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1 Anatomy and Physiology of Phonation

Summary
This chapter describes the anatomy and physiology of key laryngeal structures involved in *phonation* (i.e., vocal fold vibration). A description of the cartilaginous framework, connective tissues, intrinsic and extrinsic laryngeal muscles, and details of neural innervation are provided and physiological principles underlying vocal fold oscillation are explained. Because the process of phonation is a combination of respiratory and laryngeal function, a basic description of inspiratory and expiratory activity and control will also be provided. Finally, the relationship between normal anatomy and physiology to disordered conditions is presented.

Keywords: larynx, phonation, respiration, voice, dysphonia

1.1 Learning Objectives

At the end of this chapter, learners will be able to
- Identify the cartilaginous, soft tissue, and neurological substrates of the larynx.
- Compare and contrast the roles of intrinsic and extrinsic laryngeal muscles.
- Discuss the aerodynamic and muscular forces underlying vocal fold vibration during phonation.
- Compare and contrast muscular and neurological control during adjustments of vocal fundamental frequency and intensity.
- Identify key musculature involved in inspiration and expiration and describe the necessary coordination between respiratory and laryngeal function to produce phonation.
- Describe the key characteristics of the sound wave produced at the vocal fold level and how supraglottal function affects and transforms this sound.

1.2 Introduction

Phonation is primarily the result of aerodynamic forces acting on the inherently elastic tissue of the vocal folds, setting them into vibration and creating acoustic energy which we call "**voice.**" The characteristics of this vibration (e.g., the frequency of vibration) may be modified by muscular forces which influence the effective mass and tension of the vibrating folds. We are constantly modifying the aerodynamic and muscular forces underlying voice production to result in the wide variations of pitch and loudness produced in typical speech or in other aspects of voice function such as singing.

When laryngeal structure or physiology is impaired, the result may be a negative impact on laryngeal function, the efficiency of phonation, communication effectiveness, and subsequent perceptual characteristics of voice quality. The negative impacts on phonation and voice quality are perceptually labeled as **dysphonia**, which many speech–language pathologists will encounter during clinical practice. The purpose of this chapter is to provide an overview of the anatomy and physiology underlying normal, healthy phonation as a foundation to facilitate advanced understanding of impairments that result in dysphonia and their assessment, diagnosis, and treatment, which subsequent chapters will cover.

1.3 Evolution and Biological Roles of the Larynx

Voice production is considered an "overlaid" or nonbiological function of the larynx, taking a backseat to respiration, airway protection, and the generation of lung pressure to fixate the thorax during physical activity. According to Hirose, the larynx evolved as a simple muscular sphincter atop the primitive lung that would protect against entry of water or food.[1] This sphincter evolved into a more complicated valve capable of abduction (separation) or adduction (combination) at various levels within the larynx. Eventually, in mammals this protective structure also became used as a type of flutter valve that would vibrate during the controlled expiration.

During passive and active respiration, the **glottis** (the space between the two vocal folds) acts as an air valve. At rest, the two vocal folds lie in an **abducted** (away from midline) position creating an open glottis and a continuous passageway from the lungs to the oral cavity. During inspiratory cycles, the glottis will widen and then return to a resting open position during expiration. Deep, forceful inhalations will be accompanied by an even wider glottis. The vocal folds can be adjusted to an **adducted** (toward midline) position, so that the glottis will close forcefully to allow for the buildup of subglottal air pressure, which is required for coughing and clearing material that inadvertently falls into or irritates the larynx, trachea, or lungs. By closing the glottis in this manner, an individual can also fixate the thorax to direct muscular effort to the limbs for lifting and exercise. In a similar manner, glottal closure allows for the generation of abdominal pressures for bodily functions such as defecation.

Phonation for speech is an intentional (voluntary) behavior, but a number of involuntary reflexes mediated by the nervous system can override phonation, interrupting ongoing voice production (e.g., causing a voice break) or preventing a speaker from initiating phonation (e.g., think about food/liquid "going down the wrong pipe" when swallowing—you will experience strong laryngeal closure and/or cough, but not be able to produce voice until the stimulus is cleared). Peripheral sensory receptors located throughout the laryngeal surfaces can elicit adductor responses and cough subsequent to noxious stimuli. Central nervous system (CNS) pattern generators also mediate tonic and phasic activity in the 10th cranial nerve, called the **vagus nerve**, during respiration and swallowing.

The **laryngeal adductor reflex** is a response triggered by activation of the sensory division of the vagus nerve, which elicits activity within a brainstem nucleus called the **solitary tract nucleus** (STN—a.k.a., nucleus tractus solitaries; ▶ Fig. 1.1) in the medulla. The STN forms a reflex loop with the motor nucleus of the vagus nerve, the **nucleus ambiguus** (NA). Upon activation, the STN relays excitatory signals to the NA which then stimulates activity in the laryngeal adductor muscles via firing of **alpha motor neurons**.

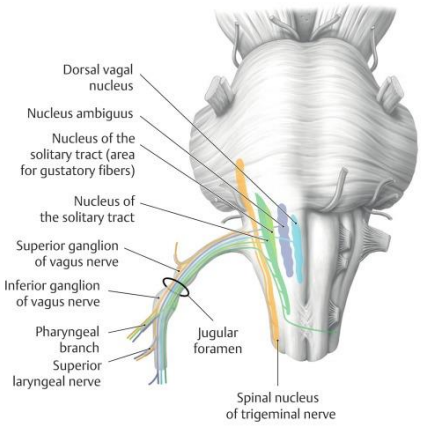

Fig. 1.1 Locations of sensory and motor nuclei of the vagus nerve in the brainstem. (From LaPointe L. Atlas of Neuroanatomy for Communication Science and Disorders, 1st ed. New York: Thieme Publishers; 2011.)

This reflex serves to forcefully close the glottis to protect the lower airway. At its most extreme, the laryngeal adductor reflex can present as **laryngospasm**, which is a prolonged tonic contraction of the laryngeal adductor muscles.

The production of **cough** can be under voluntary or involuntary control by the nervous system. Involuntary cough is triggered by stimulation of the laryngeal sensory receptors and typically serves to eliminate foreign particles or adverse sensations throughout the larynx or in the subglottic spaces.[2] The physiology of cough can be grouped into three phases: an *inspiratory phase* involving recruitment of the inspiratory muscles (diaphragm and intercostals), a *compressive phase* involving recruitment of the expiratory muscles along with strong medial compression at the glottis via recruitment of the laryngeal adductors (and subsequent buildup of subglottal pressure), and an *expulsive phase* via recruitment of additional activity in the abdominal muscles resulting in high pressure air pulses being sent through the glottis.[2] The pressure pulses flowing through the glottis help to clear the airway but also result in phonation, producing the familiar perception of a cough sound. Conscious suppression of cough is possible through cortical mechanisms; however, the neural circuitry which allows for this voluntary override of the cough reflex is not well understood.[3] Voice therapy focusing on conscious control and patterning of the respiratory muscles along with semiocclusion (narrowing) in the oral cavity during exhalation are used as a behavioral voice therapy modality to treat conditions of chronic cough.[4]

It is clear that the basic biological functions of the larynx generally have precedence over the overlaid development of behavioral voice function. In fact, the body's need to breathe or protect the trachea and lungs can interrupt phonation, which many readers of this book will have experienced when trying to speak while swallowing or after an exhausting physical exercise.

1.4 Respiratory Function

Phonation is built upon a foundation of respiration. Respiratory drive provides the power source for phonation. Speakers breathe during speech using a combination of diaphragmatic and thoracic muscular activity. Primary use of the **diaphragm** (▶ Fig. 1.2) for speech breathing is considered the most efficient method as its contraction is not resisted by bone, whereas the thoracic muscles must expand the rib cage to influence lung volume. During passive rest breathing, brainstem pattern generators control respiratory cycles at an unconscious level. However, for speech, **pyramidal (voluntary) pathways** in the CNS engage the lower motor neurons (LMN) of specific spinal nerves that form the **phrenic nerve** (nerves C3, C4, and C5) which innervates the diaphragm. Spinal intercostal **nerves** also leave the spinal cord at the thoracic level to innervate the **internal** and **external intercostal muscles** (▶ Fig. 1.3). Diaphragmatic contraction moves the lungs inferiorly, while contraction of the external intercostal muscles moves the lungs horizontally and superiorly. This has the effect of increasing lung volume and lowering air pressure within the lungs, causing air to flow in for inspiration.

Exhalation during passive breathing is accomplished by rapid relaxation of the diaphragm and/or intercostal muscles. For speech, the air must be efficiently controlled. This is accomplished through two muscular checking actions: (1) relaxation of the diaphragm and external intercostals in concert with (2) increasing activation in the abdominal and internal intercostal muscles. The abdominal muscles are antagonistic to the diaphragm, while the internal intercostals are antagonistic to the external intercostals. These checking actions help control the outflow of air from the lungs to support speech production. Exhalation is also controlled at the level of the vocal folds, which act to valve the upward flowing air. Fully adducted vocal folds will completely seal the glottis, which will cause an increase in subglottal air pressure as exhalation from the lower lungs continues.

1.4.1 Nervous System Regulation of Respiration

The primary purpose of respiration is life sustenance, with respiratory support for speech being a secondary overlaid function. Nervous system pathways and regions that modulate the activity in respiratory muscles will vary depending on whether respiration is being controlled volitionally (e.g., during speech, holding breath, and blowing) or at an unconscious involuntary level (e.g., quiet breathing while reading, watching television, and sleeping). Control from involuntary pathways will override conscious voluntary control whenever the body detects an urgent need for different blood chemical levels (e.g., oxygen).

Conscious control of the respiratory cycle (inhalation/exhalation) involves cortical motor regions which include the bilateral pyramidal pathways. Pyramidal neurons, the majority of which originate in the primary motor cortex (precentral gyrus) of the frontal lobe, are responsible for executing motor programs for volitional, planned movement including respiratory activity

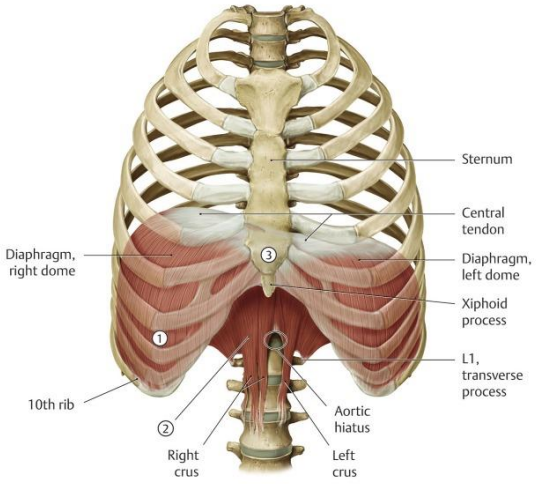

Fig. 1.2 The diaphragm. (From Baker E. Anatomy for Dental Medicine, 2nd ed. New York: Thieme Medical Publishers; 2016.)

during speech production. These neurons will travel a direct route to synapse on LMNs in the brainstem and spinal cord. Target LMNs in the spinal cord include those of the intercostal and phrenic nerve, which travel into the periphery to innervate the diaphragm, intercostal, and abdominal muscles.

The brainstem **reticular formation** contains neurons which control the involuntary patterns of respiration (▶ Fig. 1.4). Located throughout the pons and medulla, the regions of reticular formation related to involuntary respiratory patterns can be organized into four areas, two in the pons (**pneumotaxic** and **apneustic** centers) and two in the medulla (**ventral respiratory group [VRG]** and **dorsal respiratory group [**DRG**]**). The medullary DRG acts as the pacemaker for involuntary/passive respiration. These neurons also drive activity in the VRG. The DRG needs stimulation to modulate respiratory activity—this stimulation arrives in the form of sensory information provided by peripheral sensory nerves via input from the lungs and cardiovascular system (mechanoreceptors and chemoreceptors). This peripheral stimulation also influences the **apneustic center**. When neurons of the DRG depolarize and fire, they send excitatory signals to the phrenic and intercostal nerves which innervate the diaphragm and external intercostal muscles, respectively. When ventilation demands are high, such as during physical exercise, the DRG will facilitate activity in neurons of the VRG, which then recruit additional motor neuron pools to further activate the muscles of inspiration and expiration.

The pontine pneumotaxic and apneustic centers can be thought of as "fine-tuning" the activity of the DRG. The apneustic center provides stimulation to the DRG to facilitate and prolong inspiration (e.g., when out of breath and needing to take deep, prolonged inhalations). The **pneumotaxic center** acts as an "off-switch" for inspiration. Neuronal activity in the pneumotaxic center causes termination of inspiration by inhibiting activity of the DRG. This in turn causes higher respiratory frequency (e.g., breathing faster) and reduced tidal volumes.

Activity of the brainstem respiratory pattern generator results in a resting respiratory rate of **12 to 16 breaths per minute**. Inspiration usually lasts approximately 2 seconds, and expiration lasts about 3 seconds. The normal inspiratory rate and rhythm is called **eupnea**, and difficult respiration is termed **dyspnea**. Respiratory rate and patterns can be modified when respiration is brought to a conscious level by voluntary acts (e.g., speech) and during complex involuntary reflexes such as sneezing, coughing, and vomiting. In addition, respiratory activity can be affected by conditions such as emotional state via input from the limbic system, or temperature via the hypothalamus.

The vagus nerve is also influenced by brainstem respiratory pattern generators and voluntary cortical pathways, and is active during voluntary and involuntary cycles of respiration. Laryngeal muscles controlled by the vagus are activated by brainstem respiratory centers during inspiration. The abduction–relaxation phasic action of the glottis resulting from this muscular activity is visible on laryngeal endoscopy when a patient is breathing at rest. During expiration, laryngeal muscles are also activated. Voluntary cortical control of laryngeal muscles during inspiration is present for such tasks as respiratory support for speech and deep inhalations, both of which require increased activity in laryngeal muscles via the vagus nerve to widen the glottis.

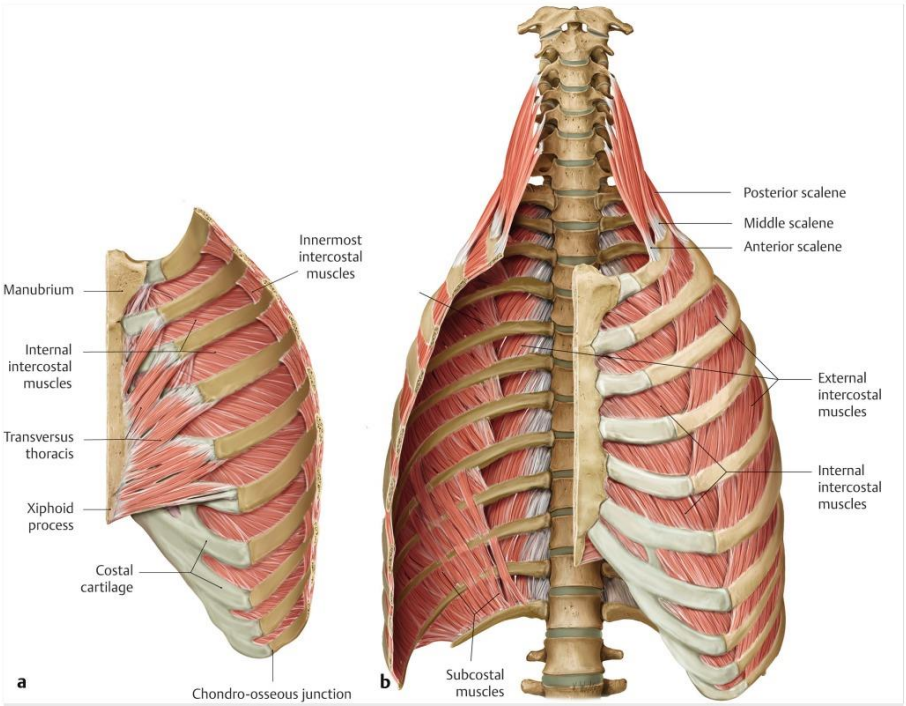

Fig. 1.3 The intercostal muscles. (From Baker E. Anatomy for Dental Medicine, 2nd ed. New York: Thieme Medical Publishers; 2016.)

1.5 Laryngeal Framework

The larynx is located in the midline of the anterior neck (▶ Fig. 1.5), suspended from the hyoid bone above and attached to the trachea below via connective tissue (membranes, ligaments, and muscles). Removal of the connective tissue within the larynx would reveal a framework of six named cartilages (some are paired and some are unpaired). Five of these cartilages articulate with at least one other laryngeal cartilage, while the remaining one is suspended in connective tissue. Although technically not part of the laryngeal framework, the influence of the hyoid bone on laryngeal position must be emphasized. It is generally accepted that vertical hyoid position, which is determined by the degree of activation in muscles which attach to it, can have significant influence on the physiology of phonation.

1.5.1 Hyoid Bone

The hyoid is a horseshoe-shaped bone in the anterior midline of the neck positioned at the level of the third cervical vertebra. The broad central portion of the hyoid is referred to as the body, to which are fused two lateral (left and right) and superior bony projections called the "greater horns" (or cornu, which is Latin for "horn") and "lesser horns" of the hyoid, respectively (▶ Fig. 1.6). The body, greater horns, and lesser horns serve as points of attachment for muscles and other connective tissue. The hyoid serves as an origin or insertion point for muscles that move the jaw, tongue, and larynx. Laryngeal muscles that connect to the hyoid can have the effect of elevating the hyoid bone in a superior and anterior direction, bringing the hyoid bone in closer approximation to the laryngeal framework, or depressing the larynx in an inferior direction.

1.5.2 Thyroid Cartilage

The singular (unpaired) thyroid cartilage is located in the anterior midline of the neck and forms a large portion of the anterior laryngeal border. The thyroid cartilage is shaped like a shield (▶ Fig. 1.6), formed by two broad plates (laminae) of hyaline cartilage which fuse at the midline. Numerous muscles attach to the thyroid lamina, influencing its vertical position. Superior and inferior thyroid horns (cornua) project from the posterior borders of the thyroid lamina and serve as point of attachment for connective tissue. At the apex of the thyroid midline, the fusion is incomplete creating the **thyroid notch**, which can be located by digital palpation in most adults. Immediately inferior to the notch