5.5.1 Discuss the role of the larynx in phonation. What other structures of the aerodigestive tract are important in phonation as well? HL

Voice is an acoustic product resulting from the semicyclical vibrations of the two vocal cord(s) (ie, vocal folds). The vibration of the vocal cords is age and gender dependent and is controlled by myoelastic properties and aerodynamic forces; the vibration is generated as the air expelled under pressure from the lungs passes between the vocal cords and sets the cords into an oscillatory motion. Normal voice is generated by the vibratory wave-generating oscillations of the membranous portion of the vocal cords (the mucosa), which slides/glides in an undulating (phase locked) manner over the underlying muscle.

- Myoelastic properties
  - Paired intrinsic laryngeal muscles (ILM), which are responsible for the size, shape, length, mass, stiffness, and tension characteristics of the vocal cords.
    - The ILM include the thyroarytenoid muscles, the pairs of lateral cricoarytenoid muscles, the posterior cricoarytenoid muscles, and the interarytenoid muscle, which consists of both transverse and oblique portions.
    - The ILM are innervated by the recurrent laryngeal nerves (RLNs) and all muscles, with the exception of the posterior cricoarytenoid muscles (the only vocal cord abductor), are responsible for vocal cord adduction and vocal cord approximation
  - Bilateral cricothyroid musculature elongates the vocal cords, produce pitch elevation.
  - The nonmuscular myoelastic properties include membranes (mucosa), ligaments, glandular elements, a blood supply, and nerves, all of which are located within the articulating cartilaginous housing that comprises the thyroid, the cricoid, and the two arytenoid cartilages.

- Abnormal vocal production achieved by subglottic and supraglottic structures (ex. False vocal folds, epiglottis) – fixing an abnormal voice producing area can cause further weakening of voice

- The entire voice box rests on the trachea and is suspended above from the hyoid bone, which communicates with the base of the tongue. When this connection is affected by as little as minor lingual tension or inappropriate vertical larynx positioning, the result may include altered voice production.

- Paired extrinsic laryngeal musculature produce vertical laryngeal motions which are crucial in phonation (singing), swallowing, respiration, and yawning, and in speech articulation. When this vertical movement is affected, voice production may be severely compromised even if the glottis looks "normal" on a routine ear, nose, and throat (ENT) exam.

Motor & Sensory Control: Motor cortex $\rightarrow$ brainstem nuclei $\rightarrow$ right and left branches vagus nerve (CN X) $\rightarrow$ motor end plate of intrinsic laryngeal mm via RLNs $\rightarrow$ vocal cord contractions $\rightarrow$ voluntary and involuntary phonation

- The entire efferent process can be accomplished within 90 ms
- Requires coordination of all vocal tract and respiratory laryngeal musculature via the central nervous system motor neurons. The coordination of these movements is achieved by a complex neural network with access to phonatory motor neuron pools that receive proprioceptive input from the various receptors associated with these three systems and by control of voluntary vocalization rather than involuntary vocalization involving different brain regions.
- The left RLN is longer than the right nerve, d/t differential axonal composition of both nerves, the efferent impulses manage to arrive at the two vocal cords almost simultaneously, causing the vocal cord vibration to be semiperiodic. This type of vibration makes the sound of the voice "human."
- The vagus nerve also branches into the left and right superior laryngeal nerves (SLNs), which mediate the afferent signals from the larynx via their internal branches.
The external branches of the SLNs innervate paired cricothyroid muscles.
The action of the cricothyroid musculature is also responsible for the motion of the vocal cords seen in paralysis of the vocal cords due to RLN involvement. When some motion of the vocal cord is observed on the paralyzed side, it must be interpreted with caution as a sign of recovery, but rather as motion secondary to the ipsilateral SLN-mediated impulses. When the SLN is out in addition to the RLN, the posterior glottis will not approximate, a wider posterior gap will be present, and the arytenoids will not touch on phonation. Observing and documenting these conditions during clinical PhFS are of paramount importance for treatment planning.

- **Because of the contra- and ipsilateral innervation of the corticobulbar tract, a unilateral corticobulbar tract lesion will not cause unilateral vocal cord paralysis.**

### Vocal Cords

- **Phonation: subdivided into muscular components (the "body") and nonmuscular components (the "cover").**
  - Body of the vocal cords is formed by the two thyroarytenoid muscles, which contain fast (adductive) and slow (eg, phonatory) fibers that determine the length, contour, and glottic closure shape of the vocal cords and that regulate the tension of the cover that slides over the body of the vocal cords to create the mucosal vibratory wave.
    - Mucosal vibratory wave undulates, proceeding from the inferior (ie, lower lip) to the superior surface (ie, upper lip) of the vocal cords.
    - The area between the upper and lower lips adjusts as pitch and loudness change; therefore, when a phonatory lesion is located within this space, its location and size determine the area of pitch and loudness dysfunction. More severe if anteriorly located lesions than by larger lesions located toward the upper lip or on the superior phonatory surfaces.
  - The cover is subdivided into the outer and the inner layers and the lamina propria; the latter consists of three layers: superficial (the Reinke space), intermediate, and deep. The vocal ligament is the free edge of the conus elasticus, belonging to the deep and intermediate layers of the lamina propria.
    - Obliteration of the Reinke space retards or prevents the mucosal vibratory wave, resulting in dysphonia of varying severity.
    - If one vocal cord nonvibratory and the other vibrates and approximates well against the nonvibrating vocal cord, the voice may be remarkably good despite the insufficiency of one cord. Therefore, it is important at times not to "repair" the stiff vocal cord but to leave it alone or even make it stiffer to improve the overall voice quality.
    - Most benign phonatory mucosal lesions are typically found within the superficial layer. If located on the superior surface of the vocal cord away from the vibratory edge, the voice may not be affected at all, even if the lesion is large. These findings are crucial in determining the extent of surgical interventions.

- **Clinical vocal cord divisions:**
  - Vibratory (membranous) and nonvibratory (cartilaginous) portions.
    - At rest, they outline a V-shaped space called the glottis (see Figure 29–1). The front of this V forms the anterior glottic commissure, and the back of the V forms the posterior glottic commissure.
    - The posterior end of each vocal cord (the thyroarytenoid muscle) inserts into the muscular process of each of the arytenoid cartilages. The maximum width of the posterior commissure occurs during inspiration or cough and measures approximately 9–12 mm, or three times the most posterior width of the muscular portion of the vocal cord at rest.
    - When the vocal cords approximate for phonation, the entire glottis is closed in a male, whereas a small posterior chink is often present in a female, giving the female voice
quality a slightly softer and airy tone. The specific shapes of glottic phonatory closure allow variations in normal voice qualities.

- Anterior, middle, and posterior thirds, with nodular lesions located at the anterior third juncture and opposite each other if bilateral. An asymmetric location of mucosal lesions is found in mixed-type organic dysphonias.

The Vibratory Process
- The two thyroarytenoid muscles, together with the other ILM and the extrinsic laryngeal muscles, control the relative elasticity and stiffness of the vocal cords, determine the shape of the mucosal vibratory wave, which in turn determines the pitch, loudness, and tone of the voice. The amplitude of the mucosal vibratory wave is wider at the lower pitches, whereas reduced mucosal vibratory wave amplitude predominates at high pitches or at any pitch level when the cover is stiff.
- The duration and shape of the mucosal vibratory wave cycle form specific opening and closing phases that determine specific vibratory modes or vocal qualities (eg, fry, normal, overpressured, breathy, or falsetto). The time interval between cycles is called the fundamental period ($F_0$), whereas in perceptual terms it is referred to as a pitch period.

The Aerodynamic Properties of Phonation
- The aerodynamic properties of phonation include the subglottic air pressure ($P_s$), the airflow (AF), the supraglottic pressure ($P^s$), the intraoral pressure ($P_{io}$), and the glottal resistance, all of which are responsible for the Bernoulli effect, which separates the approximated vocal cords during phonation.
- To generate sound, $P_s$ must reach at least 5 cm H$_2$O, but $P_s$ can exceed 50 cm H$_2$O in loud or overly pressured (ie, pathologic) phonation. Normal conversational voice is produced between 6 and 10 cm H$_2$O $P_s$ at approximately 65–70 dB, whereas a loud voice can reach 85–95 dB.
- The mean airflow in normal phonation ranges from 89 to 141 mL/s and increases as the fundamental period and the loudness are elevated. The glottal resistance cannot be measured directly, but is estimated to vary from 20 to 150 dyne/s/cm$^3$ depending on the pitch and the sound intensity.

Resonation
- When the voice ($F_0$) resonates within the entire vocal tract (ie, the larynx, trachea, pharynx, and oral and nasal cavities) and when the vocal tract articulates, speech, singing, or other forms of communication are formed.

Ornamentation in voice can result from specific vocal tract configurations and specific time-locked acoustic events, with rate approximating 5–6 Hz for vibrato or vocal tremor. It is interesting to note that tremor-like vocal oscillations having similar rate may be present in deception.

5.5.2 What is the difference between hoarseness and dysarthria? What are distinguishing features of each?

<table>
<thead>
<tr>
<th></th>
<th>Hoarseness (dysphonia)</th>
<th>Dysarthria</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition</strong></td>
<td>a disorder characterized by altered vocal quality, pitch, loudness, or vocal effort that impairs communication or reduces voice-related quality of life (QOL) alteration in the quality or character of phonation</td>
<td>the abnormal articulation of sounds or phonemes. The pathogenic mechanism in dysarthria is abnormal neuromuscular activation of the speech muscles, affecting the speed, strength, timing, range, or accuracy of movements involving speech</td>
</tr>
<tr>
<td><strong>Patient description</strong></td>
<td>Breathy, harsh, rough voice</td>
<td>distortion of consonant sounds</td>
</tr>
<tr>
<td><strong>Cause</strong></td>
<td>mechanical, a physical</td>
<td>Neurogenic, related to dysfunction of the central nervous system,</td>
</tr>
</tbody>
</table>
Disorders of the pharynx and larynx involve nerves, neuromuscular junction, or muscle, with a contribution of sensory deficits in some cases. Dysarthria can affect not only articulation but also phonation, breathing, and prosody (emotional tone) of speech. Total loss of ability to articulate is called anarthria.

### Common Etiologies

- Viral illness
- Vocal fold paralysis – unilateral: vocal fatigue, symptoms similar to LPR, aspiration, subjective shortness of breath especially with vocal use (RF: cardiac, c-spine, thyroid surgery; cancer; etc); bilateral: often iatrogenic vs. cancer
- LPR – globus sensation, cough, frequent throat clearing (no postprandial heartburn = GERD), exam: cobblestoning, edema, redness, 20% concurrently have esophageal abnormalities... image esophagus with barium swallow, etc.
- Laryngeal polyps – recurrent papillomatosis d/t HPV 6 and 11
- Allergy
- Vocal abuse
- Dysplasia, Cancer – especially in patients with hoarseness lasting >2wks, usually SCC of true vocal folds (RF: Smoking)
- Vocal polyps – d/t local tissue inflammation
- Vocal nodules (always bilateral) – due to vocal abuse
- Vocal fold granulomas – d/t extraesophageal reflux

### Mayo Clinic Classification:

<table>
<thead>
<tr>
<th>Type</th>
<th>Localization</th>
<th>Auditory Signs</th>
<th>Characteristic Disease(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flaccid</td>
<td>Lower motor neuron</td>
<td>Breathy, nasal voice, imprecise consonants</td>
<td>Stroke, myasthenia gravis</td>
</tr>
<tr>
<td>Spastic</td>
<td>Bilateral motor neuron</td>
<td>Strain-strangle, harsh voice; slow rate; imprecise consonants</td>
<td>Bilateral strokes, tumors, primary lateral sclerosis</td>
</tr>
<tr>
<td></td>
<td>Unilateral upper motor neuron</td>
<td>Consonant imprecision, slow rate, harsh voice quality</td>
<td>Stroke, tumor</td>
</tr>
<tr>
<td>Ataxic</td>
<td>Cerebellum</td>
<td>Irregular articulatory breakdowns, excessive and equal stress</td>
<td>Stroke, degenerative disease</td>
</tr>
<tr>
<td>Hypokinetic</td>
<td>Extrapyramidal</td>
<td>Rapid rate, reduced loudness, monopitch and monoloudness</td>
<td>PD</td>
</tr>
<tr>
<td>Hyperkinetic</td>
<td>Extrapyramidal</td>
<td>Prolonged phonemes, variable rate, inappropriate silences, voice stoppages</td>
<td>Dystonia, HD</td>
</tr>
<tr>
<td>Spastic and flaccid</td>
<td>Upper and lower motor neuron</td>
<td>Hypermnasality, strain-strangle, harsh voice, slow rate, imprecise consonants</td>
<td>ALS, multiple strokes</td>
</tr>
</tbody>
</table>
5.5.3 What are some objective measures of voice disorders? What is shimmer? Jitter? S-Z ratio? HL

**LARYNGEAL FUNCTION STUDIES**

1. Acoustic Measures
   
   i. Average pitch: The patient repeats the sustained vowel "ah" 3 times, each lasting at least three seconds. For these, the average pitch (in Hertz) can be measured. Adult females generally have an average pitch of 175 to 250 Hz, while male adults average 80 to 150 Hz.

   ii. Jitter perturbation: From the same vowel productions used in #1 (above), the cycle-to-cycle pitch period variation can be established. This is reflective of the frequency perturbation and normally falls below 1%.

   iii. Shimmer perturbation: As with jitter, shimmer is a measure of perturbation occurring in vowel production using the same sustained "ah" vowel. Shimmer reflects cycle-to-cycle amplitude variations in the voice waveform. Normal shimmer values are less than 5%.

   iv. Noise-to-harmonic ratio: From the same vowels, the amount of the signal coming from voicing versus noise can be measured. Normal noise-to-harmonic ratio is less than 0.2%. Normal measures of jitter, shimmer, and noise-to-harmonic ratio are influenced by algorithm, recording environment, recording technique, and recording hardware.

   v. Maximum phonation time: The maximum amount of time a person can sustain phonation of "ah" is timed. Typically, adult females sustain phonation of "ah" from 15 to 25 seconds; while males range from 25 to 35 seconds.

   vi. S/Z ratio: The patient is timed while sustaining production of "s" and "z" for as long as possible. "S" and "z" are essentially produced in the same way; however, the "z" is voiced, while the "s" is not. The ideal ratio of "1" is when the productions are sustained
for the same amount of time. If there are difficulties in glottal valving, the "s" will be phonated longer and will be reflected by ratios greater than 1.

2. Physiologic pitch range:
   i. Highest possible pitch: The patient is instructed to, "Start at a comfortable pitch and loudness, then raise your pitch gradually as high as it will go until it breaks off." The examiner demonstrates.
   ii. Lowest possible pitch: The patient is instructed to, "Start at a comfortable pitch and loudness, then lower your pitch gradually as low as it will go, as if you were yawnning or sighing." The examiner demonstrates.

**Phonatory function studies (PhFS):** composed of at least of two primary parts: (1) an acoustic portion that examines the nature of the generated sound, and (2) a visual portion that examines via stroboscopic transoral or transnasal approach the glottis and surrounding area including the subglottis. PhFS are considered a standard of modern voice care because they provide information beyond subjective clinical impressions; they also provide objective descriptions of normal and pathologic phonatory processes. These processes include (1) mapping acoustic voice characteristics, (2) correlating voice with physiologic findings, (3) providing guidelines for the development of efficacious treatment plans, (4) predicting the progress and outcomes of treatment plans, (5) providing preoperative–postoperative lesion mappings, and (6) providing documentation for medicolegal purposes. PhFS are reproducible and allow a contrast of individual results to a database specific to the patient's age and gender. The information these studies provide also allows for a frank discussion with the patient and education of the patient, including discussion of the risks and alternatives associated with various treatments.

- The acoustic portion records and analyzes the voice of the patient. This portion is of paramount value, specifically when a surgical intervention is planned and when the patient uses voice as a tool of labor. *Do not ignore* this part of the exam. An acoustic voice assessment provides information on the nature of the generated sound and should include physical voice recordings (analog, digital, or video) and an objective acoustic analysis; it should also include a subjective psychoacoustic analysis, a psychometric analysis, a phonometric analysis, or all of the above. Barring minor technical problems, either dedicated instrumentation or a computerized approach can be used for a fast, reliable, and reproducible acoustic analysis. Acoustic analysis provides information on sound duration, loudness, pitch, and spectral context, including static and dynamic pitch changes of the voice during speech.
  - **Pitch:** subjective, frequency of voice or speaking fundamental frequency. Recorded in vocal cycles/min (Hz). *Deviations in fundamental frequency expressed by jitter (pitch perturbation factor):* fundamental frequency value obtained by subtraction duration of pitch period from duration of period immediately preceeding it. Fundamental frequency is age and gender dependent. The average level of fundamental frequency for a child is approximately 250 Hz; it is 200 Hz for an adult female, and for an adult male, it is approximately 120 Hz. The maximum fundamental frequency range for both genders is from 36 to 1760 Hz, or roughly the distance from D1 to A6 on a piano. The speaking fundamental frequency of females drops over the life span, whereas this frequency becomes elevated in male geriatric populations.
  - **Loudness:** acoustict intensity, measured in Db. Dependent on subglottic air pressure and airflow exiting glottis. Reported as relative Db. Affected by fundamental frequency, normal loudness greatest at mid-frequency rantges and lowest at high/low levels of fundamental frequency. *Loudness pertubatation factors are known as Shimmer, and is used to describe acoustic intensity variation and dispersion.* Loudness of speaking 65-75 dB, values above or below this measure are pathologic.
  - **Phonetogram:** profile of fundamental frequency (dB) used to represent pitch and loudness. Reflects vocal capacity rather than glottic function.
  - **Spectral analysis:** 3-D representation of sound: time, intensity and frequency
  - **Multi-dimensional voice profile:** displays multiple vocal parameters in graphic form all at once
Rate analysis: define rate and extent of specific acoustic variations, pathologic if between 5-6 Hz, similar to vibrato rate.
Vocal cord contact area: % of vocal cord contact area, values <90% are abnormal
Vowel space: affected by fundamental frequency and loudness, examine if patient is a professional singer
Maximum phonation time: time individual can phonate per inhalation, normal 17-35 seconds (male), 12-26 seconds (female); reduction in this time due to hypofunctional glottis, prolongation in time due to overapproximated glottis

- The physiologic portion (31579) visualizes via rigid or flexible stroboscopic visualization, aerodynamics, glottography, electromyography, and special studies. and also maps the location, the extent, and the effects of phonatory lesions (when present), and their contribution to dysphonia. In addition to these two primary components, special tests may also be a part of the PhFS battery. These include delayed auditory feedback, voice load tests, nerve blocks, manual compression tests, and EMG.
  - Phonomicroscopy: Video stroboscopy, used to make >20% diagnostic and treatment planning decisions. Maps location of phonatory lesions wrt acoustic findings, provides fundamental frequency values, shows symmetry of vocal cord vibrations, reveals glottis closure configuration, shows horizontal excursion of vocal cords (i.e. amplitude), reveals appearance and working of upper and lower phonatory lips of the true vocal folds, demonstrates mucosal vibratory wave.
  - Videokymography (VKG): directly observes vocal cord vibrations by coupling a modified video camera to standard rigid endoscope and constant illumination. Less intuitive, less used in US than video stroboscopy.
  - Electroglostography: Evaluates vocal cord vibration using electrical impedance across tissue and open spaces. Electrodes are placed on the neck over the lamina of the thyroid cartilages; a weak current is passed between the electrodes, which generate an impedance curve that corresponds to the shape and nature of the vibratory cycle.
  - Aerodynamic tests: evaluate air “voice fuel” behavior during phonation. Measures subglottic and supraglottic air pressures as well as glottis air impedance and airflow at glottis including volume velocity. Interpretation difficult as tests are subject to voluntary motor responses and affected by variations in vocal intensity and register.
  - EMG: examines neuromuscular integrity of striated muscle by recording discharges of muscle, not often used to examine vocal cord paralysis, and role in examination is not well established and difficult to determine if muscles undergoing denervation or reinnervation.
  - Special physiologic tests include aerodynamic tests, manual pressure tests, and temporary denervation procedures.
    - Upper esophageal insufflation test: to test failures in acquiring voice after tracheal puncture procedures. Because sudden change in aerodynamics affects the glottic biomechanics, as does inhaling gases of other density than air (eg, helium), such tests are useful when examining a suspected psychogenic voice disorder.
    - Manual pressure test: laryngeal circumference pressure test, is useful in testing for muscular tension dysphonia as well as psychogenic dysphonia. It is also useful in assessing the viability of medialization procedures. Similarly, the head-positioning test, which can cause changes in vocal cord approximation, can be used as a predictor of the correction potential (therapeutic, surgical, or both) of breathy dysphonia. A neck pressure test can also be used to test failures in acquiring voice after esophageal injection (eg, following total laryngectomy).
    - Nerve blocks, as well as the so-called oral lidocaine bath, can be very useful in the differential diagnosis of psychogenic dysphonia. In addition, an RLN block is often crucial in testing for adductor spasmodic dysphonia and vocal tremor. A temporary block of the SLNs can be used in testing for abductor spasmodic dysphonia and in persistent postpubertal infantile dysphonia. The neural block test can also be used to test problems with air insufflation in patients after a total laryngectomy.
Radiologic PhFS include a videofluoroscopic exam of a nonfunctional phonatory segment after total laryngectomy. It also appears that neuroradiographic studies that use enhanced viewing to reveal fat deposits in vocal cords may be useful in studying nonmobile vocal cords. With additional testing, this technique may prove to be excellent in the differential diagnosis of voice disorders due to vocal cord paralysis or due to mechanical problems (eg, arytenoid joint dislocation or vocal cord fixation, or ankylosis).

5.5.4 How can laryngeal electromyography (EMG) assist in the diagnosis and management of voice disorders? CC


5.5.5 What are hyperfunctional voice disorders? Give us a list and short description of each. CC

5.5.8 Describe the clinical presentation of adductor vs. abductor spasmodic dysphonia (SD). How might you differentiate the two disorders? CC

5.5.9 What other neurologic disorders can produce a voice disorder? What is Meige’s disease? CC

5.5.13 Botulinum toxin injection is a common treatment for SD-what is the mechanism of action? Should you do unilateral vs. bilateral injection? What laryngeal muscle(s) do you inject and how do you find it? What are the outcomes of treatment, side effects of the injections, and advantages/disadvantages to other treatments?? What dosages are used? SW


5.5.14 Discuss recurrent nerve section for treatment of SD. What are advantages and disadvantages. SW

5.5.16 What systemic drugs have been used in treatment of SD and do they work? SW

5.5.17 What is paradoxical vocal fold motion? What are the organic and non-organic causes of the phenomena? AL
Paradoxical Vocal Fold Motion Disorder (Episodic Paroxysmal Laryngospasm)
Normal vocal folds maintain an open rima glottides (the space or opening between the vocal folds) at rest, abducting during inspiration and adducting slightly with expiration. Paradoxical vocal fold motion disorder (PVFMD) is a condition in which the larynx exhibits paradoxical vocal cord adduction during inspiration. Attenuation of the inspiratory component of the flow-volume loop, indicating a variable extrathoracic airway obstruction, may be seen when symptomatic (Fig. 62.5). This is a poorly defined disorder in which the clinical diagnosis relies on exclusion of other disorders with similar symptoms (e.g., asthma or subglottic or tracheal pathology). PVFMD is most common among individuals 10 to 40 years of age who are considered high achievers, educated, and competitive in their career or athletic forum. A high level of anxiety disorders is seen in this population. In adults, the disorder is predominantly found among women, but the gender difference
INTRODUCTION — Paradoxical vocal cord motion (PVCM) refers to inappropriate movement of the vocal cords, which results in functional airway obstruction and inspiratory or expiratory stridorous breathing. Patients with PVCM are often misdiagnosed with asthma, because the sound produced can be mistaken for asthmatic wheezing.

This topic will review the presentation and treatment of PVCM, also called vocal cord dysfunction, Munchausen stridor, psychogenic stridor, factitious asthma, pseudoasthma, and irritable larynx syndrome [1-3]. Features of wheezing illnesses other than PVCM are discussed separately. (See "Evaluation of wheezing illnesses other than asthma in adults" and "Diagnosis of asthma in adolescents and adults".)

ANATOMIC FINDINGS — In the normal larynx, the true vocal cords abduct on inspiration, opening the glottis, and adduct partially during expiration, closing the glottic aperture about 10 to 40 percent. Normal inspiratory abduction is controlled by the vagus nerve; inspiratory abduction can also be induced by sniffing and panting. Normal adduction of the true vocal cords occurs with phonation, swallowing, and during a Valsalva maneuver. Normal cough mechanics also involve vocal cord adduction for 0.2 seconds following the end of inspiratory phase; this is called the compressive phase as the expiratory muscles initiate shortening against a closed glottis; finally, the vocal cords abduct and forceful exhalation ensues [4].

In PVCM, adduction of the true vocal cords occurs on inspiration, expiration, or both [3,5,6]. The false vocal cords may also adduct abnormally and the posterior laryngeal wall may move anteriorly to compress the airway.

ETIOLOGIES — PVCM has been associated with psychosocial disorders, stress, exercise, perioperative airway and neurologic injury, gastroesophageal reflux, and irritant inhalational exposures. Some patients have concomitant asthma or have been misdiagnosed with asthma [7].

Psychosocial disorders and stress — PVCM has been associated with a variety of psychosocial disorders; however, it is not considered to be a form of malingering as patients do not intentionally produce their condition for secondary gain.

Several studies have reported an association of PVCM with a history of prior psychiatric illness, including depression, personality disorders, posttraumatic stress disorder, or a history of childhood sexual abuse [8-11]. In a case-control study comparing adolescents with PVCM to those with asthma, PVCM patients had higher levels of anxiety and more frequent diagnoses of generalized anxiety disorder and separation anxiety [12]. (See "Factitious disorder and Munchausen syndrome".)

Exercise — One form of PVCM occurs predominantly in young female athletes who present with dyspnea and sometimes stridor triggered by exercise [13,14]. In a group of 40 military patients with exertional dyspnea studied prospectively, 15 percent had PVCM [15].

Neurologic injury — PVCM has been demonstrated in patients after thyroid and cervical spine surgery and in one patient after a polycranial neuropathy from herpes simplex. In this setting, the presumed etiology is aberrant reinnervation or synkinesis [16].
Postoperative — PVCM may occur in the postoperative setting with the onset of dyspnea and stridor after extubation [17-20]. The use of flexible laryngoscopy in the immediate post extubation period allows differentiation between PVCM and other pulmonary etiologies of postextubation respiratory distress. It should be noted that postoperative onset of acute dyspnea and stridor is much more likely to be caused by laryngospasm rather than PVCM. Laryngospasm is usually an acute onset brief episode of sustained vocal cord adduction, often seen on emergence from general anesthesia. (See "Overview of the management of postoperative pulmonary complications").

Gastroesophageal reflux — Gastroesophageal reflux to the larynx and pharynx, also known as laryngopharyngeal reflux (LPR), has been associated with PVCM. Laryngoscopic findings consistent with LPR were present in 95 percent of juveniles with confirmed PVCM [21]. However, other studies have questioned the reliability of laryngoscopy in making the diagnosis of LPR [22]. It is unclear whether there is a causal relationship between LPR and PVCM. (See "Laryngopharyngeal reflux").

Irritants — Exposures to a variety of irritants (eg, ammonia, soldering fumes, cleaning chemicals, aerosolized machining fluids, construction dust, and smoke) have been associated with PVCM [5]. The exposures typically occurred within 24 hours of the onset of symptoms. Sometimes patients feel that ongoing problems with PVCM are related to a previous inhalational irritant exposure. While an acute exposure might cause temporary laryngeal irritation, it is unclear how a previous remote exposure would cause ongoing episodes of PVCM.

PRESENTATION — PVCM occurs most commonly in women between the ages of 20 and 40, but has also been reported in men and in the pediatric population [6,23]. Patients may present with significant respiratory distress and dramatic inspiratory stridor [15]. Often the diagnosis is suspected after multiple visits to the emergency department for these episodes, or during an evaluation for severe asthma [6,24]. Some patients may have both asthma and PVCM. (See "Evaluation of severe asthma in adolescents and adults").

In addition to dyspnea, patients may complain of throat tightness, a choking sensation, dysphonia, and cough. Less often, patients report gastroesophageal reflux, dysphagia, and rhinosinusitis. Onset of symptoms may be spontaneous or induced by exercise or irritant exposure.

During symptomatic episodes, stridorous sounds are loudest above the throat and are less audible through the chest wall, where the sound is attenuated by transmission through the airways and the pulmonary parenchyma. Typically, albuterol has minimal to no beneficial effect. While there is little information in the literature regarding the specific duration of these episodes, most patients' episodes last anywhere from several hours to several days. This distinguishes the episodes from laryngospasm which usually last seconds to a few minutes. (See 'Differential diagnosis' below.)

Some patients with PVCM experience dysphonia during or between attacks. On examination this is often associated with vocal cord hyperfunction (eg, excessive false vocal cord adduction and anterior-superior laryngeal compression during phonation). There are no reports yet of formal voice analysis in these patients. (See "Hoarseness in adults").

Rarely, acute respiratory distress may be of such severity that patients require endotracheal intubation or tracheotomy to restore airway patency before diagnostic tests can be performed. Immediately afterwards, cessation of wheezing and stridor is noted, suggesting that the airflow limitation was due to an upper airway process instead of asthma.

EVALUATION — The most useful tests in evaluating PVCM are pulmonary function tests (including flow volume loops and bronchoprovocation challenge) and laryngoscopy. In addition, patients with PVCM should be assessed for underlying psychosocial disorders.

Pulmonary function tests — Most patients have normal expiratory spirometry, and a few have mild restrictive physiology. In contrast, flow-volume curves often show flattening of the inspiratory loop and an increased ratio of forced expiratory flow to forced inspiratory flow at 50 percent vital capacity, consistent with extrathoracic airway obstruction (figure 1) [25]. However, flow-volume loops may be normal when the patient is asymptomatic between episodes.

PVCM may be induced with methacholine inhalation presumably through an irritant mechanism [26]. However,
normal flow volume loops after methacholine do not rule out PVCM. Methacholine inhalation challenge may be most helpful in conjunction with laryngoscopy to confirm that airflow limitation is due to PVCM and not asthma [26,27]. (See "Bronchoprovocation testing".)

Arterial blood gases usually do not show significant abnormalities, although mild hypercapnia may occur. The alveolar-arterial oxygen difference remains normal [3].

Imaging — Chest radiographs are generally not helpful, other than to exclude an intrathoracic cause of dyspnea. Diagnosis of PVCM by fluoroscopy and by color flow Doppler has been reported, but these techniques have not been standardized against direct visualization with flexible fiberoptic laryngoscopy [28,29].

Laryngoscopy — Visualization of the cords using a flexible fiberoptic laryngoscope confirms the diagnosis by revealing abnormal adduction of the true cords (just during inspiration, throughout the respiratory cycle, or rarely just during expiration); the glottic aperture may be obliterated except for a posterior diamond-shaped passage [3,7]. There may also be adduction or bunching of the false vocal cords [3,7,30]. Although these findings are normally seen only during an acute episode, they can often be reproduced on examination when the patient is asked to mimic what happens during an attack.

It is important that the procedure is performed by an experienced laryngoscopist who is aware of this disorder to avoid misdiagnosis of bilateral true vocal cord fixation or paralysis, laryngopharyngeal reflux, or laryngeal edema. Normal vocal cord motion may be restored by asking the patient to cough or breathe in a panting manner [30]. It has been noted that the stridor resolves completely when the patient is asleep or in some cases when the patient is unaware of being observed [1,31]. (See "Laryngopharyngeal reflux".)

DIFFERENTIAL DIAGNOSIS — In patients suspected of having PVCM, a thorough evaluation for asthma and causes of upper airway obstruction is essential. Certain clues may indicate that a patient is suffering from PVCM rather than asthma [32]. (See "Diagnosis of asthma in adolescents and adults".)

Subjectively more difficulty on inspiration than expiration

Minimal response to aggressive asthma treatment

A flattened inspiratory flow-volume loop

Normal expiratory spirometry, lung volumes, and arterial blood gas measurements

PVCM should also be differentiated from other causes of upper airway obstruction including:

Laryngeal angioedema due to anaphylaxis, angiotensin converting enzyme inhibitor therapy, and C1 inhibitor deficiency. (See "An overview of angioedema: Pathogenesis and causes".)

Paroxysmal laryngospasm usually occurs in an older patient population than does PVCM, often in the setting of a viral upper respiratory infection, or in a pediatric population in the setting of general anesthesia [33]. It is associated with aphony and a choking sensation; may awaken patients from sleep; or be initiated by coughing. Unlike PVCM, the time course of laryngospasm is so limited (eg, seconds to minutes) that it is rarely observed by the clinician. Laryngospasm may also be triggered by laryngopharyngeal reflux. (See "Laryngopharyngeal reflux".)

Structural lesions causing upper airway obstruction (eg, vocal cord polyps, glottic and tracheal tumors, tracheal stenosis) should also be excluded. (See "Diagnosis and management of central airway obstruction".)

Vocal cord paralysis as in bilateral abductor paralysis caused by recurrent laryngeal or vagus nerve injury or neuropathy [34].

5.5.18 Discuss the treatment of paradoxical vocal fold movement. What techniques may be helpful. AL

The mainstay of management includes aggressive treatment of comorbidity, such as laryngopharyngeal reflux (LPR), allergies, and asthma, in conjunction with appropriate voice therapy (see Chapter 63). Heliox and positive airway pressure, in conjunction with
anxiolytics and reassurance, may help to alleviate respiratory distress during an acute attack. Voice therapy includes focus on exhalation and abdominal breathing techniques with panting and sniffing to encourage vocal fold abduction. Laryngoscopic biofeedback has also been used with success.

Voice Therapy - Accent Method
The accent method, originally developed by Smith (12), focuses on rhythmic breathing as the primary control mechanism and pairs body movement with respiration and phonation to rebalance the system. The method trains patients to produce easy voicing, abdominal breathing movements (by alternating contraction and release), and open throat postures. These productions are achieved by using rhythmic vocalizations of consonant sounds (called accents), in combination with body movements (e.g., swaying arms or rocking back and forth), while stressing respiratory support for each accent. Proponents of this method report that patients acquire optimal respiratory support, coordination of exhalation and phonatory onset, and phonatory effort (13). Research investigating the efficacy of the accent method shows good outcomes (13,14).

Anatomic/Physiologic Explanation
The rhythmic pairing of timing with respiration and phonation may promote motor learning of new phonatory behaviors (15).

Application
The accent method may be used to treat benign lesions, MTD, vocal fatigue, paradoxical vocal fold motion (PVFM), and mild glottal incompetence.

TREATMENT — Various treatment strategies have been used for PVCM, although none have been studied in a controlled fashion. In general, therapies for asthma such as beta adrenergic agents and inhaled glucocorticoids are not beneficial. It is helpful to separate treatment approaches into acute management and long-term prevention.

Acute management — Acute management strategies that may be useful include:

- Reassurance and supportive care until the episode spontaneously resolves. Asking patients to pant can sometimes abort an episode; panting activates the posterior cricoarytenoid muscle causing abduction of the true vocal cords [35].

- Use of continuous positive airway pressure (CPAP) [7,36]. (See "Noninvasive positive pressure ventilation in acute respiratory failure in adults").

- Inhalation of a helium oxygen mixture (heliox) [3,37]. In a case series, four of five PVCM patients experienced an improvement in symptoms and in associated anxiety with heliox inhalation during acute episodes [37]. (See "Physiology and clinical use of heliox").

- Endotracheal intubation or tracheostomy is rarely needed, and is usually performed only when the diagnosis has not been confirmed [36].

Long-term prevention — Long-term prevention strategies usually employ a coordinated approach combining speech therapy and sometimes psychological counseling [23,38]. There are no published studies on the efficacy of either psychodynamic therapy or psychopharmacologic treatment for PVCM.

The first step is to convince the patient of the validity of the diagnosis, which may be difficult since the diagnosis is often delayed, and some patients have been treated chronically and aggressively for asthma. Many such patients strongly believe that their health depends upon continuing their longstanding asthma medications. When PVCM coexists with asthma, medications for asthma should be continued during treatment for PVCM.

The diagnosis of PVCM should be explained in a nonjudgmental fashion that strives to maintain patient dignity. A useful analogy is to describe how muscle spasms in the neck and shoulder can be related to stress; the muscle spasm isn't under conscious control; and nothing is structurally wrong with the neck. Similar to neck muscle spasm, muscle relaxation and psychological counseling to reduce stress are appropriate treatments for PVCM.
Success has been reported with speech/voice therapy that uses breathing, voice, and neck relaxation exercises to abort the onset of PVCM episodes [2,3,7,23,39-45]. A speech therapy intervention focusing on respiratory control in 20 adolescent female athletes yielded symptom control for 6 months and allowed continued participation in athletic endeavors [13]. In another study all five patients with documented PVCM improved with respiratory retraining [46]. Techniques including focusing attention away from the larynx and inspiration, using abdominal muscles for breathing, and relaxing the neck muscles were helpful in another series of five patients [3].

Exercise associated PVCM may respond to the above described psychological counseling and speech therapy with relaxation exercises. In addition, pretreatment with an anticholinergic medication prior to exercise may be beneficial. As an example, in an uncontrolled case series, exercise induced PVCM was prevented in six of seven patients who used ipratropium prior to exercise [47]. Successful use of inspiratory resistive training with a pressure-loaded respiratory muscle trainer has been described in case reports [48-50]. As an example, an 18-year old soccer player experienced resolution of PVCM symptoms after five weeks of a five day per week regimen (five sets of 12 breaths) of inspiratory muscle training [49]. The inspiratory resistance was set at 75 percent of maximum inspiratory capacity.

5.5.19  Describe dysphonia plica ventricularis symptoms, etiology, diagnosis and management.  AL


Abstract
OBJECTIVE/HYPOTHESIS:
Ventricular dysphonia, also known as dysphonia plica ventricularis, refers to the pathological interference of the false vocal folds during phonation. Despite its low incidence and prevalence, Vd is a well-known phenomenon in voice clinics. The present report reviews symptoms, etiology, diagnosis, and therapeutic options regarding this voice disorder.

STUDY DESIGN:
Literature review and case studies.

METHODS:
The literature pertaining to all clinical aspects of V(D) was reviewed to define diagnostic and therapeutic clinical decision making.

-Definition of dysphonia plica ventricularis: phonation using false vocal fold vibration rather than true vocal fold vibration, most commonly associated with severe muscular tension and occasionally may be an appropriate compensation for profound true vocal fold dysfunction.
-Incidence: 4-6%
-Characteristic voice: low pitch, hoarseness, voice breaks, diplphonia (double sounding voice pattern resulting from simultaneous action of true and false vocal cords)
-False cords have more mass -> slower vibration pattern -> low pitch, hoarse, harsh, rattling, rumbling, cracking voice
-Less sophisticated vibration pattern than true cords -> restricted vocal range, reduced loudness, little pitch variability, monotonous voice
-Other associated symptoms: voice fatigue, aphonia, effortful phonation, fatigability, unproductive throat clearing, pain, lump in throat
-Not associated with: dysphagia, odynophagia, stridor, dyspnea
-Etiology:
  -VD as a consequence of excessive muscular tension in the laryngeal area
  -VD as a substitute voice resulting from severe true vocal fold disease such as true vocal fold paralysis, tumor pushing the TVC apart, vertical hemilaryngectomy, congenital TVC anomaly
  -VD as a substitute for glottal incompetence
VD as a functional result of psychoemotional interference and physical and emotional tension

VD as an unexplained idiopathic phenomenon

Habitual, resulting from hyperkinetic dysphonia with continuous vocal abuse
Stress-induced, through hyperkinesis of the ventricular folds
compensatory as a reaction to laryngeal paralysis
a cerebral type signaling dysarthria (with a low, rough, intermittent, spastic, and squeezed sound)
cerebellar or midbrain (eg parkinsonism) type
vicarious as a desirable adjustment to defective true vocal folds

Supraglottic structures are recruited to compensate for underlying glottal insufficiency
previous VC surgery, surgical intubation, unilateral TVC paralysis, irritant exposure
other precipitating medical conditions: vocal abuse, spastic dysphonia, respiratory disease, neck or chest surgery

-10% had no medical correlate

Pathophysiology:
FVC are composed of soft and elastic tissue holding cells with scattered fibers of the thyroarytenoid muscle
innervation from anterior division of the RLN
possible contribution of the lateral cricoarytenoid muscle for the sphincteric contraction of the FVC’s

Diagnosis:
symptoms, perceptual evaluation of the voice, history, laryngeal examination
in hypertrophied FVF’s, TVF may not be visualized due to sphincteric contriction of the supraglottic region
Videostroboscopy: reveals deficient TVF oscillation and can record ventricular fold adduction and vibration
Videofluoroscopy
Direct laryngoscopy – disadvantage: can’t detect vocal fold anomaly
aerodynamic analysis: abnormally high subglottic pressure, high translaryngeal airflow (indicates glottis air leak)
maximum phonation time: short
acoustic analysis: severe acoustic perturbation
quantification of the restricted vocal range
EMG: no data

Treatment Scheme: essentially depends on whether the TVF’s are or are not capable of phonation
Behavioral Therapy: retraining TVF vibration. Yawn-sigh approach, glottal fry, tongue protrusion, inhalation phonation technique (phonating while inhaling).

Digital manipulation of the thyroid cartilage downward during inhalation and exhalation phonation

Labial constriction technique: displacement of the site of constriction from the larynx to the lips associated with a high expiratory flow

RESULTS:
Ventricular dysphonia is characterized by a typical rough, low-pitched voice quality resulting from false vocal fold vibration. Ventricular dysphonia may be compensatory when true vocal folds are affected (resection, paralysis). Noncompensatory types may be of habitual, psychoemotional, or idiopathic origin. Because perceptual symptoms may vary considerably, diagnosis should rely on a meticulous voice assessment, including laryngeal videostroboscopic, perceptual, aerodynamic, and acoustic evaluation. Various therapeutic approaches for the noncompensatory type of ventricular dysphonia may be considered: voice therapy, psychotherapy, anesthetic or botulinum toxin injections, or surgery.

CONCLUSION:
The study presents the state of the art with respect to ventricular dysphonia and may be helpful in diagnosis and therapeutic decision-making.