5 Evaluation of Neurogenic Voice Disorders

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Introduction

The larynx is a complex organ contributing to physiologic processes of phonation, respiration, and deglutition. Neurogenic disorders can disrupt some or all of these functions. Evaluating focal and systemic voice disturbance is best accomplished through a holistic blend of perceptual, aerodynamic, acoustic, and instrumental assessments. This chapter offers a physiologic approach to expose common pathway disruptions. Laryngologists and voice-specialized speech-language pathologists have expertise evaluating disease nuances, providing accurate diagnoses and managing symptoms. Collaboration among providers is paramount as individuals with laryngeal symptoms often consult with numerous medical specialties in pursuit of answers.

Neurologic input to the larynx is critical for vegetative functions and communicative processes. Vocal intent begins in the central nervous system. It then courses through the peripheral nervous system to lower motor neurons and engages the larynx. Healthful voice production requires intact neurologic input to the lungs, larynx, pharyngeal, and oral cavities. Vocal motor control remains a source of ongoing investigation [1–3]. Evidence supports activation of a feedback loop among the sensorimotor cortex, auditory cortex, basal ganglia, cerebellum, and periaqueductal gray matter [4, 5]. The tenth cranial nerve, the vagus nerve, is essential for voice production. Pertinent branches for vocal function include pharyngeal, superior laryngeal, and recurrent laryngeal nerves. The superior laryngeal nerve innervates the cricothyroid muscle for pitch control and supplies sensation to the laryngeal mucosa. The recurrent laryngeal nerve controls all other intrinsic laryngeal muscles [6]. As air expels from the lungs, the vocal folds close and oscillate. The vibration then filters through pharyngeal, oral, and nasal cavities to trademark a unique sound. Details of voice physiology are covered in more depth in other chapters.

Noninstrumental Assessment

Behavioral evaluation of neurogenic dysphonia will reveal task-specific disease hallmarks. Differential diagnosis hinges on physiologic trait recognition and vocal patterns. Perceptual features of functional disorders may masquerade as a neurological condition. Gradations of compensatory muscle tension also commonly overlap

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Table 5.1 Self-rating scales

Voice Handicap Index [7]
Singing Voice Handicap Index [8]
Voice-Related Quality of Life Index [9, 10]
Spasmodic Dysphonia Attributes Inventory [11]
Unified Spasmodic Dysphonia Rating Scale [12]
Voice Symptom Scale [13]
Voice Activity and Participation Profile [14]
Dyspnea Index [15]
Eating assessment Tool-10 [16]

Table 5.2 Interview

When did symptoms begin? Were the changes sudden or gradual?
Can you correlate these to any medical, medication, or personal events?
Do you have changes in swallowing, articulation or breathing?
Are the symptoms stable, worsening or getting better?
What are your vocal demands?
Are your symptoms always present?
Can you discern triggers or anything to suppress your voice issue?
Does your voice improve with alcohol?
Do you sing? Is there change to your singing voice?
Does your voice ever sound normal?
Do you find it takes excess effort to use your voice or speak?
Do you have prior history of voice issues?
Does anyone in your family have a voice problem or history of neurological disease including tremor?
Have you noticed tremor in your hands or other body parts?

and confound neurogenic presentations. Varying phonatory contexts is often revealing: voiced vs. voiceless sounds, loud vs. soft phonation, pitch variation vs. monotone, sustained phonation, singing, emotionally mediated speech, and vegetative sounds. This diversity illuminates compensatory, functional, and/or neurologic pathophysiology. In some cases, repeating tasks for potential fatigability may be a lone subtle sign of neurogenic dysfunction. An individual's perception of the disorder complements the objective clinical interaction. A variety of self-rating scales, a few of which are referenced in Table 5.1 [7–16], uncover the precise nature of voice dysfunction and reflect patient perception. The Dyspnea Index [15] and Eating Assessment Tool-10 [16] are also useful because voice disorders, particularly of neurologic origin, have higher incidence of confounding respiratory and swallowing dysfunction [17].

During the initial patient interview, questions targeting timelines and comorbidities aid diagnostic precision (Table 5.2). Symptom onset and time course of disease provide valuable insight. For example, vocal fold paralysis and paresis are more likely to have a sudden onset and then stabilize or resolve [18], whereas phonatory weakness or asthenia presents gradually in the setting of degenerative neurologic disease. Laryngeal and respiratory dystonia begin suddenly without overt provocation or insidiously over time. Dystonia has been associated with preceding viral illness, history of extensive voice use, symptom onset in middle ages, and female dominance [19, 20]. Neurologic conditions sometimes manifest with sensory abnormalities as well including laryngeal hyperresponsiveness, cough, paradoxical vocal fold movement, and functional dysphagia. The most concerning circumstances of voice change are those that are progressive and encompass the entire speech and swallowing mechanism. This could be early presentation of serious neurodegenerative diseases such as amyotrophic lateral sclerosis (ALS), multiple system atrophy (MSA), Parkinson disease (PD), and multiple sclerosis (MS). In these diseases, laryngeal, pharyngeal, and oral anomalies may present initially before other systemic ailments are apparent. As voice and swallowing clinicians are often the initial medical encounter, they must be vigilant to expediate consultation with a neurologist.

Familiarity with common perceptual features of neurogenic voice disorders will unearth clues of etiology. Breathy vocal quality is typically associated with hypofunctional voice disorders such as unilateral vocal fold paralysis/paresis, PD, and vocal fold atrophy. Incomplete glottic closure underpins this profile. Strained quality occurs with adductor spasmodic dysphonia, laryngeal spasticity, and bilateral vocal fold paralysis [21]. A non-neurological voice disorder, muscle tension dysphonia, can manifest with similar perceptual features and should be differentiated given its significant symptom overlap [22]. Strained vocal quality, also referred to as spastic dysphonia, may

be a sign of broader dysarthria, such as ALS [23] or MSA [24]. The other common trait surfacing among neurogenic profiles is tremor. Voice tremor evolves from unintended rhythmic fluctuations of pitch and loudness between 4 and 5 Hz [25]. While tremor can be isolated to the larynx, often other anatomic subsites are involved [26]. When tremor is distinct from other neurological processes, such as Parkinsonian tremor, it is called essential tremor [26]. Severe tremor can provoke complete phonatory arrests [27]. This should be differentiated from laryngeal dystonia as it can co-occur with tremor. In sustained phonation, essential voice tremor will persist [28, 29], while laryngeal dystonia more typically occurs only with sound initiation.

Instrumental Assessment

Instrumental assessment of voice production involves acoustic analyses, aerodynamic function, and endoscopic laryngeal imaging. Guidelines were developed by the American Speech-Language-Hearing Association in 2018 in attempt to standardize acquisition, synthesis, and reporting of these measures [30]. The benefit of regulated intake processes is multifold. When evaluation procedures become consistent across healthcare institutions, patients are more likely to be assigned accurate diagnoses. Furthermore, consistency in symptom profiles allows for effective communication in research and treatment design.

Acoustic and Aerodynamic Analysis

Evaluation of sound and airflow patterns are essential for determining the appropriate diagnosis and treatment recommendations. Neurogenic pathology can disrupt single and multilevel physiology. Acoustic and aerodynamic measurements help distinguish normal from pathological vocal physiology, inform treatment plans, and objectify therapeutic outcomes. Assessment of neurogenic voice disorders is based on pattern recognition of symptoms. These objective measurements compliment perceptual assessment for a comprehensive view of vocal behavior.

Vocal frequency and intensity are basic acoustic measurements that can be obtained with low technology recording equipment and sound level meters. They roughly correspond to a person's vocal pitch and loudness. Quantifying vocal quality involves more sophisticated equipment to detect degrees of noise in the acoustic signal. Normal voices vibrate at regular periodic intervals with acoustic energy organized around the fundamental frequency (F_0). Maintenance of stable F_0 requires fine motor control of the laryngeal muscles. Researchers are developing an acoustic measurement to capture this control called relative fundamental frequency – RFF [31, 32]; however, it is not currently used in clinical practice. In a disordered voice, noise energy is disorganized and distributes across multiple frequencies in an aperiodic manner. This aperiodicity may occur across an entire speech task or occur in a specific context (i.e., on voiced sounds like "w," but not unvoiced sound like "s" in spasmodic dysphonia). Consider the context of the speech sample gathered during assessment and how it reflects on speech patterns in the patient's daily life. Samples that require patients to speak in phrases or sentences are valuable to estimate dysphonia in daily speech; however, these connected speech samples cannot be used to extract acoustic values reliant on stable F_0, such as variations of pitch (jitter), loudness (shimmer), and noise to harmonic ratio [33], which require recordings of a sustained vowel.

One recently developed approach to measuring vocal quality uses the cepstrum, which can be extracted from connected speech samples [34, 35]. This method of analysis is not time-based and does not rely on a stable F_0, rather, it is completed on the frequency structure (harmonics) of the acoustic sample. A normal voice has a well-organized harmonic structure, while a disordered voice demonstrates disorganization. The peak, cepstral peak prominence (CPP), describes this structure and is of high value in normal (organized) voices and low in disordered (unorganized) voices. It is especially sensitive to the perception of breathiness. Specific descriptions of all acoustic measurements can be found in Table 5.3 [34–49].

Table 5.3 Common acoustic and aerodynamic measurements used to quantify vocal disorders are listed below. Please note that the normative values listed are only a sample of available references and not exhaustive

	Measurement	Stimuli	Definition/construct measured			
Acoustic	Fundamental frequency (F0)	Connected speech or sustained vowel	F_0 captures the lowest frequency at which the vocal folds are vibrating. This roughly corresponds to the pitch we hear. F_0 can be sampled from sustained phonation, although is most relevant during connected speech. *Normative values*: Torre and Barlow 2009 [36], 60–89 years of age, gathered from a connected speech sentence context			
					Male	**Female**
			Fundamental frequency (Hz)		122–142	164–180
	Intensity	Sustained vowel or connected speech	Measures overall energy in the acoustic sample and is helpful to sample vocal loudness and prosodic variation in speech. Measured in dB SPL or root mean square (RMS). Normative values are highly dependent on speech context.			
	Cepstral peak prominence (CPP)	Connected speech or sustained vowel	Measures general dysphonia severity, gathered from the voice cepstrum, and is particularly sensitive to changes in breathiness. *Normative values*: Heman-Ackah et al. [34], gathered from "Marvin Williams" passage; Watts and Awan 2011 [37], gathered from second sentence of "Rainbow Passage"; Iranian norms: Hasanvand et al. [38]			
					Male and female grouped together	
			CPP-smoothed [34] (dB), Marvin Williams passage		4.77 ± 0.97	
			CPP(dB) [37] rainbow passage		5.42 ± 1.38	
			CPP(dB) [37], sustained /a/		11.08 ± 1.91	
	Cepstral-spectral index of dysphonia (CSID)	Connected speech or sustained vowel	Measures overall vocal severity. It is an algorithm consisting of multiple acoustic measures of dysphonia (i.e., CPP and L/H ratio) and is intended to objectively quantify dysphonia similar to clinician perception *Normative values*: Awan et al. [35], gathered from the second and third sentences of the "Rainbow Passage"			
				Male	**Female**	**Male and Female Grouped**
			CSID	28.63 ± 23.82	28.23 ± 17.74	
	Jitter	Sustained vowel only	Captures cycle to cycle changes in fundamental frequency. Requires that voices be rated as type I (nearly completely periodic) or type II (mostly periodic with subharmonics) [39] and carries reliability concerns [40] *Normative values*: Spazzapan et al. 2018 [41], ages 50–60, gathered from sustained "ah"			
				Male	**Female**	
			Jitter	0.92 ± 0.54	0.79 ± 0.61	
	Shimmer	Sustained vowel only	Captures cycle to cycle changes in intensity. Requires that voices be rated as type I or type II [39] and carries reliability concerns [40] *Normative values*: Spazzapan et al. 2018 [41], ages 50–60, gathered from sustained "ah"			
				Male	**Female**	
			Shimmer	5.29 ± 2.4	4.04 ± 1.64	
	Noise to harmonic ratio (NHR)	Sustained vowel only	This ratio captures the energy in periodic vs. aperiodic regions of the signal and relates to overall severity of the voice disorder [42]. It is limited by reliability issues [43]. *Normative values*: Spazzapan et al. 2018 [41], ages 50–60, gathered from sustained "ah"			
				Male	**Female**	
			NHR	0.16 ± 0.06	0.14 ± 0.02	
	MDVP tremor profile (FTRI, Fftr)	Sustained vowel	This group of measurements is available in the KayPENTAX multidimensional voice profile (MDVP) software package. It is intended to capture slow modulations (i.e., tremor) in frequency and amplitude of the signal. *Normative values*: Maccallum et al. [44]			
				Male and female grouped together		
			FTRI (%)	0.157 ± 0.25		
			Fftr (Hz)	1.154 ± 1.46		

Aerodynamic	Maximum phonation time	Sustained vowel	Maximum phonation time (MPT) is an indirect measurement of respiratory function. It is thought to describe how the body uses airflow or "airflow consumption." It is taken by having the patient inhale as deeply as possible and sustain a steady vowel for as long as possible *Normative values*: Joshi 2019 [45] gathered from sustained "a"'; Zraick et al. 2012 [46] gathered from healthy older adults aged 60–89 years		
				Male	**Female**
			Max phonation time (seconds)	24.16 ± 6.14 [45] 19.94 ± 6.79 [46]	18.92 ± 4.97 [45] 20.02 ± 6.58 [46]
	S/Z ratio	Sustained speech sounds	This ratio compares the maximum amount of time the patient is able to sustain an /s/ divided by the time to sustain a /z/. it compares ventilatory to phonatory function *Normative values*: Joshi 2019 [45]; Gelfer and Pazera 2006 [47]		
				Male	**Female**
			S/Z ratio	1.07 ± 0.37 [45] 1.01 ± 0.2 [47]	1.09 ± 0.25 [45] 1.06 ± 0.27 [47]
	Rainbow passage: Breaths and time	Connected speech, reading	The rainbow passage is a text that samples voice production in varying phonetic contexts. The number of breaths and time to read the passage relates to aerodynamic performance in patients with glottic incompetence. *Normative values*: Lewandowski et al. [48]		
				Male	**Female**
			Breaths	5.08 ± 1.72	4.75 +/−1.76
			Time	23.42 seconds ± 3.23	22.63 seconds ± 0.17
	Psub (subglottal pressure) and laryngeal resistance	Repetitive productions of /pa/	Subglottal air pressure measures the pressure immediately below the glottis during phonation (indirectly for clinical purposes), which is the energy needed for speech production. Psub requires precise neuromuscular control of the vocal folds and is the basic physiology control mechanism for pitch modulation and phonation maintenance. Laryngeal resistance is the accompanying description of the subglottal pressure divided by the flow rate, and it measures respiratory driving force. *Normative references*: Joshi et al. 2019 [45]; Zraick et al. 2012 [46] (ages 60–89 years); Matheron et al. 2017 [49]		
				Male	**Female**
			Psub (cm H20)	6.9 ± 2.53 [45]	5.84 ± 1.95 [46]
				8.5 ± 0.18 [49][a]	
			Laryngeal resistance (cm H20/L/s)	55.45 ± 60.64 [45] 137.31 ± 221.5 [46]	50.43 ± 28.83 [45] 79.05 ± 52.05 [46]
	Phonatory airflow (glottal flow)	Sustained vowel or connected speech	This is the average volume of air expended per second during a speech sound *Normative reference values*: Lewandowski et al. 2018 [48]; Joshi 2019 [45]		
				Male	**Female**
			Phonatory airflow (rainbow passage) [48]	0.16 L/sec ± 0.05	0.14 L/sec ± 0.001
			Glottal flow [45] (sustained vowel)	0.14 L/ sec ± 0.10	0.17 L/ sec ± 0.06

[a]Values include both male and female participants, gathered from phrase "buy pop or pop a papa," mean age 68.9 years

Aerodynamic assessment complements acoustic analyses, as it quantifies the physiologic force underpinning healthy vocal fold vibration. Specific tasks are designed to evaluate multiple components of airflow, such as speech level breathing patterns, average airflow used during voicing, or the estimated glottic air pressure generated during vocal fold vibration. Aerodynamic assessment can be accomplished with limited equipment using maximum phonation time, S/Z ratio, and a spirometer [50]. Alternatively, the Phonatory Aerodynamic System TM (KayPENTAX, Lincoln Park, NJ) sophisticates this process by calculating airflow and pressures during functional speech. Aerodynamic patterns may arise and correspond to a specific neurological dysfunction. Examples include patients with PD disease presenting with low transglottal airflow [51], whereas patients with vocal fold paralysis can exhibit high transglottal airflow [52].

While neurologic disruptions vary, disease-specific commonalities are likely to surface across acoustic and aerodynamic measures. A variety of identifying pathways are provided in Figs. 5.1 and 5.2 [48, 52–54], and see Table 5.3. Examples include reduced pitch range, monotone, and low-volume output in PD. Isolated vocal intensity disruption may be due to discrete glottic insufficiency from a vocal fold paralysis. Strained quality combined with elevated subglottal air pressures may be indicative of dystonia. If disease features are nebulous, pair measures with perceptual ratings, self-ratings, and the patient interview. Diagnostic voice therapy is often a favorable adjunct to complement acoustic and aerodynamic measures across linguistic and behavioral contexts.

Endoscopy and Videostroboscopy

Laryngeal visualization evaluates for co-occurring anatomic irregularities, provides opportunity to scrutinize movement patterns, and permits observation of vibratory characteristics. This process leads to identification of periphery nerve insults encompassing the superior laryngeal nerve, recurrent laryngeal nerve, or both. When the abnormalities are multilevel, consider central nervous system involvement. The examination is performed with an endoscope inserted into the oral cavity (rigid endoscopy) or nasal cavity (flexible endoscopy). Neurologic disease unfolds most clearly during flexible endoscopy whereas rigid endoscopy highlights discrete mucosal abnormalities. Transnasal endoscopy is well tolerated [55], depicts velopharyngeal integrity, and provides gestalt function during speech and respiration. A small subset of larynges may demonstrate elevated hypersensitivity leading to endoscopic intolerance. Sensory neuropathy may underpin this and have concurrent evidence of motor dysfunction depending on the involved nerve [56]. During endoscopy a still light is used to examine broad movement features and positioning while stroboscopic light depicts vibratory characteristics. Otolaryngologists and voice-specialized speech-language pathologists routinely perform these procedures.

Specific movement attributes are evaluated during endoscopy, including vocal fold opening, closing, and lengthening. The anticipated laryngeal movement under normal circumstances involves complete and symmetric opening and closing of both vocal folds during inhalation and phonation. Disruption of this process implicates recurrent laryngeal nerve or vagal dysfunction, especially if the movement deficit is unilateral. This is best elicited when prompting patients to alternate “sniffing” and “eee” postures in sequence. Irregularities range from obvious immobility to subtle sluggish movements of one or both vocal folds [57]. There may also be deviation of the glottic axis [58], uneven vocal fold height [59], insufficient arytenoid rotation [60], and fatigability [61]. Observe vocal fold lengthening at rest and during pitch glissandi spanning the entire stimulable range. Inability to control pitch and loudness suggests superior laryngeal nerve injury. Pitch range extremes are useful to evoke asymmetric postures compensating for abnormal neurologic input. Capturing continuous speech and sustained modal pitch is equally telling to correlate perceptual, aerodynamic, and acoustic physiologic disruptions.

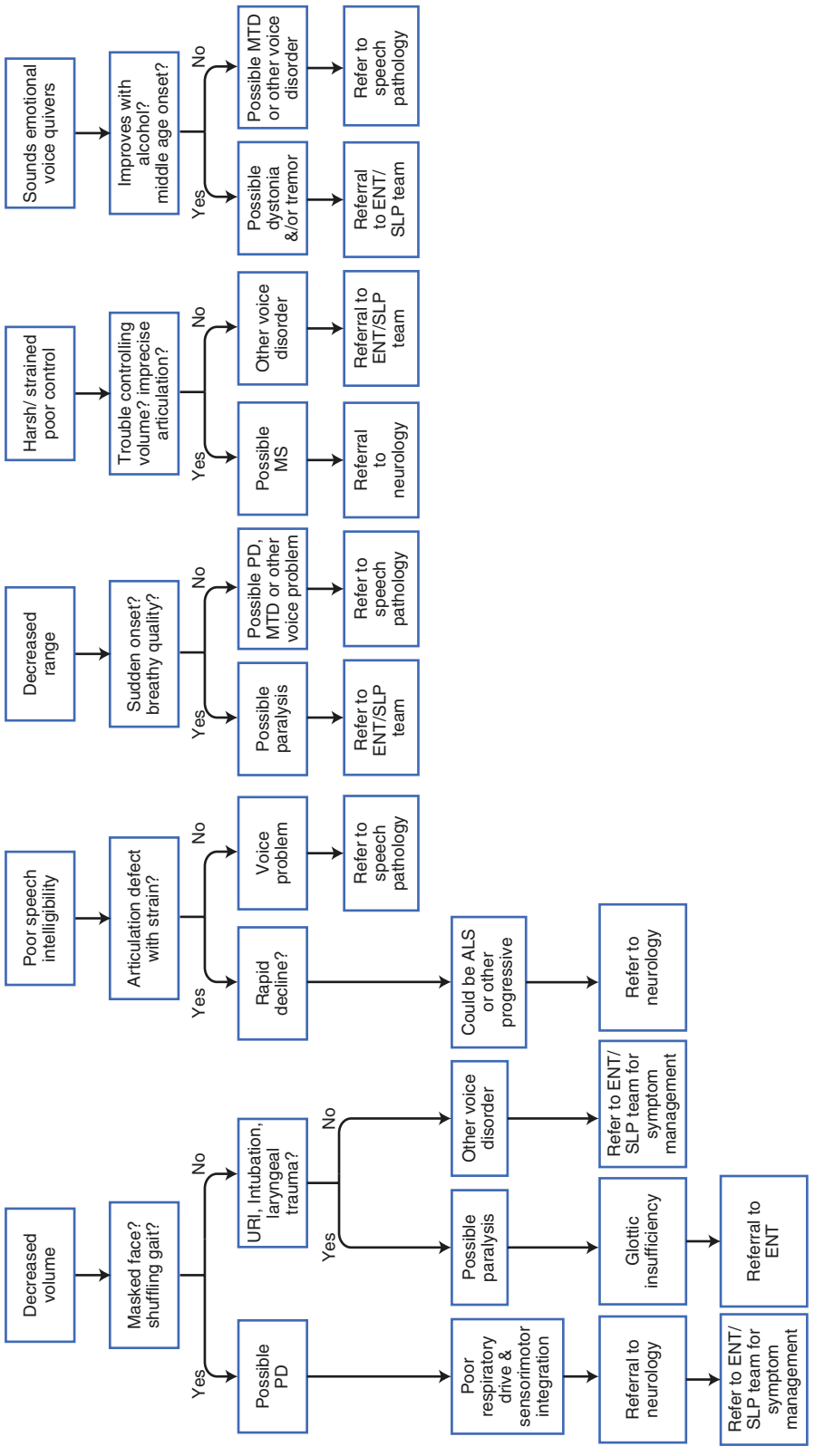


Fig. 5.1 Algorithm used by authors to obtain neurologic profile

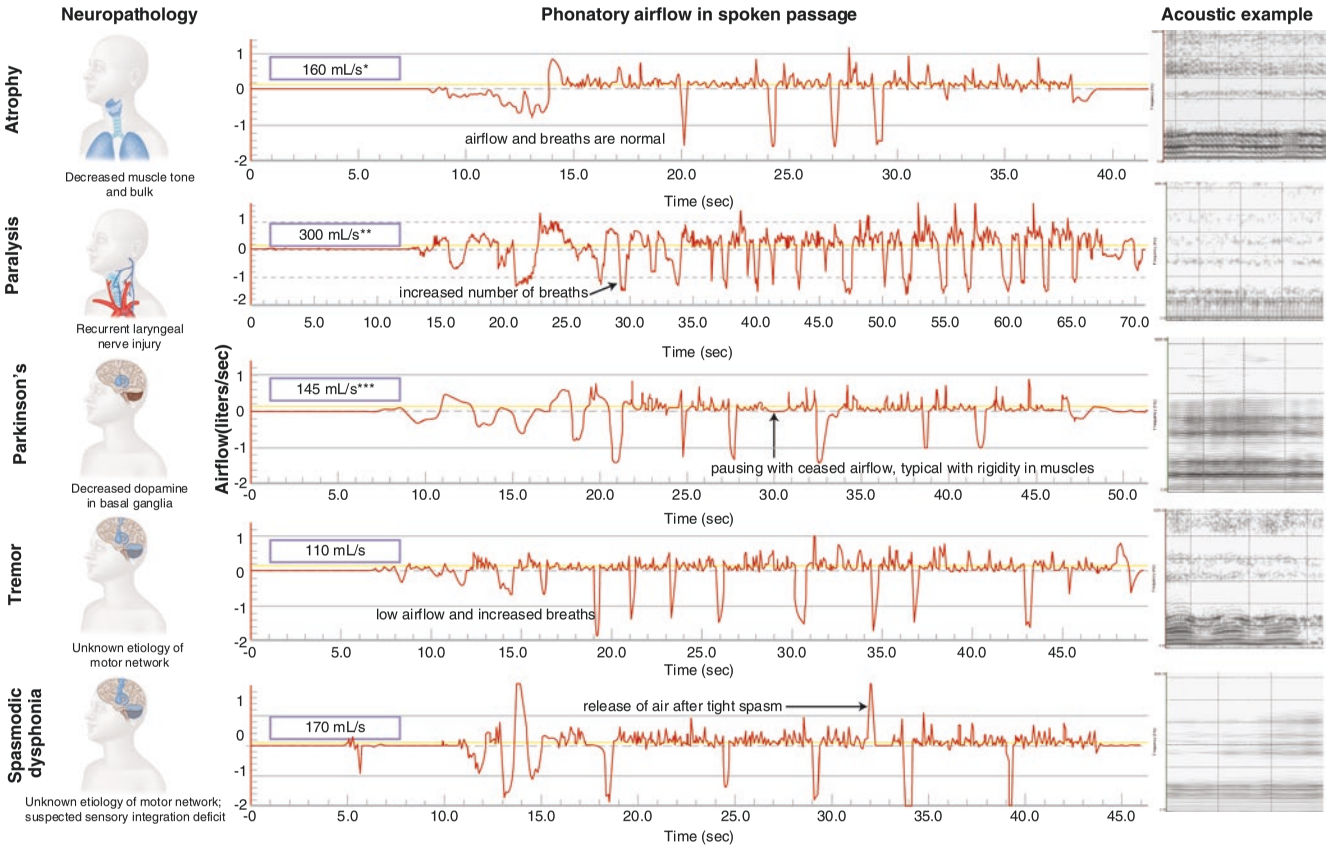


Fig. 5.2 Example acoustic and aerodynamic measurements from select patients are shown in the above diagram for the corresponding neurological disorder. These samples were chosen to depict salient features of each disorder. Red tracings depict phonatory airflow while reading the Rainbow Passage; see Gartner-Schmidt et al. ([52]) for protocol details. Average phonatory airflow is given in the box on the left; asterisk indicates normative value. Measurements for tremor and spasmodic dysphonia do not reflect normative information. Example spectrograms on the right side reflect example acoustic patterns during sustained vowel production and correspond to the same patients with airflow tracings. Varying degrees of harmonic organization can be noted in the horizontal stripes, which represent different frequencies (Hz) (i.e., paralysis – horizontal lines are not clear/disorganized). The tremor spectrogram depicts rhythmic variation in intensity (*dark* vs. *light shading*) and frequency. References: (∗Goodwin et al. [53]; ∗∗Lewandowski et al. [48]; ∗∗∗Dastolfo-Hromack et al. [54]). Measures were taken using the Phonatory Aerodynamic System (PAS) and acoustic spectrograms were generated using the Analysis of Dysphonia in Voice (ADSV) software package; both tools are created by KayPENTAX®

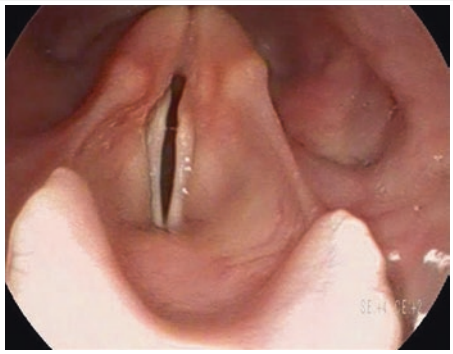
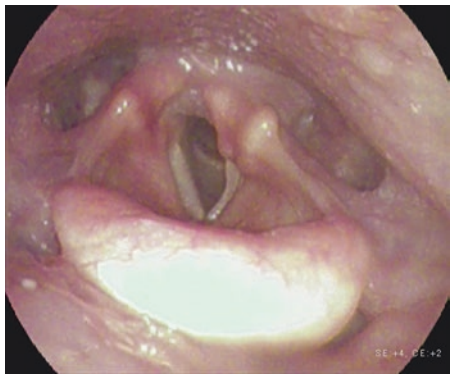
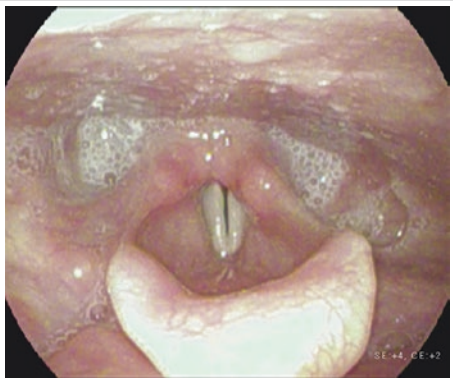
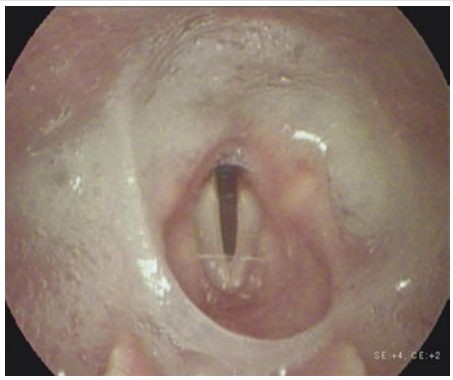
Unveiling tremor is performed in a systematic manner. The *Vocal Tremor Scoring System* was designed for endoscopic evaluation of vocal tremor. It aims to quantify affected structures including the palate, tongue base, pharyngeal walls, larynx, supraglottis, and true vocal folds [62]. This metric eases therapeutic planning and has demonstrated prediction of treatment outcomes. If tremor surfaces only with voicing, it is called a dystonic tremor. When observed within unvoiced contests, it may represent essential voice tremor. Close collaboration with neurology colleagues will facilitate the most accurate profile of tremor disorders, each of which reveals a unique pathophysiology. Researchers found the unvoiced phoneme /s/, continuous whistling, and falsetto a means of distinguishing between essential and dystonic tremor syndromes [63]. Tremor may be isolated, coexisting with dystonia or part of broad neurological disease such as PD.

Laryngeal dystonia has distinct features that are identifiable during laryngoscopy. There are a number of passages laden with voiced and voiceless phonemes that can highlight or trigger dystonic features. Examples of tasks to elicit adductor dystonia include passages such as "I'll roll you away" and "Good dogs beg in bed" [64]. Abductor spasms, the less common laryngeal dystonia variant, will become apparent with abnormally long pauses during voiceless passages such as "Hal hurt his heavy heart" and "Pick up a tasty cake" [64]. In either condition, the vocal folds may demonstrate intermittent or persistent freezing in adduction or abduction. More subtle variations of laryngeal dystonia may not be reflected during laryngeal visualization. These cases will typically surface during thorough perceptual, aerodynamic, and acoustic testing. If the tension is not contextual, consider primary muscle tension dysphonia.

Transitioning from still to stroboscopic light permits observation of vibratory function. Videostroboscopy illuminates progressive positions of the vocal cords throughout the vibratory cycle. The interaction of video and these discrete positions reveals a composite image that mimics real-time vibration. While there are limitations to gaps in vibratory function, examination of each cycle with high-speed imaging typically does not change the diagnostic impression [65]. Advantages of high-speed imaging include greater refinement of the mucosal wave, vibratory amplitude, and glottal closure patterns. This technology is cost prohibitive and not readily integrated into most voice clinics. The cyclic waves course along medial to lateral planes and are evaluated based on pliability, wave propagation, and symmetry [66]. Neurogenic anomalies may impose reduced oscillation due to poor respiratory drive, vocal fold atrophy, immobility that limits free edge contact, and vibratory asymmetries. There is also potential of discovering comorbid mucosal disturbances that further complicate the underlying disorder limiting glottic competence.

Disease-specific trends can be observed throughout the endoscopy, including hypomobility, paradoxical vocal fold movement, tremor, spasm, and dysphagia when secretions are poorly managed. These are outlined in Table 5.4. Consider that many of the individuals undergoing evaluation are in later decades of life. A wide range of dysphonia incidence occurs in the elderly, between 12% and 47% [67], and laryngoscopic incidental findings are common [68]. This can encompass vocal fold atrophy, mucosal imperfections, and inflammation. These discoveries may warrant treatment because they will likely exacerbate a neurogenic communication handicap and can obscure diagnostic symptomatology. Strategies to dissect neurological signs from potential physiological aging effects include a detailed temporal depiction of symptom presentation and associating disease-specific trends. Finally, endoscopy may be abnormal without unifying disease traits. In these circumstances initiating therapy services with a speech-language pathologist is advised. Repeating the exam at a later date can allow a disease to unfold over time and optimize complementary behavioral observations.

Table 5.4 Laryngoscopic findings commonly found in neurolaryngologic voice disorders

Neurological sign	Description of finding	Common disorders
	Incomplete glottal closure. May not close in spite of effort. Prominent vocal processes. Bowed configuration. If paired with asymmetric movement or hypokinesia, could be Parkinson disease	Atrophy, Parkinson disease
	Immobile or partially immobile vocal fold. Challenge with sniff "eee" combination and pitch manipulation.	Paresis, paralysis
	Complete or partial fixation of both vocal cords. Voice may sound normal or strained. Listen for stridor and probe respiratory complaints.	Bilateral paralysis, post radiation, multiple systems atrophy
	Hypopharyngeal pooling	Sensory deficit, dysphagia

Conclusion

The larynx is a complex organ intimately tied to human expression. Neuropathology involving this structure can be identified early by a varied roster of medical care providers, depending on symptom constellation and evaluation acumen. One such example includes a pulmonary consult for severe dyspnea in the setting of bilateral vocal fold paralysis. Another involves patients with PD referred by general practitioners to speech pathologists for seemingly idiopathic voice weakening and eventually meeting with neurology for systemic diagnosis. All practitioners have a unique opportunity to initiate appropriate care pathways based on perceptual voice impression. Skilled physiologic evaluation, with subsequent coordinated treatment among appropriate providers, greatly improves care and patient outcomes. Whether the disease lies centrally or peripherally, opportunity exists to assess integrity of this multilevel system involving respiration, phonation, articulation and deglutition.

A comprehensive neurological voice evaluation ought to involve four components: thorough history, self-rating scales, laryngeal visualization, and behavioral evaluation with acoustic, aerodynamic, and perceptual measures. Disease onset characteristics, demographics, and epidemiologic factors provide vital clues for diagnosis. Varied performance in speech and nonspeech tasks and responsiveness to technique modifications are important to disambiguate neurogenic disease from psychopathology. It also improves sensitivity to categorize the type of neurological voice disturbances. The included diagrams and questionnaires in this chapter are intended to mold and sequence interactions with patients whose case is suspect for a neurogenic laryngeal disorder. Isolating the condition as focal (paralysis, paresis, dystonia, tremor) or systemic (PD, ALS, MS, MSA) is a critical piece of this intake. Referrals to speech-language pathology, laryngology, and neurology can then refine and synthesize the symptoms with endoscopy, aerodynamic, acoustic, and perceptual analyses. Ultimately, patients will benefit when all clinicians are well-educated on laryngeal symptoms and the neurological pathology from which they originate.

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