical Concepts at the end of select chapters reflect many of the learning objectives.
- Guided Reading exercises at the end of select chapters reference key clinical articles.
- Multiple-choice questions (Preparing for the PRAXIS™) at the end of select chapters help readers master the type of content covered in the Praxis II™ examination in speech-language pathology.
- An updated and robust instructor resource package, contains a wealth of supplemental materials, including PowerPoint slides for each chapter, an instructor resource manual, and a test bank.

We are fascinated by the human voice and intrigued by the art and science of voice therapy. As the great American poet Henry Wadsworth Longfellow wrote,

"Oh, there is something in that voice that reaches the innermost recesses of my spirit!"

We invite you to join us as lifelong students of the human voice, and we hope that while you read this edition, you will share the passion we had for writing it.

Daniel R. Boone
Stephen C. McFarlane
Shelley L. Von Berg
Richard I. Zraick

Acknowledgments

We would like to thank our reviewers: Ramesh Bettagere, University of Southern Mississippi; Myna L. Burks, Jackson State University; Kenneth Tom, California State University - Fullerton; we'd also like to thank Nancy Solomon, Walter Reed National Military Medical Center, for her suggestions regarding the book's content. This book contains material produced with our valued colleagues, as follows: (i) The Laryngeal Pathology Photo Gallery and Laryngeal Pathology Video Library are from the Voice and Swallowing Center in the Department of Otolaryngology—Head and Neck Surgery at the University of Arkansas for Medical Sciences (UAMS) (Little Rock, Arkansas). We are grateful to Dr. Ozlem Tulunay-Ugur for allowing their inclusion. (ii) The Voice Facilitating Approaches videos would not have been possible without the expertise of Mark Gandolfo and his team in Media Design and Production at Teaching and Learning Technologies at the University of Nevada, Reno. The medical illustrations were generated by the gifted hands of Maury Aaseng and Eiliana Hernandez. Sara Gray assisted with manuscript preparation. Douglas Kucera provided eagle-eyed manuscript scrutiny.

Daniel R. Boone
Stephen C. McFarlane
Shelley L. Von Berg
Richard I. Zraick

Prologue

The Voice and Voice Therapy was first published by Prentice-Hall in 1971, emerging before cellphones or any hint of help from digital technology. Much of the early voice literature was dominated by otolaryngology for "organic" voice problems and by psychiatry for "functional" voice problems. Much of the voice disorders literature in the middle-1960s and earlier references were based on this organic-functional dichotomy. Prominent in the speech pathology literature were two "organic" voice disorders: (1) resonance problems with focus given to hypernasality and cleft palate, and (2) the clinical management after laryngectomy centered on the teaching of esophageal speech.

The "functional" voice disorders literature in the 1960s was based on the premise of dysphonias being caused by continuous abuse and misuse of the voice. Reactive to such excessive vocal effort, vocal fold tissue changes could develop, producing such physical lesions as nodules, polyps, or contact ulcers. However, many children and adults with such hyperfunctional voice problems seen in our clinics showed no laryngeal tissue changes. Their common voice problems were alterations in loudness, inappropriate pitch, poor voice quality, and faulty resonance.

As physicians began referring more people with either "organic" or "functional" voice disorders, the typical speech-language pathologist (SLP) experienced a larger caseload of patients with voice disorders. With this growing demand for voice therapy, many SLPs increased their search for more knowledge about voice disorders and their treatment. Literature search was supplemented by increasing attendance at voice disorder workshops scattered around the country. I can well remember attending such workshops—receiving excellent descriptions of the vocal mechanisms, but sorely lacking about voice therapy strategies and demonstrations of therapy procedures.

As a young professor in the 1960s teaching voice disorders, I developed a voice therapy file for each of three clinical voice components: respiration, phonation, and resonance disorders. My sources for the therapy procedures came from the workshops, from a scant literature including clinical texts, from other SLPs and voice scientists in my profession, from drama and singing teachers, and observations of a few superior users of voice. Since there were no smartphones, emails, web pages, nor Google-type resources available for therapy suggestions, my therapy file showed me what to do and how to do it.

I used this therapy file and other parts of my voice course materials when writing *The Voice and Voice Therapy*. I selected 25 therapy techniques, labeling them in the text as "Facilitating Techniques." Four subheadings under each technique illustrated the kind of problem for which the approach could be useful, its procedures, a case history example, and an evaluation of the approach. The SLP would apply a therapy technique with the patient. If it worked, it would remain as part of the therapy regimen. If it were not helpful, it was replaced by another of the techniques. The 25 facilitating approaches became a feature of the first edition and have proved popular in all editions since. Incidentally, Prentice-Hall informed me many years ago that this therapy chapter was probably the most photocopied chapter in their total inventory of texts.

While the varying lists of 25 voice facilitating approaches have grown over the years, the relevance of *The Voice and Voice Therapy* has grown remarkably. As each new invited coauthor brought his or her expertise to the conceptualizing and writing of the book, the text continually grew. Now, this tenth edition presents cutting-edge references supporting the latest information on normative voice function and on the present clinical evaluation/management of voice disorders. Pictorial and video clips supplement well the narrative text. The organic–functional dichotomy was modified in many chapters to show a useful blend of management effectiveness. The therapeutic challenge of evidence-based practice and its impact on vocal management and therapy becomes clearer through the praxis exams following each chapter.

Thanks to our three coauthors, I can say without reservation that we may have produced one of the best voice disorders and therapy books ever written. I could not be prouder than I am of the tenth edition of *The Voice and Voice Therapy*.

Daniel R. Boone, Ph.D., Professor Emeritus
The University of Arizona

Chapter 1
An Introduction to Voice Disorders and Their Management

Learning Outcomes

After reading this chapter, one should be able to:

1.1 List and describe the biological, emotional, and linguistic functions of the larynx.

1.2 Describe the prevalence of voice disorders in the general population.

1.3 Describe the prevalence of voice disorders in specific populations.

1.4 Describe the kinds of voice disorders seen by speech-language pathologists.

1.5 Identify the various types of management approaches to voice disorders.

When we hear the long-awaited cry of the newborn infant, we are hearing the infant's first coordination of the outgoing use of the breath stream passing between the vocal folds. More important, we are hearing the confirming evidence that a new life has been born. From that moment on, the mother and those nearby listen closely to the baby's vocalizations. The mood states of anger, loving, and hunger can be heard in the baby's vocal shadings. Similarly, for a lifetime, the sound of the voice often carries more meaning than the words that we say.

Speaking is a distinctive way of using the larynx. The act of singing is even more so. Both speaking and singing demand a combination and interaction of respiration, phonation, resonance, and speech articulation. The best speakers and singers are often those persons who, by natural gift or training, or by a studied blend of both, have mastered the art of optimally using these vocal mechanisms. For most of the population, however, we count on our voices being there when we speak, sing, cry, or laugh with very little conscious effort required.

The larynx sits at the top of the airway, and it appears that its primary function in mammals (including humans) is protecting the airway from any kind of obstruction. Production of the human voice is a secondary function. While the focus of this text is on voice habilitation and rehabilitation, whatever we do in therapy must be consistent with the primary demands of the respiratory system. We will also see that, beyond breathing

problems, voicing difficulties can be the result of anatomic deviation and disease, or of emotions overriding normal vocal function, or of a change of vocal function resulting from misuse and overuse of vocal mechanisms.

The voice evaluation by both the physician and the speech-language pathologist (SLP) attempts to identify the causal factors of a particular voice disorder. This dual evaluation of voice documents and quantifies the elements for possible vocal change. If remediation of the voice problem is indicated, it will be by medical treatment or voice therapy alone. The focus of this text is on voice therapy by the SLP, regardless of the setting (school, clinic, hospital) in which he or she works with the patient with a voice disorder.

The Biological Function of the Larynx

A description of the biological aspects of laryngeal function provides us an early hint of how the biological demands of the airway and the larynx will always take precedence over artistic or communicative vocal production. When the brain signals the body's need for renewal of oxygen in the breath cycle, we automatically take in a breath. Oxygen-laden air flows through the passages of the upper airway into the lungs, followed by the outgoing carbon dioxide-loaded air flowing out of the body through the airway. This transportation of air into and out of the lungs is the primary function of the airway. Protecting the airway for an unobstructed passage of the air supply is the larynx. The primary biological function of the larynx is to keep fluids and foods from going into the airway (aspiration).

The larynx sits in a vital site at the front, bottom of the throat (pharynx), and at the top of the windpipe (trachea). As fluids and chewed food (bolus) come down the posterior throat, they are diverted from the lower throat (hypopharynx) into the open

esophagus, where they continue their journey through the esophagus down into the stomach. As part of the swallowing act, the larynx raises high in the neck (elevating the esophagus and trachea with it). As swallowing progresses, the tongue comes back, and the epiglottis, which acts as a partial cover, closes over the open glottis (see Sidebar 1.1).

Whenever the larynx plays this sphincteric role of closing off the airway to permit the posterior passage of liquids or food, the entire laryngeal body rises. Also, in fear situations, the larynx may reflexively elevate as part of its primary role in protecting the airway. Some voice patients, sometimes those with excessive fears, will attempt voice with the larynx in its elevated "protector" posture. Such excessive laryngeal elevation is not a good posture for producing a normal voice. This may be associated with the Polyvagal theory (see Porges, 2003).

Besides the elevating capability of the larynx, which helps prevent aspiration, airway closure is aided by three laryngeal muscle valves, described in Chapter 2 as the aryepiglottic folds, the ventricular folds (false folds), and the thyroarytenoid muscles (the true vocal folds). The most vertical of these valve pairs in the larynx are the aryepiglottic folds, which are considered part of the supralarynx. Under vigorous valving conditions, such as severe coughing, they begin to approximate (adduct). Below them are the ventricular folds; only during vigorous adductory activities, like the cough, do they approach each other. The lowest and more medial of the three laryngeal valves are the thyroarytenoid muscles, the true vocal folds. During swallowing, they always adduct to prevent possible aspiration. Also, the individual has fine control of the true vocal folds, with some capability of altering their shape, length, and tension, to produce various voicing changes.

When one breathes naturally, all three valve sites are open. The vocal folds separate further on inspiration, allowing a greater volume of air to pass through quickly; on expiration, they move slightly toward (adduct) one another. As further described in Chapter 2, voice is produced when the vocal folds adduct slightly together, allowing expired air to pass between them, setting the folds into vibration. This vibration produces voice (phonation). This phonation is then resonated through various sites of the vocal tract. The resonance of the voice begins with this vibratory sound in the larynx, traveling up through the pharynx and the oral and nasal cavities above. The voice we hear, then, is produced by a combination of respiratory activation, phonation, and amplifying resonance. Although the primary role of the larynx is to protect the airway, the larynx and voice in the human play an important role in emotional and linguistic expression.

Sidebar 1.1

To appreciate this movement, put your right index finger on your Adam's apple and swallow repeatedly. You should feel the larynx rising and gliding forward, and then gliding back and lowering to its resting position.

The Emotional Function of the Larynx

The infant seems to express emotions by making laryngeal sounds. Certainly, the caregiver can soon detect differences in the emotional state of the baby by changes in the sound of the baby's vocalizations: A cry from hunger may sound different from a cry of discomfort or the vocalization of anger. Contentment (after a full stomach or being held) can be heard in the relaxed cooing responses of the baby. From early infancy throughout the lifespan, the sound of one's vocalization often mirrors one's internal emotional state.

Our voice can sound happy or sad, contented or angry, secure or unsafe, placid or passionate (see Sidebar 1.2). How one feels affectively may be heard in the sound of the voice as well as in changes of the prosodic rhythm patterns of vocalization. Our

Sidebar 1.2

To appreciate this, try to express various emotional states using just your voice. See if the person who is listening to you can detect which emotional state you are trying to convey.

emotional status plays a primary role in the control of respiration; for example, nervousness may be heard in one's shortness of breath. Our emotional state seems to dictate the vertical positioning of the larynx, the relative relaxation of the vocal folds, and the posturing and relaxation of the muscles of the pharynx and tongue.

One's emotionality can be heard in the voice, a fact that can be threatening to the professional singer, or harmful to sales for the nervous salesperson, or embarrassing to someone who sounds like he or she is crying when actually happy. Our mood state can be harmful to voice. Many voice disorders are the result of various affective excesses; for example, a young professional woman attempts to use normal conversational voice when her larynx is postured in a high, sphincter-like position, resulting in a tight, tense voice. Her problem may be more related to unchecked and unrealistic fear than it is to faulty use of the vocal mechanisms per se.

Because emotionality and vocal function are so closely intertwined, effective voice therapy often requires the treatment of the total person and not just remediation of voice symptoms. Therefore, as we will see in later chapters, getting to know the patient is an important prerequisite to taking a case history or making an instrumental-perceptual voice evaluation. Voice clinicians have long recognized that the patient in the office may not resemble the same person in play or stress settings; the patient's voice will change according to his or her mood state. To assess voice realistically, we often have to observe and listen to the patient in various life settings.

The Linguistic Function of the Voice

Voice seems to hold spoken language together. From the primitive emotional vocalization that may color what we say to the skilled use of voice stress to emphasize a particular utterance, the voicing component of spoken language plays a primary role. It is not always what we say that carries the message, but how we say it.

New interest in infant vocalization is producing a fascinating literature. By the time typical one-year-old babies utter their first word, they have already used their voice in highly elaborate jargon communication. While human babies all seem to babble about the same way from four to six months of age, babbling becomes more language-differentiated beyond that age. That is, babies no longer sound alike after six months; rather, they begin to sound like the primary language they have been hearing. The melody of the parent language, or its prosody, begins to color the vocalizations of the baby.

These prosodic vocal patterns exist far beyond the individual word or segment. Such voicing is known as suprasegmental phonation. In young babies, suprasegmental vocalization far exceeds the voicing of actual word segments. As infants speak new words, they often place them in the proper place of their ongoing voicing rhythm. If they want to say "milk," they are far more likely to say the word at the end of a jargon phrase, such as "gawa na ta milk," rather than say the word in isolation. The jargon leading up to the word is suprasegmental voicing. The jargon voice carries a noncoded message with no specific meaning but seems to convey some general meaning by the overall sound of it. The mood and need state of the baby influence the sound of the vocalization.

Although jargon speech appears to diminish after the first 18 months of life, we continue to use suprasegmental vocalization in all aspects of spoken communication. We may add vocal stress patterns to augment the meaning of what we say. The actual words we say are only part of the communication. The "how we say it" is conveyed by

various vocal stress strategies, such as changing loudness, grouping words together on one breath, changing pitch level, changing vocal quality and resonance to match our mood. These stress changes of the suprasegmentals can be produced with or without intent. That is, if it serves our purpose, we can sound angry by talking louder, or we may sound angry despite our best efforts to hide our anger from our listener. Once again, the voice carries much of the message. The same words spoken or written may convey different messages (as any lawyer taking depositions will tell you) depending on the stress patterns given the words by the speaker, with or without intent.

Considering the role of the voice in both emotional and linguistic expression, it is no wonder that people with voice disorders may find themselves handicapped in their communication. A young girl with vocal nodules, for example, may have developed them in part from excessive emotional vocalization (such as constantly yelling). Once the nodules were developed, however, she may be unable to use the vocal suprasegmentals and stress patterns she had previously used with ease in communication. As anyone knows who has ever suffered a complete loss of voice from severe laryngitis, the lack of voice prevents you from being you. Whisper and gesture somehow do not carry the communication effectiveness that normal voice allows you to add to the words you say (see Sidebar 1.3).

While a primary role of the human larynx appears to be biological (guarding the airway), laryngeal voicing plays a vital role in the expression of both emotional and linguistic communication. When we add the voicing dimensions of acting and singing as laryngeal functions, we can truly appreciate the amazing artistic capabilities of the vocal tract (that a few people are fortunate to have and sometimes use). The role of the human larynx is obviously more complex and subtler than the way the larynx functions as an airway protector in most other mammals.

Sidebar 1.3

To experience this restriction, whisper the phrase *Today is Tuesday* two different ways—once as a statement and once as a question. Repeat these phrase contrasts using a normal voice. Which is more effective: whisper or voice?

Sidebar 1.4

Incidence is a frequently used epidemiological measure of rate of occurrence of new cases of a disease or condition. Incidence is calculated as the number of *new cases* of a disease or condition in a specified time period (usually a year) divided by the size of the population under consideration who are initially disease-free (Le & Boen, 1995).

Sidebar 1.5

Prevalence is a frequently used epidemiological measure of how commonly a disease or condition occurs in a population. Prevalence measures *how much of some disease or condition occurs in a population at a particular point in time.* The prevalence is calculated by dividing the number of persons with the disease or condition at a particular point in time by the number of individuals examined (Le & Boen, 1995).

Prevalence of Voice Disorders in the General Population

It is difficult, for several reasons, to establish normative incidence and prevalence data on voice disorders (see Sidebars 1.4 and 1.5). For example, voice can become temporarily disordered from a common cold that changes laryngeal tissue vibration and may fill resonating sinuses with infected mucus; almost everyone has experienced some voice change (phonation or resonance) as a result of a cold. Or some people experience voice changes from allergies. Therefore, if we were to take a large segment of the population and determine the present and past incidence of a voice disorder, our incidence reporting would be near 100%. Such incidence data would be meaningless. Rather, if we took a segment of a population, such as airline pilots, and looked back at the occurrences of hoarseness in a certain time period, we would determine some prevalence data for that particular group. Even these data would have far more meaning if there were a comparison between the pilots' voices and the voices of matched controls (matched, for example, by gender and age).

There have been only a handful of epidemiologic studies of the prevalence and risk factors of voice disorders in the general population (see Cohen and colleagues, 2012a;

Cohen and colleagues 2017; Cohen and colleagues, 2015; Watts and Knickerbocker, 2018; Verdolini and Ramig, 2001; and Best and Fakhry, 2011, for reviews). There has been substantial variability in reported prevalence estimates across the studies (Roy and colleagues, 2005). Conflicting definitions of voice disorder and methodological differences in procedures, and patient populations and sizes, are some of the causes of variations in the overall reported prevalence (Van Houtte and colleagues, 2010). The absence of acceptable epidemiologic data makes it difficult to precisely identify specific populations at risk, delineate the causes and effects of voice disorders, develop early screening procedures to identify those at risk, estimate societal costs related to voice disorders, and plan healthcare services designed to prevent or treat such problems (Roy and colleagues, 2005, p. 1988).

Roy and colleagues (2005) conducted a cross-sectional telephone interview survey of over 1,300 adults chosen at random. They discovered that nearly 7% of respondents had a voice disorder at the time of the interview and that nearly 30% had experienced a voice disorder at least once in their lifetime. About 7% of adults had missed work for more than one day because of their voice disorder. These findings are remarkably similar to those reported by Cohen (2010), who surveyed over 850 adults seeking medical care by their primary care physician for a variety of reasons. Cohen discovered that the lifetime prevalence of dysphonia in this population was 29%, and that the point prevalence (number of persons with dysphonia at the time) was just over 7%. Four percent of patients had experienced dysphonia for more than 4 weeks, and 73% had experienced dysphonia more than once. With over 300 million people in the United States, a 7% point prevalence rate means that approximately 20 million people have a voice disorder at any given time.

Cohen and colleagues (2012b) examined the prevalence and common causes of dysphonia as diagnosed by primary care physicians (PCPs) and otolaryngologists (ENTs) and evaluated differences in etiologies offered by these care providers. A retrospective analysis of data from a nationally representative administrative U.S. claims database of 55 million individuals revealed that about 1% of patients received a diagnosis of dysphonia. It was further discovered that females were almost twice as likely as males to be diagnosed with dysphonia, and that adults over the age of 70 years were two-and-a-half times more likely than those under age 70 years to be diagnosed with a voice disorder. The most frequent diagnoses overall were acute laryngitis, nonspecific dysphonia, benign vocal fold lesions, and chronic laryngitis. Some trends noted by Cohen were that prevalence decreased slightly after age nine years, and increased after the age of 30 years, peaking among those greater than 70 years old (p. 344). Also, within age categories, males had a higher prevalence rate in zero- to nine-year-olds, and females had a higher prevalence beginning with puberty and persisting until age 70 years or older (p. 345).

In a similar study using the same database, Cohen and colleagues (2012b) reported that nearly three-fourths of patients who received a diagnosis of dysphonia received medical treatment for 12 months or more. More women than men required follow-up treatment. These clinical researchers estimated that the total annual direct costs of caring for such persons ran into the hundreds of millions of dollars, which is comparable to other chronic disease states. The mean cost per patient over 12 months was between approximately $500 and $1,000. In a 2017 follow-up to this study, Cohen and colleagues noted that these costs nearly quadrupled among patients seeking prolonged periods (defined as 30 days or more) of voice-related care. Watts and Kinckerbocker (2018) added to this point by indicating that after a 30-day period of voice-related care by a

non-ENT physician, patients became more likely to engage in prolonged voice-related care. In an effort to identify how ENTs manage such patients, Cohen, Pittman, and colleagues (2012) surveyed 1,000 ENTs about their practice patterns. Approximately 300 physicians responded, and they reported that prescribing medication to control laryngopharyngeal reflux disease was their most common approach, followed closely by referral to speech-language pathology for voice therapy. The most common laryngeal conditions leading to voice therapy referral were vocal fold nodules and muscle tension dysphonia. In 2015, Cohen and colleagues went on to reveal that of 168,444 adults who visited an ENT, 6.1% of these patients sought a videolaryngostroboscopic evaluation. Half of these individuals received a change in their laryngeal-related diagnosis—indicating the importance of ENT input in the diagnosis and treatment of laryngeal disorders.

Prevalence of Voice Disorders in Special Populations

While the information already summarized in this chapter is helpful, a look at the prevalence of voice disorders in particular segments of the population may be more meaningful than looking at the population as a whole. Doing so may help clinicians focus their prevention, screening, and intervention efforts. In the following sections, we summarize the prevalence data on voice disorders in children and older adults, and across occupations (see also Chapter 8).

Prevalence of Voice Disorders in Children

The actual prevalence of voice disorders in children is difficult to determine. As summarized in McKinnon and colleagues (2007), a variety of different methods have been used to establish the presence of a communication disorder. The methods depend on both the age of the individual and the setting. Both direct (face-to-face assessment including screening and diagnostic techniques) and indirect (parent or teacher report) methods have been used extensively. Parent report measures are commonly used with preschool-age children while teacher report measures are commonly used with school-age children. Lower prevalence rates are typically derived from indirect methods in comparison to direct methods. Methodological challenges notwithstanding, it is very important that children be identified because it has been shown that a communication disorder such as dysphonia can negatively affect academic achievement and affect vocational choices later in adulthood (Sedehi and colleagues, 2016; Şenkal & Çiyiltepe, 2013; Pack, 2008; Ruben, 2000).

A number of researchers have concluded conservatively that between 6% and 9% of school-age children may have a voice disorder (Andrews & Summers 2002; Carding and colleagues, 2006; Cornut and Troillet-Cornut, 1995). Some studies, such as the one conducted by Duff and colleagues (2004), have reported a slightly lower prevalence rate of 4%. Other studies have reported that the prevalence rate may actually be as high as 20% to 30% (Angelillo and colleagues, 2008; Boyle, 2000; Faust, 2003; Silverman & Zimmer,

1975). According to the U.S. Census Bureau (2010), there are approximately 74 million persons between the ages of 0 and 18 years; this is approximately 25% of the total U.S. population. Using a conservative prevalence rate of 6% to 9% suggests that there are between 4.5 and 6.6 million children who may have a voice disorder (for information about voice-disorder prevalence rates for other countries, see Akif Kilic and colleagues, 2004; Milutinović, 1994). Perhaps even more alarming than this actual number is the fact that most of these children are not receiving voice therapy (Andrews & Summers, 2002). This is consistent with school SLPs who report a much higher incidence. In 2017, Bainbridge and colleagues analyzed data from the National Longitudinal Study of Adolescent to Adult Health and conducted a cross-sectional analysis which revealed that, of 14,794 young adults aged 24 to 34 years, 6% experienced a voice disorder. Of these individuals who experienced a voice disorder, comorbid conditions included anxiety, tinnitus, and hypertension. This work, while important, highlights a lack available data on the incidence and prevalence of voice disorders among students in middle school and high school.

Prevalence of Voice Disorders in the Elderly

According to the U.S. Census Bureau (2010), there are approximately 40 million persons age 65 years or older, comprising about 13% of the total U.S. population. In spite of this large number (which is expected to grow), there are very few studies of the prevalence, risk factors, and psychosocial impact of dysphonia in the elderly. Studies of the incidence and prevalence of dysphonia in this population have been restricted solely to investigations of those seeking treatment; thus, the true prevalence of voice disorders in the general elderly population remains largely unknown. Three separate studies examining the prevalence of voice disorders in a non-treatment-seeking population over age 65 years (Cohen & Turley, 2009; Golub and colleagues, 2006; Roy and colleagues, 2007) discovered that between 20% and 30% of persons completing a survey about their voices reported having a current voice disorder, and that more than half these persons experienced significant quality-of-life impairment resulting from their dysphonia. The prevalence data reported by Roy and colleagues and Golub and colleagues are supported by the finding reported by Cohen and colleagues (2012a) that adults over the age of 70 years were two-and-a-half times more likely than those under the age of 70 years to be diagnosed with a voice disorder; in fact, adult males over the age of 70 years were the most likely persons to experience a voice disorder. The risk of an elderly person having a voice disorder is greater if the person also has a hearing loss, and having either disorder is more likely to lead to depression (Cohen & Turley, 2009). Similarly, Roy and colleagues (2016) found that a variety of variables including physician type, age, gender, geographic location, and comorbid conditions were significantly affiliated with voice disorder diagnoses within populations aged 65 years or older.

Prevalence of Voice Disorders in Teachers and Student Teachers

It has been estimated that 5% to 10% of the U.S. workforce are "heavy occupational voice users" (Titze and colleagues, 1997). Within this group are over 3.7 million primary and secondary school teachers—the largest group of professionals who use their voice as a primary tool of their trade (U.S. Department of Labor, Bureau of Labor Statistics, 2016; U.S. Census Bureau, 2010). As described in Vanhoudt and colleagues (2008,

p. 371), these teachers are at risk for having a voice disorder due to vocal load (hours of voice use, number of communication partners), physical factors (physical condition and mucosal problems), psycho-emotional factors (stress, emotions, and work pressure), and environmental factors (acoustics, humidity, and environmental pollutants) (see also Da Costa and colleagues, 2012; Ferrand, 2012).

The prevalence of voice disorders in U.S. teachers has been studied quite extensively, yielding prevalence rates ranging from 4% to 57.7% or higher (Munier & Kinsella, 2008; Roy and colleagues, 2004a, 2004b; Martins and colleagues, 2014), with most studies indicating a prevalence rate higher than that for comparable persons from the general population. For example, Roy and colleagues (2004a) surveyed about 1,200 teachers and 1,300 nonteachers and reported a prevalence rate for teachers of 11% compared to 6% for nonteachers; furthermore, 57% of these teachers had experienced a voice disorder at some point in their lives, compared to 26% for the nonteachers. Of those teachers who had experienced a voice disorder at some point in their lives, only 14% sought help from physicians or SLPs. Roy and colleagues also examined risk factors, and discovered that being a teacher, being a woman, being between 40 and 59 years of age, having 16 or more years of education, and having a family history of voice disorders were each positively associated with having experienced a voice disorder. In a related survey study of the same two groups, Roy and colleagues (2004b) discovered that teachers, compared with nonteachers, had missed more workdays over the preceding year because of voice problems and were more likely to consider changing occupations because of their voice (p. 542). To address this issue, vocal training programs targeting vocal health in teachers have been developed (see Roy and colleagues, 2001, for an example). The problem of voice disorders in teachers is not limited to those working in the United States; similar problems have been reported by teachers working in other developed and developing countries (see de Medeiros and colleagues, 2011, for a review; see Seifpanahi and colleagues, 2016, for preliminary data regarding developing countries such as Iran).

Student teachers are also at risk for developing a voice disorder. Thomas and colleagues (2007) compared the incidence of voice complaints in student teachers versus students from nonteaching disciplines, and reported an incidence rate of 17% for student teachers compared to slightly less than 10% for their peers. Timmermans and colleagues (2005) reported that student teachers experience significantly more symptoms of dysphonia than their peers, including throat clearing, coughing, hoarseness, pain in the throat, fatigue, and difficulty being heard. One study reported that 90% of future teachers who experienced voice problems during their education experienced voice problems later in their teaching career (de Jong and colleagues, 2006). To address this issue, individual and group training programs have been developed to address vocal health in student teachers (see Simberg and colleagues, 2006, Timmermans and colleagues, 2005, Van Leider and colleagues, 2010, and for examples). In Chapter 8, we discuss the issue of voice disorders in teachers and student teachers in greater detail.

Prevalence of Voice Disorders in SLPs and Future SLPs

SLPs are professionals who also rely on healthy voices. SLPs and those in training to be SLPs have high vocal loads and often use their voices in emotional or stressful contexts, such as therapy, counseling, conferencing, and public speaking. They must also demonstrate and model appropriate voice use. Gottliebson and colleagues (2007) investigated the prevalence of voice disorder in 104 U.S. student SLPs (94% female) and

reported that 12% had perceptual features of dysphonia in their habitual voice—a higher prevalence rate than that of the general population of students, and one that is similar to that of student teachers (see Thomas and colleagues, 2007). These results were later confirmed by Tafiadis and colleagues (2017), who found that speech-language pathology students generally had a significantly higher score on the Voice Handicap Index (VHI) than those of students in other health professions. Because of the risk to this population, intervention programs have been developed to address vocal health and performance (see Van Lierde and colleagues, 2011, 2012, and Meerschman and colleagues, 2017 for examples).

Kinds of Voice Disorders

When we talk about "kinds of voice disorders," we are usually talking about classifying the cause of voice disorders. This kind of classification over time has led to the historic causal simplification: the organic and functional dichotomy. In most classification systems, there is a mixture of etiologic causations and descriptive names of conditions, such as *cancer* as a causative form of an organic disorder, and *dysphonia* as the name of a condition that may have organic or functional origins.

Let us look at a few literature presentations of voice disorder classifications. The Classification Manual for Voice Disorders–I (Verdolini and colleagues, 2006) describes seven distinct causal classifications: laryngeal problems related to structural

(1) pathologies, (2) inflammatory conditions, and (3) trauma or injury; (4) systemic conditions; (5) nonlaryngeal aero digestive disorders; (6) psychiatric-psychological disorders; and (7) neurological disorders. The manual also offers two other categories ("other disorders" and "undiagnosed"). Under each of the causative categories is specific information about etiology, behavioral description of the voice disorder, severity criteria, and so on, all of which can be most helpful to the SLP. Such diversity of nomenclature generates many categories of voice patient groups, however, complicating the task for generating evidence-based data.

Addressing the need to develop useful outcome data, an Australian diagnostic system (Baker and colleagues, 2007) presents a modified classification system as part of an inter-rater reliability study. Baker and colleagues basically modify the historic two broad categories of voice disorders, organic and functional. The organic classification of voice disorder causation combines structural changes of the vocal folds or cartilages or by "interruption of neurological innervations of the laryngeal mechanism." Such a combination of organic problems under one heading may present a real hindrance in evaluating treatment outcome effectiveness. Study of clinical effectiveness would probably be simpler if the organic causations and neurogenic categories were separated. Similarly, the functional voice disorder categories might be separated into two separate classifications: psychogenic voice disorders (PVD) and muscle tension voice disorder (MTVD, otherwise known as muscle tension dysphonia). While both PVD and MTVD are both functional voice disorders, they have distinctly different origins.

A different etiologic classification for causes of voice disorders was introduced by Stemple (2007) who presented these four pathology classifications: congenital laryngeal pathologies, pathologies of the vocal fold cover, neurogenic laryngeal pathologies, and pathologies of muscular dysfunction. The first category, congenital, includes only five relatively rare congenital conditions, such as congenital web or congenital cyst. The vocal cover category lists 15 various laryngeal conditions, from nodules to papilloma to sulcus vocalis. The neurogenic category does not include degenerative diseases and their possible influence on vocal fold function.

In this text, we have made an effort to use a classification system that allows easy identification of a voice disorder population—one that will promote valid and reliable clinical research. There appear to be three distinct categories of voice problems, one of them functional in causation and two of them organic/neurogenic in origin. Under the first category, functional voice disorders, there appears to be two subcategories: muscle tension voice disorders, which can develop from excessive muscle tension and use, and psychogenic voice disorders, which are caused by psychosocial factors. The second category, organic voice disorders, includes any organic structural deviation that affects vocal fold function. The third category, neurogenic voice disorders, relates to neurological conditions that cause faulty vocal fold closure from either paralysis (or weakness) or from neurological disease. In summary, these three categories—functional voice disorders, organic voice disorders, and neurogenic voice disorders—appear clear and distinct.

Functional Voice Disorders

There are basically two types of functional voice disorders: muscle tension dysphonia and psychogenic voice disorders. Each has a different etiology requiring different management and therapy approaches.

MUSCLE TENSION DYSPHONIA. Muscle tension dysphonia (MTD) is the most common voice disorder seen in both children and adults. MTD is the most common manifestation of vocal hypertension—using too much muscular effort to phonate. This overuse of the respiratory, laryngeal, and supralaryngeal systems when voicing usually begins gradually. After talking awhile, the individual may begin to experience some pain and discomfort in the throat area. Before any dysphonia can be heard in the voice, the patient may experience fatigue and effort that increases with voice use. Children's loud voices and yelling over time seems to produce some hoarseness of voice. Also, adults may experience more hoarseness after prolonged voice use. Physical examination of the larynx shows no organic pathologies, and the voice problem is considered functional in origin. Baker and colleagues (2007) classify the patient's discomfort coupled with hoarseness (and normal laryngeal structures) as representing primary muscle tension dysphonia. With continued misuse of the voice over time, however, children and adults may develop secondary tissue changes related to this vocal hyperfunction, such as vocal fold changes (swelling, thickening, nodules, polyps, and so on). Both primary and secondary forms of MTD can usually be minimized with voice therapy that is designed to reduce excessive tension by restoring the normal balance among the respiratory, phonatory, and resonance systems.

PSYCHOGENIC VOICE DISORDERS. Some children and adults experience severe emotional trauma or conflict that shows itself in some kind of physical alteration. Reaction to the trauma may manifest itself in a complete loss of voice, often labeled as a conversion aphonia. More commonly, the emotional reaction may show itself in a functional dysphonia—a hoarseness that has no physical cause. Or excessive emotionality may cause an alteration of voice pitch or speaking style that has no physical cause. In aphonia, the patient usually continues to whisper and attempts to speak. The complete lack of voice prevents the patient from having normal conversational interactions and may have devastating effects vocationally. Clinically, we have seen aphonic teachers unable to teach, an airline pilot unable to fly, a politician unable to continue a political campaign. Similarly, patients with a psychogenic dysphonia may suffer severe social and vocational limitations. Conversion-type voice problems vary dramatically, perhaps showing only in particular emotional or physical situations. For example, a Jesuit priest could interact with a normal voice with his Jesuit teaching peers, but he may lose his voice completely when he enters the classroom. In psychogenic aphonia or dysphonia, the patients are not willfully experiencing voice limitation. Rather, their vocal symptoms are often the result of long-term or recent psychologically damaging circumstances, such as a loss of a loved one or from continued sexual abuse. Although the patient with a psychogenic voice disorder may experience some voice improvement from direct voice therapy, in most cases, the voice disorder will not resolve unless there is some concomitant counseling or psychotherapy to address the underlying emotional problem.

Organic Voice Disorders

Organic voice disorders are related to structural deviations of the vocal tract (lungs, muscles of respiration, larynx, pharynx, and oral cavity) or to diseases of specific structures of the vocal tract. An example of a structural deviation is cleft palate, where there is abnormal coupling of the oral and nasal cavities, producing hypernasality during voicing attempts. An example of a vocal tract disease is viral papilloma of the larynx, where the child or adult experiences additive growths in the larynx, which might compromise the airway and interfere with vocal fold vibration. In Chapters 3 to 5 and Chapters 8 to 10, we will consider various organic diseases that may affect voice.

Although the SLP may play an active role in the identification and evaluation of the patient with an organic voice disorder, the primary treatment of the disorder is often medical, dental, or surgical. Treatment by the SLP may have several goals, such as helping to improve the physiologic function of a damaged larynx. When the structural problem is controlled or stabilized, the SLP works with the patient to develop the best voice possible using various therapy methods.

Neurogenic Voice Disorders

The muscle control and innervation of the muscles of respiration, phonation, resonance, and articulation may be impaired from birth or from injury or disease of the peripheral or central nervous systems occurring at any age. For example, the SLP may work closely with a young child with cerebral palsy, perhaps working on both respiratory–voice control and helping the child to develop language. Or the SLP may work with the adult patient with a motor speech disorder acquired after a stroke, not only to improve respiration, voice, and articulation, but also to address concomitant swallowing problems. The tight, spasmodic voices of patients diagnosed with adductor spasmodic dysphonia appear to have unspecified neurogenic origins. Most of the neurological diseases presented in Chapter 5 alter normal voice in some way. The SLP plays a vital role in the assessment and management of the patient with a neurological voice disorder, such as assessing the patient's respiratory volumes and expiratory control, or visualizing through endoscopy a paralyzed vocal fold, or applying diagnostic probes to the Parkinson's disease patient to determine which therapy approaches produce a better voice.

While the majority of neurological impairments and diseases that impair swallowing, breathing, voice, and resonance cannot be cured or eradicated, the SLP frequently plays a vital role in maximizing function to as near normal levels as possible. For many neurologically impaired patients, there is a functional margin of disability that can be minimized by improved patient management and direct therapy intervention.

Management and Therapy for Voice Disorders

Probably no client/patient groups seen by the SLP are more responsive to management and therapy than children and adults with voice disorders. Successful intervention for a voice disorder first requires the identification of the cause of the disorder. We have grouped causal factors of voice disorders into three etiologic categories: functional, organic, and neurogenic. The typical history shows the patient experiencing some problem in respiration, voice, and resonance. Breathing and resonance problems are often long-standing, perhaps experienced by the patient over time. Voice or phonation problems, such as hoarseness, are more likely to develop more recently. The SLP often counsels patients that, unless they are experiencing hoarseness (dysphonia) as part of an allergy or upper respiratory infection (URI), they should wait no more than seven days to have a medical evaluation of the hoarseness. The SLP and otolaryngologist (ear-nose-throat physician, or ENT) have a close working relationship (Thibeault, 2007), and the SLP would refer the hoarse patient to the ENT for identification of the problem and possible medical treatment.

The ENT often refers the patient to the SLP who, by training is able to evaluate and diagnose the voice problem. The SLP takes a detailed history, observes the patient closely, and uses instrumental and noninstrumental assessment approaches. The SLP collects measurement values related to respiratory volumes and performance; performs acoustic measurement of vocal function; visualizes the larynx; and determines resonance function, particularly as related to velopharyngeal closure. Added to these observations and measurements of structure and function, the SLP determines how the patient feels about his or her voice problem, its effect on personality and interaction with others, and the influence of the voice problem on vocational performance. For many voice problems at the time of the evaluation, the SLP tests voice stimulability by introducing a few voice therapy approaches, often called diagnostic probes, with the patient. The SLP determines the outcome from using a particular technique, such as opening one's mouth more or speaking in a louder voice. If such an approach facilitates the production of a target voice, it might well be among the first used in therapy.

Many forms of voice therapy are available for the SLP to use with different kinds of voice problems. The client's response to the diagnostic probe may indicate the general direction of the therapy to be provided. For example, changing the loudness of the patient's voice may be an option. Perhaps with two young boys with vocal nodules, the SLP would recommend using a softer voice, such as "the voice to use when not wanting to awaken a sleeping person, a quiet voice" (Boone and colleagues, 2009, p. 190). Such a quiet voice is also presented by Casper (2000) as the "confidential voice." Or increasing loudness with an aging patient with Parkinson's disease may improve both

articulation and voice, achieved by using a voice loud enough to be heard "20 or 30 feet away" (Boone & Wiley, 2000). Working on loudness changes—developing a softer voice or a louder voice—may go either way.

We have a professional need to develop scientific evidence to support what we do in therapy, but group data are difficult to gather because voice therapy is so individualized. Two patients with the same causative voice problem may require a distinctively different combination of therapy procedures. It is possible, however, to look at therapy effectiveness using different levels of evidence, as suggested by Butler and Darrah (2001) in their determination of treatment outcomes with patients with cerebral palsy. Their five levels of evidence range from Type I (clinic versus control group study) to Type V (descriptive case series/case reports.) The construct validity of each level of typing may have to be questioned. The effectiveness of "Voice Facilitating Approaches" presented in Chapter 7 can probably best be determined by comparing pre-therapy evaluation measurement with post-therapy evaluation by the same measurements. As we shall see when we discuss evaluating the outcome of therapy techniques in Chapter 7, validity of outcome data are often compromised by limitations in the number of voice patients available, SLP fiscal requirements, patient fee difficulties, and the demand for rapid progress.

For organic voice problems, the SLP may work closely with other specialists, such as in physical medicine or respiratory therapy, depending on the particular problem. For example, a preschool youngster with a growth in the airway called papilloma (caused by a virus) may require the clinical services of a surgeon; a respiratory therapist; extra nursing care; and the close watching by the SLP, who may need to work with the child to address breath support and voice. The voice therapy goal with such a child may be solely to develop the best voice possible with a vocal mechanism heavily laden with multiple papilloma growths. When such papillomas become large enough to impinge on the airway, the surgeon excises or reduces them, as needed, to improve breathing function.

Neurogenic voice problems come in many different forms, as we will see in Chapter 5. The SLP may be the first healthcare professional to see a patient who is just beginning to experience voice symptoms. For example, a recent patient was seen in our clinics because he felt he was experiencing a new problem pronouncing occasional words. A subsequent evaluation by the SLP found evidence of tongue tremor (fasciculations) and a problem in tongue diadochokinesis (he could not move his tongue rapidly when making alternating movements, such as saying "ta-ka" in a rapid series). A subsequent referral by the SLP to a neurologist confirmed that the patient was showing beginning signs of amyotrophic lateral sclerosis. Or the neurologist may be the first professional to see the patient with a neurogenic disorder and then refer the patient to the SLP for detailed assessment of breathing, phonation, and speech. The SLP sends his or her evaluation back to the neurologist, and together they may develop a management plan for the patient. Such a plan frequently includes medications for improving the patient's motor functions, with specific goals for the patient to achieve with the SLP in voice resonance and articulation therapy.

The SLP often works with actors, singers, and teachers who want to improve their speaking voices. The role of the SLP in improving the speaking voice of the professional user of voice was defined in a joint statement published by American Speech–Language–Hearing Association (ASHA), the National Association of Teachers of Singing (NATS), and the Voice and Speech Trainers Association (VASTA) (ASHA/NATS/VASTA, 2006). This joint statement defines how the SLP would work collaboratively with members of NATS and VASTA. Gerhard (2016) qualifies this statement by pointing out that, while there is no overt accrediting organization for a holistic vocal

rehabilitation training program, there are avenues for vocal training and rehabilitation that incorporate various professionals including the SLP who specializes in voice. Since the establishment of roles for each voice specialist, there has been an increase in cross-referrals among the three voice specialties. Despite these cross-referrals, Manning and Blanchett found that university vocal performance students report their voice or choral instructors as primary source for information about vocal function, hygiene, and care for their voice (Manning, 2014 and Blanchett, 2014). For the actor and singer, the SLP can often offer vocal hygiene and voice therapy techniques that the professional can use in a normal day of voice usage when not acting or singing. For a thorough review of extant literature and a proposed definition of the "singing voice specialist", refer to Gilman, 2010.

The SLP often works closely with the counselor, psychologist, or psychiatrist with patients with psychogenic voice problems. Working on voice symptoms alone often needs to be supplemented with psychological therapy to deal with underlying emotional problems that may be driving the voice problem. While loss of voice (aphonia) can often be treated successfully by symptomatic voice therapy, the vocal gains may only be temporary, or they may recur in particular environmental situations. Similarly, long-term dysphonias without identifiable physical causes often resist successful resolution because the voice symptoms serve the patient in some way. Mutational falsetto, or continuing to use a higher-sounding voice after puberty (puberphonia), is often thought to be a psychogenic voice disorder among our psychology colleagues. The experience of most SLPs, however, is that the higher voice in the postpubertal male is usually changed in one or two voice therapy visits. Most young men seem to be trapped by habit in using the higher voice and are dramatically relieved with the discovery of the adult male voice. On occasion, the SLP may still refer these adolescents for some counseling or psychological therapy.

Most functional voice disorders result from the patient using excessive effort and are classified as muscle tension dysphonia (MTD). Both children and adults with MTD often abuse, overuse, or misuse their voices. The primary focus of the SLP with these patients is to identify their vocal excesses; once identified, vocal misuse can be reduced and often eliminated by voice therapy alone (Cohen & Garret, 2007). In Chapters 7 and 8, we will cite numerous other successful voice therapy outcome studies, similar to the seminal study of Benninger and Jacobson (1995). These clinical researchers followed 115 MTD patients who eventually developed vocal nodules or polyps from continued vocal excesses. After receiving appropriate voice therapy to reduce their vocal excesses, 94% of the patients experienced resolution of their problem. The authors concluded that, with appropriate voice modification and therapy provided by the SLP, "nodules will generally resolve with return of normal vocal function" (p. 326). Voice therapy for patients with MTD is usually a collection of Voice Facilitating Approaches that result in the proper balance of respiratory, voice, and resonance behaviors.

Progress in voice therapy can be determined from pre- and post-treatment measures of respiratory function, acoustic comparisons, and voice quality and resonance changes. The voice patient's self-perception (Bogaardt and colleagues, 2007) of any change in voice and its impact on quality of life adds needed outcome data. Useful outcome data also come from follow-up contacts and measures by the SLP over a specified period of time.

Summary

In this chapter, we looked at voice and the larynx in the biologic viability of the individual as a tool in emotional expression, and in its complicated and extensive role in spoken human communication. We reviewed the prevalence of voice disorders in the general population and in specific subpopulations. We saw that there appear to be three causal factors in the development and maintenance of voice disorders: functional, organic, and neurogenic. The child or adult with a voice problem is evaluated by the SLP who uses instrumental and noninstrumental approaches for various respiratory and acoustic measures in the attempt to identify causal factors and define aspects of voice production. Diagnostic probes, the application of trial therapy approaches, are then used to determine the efficacy of a particular therapy technique for improving the patient's voice productions. The patient's self-perception of the handicaps presented by the voice disorder in his or her life is then recorded. If evaluation measures indicate that the patient can profit from therapy, the SLP then provides needed voice therapy. At the conclusion of voice therapy, therapy success is determined by comparing pre- and post-therapy measures, providing needed outcome data.

Clinical Concepts

The following clinical concepts correspond with many of the objectives at the beginning of this chapter:

1. Some voice-disordered patients will come to you because they have a larynx that cannot adequately protect their upper airway from aspiration of food or liquids, or the larynx cannot adequately regulate the flow of air through the glottis in order to produce normal voice. Some examples include a speaker with a paralyzed vocal fold (see Chapter 5), a patient with dysarthria (see Chapter 5), a speaker with adductor spasmodic dysphonia (see Chapter 5), a speaker who has a mass on his or her vocal fold (see Chapters 4 and 9), a speaker with paradoxical vocal fold movement (see Chapter 8), and a patient who has undergone a partial laryngectomy (see Chapter 9).
2. Some voice-disordered patients will come to you because they cannot use their voice to express emotion or linguistic subtlety. Some examples include a speaker with muscle tension dysphonia (see Chapter 3), a speaker with psychogenic dysphonia (see Chapter 3), a speaker with a paralyzed vocal fold (see Chapter 5), a patient with dysarthria (see Chapter 5), a speaker with vocal fold nodules (see Chapter 3), a speaker with adductor spasmodic dysphonia (see Chapter 5), and a speaker with hearing loss (see Chapter 8).
3. Obtaining valid data about the incidence and prevalence of voice disorders entails theoretical considerations and practical challenges. It is probable that many persons with voice disorders are underserved because they are either not identified or not referred for voice therapy.
4. There is no universally accepted classification of voice disorders. Regardless of whether or not one adopts the classification system put forth in this text, the voice clinician must have a thorough understanding, not only of the cause of an individual's voice disorder, but also of the personal and environmental factors that may perpetuate the voice disorder. This requisite knowledge is needed in order to plan and deliver effective treatment.

Guided Reading

Read the following article:

Baker, J., Ben-Tovim, D. I., Butcher, A., Esterman, A., & McLaughlin, K. (2007). Development of a modified diagnostic classification system for voice disorders with inter-rater reliability study. *Logopedics Phoniatrics Vocology, 32,* 99–112.

What are the pros and cons of using Baker's classification system compared to the one proposed in this text?

Read the following article:

Cohen, S. M., Kim, J., Roy, N., Asche, C., & Courey, M. (2012a). Prevalence and causes of dysphonia in a large treatment-seeking population. *Laryngoscope, 122,* 343–348.

What voice disorder incidence and prevalence data are still lacking? Explain.

Preparing for the Praxis™

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Directions: Please read the case study and answer the five questions that follow.

> *Aisha is a student teacher who has just begun her fall semester's teaching practicum at a local elementary school. Aisha finds herself spending 10 hours a day or more teaching, staffing with colleagues, meeting with parents and then transitioning to night classes, where she is required to participate in small groups. After a month, Aisha notices that her voice is becoming progressively hoarse and that she feels a sense of fullness and tightness at the level of the larynx. She also reports vocal fatigue, especially at the end of the day. At her medical appointment, she reports to the otolaryngologist that she has no history of allergies and she does not feel flu-like symptoms; just the tightness and fullness.*

1. The otolaryngologist's findings from laryngovideostroboscopy would most likely show

- **A.** Swelling and thickening of the vocal folds
- **B.** Vocal folds that almost medialize for voicing, but do not vibrate.
- **C.** The inability to move the left vocal fold to the midline for phonation
- **D.** A polyp

2. The otolaryngologist writes a prescription for voice therapy and refers Aisha to a speech-language pathologist. The most likely diagnosis is

- **A.** Muscle tension dysphonia
- **B.** Puberphonia
- **C.** Presbyphonia
- **D.** Papilloma

3. Once the speech-language pathologist has greeted Aisha, the SLP next

- **A.** Collects a case history, observes Aisha closely, asks about vocal load and asks about how Aisha feels about her voice problem
- **B.** Begins voice therapy
- **C.** Suggests complete voice rest
- **D.** Encourages Aisha to whisper

4. During the interview, the SLP models a number of behaviors and asks Aisha to replicate them. After a few minutes, Aisha senses a relaxation of the throat and neck muscles and feels and hears a better and more easily produced voice. These diagnostic probes introduced by the SLP are

- **A.** Also known as stimulability probes
- **B.** Supported by multiple group controlled studies
- **C.** Difficult to explain to the client
- **D.** Difficult to learn

5. Aisha asks how common it is for other student teachers to experience voice disorders. The SLP informs Aisha that

- **A.** Student teachers are about twice as likely to report voice complaints as their nonteaching counterparts
- **B.** She is an exception; student teachers rarely experience voice complaints
- **C.** Student teachers experience voice disorders with the same prevalence as their nonteaching peers
- **D.** She shouldn't worry, because once she is a full-fledged teacher, the risk for dysphonia lowers

Chapter 2
Normal Voice

Anatomy and Physiology Throughout the Lifespan

Learning Outcomes

After reading this chapter, one should be able to:

2.1 List and describe the normal aspects of voice production.

2.2 Identify the structures of and describe the mechanics of respiration.

2.3 Describe the phonatory system.

2.4 Describe the principles of phonation and factors involved in changing vocal pitch, loudness, and quality.

2.5 Identify the structures of and mechanics of resonance.

This chapter describes the normal voice and how it is produced. It is important to understand the normal voice in order to be able to diagnose and treat the abnormal voice. Few things are so difficult to define or understand as "what is normal and what constitutes normal limits?" Because of maladaptive vocal behaviors or disease, many patients present to the clinician with overworked voice production systems. To help the patient to abandon the abnormal functions, the clinician must know how normal voice is produced and be able to communicate this information appropriately to the patient. Modern-day voice evaluation often involves consultation with medical professionals who are well-grounded in human anatomy and physiology. For these reasons and more, voice clinicians must have a good working knowledge of the structures and functions serving normal and abnormal voice production throughout the lifespan.

Normal Aspects of Voice

Normal voice may be characterized by five aspects, each related to function. First, the voice must be loud enough to be heard. We may refer to this as adequate carrying power. This means that the voice can be heard, and speech can be understood over the noise of most everyday environmental sounds such as the television, air conditioning, keyboard typing, transportation noises, and so on. Second, the voice must be produced in a manner that is hygienic and safe, that is, without vocal trauma and resulting laryngeal lesions. Third, the voice should have a pleasant quality, one that is not distracting and thus interferes with verbal communication. Fourth, the normal voice should be flexible enough to accurately express emotion. The human voice can be thought of as a

"window into the soul" in that we sometimes judge how a loved one or close friend feels based on the sound of his or her voice. We often think we know if the person is happy, sad, sick, excited, or nervous. Likewise, we sometimes find it hard to mask our own emotional state with our voice. We can also change the meaning of a verbal message by changing the emotional tone of our voice. The sentence, "I am so happy for you," can be said in such a manner as to be sincere or sarcastic just by the tone of voice, even while the words remain the same. The expression, "Oh wonderful," can be said with excitement or with scorn. Last, the voice should represent the speaker well in terms of age and gender. We should not be surprised to meet someone for the first time after speaking to him or her on the phone. Our voice should not portray us as either older, younger, or as less mature than we are. Nor will we likely be pleased if we are mistaken for the opposite gender. The normal voice should represent the speaker faithfully.

Keep the five aspects of loudness, hygiene, pleasantness, flexibility, and representation in mind as we explain in the next section how the normal voice is produced. That the voice can serve us so well through our lifespan is a true testament to the uniquely human aspects of voice we just described.

Normal Processes of Voice Production

Separating the normal speaking voice into three individual processes (respiration, phonation, and resonance) for purposes of study is helpful, but we must remember that these three components of voice production are highly interdependent. For example, without the expiratory phase of respiration, there would be no phonation or resonance. Without adequate functioning of the velopharyngeal mechanism, there would be an imbalance of oral-nasal resonance. Also, these three processes are constantly changing simultaneously. Let us first consider the structures and function of respiration, particularly as they relate to production of voice.

The Respiratory System

For speech to be possible, humans have learned to use respiration for the purpose of phonation. Both speaking and singing require an exhalation (outgoing air stream) capable of activating vocal fold vibration. When training their voice, speakers or singers frequently focus on developing conscious control of the breathing mechanism. This conscious control must not conflict, however, with the physiological air requirements of the individual. When a problem occurs with respiration, it is often the conflict between the physiological needs and the speaking-singing demands for air that causes faulty usage of the vocal mechanism. Our dependence on the constant renewal of oxygen supply imposes certain limitations on how many words we can say, how many phrases we can sing, or how much loud emphasis we can use on one expiration.

Structures of Respiration

Respiration is a vital life-sustaining and voice-enabling process that results from the movement of support structures within the musculoskeletal system. While a complete review of the musculoskeletal components for speech and voice is beyond the scope of this text, an overview is presented here that will provide the student or clinician with

core information that can be applied to the diagnosis and management of persons with respiratory-based voice disorders.

The Bony Thorax

The bony thorax includes the vertebrae and vertebral column, the thoracic cage (ribs, sternum, and associated muscles), the pectoral girdle, and the pelvic girdle. The thorax is suspended from the vertebral (spinal) column, a strong but flexible structure that serves many purposes. The vertebral column consists of 33 individual vertebrae stacked loosely on top of each other, forming a strong pillar for the support of the head and trunk. The vertebrae can be grouped into five regions. The 31 pairs of spinal nerves emerge and enter the spinal cord through spaces between each pair of vertebrae, beginning at the thoracic level.

The seven cervical vertebrae are smaller and more delicate than the remaining vertebrae, and within these vertebrae the left and right vertebral arteries course superiorly on their way to joining the basilar artery at the base of the brain. Two of the cervical vertebrae (C1—Atlas and C2—Axis) are unique, because they connect the skull to the spinal column and allow for diverse head movement (e.g., rotate side to side, bend forward, and bend backward). The C7 vertebra—the cervical prominence—can be felt if you pass your fingers down the back of your neck, in between the base of your skull and the top of your spine. In terms of adult landmarks, the base of the nose and the hard palate corresponds to C1, the teeth (when mouth remains closed) correspond to C2, the mandible and hyoid bone corresponds to C3, the thyroid cartilage spans from C4 to C5, and the cricoid cartilage spans from C6 to C7. In infants and children, the laryngeal landmarks are situated higher at C1 to C3.

The 12 thoracic vertebrae are intermediate in size between those of the cervical and lumbar regions; they increase in size as one proceeds down the spine, the upper vertebrae being much smaller than those in the lower part of the region. The first 12 pairs of spinal nerves emerge from between the thoracic vertebrae. The thoracic vertebrae provide the basis for the respiratory framework because they form the posterior point of attachment for the ribs (Seikel and colleagues, 2010). The thoracic spine's range of motion is limited due to the many rib and vertebrae connections.

The five lumbar vertebrae graduate in size from L1 through L5. These vertebrae bear much of the body's weight and related biomechanical stress. They provide direct or indirect attachment for a number of back and abdominal muscles, as well as for the posterior fibers of the diaphragm (Seikel and colleagues, 2010). The lumbar vertebrae allow significant forward and backward bending at the waist, moderate side bending, and a small degree of rotation.

The sacrum is a large, triangular bone at the base of the spine and at the upper and back part of the pelvic cavity, where it is inserted like a wedge between the two hip bones. Its upper part connects with the last lumbar vertebra, and the bottom part connects with the coccyx (tailbone). It consists of usually five initially nonfused vertebrae that begin to fuse between the ages of 16 and 18 years and are usually completely fused into a single bone by age 34.

The coccyx, commonly referred to as the tailbone, is the final segment of the vertebral column. Comprising three to five separate or fused vertebrae (the coccygeal vertebrae) below the sacrum, it is attached to the sacrum by a joint that permits limited movement between the sacrum and the coccyx.

The thoracic (rib) cage is formed by the thoracic vertebral column, 12 pairs of ribs and their costal cartilages, the sternum, and the internal and external intercostal

muscles. Each of the ribs is made up of bone and cartilage, allowing for both strength and mobility. The 12 pairs of ribs can be subdivided into three general classes: true ribs (the first seven pairs), false ribs (the next three pairs) and floating ribs (the last two pairs). The true and false attach to the vertebral column and the sternum, while the floating ribs attach only to the vertebral column. The ribs connected to the thoracic vertebral column and their connecting muscles play an important role in respiration, as we shall see when we discuss respiratory function.

The pectoral girdle is formed by the clavicle and scapula (shoulder blade). It supports the upper limbs. It is suspended from the head and neck by the support fibers of the trapezius muscles, which descend from the cervical vertebrae and the skull and are attached to both the clavicle and the scapula.

The pelvic girdle is formed by the ilium, sacrum, pubic bone, and ischium. The pelvic girdle provides a strong structure for attaching the legs to the vertebral column. By means of this structure, forces generated through movement of the legs are distributed across a mass of bone which, in turn, is attached to the vertebral column (Seikel and colleagues, 2010).

The Muscles of Respiration

We now need to consider the muscles of respiration (see Figures 2.1 and 2.2). When discussing these muscles, it is helpful to think of three major categories: (1) the muscles of the rib cage, (2) the diaphragm, and (3) the muscles of the abdominal wall.

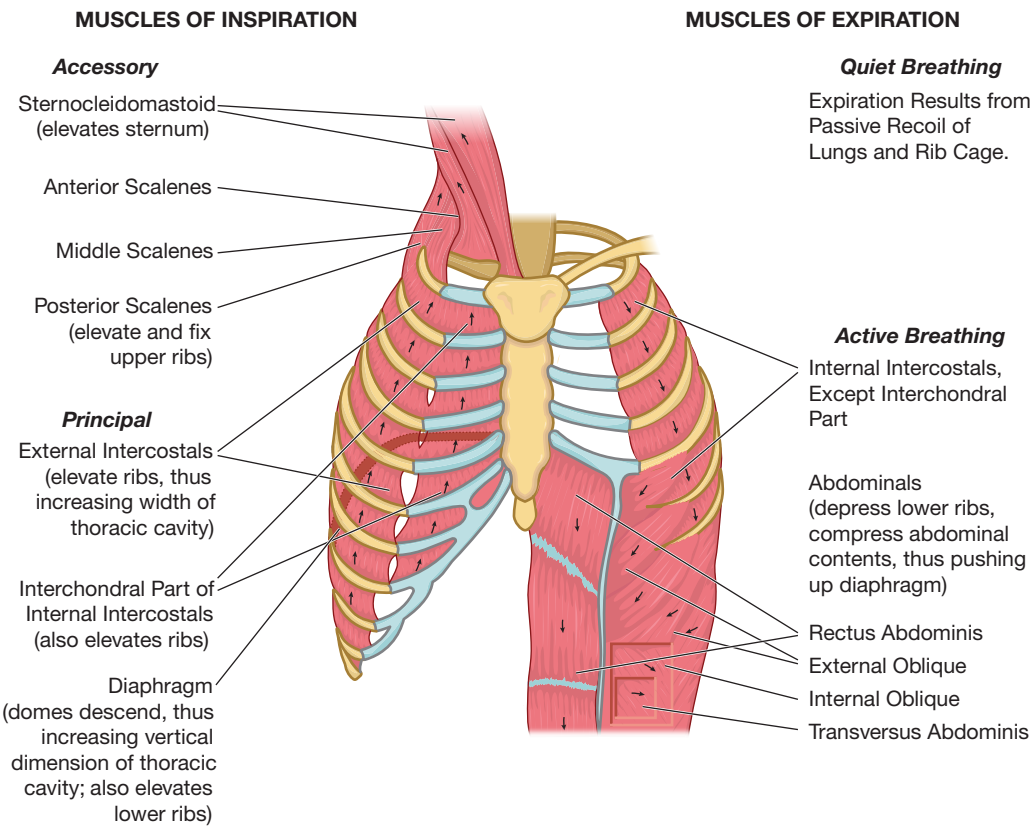
Figure 2.1 A List of Muscles of Respiration

Muscles of the Rib Cage Wall
Sternocleidomastoid
Scalenus group (anterior, medial, posterior)
Pectoralis major
Pectoralis minor
Subclavius
Serratus anterior
External intercostals
Internal intercostals
Transversus thoracis
Lattisimus dorsi
Serratus posterior superior
Serratus posterior inferior
Lateral iliocostals
Levatores costarum
Quadratus lumborum
Subcostals

Muscles of the Diaphragm
Diaphragm

Muscles of the Abdominal Wall
Rectus abdominus
External oblique
Internal oblique
Transversus abdominis

Figure 2.2 Muscles of Respiration



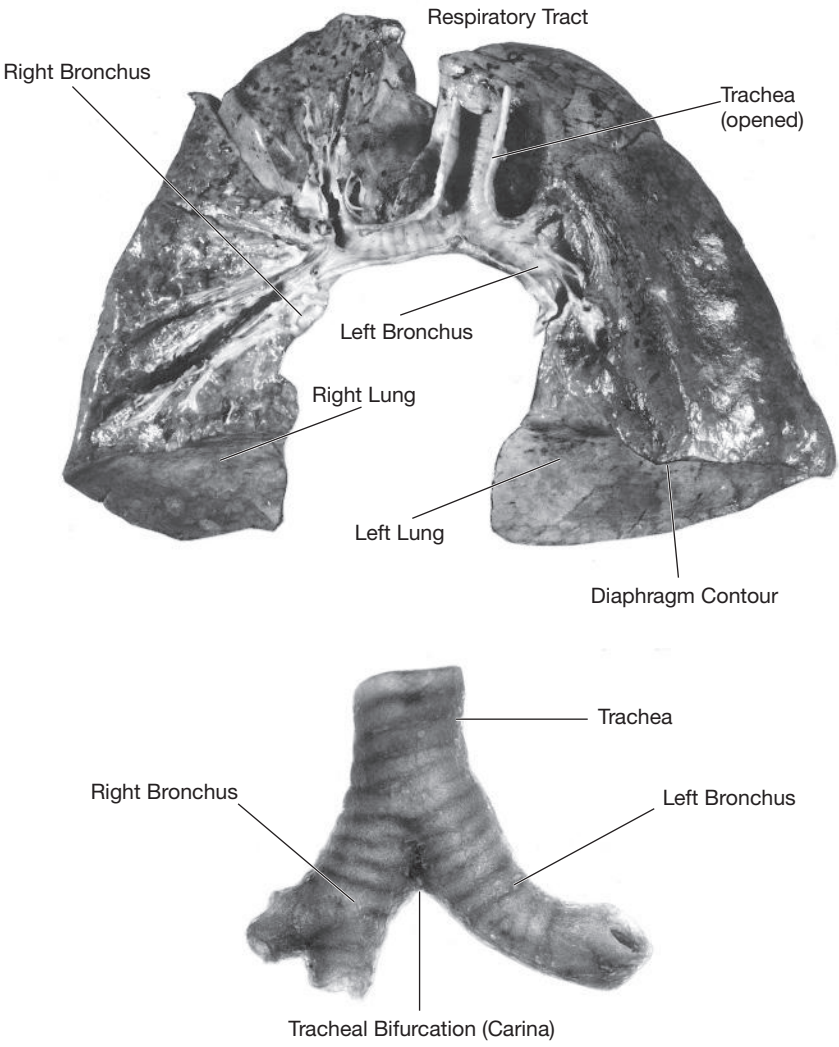
Thinking of the muscles of respiration in this way makes it easier to discuss the movements that occur due to the passive and active forces to be discussed later in this chapter, and the adjustment capabilities of the respiratory system for speech. Keep in mind now that the action of the respiratory muscles changes the dimensions of the thoracic cavity, which in turn changes the pressure within the thoracic cavity. The resulting changes in pressure result in the inspiratory-expiratory cycle with which we are all familiar.

The Inspiratory Muscles

The inspiratory muscles can be found within the thorax, back, neck, and upper limbs. The primary inspiratory muscles of the thorax are the diaphragm and external intercostal muscles. These muscles are assisted by accessory muscles in the neck, back, and upper limbs.

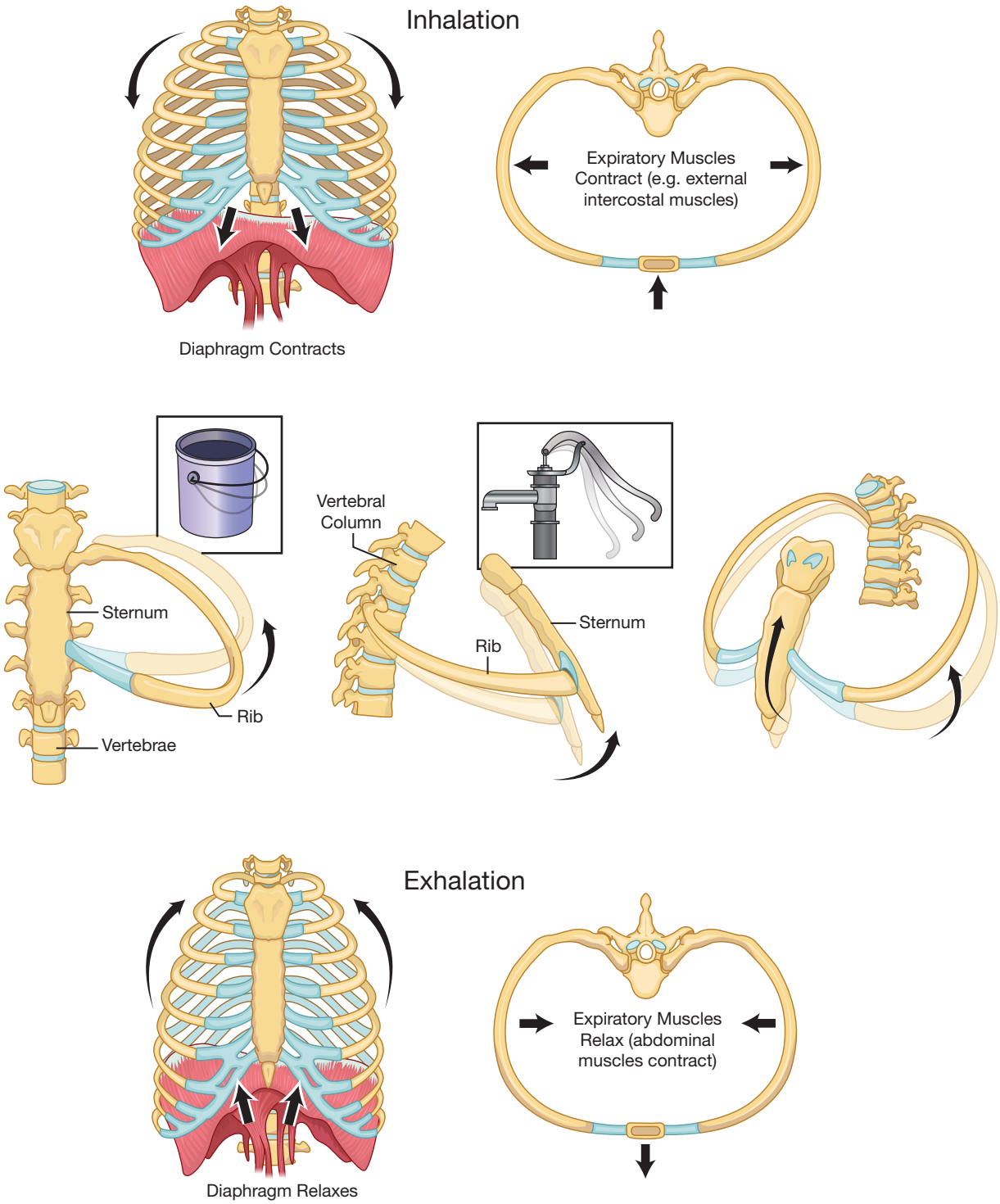
THE DIAPHRAGM. The diaphragm is a large (about 250 cm^2 in surface area) dome-shaped muscle that separates the thorax from the abdominal cavity, with openings for the esophagus and assorted arteries (see Figure 2.3 on page 24). The muscle fibers of the diaphragm insert into the sternum and the six lower ribs and their cartilages and into the first three of four lumbar vertebrae. The other ends of these muscle fibers converge to attach to the fibrous central tendon, which is also attached to the pericardium on its upper surface. At rest, the diaphragm is shaped like an inverted bowl. When you

Figure 2.3 Lungs and Tracheal Bifurcation



are standing and in the mid-phase of respiration, the dome of the diaphragm is at about the same level as the sixth rib. As described in Levitzky (2007), during normal quiet breathing, contraction of the diaphragm causes its dome to descend 1 to 2 cm into the abdominal cavity, with little change in its shape. This elongates the thorax in the cephalacaudal (top to bottom) dimension and increases its volume. These small downward movements of the diaphragm are possible because the abdominal viscera can push out against the relatively compliant abdominal wall. During a deep inspiration, the diaphragm can descend as much as 10 cm. With such a deep inspiration, the limit of the compliance of the abdominal wall is reached, abdominal pressure increases, and the central tendon becomes fixed against the abdominal contents. After this point, contraction of the diaphragm against the fixed central tendon elevates the lower ribs. When a person is in the supine position, the diaphragm is responsible for about two-thirds of the air that enters the lungs during normal quiet breathing. When a person is standing or seated in an upright posture, the diaphragm is responsible for only about one-third to one-half of the tidal volume. Motor and sensory innervation for the diaphragm comes primarily from the two phrenic nerves. These nerves originate in the cervical plexus

Figure 2.4 Movements of the Thoracic Cavity



(grouping) of spinal nerves C3 through C5 of both sides of the spinal cord (Seikel and colleagues, 2010). Thus, innervation to the diaphragm is bilateral, an indication of its biological importance. The diaphragm is under primary control of the autonomic nervous system, although one can place the diaphragm under voluntary control, albeit only temporarily (such as when holding one's breath).

THE EXTERNAL INTERCOSTAL MUSCLES. The eleven external intercostal muscles run downward and forward from the lower border of the rib cage above to the upper border of the rib cage below (see Sidebar 2.1). Together, these muscles form a large sheet of muscle that attaches the ribs to one another (Hixon and colleagues, 2008). The external intercostal muscles are positioned so that, when they contract, the entire rib cage elevates and expands (Seikel and colleagues, 2010). As can be seen in Figure 2.4, the true/upper ribs move with a pump-handle motion about the vertebrae. This motion increases the anterior-posterior dimension of the thoracic cavity. That is, it increases the distance between the sternum and the vertebral column (see Sidebar 2.2). The distance front to back is increased, leading to an overall increase in volume. The false/middle ribs move with a bucket-handle motion about the vertebrae. This motion increases the horizontal dimension of the thoracic cavity; that is, it makes the chest slightly wider, increasing the volume within, enabling breathing (see Sidebar 2.2). The floating ribs move with a caliper motion.

THE ACCESSORY MUSCLES OF INSPIRATION. The accessory muscles of respiration are muscles in the trunk and lower neck that can be called into action to assist with breathing. These muscles usually have other primary functions and assist with respiration only under certain circumstances—usually when more deep or rapid breathing is needed. The accessory muscles include the scalenes and the sternocleidomastoid muscles in the neck, the serratus anterior and the pectoral muscles in the upper trunk, the upper trapezius and latissimus dorsi muscles of the trunk, and the erector spinae muscles of the back. There are also some smaller, deeper muscles that lie against the rib cage that can also be recruited to assist with respiration.

The Expiratory Muscles

The expiratory muscles can be found within the thorax, back, upper limbs, and abdomen. The expiratory muscles include the internal intercostal muscles and the abdominal muscles. During active expiration, the most important muscles are those of the abdominal wall (including the rectus abdominus, internal and external obliques, and transversus abdominus). Contraction of these muscles forces the abdominal organs up against the diaphragm and further decreases the volume of the thorax. This results in increased intra-abdominal pressure, which in turn drives air out. The internal intercostal muscles lie deep between the ribs and are oriented at a right angle to the external intercostal muscles, continuous with the internal oblique muscles (see Sidebar 2.3). Contraction of the internal intercostal muscles assists with active expiration by depressing the rib cage, thus decreasing thoracic volume.

The Tracheobronchial Tree

For quiet breathing, inspired air enters through the nostrils and passes into the nasal cavities and into the nasopharynx through the open velopharyngeal port into the oropharynx. For mouth breathers and for speaking purposes, the air enters through the open mouth and passes through the oral cavity into the oropharynx. The air then flows through the hypopharynx. From the hypopharynx, the air flows into the larynx and passes down between the ventricular (false) vocal folds and further down between the true vocal folds into the trachea (windpipe).

The trachea is a hollow and flexible cylindrical-shaped tube formed by a series of 16 to 20 C-shaped hyaline cartilage rings that are closed anteriorly and open

Sidebar 2.1

These muscles are sometimes referred to as the *front pocket muscles* because the fibers mimic the direction a hand would enter a front pocket.

Sidebar 2.2

To appreciate these movement dynamics, do the following: (1) Place the palm of your right hand on your sternum/chest and the back of your left hand on your spine at the level of your shoulder blades. Look down at your right hand as you take a breath, and you will notice that the distance between your hands increases. That is, your thorax expands from front to back. (2) Place the thumb of your right hand on the top of the uppermost rib on your right. Then, put the middle finger of your left hand on the bottommost rib on your right. Look down at your right hand as you take a breath, and you will notice that the distance between your hands increases. That is, your thorax expands from top to bottom.

Sidebar 2.3

These muscles are sometimes referred to as the *back pocket muscles* because the fibers mimic the direction a hand would enter a back pocket.

posteriorly. The tracheal rings are 2 to 3 cm in diameter and are connected by a continuous mucous membrane lining. The gap between the rings is spanned by smooth muscle. The trachea runs from the inferior border of the larynx for about 11 to 12 cm, and then it bifurcates (divides) into two mainstem or primary bronchi at a point known as the carina, which is near the level of the fifth thoracic vertebra (see Figure 2.3). Each mainstem bronchus divides into smaller divisions known as the secondary (lobar) and tertiary (segmental) bronchi. The tertiary bronchi continue to branch and divide into smaller and smaller tubes and eventually branch into terminal (end) respiratory bronchioles. The respiratory bronchioles open into alveolar ducts. These ducts lead to outpouchings called alveolar sacs, which lead to microscopic alveoli (air sacs) inside capillary networks. In the alveolar sacs, gas exchange between oxygen and carbon dioxide occurs. Some of the bronchioli are visible in the upper picture of Figure 2.3, but most of the bronchioli and all the alveoli are covered by the pleural membrane that covers the lungs.

Control of Breathing

As described in Moini (2012, p. 255), respiratory control has both involuntary and voluntary components. The involuntary centers of the brain regulate respiratory muscles and control the depth and frequency of pulmonary ventilation. This occurs in response to sensory information that arrives from the lungs, the respiratory tract, and other sites. The voluntary control of respiration reflects activity in the cerebral cortex that affects either the output of the respiratory center in parts of the brainstem or the output of motor neurons in the spinal cord that control respiratory muscles (see Sidebar 2.4). Control of breathing for vocalization and speech has been associated with the primary motor and sensory cortex, supplemental motor area, cerebellum, thalamus, and limbic system.

Sidebar 2.4

Try holding your breath or slowing down your breathing rhythm. You are able to do this for only a limited time or to a limited degree because autonomic brainstem control takes over when blood oxygen decreases and carbon dioxide increases.

The Respiratory Cycle (Inhalation and Exhalation)

The respiratory tract functions much like a bellows. When we move the handles on the bellows apart, the bellows becomes larger and the air within it becomes less dense than the air outside it. The outside air rushes in due to the lower pressure of the less-dense air in the bellows and the greater pressure in the more-dense outside air. The inspiration of air into the bellows is achieved by active enlargement of the bellows' body. Similarly, in human respiration, the inspiration of air is achieved by active movement of muscles that enlarge the thoracic cavity. When the thorax enlarges, the lungs within the thorax enlarge. The air within the lungs becomes less dense than atmospheric air, and inspiration begins. The air is expired from the lungs by decreasing the size of the chest, thus compressing the air and forcing it to rush out. In human respiration, however, much of expiration is achieved by passive collapse of the thorax and not by active muscle contraction. This is an extremely important fact and can be valuable information for voice clinicians. Much of expiration is passive. Hixon and Hoit (2005) have described human respiration as having two types of forces that are always present: passive, nonvolitional forces and active, volitional forces.

Sidebar 2.5

To demonstrate this, simply take about a half-breath and produce the vowel /i/ for five seconds at a medium loudness level. Then listen to the vocal quality. Now take a very deep breath and produce the same /i/ vowel for five seconds at medium loudness. The vocal quality will generally be poorer because of the increased effort required to control the greater air volume and higher air pressure. Finally, produce the same /i/ vowel for five seconds at medium loudness immediately after releasing three-fourths of your air supply. Again, the vocal quality will suffer as you try to compensate, often by hyperadducting the vocal folds, for the low air pressure and low lung volume.

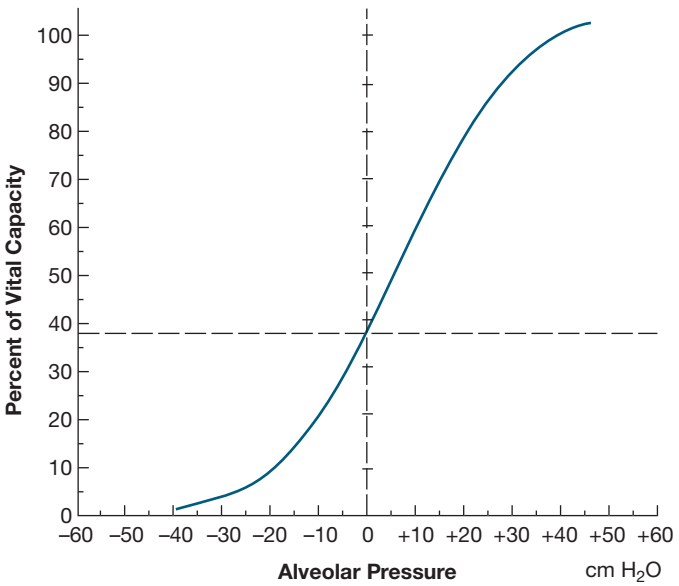
Passive Forces

Much of the power required for normal speech can be supplied by the passive forces of respiration (passive exhalation). These forces include the natural recoil of muscles, cartilages, ligaments and lung tissue, the surface tension of a special film that lines the alveoli, and the pull of gravity. These forces reduce the size of the thorax during expiration (in a manner analogous to the recoil of a stretched spring) and thus contribute to outward airflow from the lungs, which may be used in speech. The mechanism of passive force can be understood by examining the concept of relaxation pressure.

RELAXATION PRESSURE. Vocal quality is affected by extremely high or low air pressure at high or low lung volume (see Figure 2.5). The most efficient and most pleasing voice is produced at mid-air-pressure levels and mid-lung-volume levels of air (see Sidebar 2.5). Knowledge of this translates into an excellent clinical stimulation technique. We can often change the vocal quality of our dysphonic patients by instructing them to use the midrange of air pressure and lung volume. Teaching a shortened phrasing pattern may be important to teaching breath-stream management. Except in singers or actors, this generally is all the respiration training that needs to be given by the speech-language pathologist (SLP). All the emphasis placed on breathing exercises and respiration training in the past seems unproductive and unnecessary for nearly all of our patients with dysphonia. The clinical facilitation technique of glottal fry (discussed in Chapter 7) makes use of this information, because glottal fry is produced with little air pressure and little airflow.

Figure 2.5 The Relaxation Pressure Curve

The passive forces of exhalation tend to generate force during inhalation that works to restore the lung and rib cage system to the normal resting state or equilibrium. After active inhalation, these passive forces of exhalation rebound to provide some of the expiratory force needed for speech. There is a nearly linear relationship between relaxation pressure and lung volume in the range between 20% and 70% of the vital capacity. This curve represents the pressure generated by the passive factors of the respiratory system.



Active Forces

Additional power required for normal speech can be supplied by the active forces of respiration (active exhalation). These forces include the strength of the muscles within the chest wall, their patterns of movement, and the amount of air contained in the lungs. As described by Hixon and Hoit (2005, p. 18), the more air the lungs contain, the greater the force that can be produced to decrease the size of the thorax (i.e., the greater the expiratory force that can be generated). By contrast, the less air the lungs contain, the greater the force that can be produced to increase the size of the thorax (i.e., the greater the inspiratory force that can be generated).

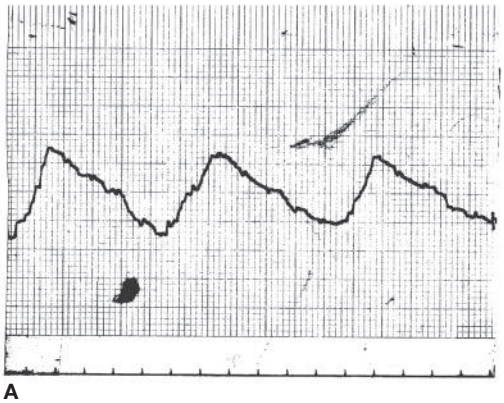
A key problem for many voice-disordered patients is the tendency to squeeze the glottis closed in order to produce the needed power rather than to increase air pressure and airflow by contracting the abdominal muscles. We can better understand this poor technique by a simple analogy. If we are watering flowers in a garden and we want to reach the far row of plants, we can either place a thumb over the end of the hose and squirt the water further (increase the power), or we can increase the water power by turning the faucet on further. When we squeeze the glottis closed, we are "putting a thumb over the end of the hose." When we contract the abdominal muscles, we are "turning the faucet further on" and increasing the airflow. Even though squeezing the glottis tends to increase the vocal power, vocal quality is diminished because the voice sounds strained. If this method is habitual, the excessive effort becomes the basis of a hyperfunctional voice disorder. Such effort may lead to laryngeal changes that can result in abnormal voice. When we need increased power to speak louder, to stress words, or to extend a phrase when singing or speaking, we should use the larger muscles of the abdomen and "turn on the faucet" controlling the source of air. Thus, the pressure at the valve (the larynx) is not excessive, and vocal quality is improved with delicate laryngeal tissue not subjected to stress and strain, which produces laryngeal edema and laryngitis. Vocal quality is not diminished, and adverse tissue change is avoided. Voice

clinicians can use this water analogy to teach patients how to monitor breath control by properly using expiratory reserve volume (see definition of terms in Table 2.1) via the abdominal muscles, rather than using excessive glottal valving in the larynx.

Figure 2.6, the simple tracings of a pneumotachometer, shows the relative time for inspirations-expiration for a passive, tidal breath, for saying the numbers "1, 2, 3, 4, 5," and for singing the musical passage, "I don't want to walk without you, baby," from the old song by that title. Note that the inspiratory time during normal tidal breathing

Figure 2.6 Pneumotachometer Tracings

Note the relative time for inspiration as opposed to expiration for three conditions: tracing A (three tidal breaths) produces an inspiration–expiration time ratio of about 1:2; tracing B (counting from one to five on eight trials) produces a ratio of about 1:3; tracing C (singing twice, "I don't want to walk without you, baby") yielded an inspiration–expiration ratio of approximately 1:10.



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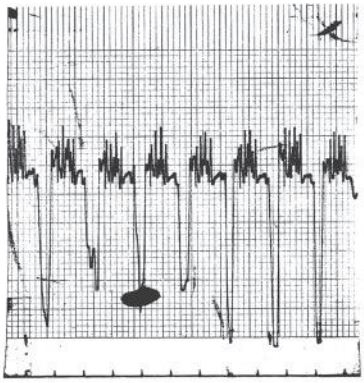

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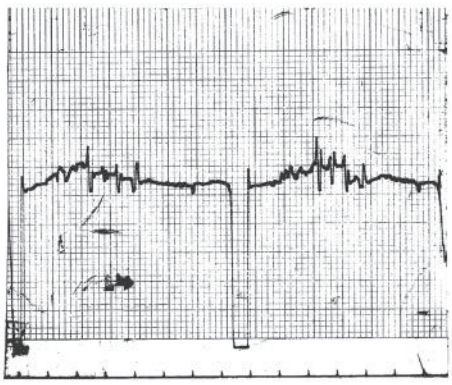


C

Table 2.1 Respiratory Volumes and Capacities

Type	Description	Calculation	Volume
Tidal volume (TV)	The amount of air inspired and expired during a single respiratory cycle	Measured	500 mL
Inspiratory reserve volume (IRV)	The maximum volume of air that can be inspired beyond the end of a tidal inspiration	Measured, or VC – (TV + ERV)	3,000 mL
Expiratory reserve volume (ERV)	The maximum volume of air that can be expired beyond the end of a tidal expiration	Measured	1,100 mL
Residual volume (RV)	The volume of air that remains in the lungs after a maximum expiration	Measured	1,200 mL
Inspiratory capacity (IC)	The maximum volume of air that can be inspired	TV + IRV	3,500 mL
Functional residual capacity (FRC)	The volume of air remaining in the lungs and airways at the end of a resting tidal exhalation	ERV + RV	2,300 mL
Vital capacity (VC)	The maximum volume of air that can be expired following a maximum inspiration	TV + IRV + ERV	4,600 mL
Total lung capacity (TLC)	The total volume of air contained in the lungs and airways after a maximum inspiration	TV + IRV + ERV + RV	5,800 mL

is much longer than the quick inspiration for speech and singing. This is indicated by the rapid rise of the tracing from a resting baseline in an almost vertical move. In the tidal breath, the rise from the baseline is gradual and sloped rather than vertical.

We will now define the terms we will use in following discussions to describe aspects of respiration. Methods for evaluating respiratory volumes and capacities will be discussed in Chapter 6.

Respiratory Volumes and Capacities

Only a small amount of the air in the lungs is exchanged during a single quiet respiratory cycle. The total volume of the lungs can be divided into *volumes* and *capacities* (see Table 2.1). Respiratory volumes refer to the amount of air in the lungs at a given time and how much of that air is used for various purposes, including speech (Solomon and Charron, 1998). Respiratory volumes include *tidal volume, inspiratory reserve volume, expiratory reserve volume,* and *residual volume*. Lung capacities combine two or more of the respiratory volumes and include *inspiratory capacity, vital capacity, functional residual capacity,* and *total lung capacity*. These volumes and capacities are useful for diagnosing problems with pulmonary ventilation (Moini, 2012). Respiratory volumes and capacities vary depending on the patient's age, gender, level of physical exertion, and vocal training. Data reported by Hoit and Hixon (1987) and Hoit and colleagues (1989, 1990) indicate that, in general, lung volumes and capacities increase from infancy through puberty and then remain stable until advancing age, when they decrease slightly (see Table 2.2).

The Effects of Aging on the Respiratory System

It has been reported that changes in general pulmonary functioning with aging become measurable at around age 40 years (Rochet, 1991). As reported by Weismer and Liss (1991), there is increased stiffness of respiratory structures, resulting in increased

Table 2.2 Group Means of Seven Age Groups of Subjects for Some Lung Volumes and Capacities (in Cubic Centimeters)

Age Group	7	10	13	16	25	50	75
Males							
TLC	2120	3140	4330	6200	6740	7050	6630
VC	1670	2510	3550	5080	5350	5090	4470
FRC	980	1400	1970	2940	3120	3460	3440
ERV	530	770	1180	1810	1730	1500	1280
Females							
TLC	2070	2980	3740	4980	5030	5310	4860
VC	1580	2340	2999	3780	3930	3600	2940
FRC	970	1430	1690	2560	2420	2930	2590
ERV	480	780	940	1350	1320	1220	670

SOURCE: Adapted from Hoit and Hixon (1987); Hoit, Hixon, Altman, and Morgan (1989); and Hoit, Hixon, Watson, and Morgan (1990).

relaxation pressure at corresponding lung volumes. Muscle weakness, muscle atrophy, and increased fibrotic content of muscle result in diminished pressure-generating capability at a given lung volume. Degeneration of nerve fibers and sensory receptors result in a loss of appreciation of absolute lung volumes, lung volume changes, and precision of motor commands for breathing. Less efficient gas exchange results in higher breathing frequency.

The first comprehensive studies reporting age-related changes in speech breathing were conducted in the late 1980s by Hoit and colleagues (1987, 1989, 1990). The major findings of the 1987 study were that elderly males demonstrated larger rib cage volume initiations, larger lung volume excursions, and larger lung volume expenditures per syllable than younger men, particularly during extemporaneous speaking. The major findings of the 1989 study were that, compared to younger women, elderly females demonstrated larger rib cage excursions during reading out loud, increased frequency of inhalation during reading out loud, increased air expenditure during nonphonated intervals during reading out loud, and larger lung volume initiations during extemporaneous speaking. (For a complete review of the speech and voice changes of geriatric speakers, see Zraick and colleagues, 2006).

Breathing for Life Versus Breathing for Speech

In breathing for life (that is, quiet breathing), the ratio of time for inhalation versus exhalation is nearly equal, with exhalation time just slightly longer than inhalation time. During breathing for speech, we have a bias toward longer exhalations, which is quite compatible with the need to extend expiration for purposes of speech (see Sidebar 2.6). Influences on speech breathing include the speaker's body position, body type, and age, as well as the type of utterance being produced, interactions between the speaker and the listener, the background noise in the setting, and so forth. As summarized by Hixon and Hoit (2005, pp. 105–106): (1) speech breathing while upright is different than speech breathing while lying down due to the effect of gravity on relaxation pressure and chest wall movements (see Sidebar 2.7); (2) body type influences speech breathing

Sidebar 2.6

To appreciate these timing differences, do the following: (1) place the tip of your right index finger just in front of, but not touching, your lips and breathe quietly through your mouth. Close your eyes and pay attention to how long it takes you to inhale and how long you feel the exhaled air on your fingertip. (2) Keeping your fingertip in place, close your eyes and talk out loud for about 30 seconds. Pay attention to how short the inhalation time is and how you feel the exhaled air on your fingertip throughout your utterance.

Sidebar 2.7

To appreciate the effect of gravity, do the following: Say the Pledge of Allegiance out loud while sitting in a chair versus lying flat on your back. What are the differences you note?

because of the effect of body fat on the movements of the abdominal wall and rib cage wall; (3) advanced age (seventh or eighth decade of life) brings changes in valving of the larynx, which result in larger lung volumes and rib cage wall excursions and greater average expenditures of air per syllable; (4) speech breathing patterns are highly variable until age three years and undergo refinement throughout childhood and adolescence; and (5) cognitive-linguistic factors affect when an inspiration occurs and how long it will be, how long the following expiration will be, how often silent pauses will occur, and how much speech is produced per breath group (see Sidebar 2.8). In addition to a change in the ratio times of inhalation–exhalation, there are changes in the volume of air inhaled and exhaled. During quiet breathing, the volume of air is approximately 10% of vital capacity, while during speech breathing it can be as high as 25% of vital capacity, depending on the length and loudness of the utterance. As described earlier in this chapter, muscle activity for exhalation is passive during quiet breathing, while it is active during speech breathing. Last, the abdomen is displaced outward relative to the rib cage during quiet breathing, while during speech breathing, it is displaced inward relative to the rib cage (see Sidebar 2.9).

The SLP must take the aforementioned factors into consideration when conducting a speech breathing evaluation (more on this topic in Chapter 6). For example, when breath support or perhaps breath control is a problem in a voice-disordered patient, it is often related to failure to take breaths at appropriate places. At other times, the tendency to push too hard in extending the expiratory reserve volume results in a strained vocal quality. Understanding lung volumes and capacities, and the difference between breathing for life versus breathing for speech, is the foundation for identifying voice-disordered patients whose dysphonia is due in part to abnormal respiratory function.

Sidebar 2.8

To appreciate these influences, do the following: (1) determine the number of quiet breaths you take per minute, then (2) silently read a passage for one minute and note the number of breaths you take. (3) Repeat the reading, this time aloud, and note the number of breaths you take. Was there a difference across conditions?

Sidebar 2.9

To appreciate these movement dynamics, do the following: (1) put the palm of your right hand on your sternum/chest and the palm of your left hand on your belly. Look down at your hands as you take a quiet breath, and you will notice that your belly expands front to back to a greater degree than your chest. (2) Keeping your hands in the same locations, look down as you are talking out loud for about 30 seconds. Notice that each time you take a breath during speaking, your belly moves inward relative to your chest.

The Phonatory System

The phonatory system is the source of voiced sound. Normal phonation (voice production) results from normal expiratory airflow, normal vocal fold structure and function, normal supraglottic structure and function, and normal nervous system control. One's voice can be heard while one is talking, singing, laughing, crying, or screaming. To quote Lord Byron in his poetic masterpiece *Don Juan* (1824), "The devil hath not, in all his quiver's choice, an arrow for the heart like a sweet voice" (Canto XV, Stanza 13).

Anatomy of Phonation

The larynx, positioned atop the trachea, is the gateway to the respiratory tract. The larynx serves important biological functions, which include allowing air into and out of the lungs for life-sustaining breathing, protecting the airway from infiltration of food or liquid during swallowing, protecting the airway from infiltration of foreign bodies, and fixing the thorax during activities demanding highly elevated abdominal pressures (such as forced bowel and bladder evacuation, childbirth, and heavy lifting). Central to these functions is the ability of the vocal folds to *abduct* (move away from each other,

starting together at midline) or *adduct* (move toward each other, ending together at midline), essentially serving as a valve between the speech tract and the respiratory tract. Using this valve to generate voice (to phonate) has required the development of intricate neural controls that permit humans to set the vocal folds into precise vibration for speaking and singing. Vocal fold vibration is possible because (1) the vocal folds are located within a fixed laryngeal framework; (2) muscles within the larynx (intrinsic laryngeal muscles) facilitate vocal fold abduction and adduction; (3) some of these intrinsic laryngeal muscles cause changes in the elastic properties of the vocal folds, thus affecting their rate of vibration; and (4) an outgoing airstream also affects vocal fold vibration. The myoelastic aerodynamic theory of phonation takes these factors into account and will be described later in this chapter. First, however, we turn our attention to providing an overview of laryngeal anatomy, including changes affecting voice across the lifespan.

The Laryngeal Framework

The larynx is a constricted tube with a smooth surface. It is located deep within the strap muscles of the neck and is situated vertically at the level of vertebrae C4–C6 in adults, but is higher in children, at the level of vertebrae C1–C3. The larynx is approximately 44 mm long (1.7 inches) in adult males and approximately 36 mm long (1.5 inches) in adult females. The circumference of the larynx in adults is approximately 120 mm (5 inches). A framework of cartilage, ligaments, membranes, and folds gives the larynx form. Connected to this framework are extrinsic and intrinsic laryngeal muscles that facilitate movement of either the laryngeal frame (in the case of the extrinsic muscles) or the vocal folds within (in the case of the intrinsic muscles).

Ligaments and membranes connect the larynx superiorly to the hyoid bone, inferiorly to the cricoid cartilage, and anteriorly to the epiglottis. These attachments of the larynx loosely position it at midline in the neck. Because the larynx is not rigidly fixed in the neck, it is capable of limited up-down and side-to-side movements (see Sidebar 2.10). The vertical and horizontal movements of the larynx are considered normal, and lack of such movements during a head and neck examination can be indicative of neurological damage, degenerative changes, blunt force trauma, the presence of a tumor or other mass, or possibly muscle tension dysphonia.

Sidebar 2.10

To illustrate, put the first three fingers of your right hand on your larynx and swallow. You should feel the larynx elevate, move forward slightly, and then glide back to its resting position. If you apply a little firmer grip, you can move the larynx from side to side. Also, if you raise your tongue within the oral cavity, or protrude your tongue, you will feel the larynx elevate slightly; this is partially because the base of the tongue inserts into the hyoid bone. As the tongue contracts, it lifts the hyoid bone slightly.

The Extrinsic Laryngeal Muscles

The extrinsic laryngeal muscles have one attachment to the larynx and another attachment to some structure external to the larynx. Along with the hyoid bone, they are located in an area of the neck called the anterior triangle, which is bounded by the mandible, the sternocleidomastoid muscles, and the midline platysma muscle. The extrinsic laryngeal muscles have a role in supporting and stabilizing the larynx and in changing its position within the neck. Muscles within this group include the sternothyroid, thyrohyoid, and inferior constrictors.

The Supplementary Laryngeal Muscles

In addition to the extrinsic laryngeal muscles, two major groups of muscles attach to the hyoid bone and originate from either above or below the hyoid bone. The suprahyoid muscle group includes the digastric, stylohyoid, mylohyoid, geniohyoid, hyoglossus, and genioglossus. The infrahyoid muscle group includes the sternohyoid and omohyoid.

It is clinically useful to understand how each extrinsic and supplementary laryngeal muscle contributes to raising or lowering the larynx and moving it forward and backward, as listed in the following table:

Raise	Lower	Forward	Backward
Digastrics	Omohyoid	Sternothyroid	Omohyoid
Geniohyoid	Sternohyoid	Digastric	Digastric
Mylohyoid	Sternothyroid	Mylohyoid	Stylohyoid
Stylohyoid		Geniohyoid	
Genioglossus		Genioglossus	
Hyoglossus			
Thyrohyoid			

The raising and lowering of the larynx is observable and noted mainly during the pharyngeal stage of swallowing. These movements serve primarily to help protect the airway from aspiration of food or liquid (see Sidebar 2.11). The extrinsic laryngeal muscles also come into play slightly during production of higher and lower pitches (especially in untrained singers) (see Sidebar 2.12). Conversely, if you lower your pitch dramatically, your larynx will lower. A good speaking voice does not apparently require much active muscle involvement of the extrinsic laryngeal muscles. Trained singers keep the height of the larynx nearly constant while singing a range of high and low notes (Sataloff, 1981).

Sidebar 2.11

To illustrate, take your index finger and place it on your Adam's apple. Swallow and you will feel the larynx rise and then return to its resting position.

Sidebar 2.12

To illustrate, again place your index finger on your Adam's apple and count from 1 to 10 at your normal pitch and loudness. You should not feel any appreciable raising or lowering of the larynx. Repeat this exercise, but when you get to the numbers 6 to 10, raise your pitch dramatically. You should feel the larynx rise.

Laryngeal Cartilages

Five major laryngeal cartilages are important for voice production and airway protection: the cricoid, the thyroid, the paired arytenoids, and the epiglottis. These are shown in Figures 2.7 through 2.9. Two other small paired cartilages, the corniculates (cone-shaped, elastic cartilages on the apex of the arytenoids extending into the aryepiglottic folds) and the cuneiforms (cone-shaped, elastic nodules located in the aryepiglottic folds), apparently play only a minimal role in the phonatory functions of the larynx. The larynx develops in utero from paired branchial arches, so slight asymmetries in structure are often observed, particularly as one ages (Lindestadt and colleagues, 2004). Such asymmetries typically do not affect voice, though.

The cricoid, thyroid, and arytenoid cartilages are intricately connected to one another by joints, ligaments, membranes, and muscles (we will describe these connecting structures in later sections of this chapter). The cricoid is the second largest of the three cartilages and is a signet-shaped ring connected to the first tracheal ring. The two pyramid-shaped arytenoid cartilages sit atop its high posterior wall. The base of each arytenoid cartilage has two concave and smooth surfaces, called processes, to which muscles attach. One of these surfaces, the muscular process, is laterally directed and is the attachment for those intrinsic laryngeal muscles that cause the arytenoid cartilage to rock, rotate, and slide on the cricoid cartilage. The other process, the vocal process, is anteriorly directed and is the posterior attachment for the vocal ligament and vocalis muscle. The remaining major cartilage, the thyroid cartilage, is the largest of the three listed. It has several parts: two laminae, a superior thyroid notch, two superior horns, two inferior horns, and two oblique lines. The two laminae fuse anteriorly in the midline and form the laryngeal prominence (commonly called the Adam's apple). In postpubertal males, the angle of the laryngeal prominence is approximately 90°, and in

Figure 2.7 Anterior View of the Laryngeal Cartilages, Hyoid Bone, and Epiglottis

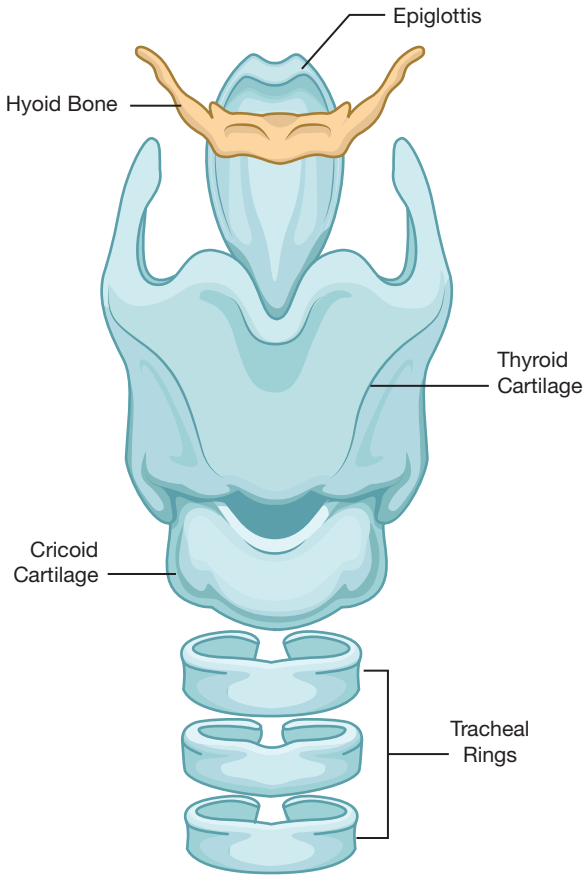
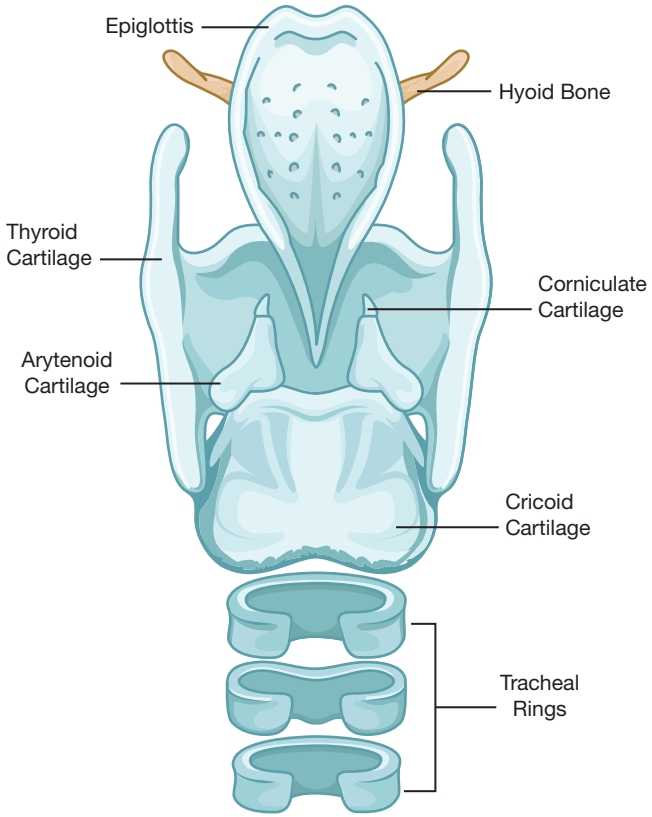


Figure 2.8 Posterior View of the Laryngeal Cartilages, Hyoid Bone, and Epiglottis



females, the angle is approximately 120°. If you feel your Adam's apple with your middle finger oriented horizontally and then place your index finger right next to it, you should feel a v-shaped notch between the laminae; this is the superior thyroid notch, present because the laminae are incompletely fused. The superior horns are posterior points of attachment for ligaments connecting the thyroid cartilage to the hyoid bone above. The inferior horns are also posterior points of attachment and connect the thyroid cartilage to the cricoid cartilage below via a joint. Each laminae also contains an oblique line, a ridge that descends diagonally from superior to inferior; this ridge is a line of attachment for some of the extrinsic laryngeal muscles and the inferior pharyngeal constrictor muscle. Similar to cartilage throughout the skeletal system, all the laryngeal cartilages are coated with a tough leathery covering (the perichondrium), which gives the larynx a waxy look. This perichondrium is thicker on the outside than the inside of the larynx.

Extrinsic Laryngeal Ligaments and Membranes

Ligaments and membranes also connect parts of the larynx to adjacent support structures. Some of these ligaments and membranes connect laryngeal cartilages to the epiglottis, some connect laryngeal cartilages to the hyoid bone, some connect the epiglottis to the hyoid bone or tongue, and some connect laryngeal cartilages to the trachea. It is helpful in gaining an understanding of some of these ligaments and membranes if one