

GUIDELINE

Clinical practice guideline: Hoarseness (Dysphonia)

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ABSTRACT

OBJECTIVE: This guideline provides evidence-based recommendations on managing hoarseness (dysphonia), defined as a disorder characterized by altered vocal quality, pitch, loudness, or vocal effort that impairs communication or reduces voice-related quality of life (QOL). Hoarseness affects nearly one-third of the population at some point in their lives. This guideline applies to all age groups evaluated in a setting where hoarseness would be identified or managed. It is intended for all clinicians who are likely to diagnose and manage patients with hoarseness.

PURPOSE: The primary purpose of this guideline is to improve diagnostic accuracy for hoarseness (dysphonia), reduce inappropriate antibiotic use, reduce inappropriate steroid use, reduce inappropriate use of anti-reflux medications, reduce inappropriate use of radiographic imaging, and promote appropriate use of laryngoscopy, voice therapy, and surgery. In creating this guideline the American Academy of Otolaryngology—Head and Neck Surgery Foundation selected a panel representing the fields of neurology, speech-language pathology, professional voice teaching, family medicine, pulmonology, geriatric medicine, nursing, internal medicine, otolaryngology—head and neck surgery, pediatrics, and consumers.

RESULTS: The panel made *strong recommendations* that 1) the clinician should not routinely prescribe antibiotics to treat hoarseness and 2) the clinician should advocate voice therapy for patients diagnosed with hoarseness that reduces voice-related QOL. The

panel made *recommendations* that 1) the clinician should diagnose hoarseness (dysphonia) in a patient with altered voice quality, pitch, loudness, or vocal effort that impairs communication or reduces voice-related QOL; 2) the clinician should assess the patient with hoarseness by history and/or physical examination for factors that modify management, such as one or more of the following: recent surgical procedures involving the neck or affecting the recurrent laryngeal nerve, recent endotracheal intubation, radiation treatment to the neck, a history of tobacco abuse, and occupation as a singer or vocal performer; 3) the clinician should visualize the patient's larynx, or refer the patient to a clinician who can visualize the larynx, when hoarseness fails to resolve by a maximum of three months after onset, or irrespective of duration if a serious underlying cause is suspected; 4) the clinician should not obtain computed tomography or magnetic resonance imaging of the patient with a primary complaint of hoarseness prior to visualizing the larynx; 5) the clinician should not prescribe anti-reflux medications for patients with hoarseness without signs or symptoms of gastroesophageal reflux disease; 6) the clinician should not routinely prescribe oral corticosteroids to treat hoarseness; 7) the clinician should visualize the larynx before prescribing voice therapy and document/communicate the results to the speech-language pathologist; and 8) the clinician should prescribe, or refer the patient to a clinician who can prescribe, botulinum toxin injections for the treatment of hoarseness caused by adductor spasmodic dysphonia. The panel offered as *options* that 1) the clinician may perform laryngoscopy at any time in a patient with hoarseness, or may refer the patient to a clinician who can visualize the larynx; 2) the clinician may prescribe anti-reflux medi-

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cation for patients with hoarseness and signs of chronic laryngitis; and 3) the clinician may educate/counsel patients with hoarseness about control/preventive measures.

DISCLAIMER: This clinical practice guideline is not intended as a sole source of guidance in managing hoarseness (dysphonia). Rather, it is designed to assist clinicians by providing an evidence-based framework for decision-making strategies. The guideline is not intended to replace clinical judgment or establish a protocol for all individuals with this condition, and may not provide the only appropriate approach to diagnosing and managing this problem.

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Nearly one-third of the population has impaired voice production at some point in their lives.^{1,2} Hoarseness is more prevalent in certain groups, such as teachers and older adults, but all age groups and both genders can be affected.¹⁻⁶ In addition to the impact on health and quality of life (QOL),^{7,8} hoarseness leads to frequent health care visits and several billion dollars in lost productivity annually from work absenteeism.⁹ Hoarseness is often caused by benign or self-limited conditions, but may also be the presenting symptom of a more serious or progressive condition requiring prompt diagnosis and management.

The terms hoarseness and dysphonia are often used interchangeably, although hoarseness is a *symptom* of altered voice quality and dysphonia is a *diagnosis*. Dysphonia may be broadly defined as an alteration in the production of voice that impairs social and professional communication. In contrast, hoarseness is a coarse or rough quality to the voice. Although the two terms are not synonymous, the guideline working group decided to use the term *hoarseness* for this guideline because it is more recognized and understood by patients, most clinicians, and the lay press.

The target patient for this guideline is anyone presenting with hoarseness (dysphonia).

- *Hoarseness (dysphonia)* is defined as a disorder characterized by altered vocal quality, pitch, loudness, or vocal effort that impairs communication or reduces voice-related QOL.
- *Impaired communication* is defined as a decreased or limited ability to interact vocally with others.
- *Reduced voice-related QOL* is defined as a self-perceived decrement in physical, emotional, social, or economic status as a result of voice-related dysfunction.

This working definition, developed by the guideline panel, assumes that hoarseness affects people differently. Some individuals may have altered voice quality, vocal effort, pitch, or loudness; others may experience problems with communication and diminished voice-related QOL.

The guideline is intended for all clinicians who are likely to diagnose and manage patients with hoarseness and applies to any setting in which hoarseness would be identified, monitored, treated, or managed. The guideline *does not apply* to patients with hoarseness with the following conditions: history of laryngectomy (total or partial), craniofacial

anomalies, velopharyngeal insufficiency, and dysarthria (impaired articulation). However, the guideline will discuss the relevance of these conditions in managing patients with hoarseness.

There are a number of patients with modifying factors for whom many of the recommendations of the guideline may not apply. There is some discussion of these factors and how they might modify management. A partial list includes prior laryngeal surgery, recent surgical procedures involving the neck or affecting the recurrent laryngeal nerve, recent endotracheal intubation, radiation treatment to the neck, and patients who are singers or performers.

GUIDELINE PURPOSE

The primary purpose of this guideline is to improve the quality of care for patients with hoarseness based on current best evidence. Expert consensus to fill evidence gaps, when used, is explicitly stated, and is supported with a detailed evidence profile for transparency. Specific objectives of the guideline are to reduce inappropriate variations in care, produce optimal health outcomes, and minimize harm.

The guideline is intended to focus on a limited number of quality improvement opportunities, deemed most important by the working group, and is not intended to be a comprehensive, general guide for managing patients with hoarseness. In this context, the purpose is to define actions that could be taken by clinicians, regardless of discipline, to deliver quality care. Conversely, the statements in this guideline are not intended to limit or restrict care provided by clinicians based on assessment of individual patients.

While there is evidence to guide management of certain causes of hoarseness, there are currently no evidence-based clinical practice guidelines. There are variations in the use of the laser, voice therapy, steroids, and postoperative voice rest and in the treatment of reflux-related laryngitis.¹⁰⁻¹³ Differences in training, preference, and resource availability influence management decisions. A guideline is necessary given this practice variation and the significant public health burden of hoarseness.

This guideline addresses the identification, diagnosis, treatment, and prevention of hoarseness (dysphonia) (Table 1). In addition, it highlights needs and management options in special populations or in patients who have modifying factors. Furthermore, this guideline is intended to enhance the accurate diagnosis of hoarseness (dysphonia), promote appropriate intervention in patients with hoarseness, highlight the need for evaluation and intervention in special populations, promote appropriate therapeutic options with outcomes assessment, and improve counseling and education for prevention and management of hoarseness. This guideline may also be suitable for deriving a performance measure on hoarseness.

Table 1
Interventions considered in hoarseness guideline development

Diagnosis	Targeted history Physical examination Laryngoscopy Stroboscopy Computed tomography (CT) Magnetic resonance imaging (MRI)
Treatment	Watchful waiting/observation Education/information Voice therapy Anti-reflux medications Antibiotics Steroids Surgery Botulinum toxin (BOTOX)
Prevention	Voice training Vocal hygiene Education Environmental measures

BURDEN OF HOARSENESS

Hoarseness has a lifetime prevalence of 29.9 percent (percentage of people affected at some point in their life) and a point prevalence of 6.6 percent (percent of people affected at a given point in time) in adults aged 65 years or under.¹ Other cross-sectional studies have found a similar high lifetime prevalence of voice complaints of 28.8 percent in the general population.² Higher prevalence rates of hoarseness have been shown in telemarketers (31%),⁴ aerobics instructors (44%),⁵ and teachers (58%).^{2,6} Women are more frequently affected than men, with a 60:40 F:M ratio.^{1,3,14}

Hoarseness may affect all age groups. Among children, prevalence rates vary from 3.9 percent to 23.4 percent,¹⁵⁻¹⁷ with the most affected age range of 8 to 14 years.¹⁸ Voice problems persist four years or longer after identification in 38 percent of children with a voice disorder, suggesting an opportunity for early intervention.¹⁹ In addition, older adults are also at particular risk,³ with a point prevalence of 29 percent²⁰ and a lifetime incidence up to 47 percent.^{20,21}

Hoarseness has significant public health implications. Patients suffer social isolation, depression, and reduced disease-specific and general QOL.^{1,8,22,23} For example, patients with hoarseness caused by neurologic disorders (Parkinson disease, spasmodic dysphonia, vocal tremor, or vocal fold paralysis) reported severe levels of voice handicap and reduced general health-related QOL, comparable to impairments observed in patients with congestive heart failure, angina, and chronic obstructive pulmonary disease.^{7,8}

Hoarseness may also impair work-related function. Approximately 28 million US workers have occupations that require use of voice.⁹ In the general population, 7.2 percent of individuals surveyed missed work for one or more days within the preceding year because of a problem with their voice.¹ Among teachers this rate increases to 20

percent,^{6,14} resulting in a \$2.5 billion loss among US adults because of missed work annually.⁹

Medical, surgical, and behavioral treatment options exist for managing hoarseness. Among the general population, however, only 5.9 percent of those with hoarseness sought treatment.¹ Similarly, only 14.3 percent of teachers had consulted a physician or speech-language pathologist for hoarseness, even though voice function is essential to their profession.² In some circumstances, complete resolution of hoarseness may not be achieved and the clinician's responsibilities will include minimizing hoarseness and optimizing patient function as well as assisting the patient in developing understanding and realistic expectations.

Lack of awareness about hoarseness and its causes are potential barriers to appropriate care. Among older adults, individuals commonly attribute their hoarseness to advancing age. Such assumptions may prevent or delay those with hoarseness from obtaining treatment. Improved education among all health professionals²⁴ and efficient medical care are essential for reducing the health burden of hoarseness.²⁵ Inadequate insurance coverage has been cited as a cause of failure to seek treatment for both functional voice problems, as seen in singers,²⁵ and life-threatening ones, as seen in cancer patients.²⁶

The primary outcomes considered in this guideline are improvement in vocal function and change in voice-related QOL. Secondary outcomes include complications and adverse events. Economic consequences, adherence to therapy, global QOL, return to work, improved communication function, and return health care visits were also considered. The high prevalence, significant individual and societal implications, diversity of interventions, and lack of consensus make this an important condition for an up-to-date, evidence-based practice guideline.

GENERAL METHODS AND LITERATURE SEARCH

The guideline was developed using an explicit and transparent *a priori* protocol for creating actionable statements based on supporting evidence and the associated balance of benefit and harm.^{27,28} The multidisciplinary guideline development panel was chosen to represent the fields of neurology, speech-language pathology, professional voice teaching, family medicine, pulmonology, geriatric medicine, nursing, internal medicine, otolaryngology–head and neck surgery, pediatric medicine, and consumers. Several group members had significant prior experience in developing clinical practice guidelines.

Several initial literature searches were performed through November 17, 2008 by AAO-HNSF staff using MEDLINE, The National Guidelines Clearinghouse (NGC) (www.guideline.gov), The Cochrane Library, Guidelines International Network (GIN), The Cumulative Index to Nursing and Allied Health Literature (CINAHL), and

EMBASE. The initial broad MEDLINE search using “hoarseness[mh]” or “dysphonia[tw]” or “voice disorders[mh]” in any field showed 6032 potential articles:

- 1) Clinical practice guidelines were identified by a GIN, NGC, and MEDLINE search using “guideline” as a publication type or title word. The search identified eight guidelines with a topic of hoarseness or dysphonia. After eliminating articles that did not have hoarseness or dysphonia as the primary focus, no guidelines met quality criteria of being produced under the auspices of a medical association or organization and having an explicit method for ranking evidence and linking evidence to recommendations.
- 2) Systematic reviews were identified in MEDLINE using a validated filter strategy.²⁹ That strategy initially yielded 92 potential articles. The final data set included 14 systematic reviews or meta-analyses (including two Cochrane systematic reviews) on hoarseness or dysphonia that were distributed to the panel members.
- 3) Randomized controlled trials were identified through the Cochrane Library (Cochrane Controlled Trials Register) and totaled 256 trials with “hoarseness” or “dysphonia” in any field.
- 4) Original research studies were identified by limiting the MEDLINE, CINAHL, and EMBASE search to articles on humans published in English. The resulting data set of 769 articles yielded 262 related to therapy, 256 to diagnosis, 205 to etiology, and 46 to prognosis.

Results of all literature searches were distributed to guideline panel members at the first meeting, including electronic listings with abstracts (if available) of the searches for randomized trials, systematic reviews, and other studies. This material was supplemented, as needed, with targeted searches to address specific needs identified in writing the guideline through February 8, 2009.

In a series of conference calls, the working group defined the scope and objectives of the proposed guideline. During the nine months devoted to guideline development ending in 2009, the group met twice, with interval electronic review and feedback on each guideline draft to ensure accuracy of content and consistency with standardized criteria for reporting clinical practice guidelines.³⁰

AAO-HNSF staff used GEM-COGS,³¹ the Guideline Implementability Appraisal and Extractor, to appraise adherence of the draft guideline to methodological standards, to improve clarity of recommendations, and to predict potential obstacles to implementation. Guideline panel members received summary appraisals in April 2009 and modified an advanced draft of the guideline.

The final draft practice guideline underwent extensive, multidisciplinary, external peer review. Comments were compiled and reviewed by the group chairpersons, and a modified version of the guideline was distributed and approved by the development panel. The recommendations contained in the practice guideline are based on the best

available published data through February 2009. Where data were lacking, a combination of clinical experience and expert consensus was used. A scheduled review process will occur at five years from publication, or sooner if new compelling evidence warrants earlier consideration.

Classification of Evidence-Based Statements

Guidelines are intended to reduce inappropriate variations in clinical care, to produce optimal health outcomes for patients, and to minimize harm. The evidence-based approach to guideline development requires that the evidence supporting a policy be identified, appraised, and summarized and that an explicit link between evidence and statements be defined. Evidence-based statements reflect both the *quality of evidence* and the *balance of benefit and harm* that is anticipated when the statement is followed. The definitions for evidence-based statements³² are listed in Tables 2 and 3.

Guidelines are never intended to supersede professional judgment; rather, they may be viewed as a relative constraint on individual clinician discretion in a particular clinical circumstance. Less frequent variation in practice is expected for a “strong recommendation” than might be expected with a “recommendation.” “Options” offer the most opportunity for practice variability.³³ Clinicians should always act and decide in a way that they believe will best serve their patients’ interests and needs, regardless of guideline recommendations. They must also operate within their scope of practice and according to their training. Guidelines represent the best judgment of a team of experienced clinicians and methodologists addressing the scientific evidence for a particular topic.³²

Making recommendations about health practices involves value judgments on the desirability of various outcomes associated with management options. Values applied by the guideline panel sought to minimize harm and diminish unnecessary and inappropriate therapy. A major goal of the committee was to be transparent and explicit about how values were applied and to document the process.

Financial Disclosure and Conflicts of Interest

The cost of developing this guideline, including travel expenses of all panel members, was covered in full by the AAO-HNS Foundation. Potential conflicts of interest for all panel members in the past five years were compiled and distributed before the first conference call. After review and discussion of these disclosures,³⁴ the panel concluded that individuals with potential conflicts could remain on the panel if they: 1) reminded the panel of potential conflicts before any related discussion, 2) recused themselves from a related discussion if asked by the panel, and 3) agreed not to discuss any aspect of the guideline with industry before publication. Lastly, panelists were reminded that conflicts of interest extend beyond financial relationships and may include personal experiences, how a participant earns a living, and the participant’s previously established “stake” in an issue.³⁵

Table 2
Guideline definitions for evidence-based statements

Statement	Definition	Implication
Strong recommendation	A strong recommendation means the benefits of the recommended approach clearly exceed the harms (or that the harms clearly exceed the benefits, in the case of a strong negative recommendation) and that the quality of the supporting evidence is excellent (Grade A or B*). In some clearly identified circumstances, strong recommendations may be made based on lesser evidence when high-quality evidence is impossible to obtain and the anticipated benefits strongly outweigh the harms.	Clinicians should follow a strong recommendation unless a clear and compelling rationale for an alternative approach is present.
Recommendation	A recommendation means the benefits exceed the harms (or that the harms exceed the benefits, in the case of a negative recommendation), but the quality of evidence is not as strong (Grade B or C*). In some clearly identified circumstances, recommendations may be made based on lesser evidence when high-quality evidence is impossible to obtain and the anticipated benefits outweigh the harms.	Clinicians should also generally follow a recommendation, but should remain alert to new information and sensitive to patient preferences.
Option	An option means either that the quality of evidence that exists is suspect (Grade D*) or that well-done studies (Grade A, B, or C*) show little clear advantage to one approach vs another.	Clinicians should be flexible in their decision making regarding appropriate practice, although they may set bounds on alternatives; patient preference should have a substantial influencing role.

*See Table 3 for definition of evidence grades.

HOARSENESS (DYSPHONIA) GUIDELINE ACTION STATEMENTS

Each action statement is organized in a similar fashion: statement in boldface type, followed by an italicized statement on the strength of the recommendation. Several paragraphs then discuss the evidence base supporting the statement, concluding with an “evidence profile” of aggregate evidence quality, benefit-harm assessment, and statement of costs. Lastly, there is an explicit statement of the value judgments, the role of patient preferences, and a repeat statement of the strength of the recommendation. An overview of evidence-based statements in the guideline and their interrelationship is shown in Table 4.

The role of patient preference in making decisions deserves further clarification. For some statements the evidence base demonstrates clear benefit, which would minimize the role of patient preference. If the evidence is weak or benefits are unclear, however, not all informed patients might opt to follow the suggestion. In these cases, the practice of shared decision making, where the management decision is made by a collaborative effort between the clinician and the informed patient, becomes more useful.

Factors related to patient preference include (but are not limited to) absolute benefits (number needed to treat), adverse effects (number needed to harm), cost of drugs or tests, frequency and duration of treatment, and desire to take or avoid antibiotics. Comorbidity can also impact patient preferences by several mechanisms, including the potential for drug-drug interactions when planning therapy.

STATEMENT 1. DIAGNOSIS: Clinicians should diagnose hoarseness (dysphonia) in a patient with altered voice quality, pitch, loudness, or vocal effort that impairs communication or reduces voice-related QOL. *Recommendation based on observational studies with a preponderance of benefit over harm.*

Supporting Text

The purpose of this statement is to promote awareness of hoarseness (dysphonia) by all clinicians as a condition that may require intervention or additional investigation. The proposed diagnosis (dysphonia) is based on strictly clinical criteria, and does not require testing or additional investigations. Hoarseness is a symptom reported by the patient or proxy, identified by the clinician, or both.

Table 3
Evidence quality for grades of evidence

Grade	Evidence quality
A	Well-designed randomized controlled trials or diagnostic studies performed on a population similar to the guideline's target population
B	Randomized controlled trials or diagnostic studies with minor limitations; overwhelmingly consistent evidence from observational studies
C	Observational studies (case-control and cohort design)
D	Expert opinion, case reports, reasoning from first principles (bench research or animal studies)
X	Exceptional situations where validating studies cannot be performed and there is a clear preponderance of benefit over harm

Some patients with objectively minor changes may be unable to work and have a significant decrement in QOL. Others with significant disease such as malignancy may have minimal functional impairment of their voice. Of patients with laryngeal cancer, 52 percent thought their hoarseness was harmless and delayed seeing a physician.³⁶ Accordingly, patients with minimal objective voice change and significant complaints as well as patients with limited

complaints but with objective alterations of voice quality warrant evaluation.

Patients with hoarseness may experience discomfort with speaking, increased phonatory effort, and weak voice, as well as altered quality such as wobbly or shaky voice, breathiness, and raspiness.^{20,37,38} While a breathy voice may signify vocal fold paralysis or another cause of incomplete vocal fold closure, a strained voice with altered pitch or pitch breaks is common in spasmodic dysphonia.³⁹ Changes in voice quality may be limited to the singing voice and not affect the speaking voice. Among infants and young children, an abnormal cry may signify underlying pathology including vocal fold paralysis, laryngeal papilloma, or other systemic conditions.

Listening to the voice (perceptual evaluation) in a critical and objective manner may provide important diagnostic information. Characterizing the patient's complaint and voice quality is important for assessing hoarseness severity and for differentiating among specific causes of hoarseness, such as muscle tension dysphonia and spasmodic dysphonia.^{40,41}

Hoarseness may impair communication. Difficulty being heard and understood while using the telephone has been reported in the geriatric population.^{20,38} Trouble being heard in groups and problems being understood are also common complaints among hoarse patients.³⁷ Consequently, patients describe less confidence, decreased socialization, and impaired work-related function.^{1,37}

Hoarseness may lead to decreased voice-related QOL and a decrement in physical, social, and emotional aspects

Table 4
Outline of guideline action statements

Hoarseness (dysphonia) (<i>statement number</i>)	Statement strength
I. Diagnosis	
a. Diagnosis (<i>Statement 1</i>)	Recommendation
b. Modifying factors (<i>Statement 2</i>)	Recommendation
c. Laryngoscopy and hoarseness (<i>Statement 3A</i>)	Option
d. Indications for laryngoscopy (<i>Statement 3B</i>)	Recommendation
e. Imaging prior to laryngoscopy (<i>Statement 4</i>)	Recommendation against
II. Medical therapy	
a. Anti-reflux therapy for hoarseness in the absence of GERD or chronic laryngitis (<i>Statement 5A</i>)	Recommendation against
b. Anti-reflux therapy with chronic laryngitis (<i>Statement 5B</i>)	Option
c. Corticosteroid therapy (<i>Statement 6</i>)	Recommendation against
d. Antimicrobial therapy (<i>Statement 7</i>)	Strong recommendation against
III. Voice therapy	
a. Laryngoscopy prior to beginning (<i>Statement 8A</i>)	Recommendation
b. Advocating for (<i>Statement 8B</i>)	Strong recommendation
IV. Invasive therapies	
a. Advocating surgery in selected patients (<i>Statement 9</i>)	Recommendation
b. Botulinum toxin for adductor spasmodic dysphonia (<i>Statement 10</i>)	Recommendation
V. Prevention (<i>Statement 11</i>)	Option

Table 5
Pertinent medical history for assessing a patient with hoarseness⁴⁸⁻⁵⁰

Voice-specific questions
Did your problem start suddenly or gradually?
Is your voice ever normal?
Do you have pain when talking?
Does your voice deteriorate or fatigue with use?
Does it take more effort to use your voice?
What is different about the sound of your voice?
Do you have a difficult time getting loud or projecting?
Have you noticed changes in your pitch or range?
Do you run out of air when talking?
Does your voice crack or break?
Symptoms
Globus pharyngeus (persisting sensation of lump in throat)
Dysphagia
Sore throat
Chronic throat clearing
Cough
Odynophagia (pain with swallowing)
Nasal drainage
Post-nasal drainage
Non-anginal chest pain
Acid reflux
Regurgitation
Heartburn
Waterbrash (sudden appearance of salty liquid in the mouth)
Halitosis ("bad breath")
Fever
Hemoptysis
Weight loss
Night sweats
Otalgia (ear pain)
Difficulty breathing
Medical history relevant to hoarseness
Occupation and/or avocation requiring extensive voice use (ie, teacher, singer)
Absenteeism from occupation due to hoarseness
Prior episode(s) of hoarseness
Relationship of instrumentation (intubation, etc) to onset of hoarseness
Relationship of prior surgery to neck or chest to onset of hoarseness
Cognitive impairment (requirement for proxy historian)
Anxiety
Acute conditions
Infection of the throat and/or larynx: viral, bacterial, fungal
Foreign body in larynx, trachea, or esophagus
Neck or laryngeal trauma
Chronic conditions
Stroke
Diabetes
Parkinson's disease
Diseases from the Parkinson's Plus family (progressive supranuclear palsy, etc)
Myasthenia gravis
Multiple sclerosis
Amyotrophic lateral sclerosis (ALS)
Testosterone deficiency

Table 5
continued

Allergic rhinitis
Chronic rhinitis
Hypertension (because of certain medications used for this condition)
Schizophrenia (because of anti-psychotics used for mental health problems)
Osteoporosis (because of certain medications used for this condition)
Asthma, chronic obstructive pulmonary disease (because of use of inhaled steroids)
Aneurysm of thoracic aorta (rare cause)
Laryngeal cancer
Lung cancer (or metastasis to the lung)
Thyroid cancer
Hypothyroidism and other endocrinopathies
Vocal fold nodules
Vocal fold paralysis
Vocal abuse
Chemical laryngitis
Chronic tobacco use
Sjögren syndrome
Alcohol (moderate to heavy use or abuse)

of global QOL similar to those associated with other chronic diseases, such as congestive heart failure and chronic obstructive pulmonary disease.^{7,8}

Clinicians should consider input from proxies when diagnosing hoarseness (dysphonia). Of patients with vocal fold cancer, 40 percent waited three months before seeking medical attention for their hoarseness. Furthermore, 16.7 percent only sought treatment after encouragement from other people.³⁶ These data highlight the fact that hoarseness may not be recognized by the patient.

Children and patients with cognitive impairment or severe emotional burden may be unaware or unable to recognize and report on their own hoarseness.⁴² QOL studies in older adults have required proxy input in approximately 25 percent of the geriatric population.⁴³ While self-report measures for hoarseness are available, patients may be unable to complete them.⁴⁴⁻⁴⁶ In these cases, proxy judgments by significant others about QOL are a good alternative.⁴² Moderate agreement has been shown between adult patients and their communication partners on the Voice Handicap Index. Parent proxy self-report measures have also been validated for use in the pediatric population.^{38,47}

When evaluating a patient with hoarseness, the clinician should obtain a detailed medical history (Table 5) and review current medications (Table 6) as this information may identify the cause of the hoarseness (dysphonia) or an alternative underlying condition that may warrant attention.

Evidence profile for Statement 1: Diagnosis

- Aggregate evidence quality: Grade C, observational studies for symptoms with one systematic review of QOL in

Table 6
Medications that may cause hoarseness

Medication	Mechanism of impact on voice
Coumadin, thrombolytics, phosphodiesterase-5 inhibitors	Vocal fold hematoma ⁵¹⁻⁵³
Biphosphonates	Chemical laryngitis ⁵⁴
Angiotensin-converting enzyme inhibitors	Cough ⁵⁵
Antihistamines, diuretics, anticholinergics	Drying effect on mucosa ^{56,57}
Danocrine, testosterone	Sex hormone production/utilization alteration ^{58,59}
Antipsychotics, atypical antipsychotics	Laryngeal dystonia ^{60,61}
Inhaled steroids	Dose-dependent mucosal irritation, ⁶² fungal laryngitis

voice disorders and two systematic reviews on medication side effects

- Benefit: Identify patients who may benefit from treatment or from further investigation to identify underlying conditions that may be serious, promote prompt recognition and treatment, and discourage the perception of hoarseness as a trivial condition that does not warrant attention
- Harm: Potential anxiety related to diagnosis
- Cost: Time expended in diagnosis, documentation, and discussion
- Benefits-harm assessment: Preponderance of benefits over harm
- Value judgments: None
- Role of patient preference: Limited
- Intentional vagueness: None
- Exclusions: None
- Policy Level: Recommendation

STATEMENT 2. MODIFYING FACTORS: Clinicians should assess the patient with hoarseness by history and/or physical examination for factors that modify management such as one or more of the following: recent surgical procedures involving the neck or affecting the recurrent laryngeal nerve, recent endotracheal intubation, radiation treatment to the neck, a history of tobacco abuse, and occupation as a singer or vocal performer. *Recommendation based on observational studies with a preponderance of benefit over harm.*

Supporting Text

The term “modifying factors” as used in this recommendation refers to details elicited by history taking or physical examination that provide a clue to the presence of an important underlying etiology of hoarseness (dysphonia) that

may lead to a change in management. The history and physical examination of the patient with hoarseness may provide insight into the nature of the patient’s condition prior to the initiation of a more in-depth evaluation.

Surgery on the cervical spine via an anterior approach has been associated with a high incidence of voice problems. Recurrent laryngeal nerve paralysis has been reported to range from 1.27 percent to 2.7 percent.⁶³⁻⁶⁵ Assessment with laryngoscopy suggests an even higher incidence.⁶⁶ The incidence of hoarseness immediately following anterior cervical spine surgery may be as high as 50 percent.⁶⁷ Hoarseness resulting from anterior cervical spine surgery may or may not resolve over time.^{68,69}

Thyroid surgery has been associated with voice disorders. Patients with thyroid disease requiring surgery may have hoarseness and identifiable abnormalities on indirect laryngoscopy prior to surgery.⁷⁰ Thyroidectomy may cause hoarseness as a result of recurrent laryngeal nerve paralysis in up to 2.1 percent of patients.⁷¹ Surgery in the anterior neck can also lead to injury to the superior laryngeal nerve with resulting voice alteration, although this is uncommon.⁷²

Carotid endarterectomy is frequently associated with postoperative voice problems⁷³ and may result in recurrent laryngeal nerve damage in up to 6 percent of patients.^{74,75} Surgery to achieve an urgent airway or on the larynx directly may alter its structure, resulting in abnormal voice.^{76,77}

Surgical procedures not involving the neck may also result in hoarseness (dysphonia). Hoarseness following cardiac surgery is a common problem, occurring in 17 percent to 31 percent of patients.^{78,79} Hoarseness may result from changes in position or manipulation of the endotracheal tube or from lengthy procedures.⁷⁸ Recurrent laryngeal nerve injury occurs in about 1.4 percent of patients during cardiac surgery.⁷⁸ The left recurrent laryngeal nerve is damaged more commonly than the right as it extends into the chest and loops under the arch of the aorta. Damage may result from direct physical injury to the nerve or hypothermic injury due to cold cardioplegia.⁸⁰

Surgery for esophageal cancer frequently results in damage to the recurrent laryngeal nerve with subsequent hoarseness. In one study, 51 of 141 patients undergoing esophagectomy for cancer had laryngeal nerve paralysis, with 30 of these patients having persistent paralysis one year following surgery.⁸¹ The implantation of vagal nerve stimulators for intractable seizures has been associated with hoarseness in as many as 28 percent of patients.⁸²

Prolonged endotracheal intubation has been associated with hoarseness. Direct laryngoscopy of patients intubated for more than four days (mean nine days) demonstrates that 94 percent of patients have laryngeal injury.⁸³ The injury patterns seen in the patients with prolonged intubation include laryngeal edema and posterior and medial vocal fold ulceration. As many as 44 percent of patients with prolonged intubation may develop vocal fold granulomas within four weeks of being extubated. In this study, 18

percent of patients had prolonged true vocal fold immobility for at least four weeks after extubation.⁸⁴ Another study following a large group of patients for several years found chronic phonatory dysfunction in many patients after long-term intubation.⁸⁵

Short-term intubation for general anesthesia may result in hoarseness and vocal fold pathology in over 50 percent of cases.⁸⁶ While most symptoms resolved after five days, prolonged symptoms may result from vocal fold granuloma. If hoarseness persists, the remoteness of the index event may confound the evaluating clinician. Use of a laryngeal mask airway may reduce postsurgical complaints of discomfort, but does not objectively reduce hoarseness.⁸⁷

Long-term intubation of neonates may result in voice problems related to arytenoid and posterior commissure ulceration and cartilage erosion.⁸⁸ Children with a history of prolonged intubation may have long-term complications of hoarseness and arytenoid dysfunction.

Voice disorders are common in older adults and significantly affect the QOL in these patients.²¹ Vocal fold atrophy with resulting hoarseness (dysphonia) is a common disorder of older adults and is frequently undiagnosed by primary care providers.^{89,90} Hoarseness resulting from neurologic disorders such as cerebral vascular accident and Parkinson disease is also more common in elderly patients.⁹¹⁻⁹⁴ Multiple sclerosis can lead to hoarseness in patients of any age.⁹⁵

Chronic hoarseness (dysphonia) is quite common in young children and has an adverse impact on QOL.⁹⁶ Prevalence ranges from 15 percent to 24 percent of the population.^{17,97} In one study, 77 percent of hoarse children had vocal fold nodules.¹⁷ These may persist into adolescence if not properly treated.⁹⁸ Craniofacial anomalies such as orofacial clefts are associated with abnormal voice,⁹⁹ but these are frequently resonance disorders requiring very different therapies than for hoarse children with normal anatomical development.

Hoarseness or dysphonia in infants may be recognized only by an abnormal cry, and suspicion of such symptoms should prompt consultation with an otolaryngologist.¹⁰⁰ When infants do present with hoarseness, underlying etiologies such as birth trauma, an intracranial process such as Arnold-Chiari malformation or posterior fossa mass, or mediastinal pathology should be considered.¹⁰¹

Hoarseness in tobacco smokers is associated with an increased frequency of polypoid vocal fold lesions and head and neck cancer.¹⁰² Accordingly, this requires an expedient assessment for malignancy as the potential cause of hoarseness. In addition, in patients treated with external beam radiation for glottic cancer, radiation treatment is associated with hoarseness in about 8 percent of cases.^{103,104}

Patients who use inhaled corticosteroids for the treatment of asthma or chronic obstructive pulmonary disease may present to a clinician with hoarseness that is a side effect of therapy either from direct irritation or from a fungal infection of the larynx.¹⁰⁵

Singers or vocal performers should be identified by the clinician when eliciting a history from the hoarse patient. These patients have significant impairment with symptoms that may be subclinical in other patients. They may be more subject to voice over-use or have a different etiology for their symptoms, and hoarseness may have a more significant impact on their QOL or ability to earn income. For example, while hoarseness is relatively rare following thyroid surgery, there are objective, measurable changes in the voice of most patients that could affect pitch and the ability to sing.¹⁰⁶ Singers are also prone to develop microvascular ectasias that affect voice and require specific therapy.¹⁰⁷

To a slightly lesser degree, individuals in a number of other occupations or avocations, such as teachers and clergy, depend on voice use. As an example, over 50 percent of teachers have hoarseness, and vocal overuse is a common, but not exclusive, etiologic factor.¹⁰⁸ Clinicians should inquire about an individual's voice use in order to determine the degree to which altered voice quality may impact the individual professionally.

Evidence profile for Statement 2: Modifying Factors

- Aggregate evidence quality: Grade C, observational studies
- Benefit: To identify factors early in the course of management that could influence the timing of diagnostic procedures, choice of interventions, or provision of follow-up care
- Harm: None
- Cost: None
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: Importance of history taking and identifying modifying factors as an essential component of providing quality care
- Role of patient preferences: Limited or none
- Intentional vagueness: None
- Exclusions: None
- Policy level: Recommendation

STATEMENT 3A. LARYNGOSCOPY AND HOARSENESS: Clinicians may perform laryngoscopy, or may refer the patient to a clinician who can visualize the larynx, at any time in a patient with hoarseness. *Option based on observational studies, expert opinion, and a balance of benefit and harm.*

STATEMENT 3B. INDICATIONS FOR LARYNGOSCOPY: Clinicians should visualize the patient's larynx, or refer the patient to a clinician who can visualize the larynx, when hoarseness fails to resolve by a maximum of three months after onset, or irrespective of duration if a serious underlying cause is suspected. *Recommendation based on observational studies, expert opinion, and a preponderance of benefit over harm.*

Table 7
Conditions leading to suspicion of a “serious underlying cause”

Hoarseness with a history of tobacco or alcohol use
Hoarseness with concomitant discovery of a neck mass
Hoarseness after trauma
Hoarseness associated with hemoptysis, dysphagia, odynophagia, otalgia, or airway compromise
Hoarseness with accompanying neurologic symptoms
Hoarseness with unexplained weight loss
Hoarseness that is worsening
Hoarseness in an immunocompromised host
Hoarseness and possible aspiration of a foreign body
Hoarseness in a neonate
Unresolving hoarseness after surgery (intubation or neck surgery)

Supporting Text

The purpose of these statements is to highlight the important role of visualizing the larynx and vocal folds in managing a patient with hoarseness, especially if the hoarseness fails to improve within three months of onset (Statement 3B). Patients with persistent hoarseness may have a serious underlying disorder (Table 7) that would not be diagnosed unless the larynx was visualized. This does not, however, imply that all patients must wait three months before laryngoscopy is performed, because, as outlined below, early assessment of some patients with hoarseness may improve management. Therefore, clinicians may perform laryngoscopy, or refer to a clinician for laryngoscopy, at any time (Statement 3A) if deemed appropriate based on the patient’s specific clinical presentation and modifying factors.

Laryngoscopy and Hoarseness

Visualization of the larynx is part of a comprehensive evaluation for voice disorders. While not all clinicians have the training and equipment necessary to visualize the larynx, those who do may examine the larynx of a patient presenting with hoarseness at any time if considered appropriate. Although most hoarseness is caused by benign or self-limited conditions, early identification of some disorders may increase the likelihood of optimal outcomes.

There are a number of conditions where laryngoscopy at the time of initial assessment allows for timely diagnosis and management. Laryngoscopy can be used at the bedside for patients with hoarseness after surgery or intubation to identify vocal fold immobility, intubation trauma, or other sources of postsurgical hoarseness. Laryngoscopy plays a critical role in evaluating laryngeal patency after laryngeal trauma where visualization of the airway allows for assessment of the need for surgical intervention and for following patients in whom immediate surgery is not required.^{109,110}

Laryngoscopy is used routinely for diagnosing laryngeal cancer. The usefulness of laryngoscopy for establishing the

diagnosis and the benefit of early detection have led the British medical system to employ fast-track screening clinics for laryngeal cancer that mandate laryngoscopy within 14 days of suspicion of laryngeal cancer.^{111,112} Fungal laryngitis from inhalers and other causes is best diagnosed with laryngoscopy and must be distinguished from malignancy.¹¹³

Unilateral vocal fold paralysis causes breathy hoarseness and is often caused by thoracic, cervical, or brain tumors that either compress or invade the vagus nerve or its branches that innervate the larynx. Stroke may also present with hoarseness due to vocal fold paralysis. Vocal fold paralysis is routinely identified, characterized, and followed by laryngoscopy.^{79,114}

In patients with cranial nerve deficits or neuromuscular changes, laryngoscopy is useful to identify neurologic causes of vocal dysfunction.¹¹⁵ Benign vocal fold lesions such as vocal fold cysts, nodules, and polyps are readily detected on laryngoscopy. Visualization of the larynx may also provide supporting evidence in the diagnosis of laryngopharyngeal reflux.¹¹⁶

Hoarseness caused by neurologic or motor neuron disease such as Parkinson disease, amyotrophic lateral sclerosis, and spasmodic dysphonia may have laryngoscopic findings that the clinician can identify to initiate management of the underlying disease.¹¹⁷ Office laryngoscopy is also a critical tool in the evaluation of the aging voice.

Neonates with hoarseness should undergo laryngoscopy to identify vocal fold paralysis,¹¹⁸ laryngeal webs,¹¹⁹ or other congenital anomalies that might affect their ability to swallow or breathe.¹²⁰

Hoarseness in children is rarely a sign of a serious underlying condition and is more likely the result of a benign lesion of the larynx such as a vocal fold polyp, nodules, or cyst.¹²¹ However, determining if laryngeal papilloma is the etiology of hoarseness in a child is particularly important given the high potential for life-threatening airway obstruction and the potential for malignant transformation.¹²² A hoarse child with other symptoms such as stridor, airway obstruction, or dysphagia may have a serious underlying problem such as a Chiari malformation,¹²³ hydrocephalus, skull base tumors, or a compressing neck or mediastinal mass. Persistent hoarseness in children may be a symptom of vocal fold paralysis with underlying etiologies that include neck masses, congenital heart disease, or previous cardiothoracic, esophageal, or neck surgery.¹²⁴

Indications for Laryngoscopy

Laryngoscopy is indicated for the assessment of hoarseness if symptoms fail to improve or resolve within three months, or at any time the clinician suspects a serious underlying disorder. In this context “serious” describes an etiology that would shorten the lifespan of the patient or otherwise reduce professional viability or voice-related QOL. If the clinician is concerned that hoarseness may be caused by a serious underlying condition, the optimal way to address this con-

cern is by visualization of the vocal folds with laryngoscopy.

The major cause of community-acquired hoarseness is viral. Symptoms from viral laryngitis typically last 1 to 3 weeks.^{125,126} Symptoms of hoarseness persisting beyond this time warrant further evaluation to insure that no malignancy or morbid conditions are missed and to allow further treatment to be initiated based on specific benign pathologies if indicated. One population-based cohort study¹²⁷ and one large case-control study¹²⁸ have shown that delays in diagnosis of laryngeal cancer lead to higher stages of disease at diagnosis and worse prognosis. In the cohort study, delay longer than three months led to poorer survival.

The expediency of laryngoscopy also depends on patient considerations. Singers, performers, and patients whose livelihood depends upon their voice will not be able to wait several weeks for their hoarseness to resolve as they may be unable to work in the interim. In fact, a number of professionals with high vocal demands may benefit from immediate evaluation.

Even in the absence of serious concern or patient considerations indicating immediate laryngoscopy, persistent hoarseness should be evaluated to rule out significant pathology such as cancer or vocal fold paralysis. In the absence of immediate concern, there is little guidance from the literature on the proper length of time a hoarse patient can or should be observed before visualization of the larynx is mandated. The working group weighed the risk of delayed diagnosis against the potential over-utilization of resources and selected a fairly long window of three months prior to mandating laryngoscopy. This safety net approach, based on expert opinion, was designed to address the main concern of the working group that many patients with persistent hoarseness are currently experiencing delayed diagnosis or are not undergoing laryngoscopy at all.

Techniques for Visualizing the Larynx

Different techniques are available for laryngoscopy and confer varying levels of risk. The working group does not have recommendations as to the preferred method. Choice of method is at the discretion of the evaluating clinician.

Office laryngoscopy can be performed transorally with a mirror or rigid endoscope, transnasally with a flexible fiberoptic or distal-chip laryngoscope, and with either halogen light or stroboscopic light application.¹²⁹ The surface and mobility of the vocal folds are well assessed with these tools.

Stroboscopy is used to visualize the vocal folds as they vibrate, allowing for an assessment of both anatomy and function during the act of phonation.¹³⁰ When hoarseness symptoms are out of proportion to the laryngoscopic examination, stroboscopy should be considered. The addition of stroboscopic light allows for an assessment of the pliability of the vocal folds, making additional pathologies such as vocal fold scar easy to identify. Stroboscopy has resulted in altered diagnosis in 47 percent of cases,¹³¹ and stroboscopic parameters aid in the differentiation of specific vocal fold

pathology, such as polyps and cysts.¹³² Surgical endoscopy with magnification (microlaryngoscopy) is utilized more often when more detailed examination, manipulation, or biopsy of the structures is required.¹³³

In the adult, visualization by indirect mirror examination may be limited by patient tolerance and photo documentation is not possible. Discomfort in transnasal laryngoscopy is usually mitigated by the application of topical decongestant and/or anesthetic such as lidocaine. A study of 1208 patients evaluated by fiberoptic laryngoscopy for assessment of vocal fold paralysis after thyroidectomy showed no significant adverse events.¹³⁴ No other reports of significant risks of fiberoptic laryngoscopy were found in a detailed MEDLINE search using key words: laryngoscopy, complications, risk, and adverse events. Transoral examinations of the larynx may be preceded by topical lidocaine to the throat and carries similarly minimal risk.

Operative laryngoscopy carries more substantial risk but generally allows for ease of tissue manipulation and biopsy. Risks associated with direct laryngoscopy with general anesthesia, include airway distress; dental trauma; oral cavity, oropharyngeal, and hypopharyngeal trauma; tongue dysesthesia; taste changes; and cardiovascular risk.¹³⁵⁻¹³⁷ The cost of direct laryngoscopy is substantially greater than that of office-based laryngoscopy due to the additional costs of staff, equipment, and additional care required.¹³⁸⁻¹⁴⁰

Special consideration is given to children for whom laryngoscopy requires either advanced skill or a specialized setting. With the advent of small-diameter flexible laryngoscopes, awake, flexible laryngoscopy can be employed in the clinic in children as young as newborns but is subject to the skill of the clinician and comfort with children. The advantage is that this examination allows for evaluation of both anatomy and function of the larynx in the hoarse child. Direct laryngoscopy under anesthesia with or without a microscope may be used to verify flexible fiberoptic findings, manage laryngeal papillomas or other vocal fold lesions, and further define laryngeal pathology such as congenital anomalies of the larynx. Intraoperative palpation of the cricoarytenoid joint may also help differentiate between vocal fold paralysis and fixation.

Evidence profile for Statement 3A: Laryngoscopy and Hoarseness

- Aggregate evidence quality: Grade C, based on observational studies
- Benefit: Visualization of the larynx to improve diagnostic accuracy and allow comprehensive evaluation
- Harm: Risk of laryngoscopy, patient discomfort
- Cost: Procedural expense
- Benefits-harm assessment: Balance of benefit and harm
- Value judgments: Laryngoscopy is an important tool for evaluating voice complaints and may be performed at any time in the patient with hoarseness
- Intentional vagueness: None

- Role of patient preferences: Substantial; the level of patient concern should be considered in deciding when to perform laryngoscopy
- Exclusions: None
- Policy level: Option

Evidence profile for Statement 3B: Indications for Laryngoscopy

- Aggregate evidence quality: Grade C, observational studies on the natural history of benign laryngeal disorders; grade C for observational studies plus expert opinion on defining what constitutes a serious underlying condition
- Benefit: Avoid missed or delayed diagnosis of serious conditions in patients without additional signs or symptoms to suggest underlying disease; permit prompt assessment of the larynx when serious concern exists
- Harm: Potential for up to a three-month delay in diagnosis; procedure-related morbidity
- Cost: Procedural expense
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: A need to balance timely diagnostic intervention with the potential for over-utilization and excessive cost. The guideline panel debated on the maximum duration of hoarseness prior to mandated evaluation and opted to select a “safety net approach” with a generous time allowance (three months), but options to proceed promptly based on clinical circumstances
- Intentional vagueness: The term “serious underlying concern” is subject to the discretion of the clinician. Some conditions are clearly serious, but in other patients, the seriousness of the condition is dependent on the patient. Intentional vagueness was incorporated to allow for clinical judgment in the expediency of evaluation
- Role of patient preferences: Limited
- Exclusions: None
- Policy level: Recommendation

STATEMENT 4. IMAGING: Clinicians should not obtain computed tomography (CT) or magnetic resonance imaging (MRI) of the patient with a primary complaint of hoarseness prior to visualizing the larynx. *Recommendation against imaging based on observational studies of harm, absence of evidence concerning benefit, and a preponderance of harm over benefit.*

Supporting Text

The purpose of this statement is not to discourage the use of imaging in the comprehensive work-up of hoarseness, but rather to emphasize that it should be used to assess for specific pathology after the larynx has been visualized.

Laryngoscopy is the primary diagnostic modality for evaluating patients with hoarseness. Imaging studies, including CT and MRI, have also been used, but are unnecessary in most patients because most hoarseness is self-limited or caused by pathology that can be identified by

laryngoscopy. The value of imaging procedures before laryngoscopy is undocumented; no articles were found in the systematic literature review for this guideline regarding the diagnostic yield of imaging studies prior to laryngeal examination. Conversely, the risk of imaging studies is well documented.

The risk of radiation-induced malignancy from CT scans is small but real. More than 62 million CT scans per year are obtained in the United States for all indications, including 4 million performed on children (nationwide evaluation of x-ray trends). In a study of 400,000 radiation workers in the nuclear industry who were exposed to an average dose of 20 mSVs (a typical organ dose from a single CT scan for an adult), a significant association was reported between the radiation dose and mortality from cancer in this cohort. These risks were quantitatively similar to those reported for atomic bomb survivors.¹⁴¹ Children have higher rates of malignancy and a longer lifespan in which radiation-induced malignancies can develop.^{142,143} It is estimated that about 0.4 percent of all cancers in the United States may be attributable to the radiation from CT studies.^{144,145} The risk may be higher (1.5% to 2%) if we adjust this estimate based on our current use of CT scans.

There are also risks associated with IV contrast dye used to increase diagnostic yield of CT scans.¹⁴⁶ Allergies to contrast dye are common (5% to 8% of the population). Severe, life-threatening reactions, including anaphylaxis, occur in 0.1 percent of people receiving iodinated contrast material, with a death rate of up to one in 29,500 people.^{147,148}

While MRI has no radiation effects, it is not without risk. A review of the safety risks of MRI¹⁴⁹ details five main classes of injury: 1) projectile effects (anything metal that gets attracted by the magnetic field); 2) twisting of indwelling metallic objects (cerebral artery clips, cochlear implants, or shrapnel); 3) burning (electrical conductive material in contact with the skin with an applied magnetic field, ie, EKG electrodes or medication patches); 4) artifacts (radio-frequency effects from the device itself simulating pathology); and 5) device malfunction (pacemakers will fire inappropriately or work at an elevated frequency, thus distorting cardiac conduction).¹⁵⁰

The small confines of the MRI scanner may lead to claustrophobia and anxiety.¹⁵¹ Some patients, children in particular, require sedation (with its associated risks). The gadolinium contrast used for MRI rarely induces anaphylactic reactions,^{152,153} but there is recent evidence of renal toxicity with gadolinium in patients with pre-existing renal disease.¹⁵⁴ Transient hearing loss has been reported, but this is usually avoided with hearing protection.¹⁵⁵ The costs of MRI, however, are significantly more than CT scanning. Despite these risks and their considerable cost, cross-sectional imaging studies are being used with increasing frequency.¹⁵⁶⁻¹⁵⁸

After laryngoscopy, evidence does support the use of imaging to further evaluate 1) vocal fold paralysis or 2) a

mass or lesion of the vocal fold or larynx that suggests malignancy or airway obstruction.¹⁵⁹ If vocal fold palsy is noted and recent surgery can explain the cause of the paralysis, imaging studies are generally not useful. If the health care provider suspects a lesion along the recurrent laryngeal nerve, imaging studies are indicated.

Unexplained vocal fold paralysis found on laryngoscopy warrants imaging the skull base to the thoracic inlet/arch of the aorta. Including these anatomic areas allows for evaluation of the entire path of the recurrent laryngeal nerve as it loops around the arch of the aorta on the left side. On the right, it will show any lesions in the lung apex along the course of the right recurrent laryngeal nerve as it loops around the subclavian artery. One study showed that a complete radiographic work-up improved rates of diagnosis,¹⁶⁰ but there is no consensus on whether CT or MRI is better for evaluating the recurrent laryngeal nerve.^{161,162} Lesions at the skull base and brain are best evaluated using an MRI of the brain and brain stem with gadolinium enhancement. If a patient presents with additional lower cranial nerve palsy, the skull base, particularly the jugular foramen (CN IX, X, XI), should be evaluated.¹⁵⁹

Primary lesions of the larynx, pharynx, subglottis, thyroid, and any pertinent lymph node groups can also be evaluated by imaging the entire area. Intravenous contrast may help to distinguish vascular lesions from normal pathology on CT. Due to the substantial dose of ionizing radiation delivered to the radiosensitive thyroid gland,¹⁶³ CT examination in children is cautioned when MRI is available.

There is still significant controversy whether MRI or CT is the preferred study to evaluate invasion of laryngeal cartilage. Before the advent of the helical CT, MRI was the preferred method.¹⁶⁴ The extent of bone marrow infiltration by malignant tumors (ie, nasopharyngeal carcinoma) can be assessed with MRI of the skull base.¹⁶⁵ MRI is preferred in children and can easily be extended to include the mediastinum to help evaluate congenital and neoplastic lesions. For those patients who have absolute contraindications to MRI such as pacemaker, cochlear implants, heart valve prosthesis, or aneurysmal clip, CT is a viable alternative.

Imaging studies are valuable tools in diagnosing certain causes of hoarseness in children. A plain chest radiograph will aid in the diagnosis of a mediastinal mass or foreign body. A CT scan can elucidate more detail if the initial radiography fails to show a lesion. A soft tissue radiograph of the neck can aid in the diagnosis of an infectious or allergic process.¹⁶⁶ CT imaging has been the test of choice for congenital cysts, laryngeal webs, solid neoplasms, and external trauma, as it provides adequate resolution without having to sedate the patient as may be necessary for MRI. The risk of radiation must be weighed against these benefits. MRI is the better option for imaging the brain stem.¹⁶⁶

FDG-PET imaging is used increasingly to assess patients with head and neck cancer. PET scans may help identify mediastinal or pulmonary neoplasms that cause vocal fold

paralysis.¹⁶⁷ PET scanning is very costly, however, and may give false-positive results in patients with vocal fold paralysis. FDG activity in the normal vocal fold can be misinterpreted as a tumor.¹⁶⁸

Evidence profile for Statement 4: Imaging

- Aggregate evidence quality: Grade C, observational studies regarding the adverse events of CT and MRI; no evidence identified concerning benefits in patients with hoarseness before laryngoscopy
- Benefit: Avoid unnecessary testing; minimize cost and adverse events; maximize the diagnostic yield of CT and MRI when indicated
- Harm: Potential for delayed diagnosis
- Cost: None
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: Avoidance of unnecessary testing
- Role of patient preferences: Limited
- Intentional vagueness: None
- Exclusions: None
- Policy level: Recommendation against

STATEMENT 5A. ANTI-REFLUX MEDICATION AND HOARSENESS. Clinicians should not prescribe anti-reflux medications for patients with hoarseness without signs or symptoms of gastroesophageal reflux disease (GERD). *Recommendation against prescribing based on randomized trials with limitations and observational studies with a preponderance of harm over benefit.*

STATEMENT 5B. ANTI-REFLUX MEDICATION AND CHRONIC LARYNGITIS. Clinicians may prescribe anti-reflux medication for patients with hoarseness and signs of chronic laryngitis. *Option based on observational studies with limitations and a relative balance of benefit and harm.*

Supporting Text

The primary intent of this statement is to limit widespread use of anti-reflux medications as empiric therapy for hoarseness without symptoms of GERD or laryngeal findings consistent with laryngitis, given the known adverse effects of the drugs and limited evidence of benefit. The purpose is not to limit use of anti-reflux medications in managing laryngeal inflammation, when inflammation is seen on laryngoscopy (eg, laryngitis denoted by erythema, edema, redundant tissue, and/or surface irregularities of the interarytenoid mucosa, arytenoid mucosa, posterior laryngeal mucosa, and/or vocal folds). To emphasize these dual considerations, the working group has split the statement into part A, a recommendation against empiric therapy for hoarseness, and part B, an option to use anti-reflux therapy in managing properly diagnosed laryngitis.

Anti-Reflux Medications and the Empiric Treatment of Hoarseness

The benefit of anti-reflux treatment for hoarseness in patients without symptoms of esophageal reflux (heartburn and regurgitation) or evidence for esophagitis is unclear. A Cochrane systematic review of 302 eligible studies that assess the effectiveness of anti-reflux therapy for patients with hoarseness did not identify any high-quality trials meeting the inclusion criteria.¹⁶⁹ For example, a nonrandomized study on treating patients with documented reflux of stomach contents into the throat (laryngopharyngeal reflux) with twice-daily proton pump inhibitors (PPIs) could not be included in the review because hoarseness was only one component of the reflux symptom index and not an outcome separate from heartburn.¹⁷⁰ One randomized, placebo-controlled trial was also not included because it did not separate hoarseness as an outcome from other laryngeal symptoms.¹⁷¹ However, the response rate for the laryngeal symptoms was 50 percent in the PPI group compared to 10 percent in the placebo group.

A randomized trial published after the Cochrane review of anti-reflux treatment for hoarseness included 145 subjects with chronic laryngeal symptoms (throat clearing, cough, globus, sore throat, or hoarseness and no cardinal GERD symptoms) and laryngoscopic evidence for laryngitis (erythema, edema, and/or surface irregularities of the interarytenoid mucosa, arytenoid mucosa, posterior laryngeal mucosa, and/or vocal folds).¹⁷² Subjects received either esomeprazole 40 mg twice daily or placebo for 16 weeks. There was no evidence for benefit in symptom score or laryngopharyngeal reflux health-related QOL score between the groups at the end of the study. However, this study included patients with one of many possible laryngeal symptoms and excluded patients with heartburn three or more days per week.¹⁷²

The benefits of anti-reflux medication for control of GERD symptoms are well documented. High-quality controlled studies demonstrate that PPIs and H2RA (histamine-2 receptor antagonist) improve important clinical outcomes in esophageal GERD over placebo, with PPIs demonstrating superior response.^{173,174} Response rates for esophageal symptoms and esophagitis healing are high (approximately 80% for PPIs).^{173,174}

In patients with hoarseness *and* a diagnosis of GERD, anti-reflux treatment is more likely to reduce hoarseness. Anti-reflux treatment given to patients with GERD (based on positive pH probe, esophagitis on endoscopy, or presence of heartburn or regurgitation) showed improved chronic laryngitis symptoms, including hoarseness, over those without GERD.¹⁷⁵

There is some evidence supporting the pharmacological treatment of GERD without documented esophagitis, but the number needed to treat tends to be higher.¹⁷³ These studies have esophageal symptoms and/or mucosal healing as outcomes, not hoarseness.

While generally safe for therapy shorter than two months, prolonged therapy with PPIs and H2RAs for greater than three months has been associated with significant risk. H2RAs are associated with impaired cognition in older adults.^{176,177} PPI use may increase the risk of bacterial gastroenteritis, specifically campylobacter and salmonella¹⁷⁸ and possibly clostridium difficile.¹⁷⁹ Epidemiological studies also associate PPIs with community-acquired pneumonia.^{180,181} Although patients with primary voice disorders may differ from those in the above mentioned studies, the treating clinician needs to consider these adverse events. Furthermore, PPIs may impair the ability of clopidogrel to inhibit platelet aggregation activity,¹⁸² to varying degrees depending upon the particular PPI.

Higher doses such as the twice-daily PPI therapy may carry a higher risk than once-daily therapy, and older adults may be more likely than younger adults to be harmed.¹⁸³ Although pneumonia is more common in young children using PPIs, the prevalence of profound regurgitation and swallowing disorders is high in that population, so it is difficult to draw conclusions about the effect of the drug itself.¹⁸⁴

Use of PPI may interfere with calcium absorption and bone homeostasis. PPI use is associated with an increased risk for hip fractures in older adults.¹⁸⁵ PPIs decrease vitamin B₁₂ (cobalamin) absorption in a dose-dependent manner,¹⁸⁶ and serum vitamin B₁₂ levels may underestimate the resulting serum cobalamin deficiency.¹⁸⁷ PPI use also decreases iron absorption and may cause iron deficiency anemia.¹⁸⁸ Additionally, acid-suppressing drugs (both H2RAs and PPIs) were associated with an increased risk of pancreatitis in a case-controlled study, not explained by the slightly higher risk of pancreatitis seen in patients with GERD symptoms alone.¹⁸⁹

For patients with hoarseness and GERD, a trial of anti-reflux therapy may be prescribed. If hoarseness does not respond or if symptoms worsen, then pharmacological therapy should be discontinued and a search for alternative causes of hoarseness should be initiated with laryngoscopy.

Anti-Reflux Medications and Treatment of Chronic Laryngitis

Laryngoscopy is helpful in determining whether anti-reflux treatment should be considered in managing a patient with hoarseness. Increased pharyngeal acid reflux events are more common in patients with vocal process granulomas compared to controls.¹⁹⁰ Also, erythema in the vocal folds, arytenoid mucosa, and posterior commissure has improved with omeprazole treatment in patients with sore throat, throat clearing, hoarseness, and/or cough.¹⁹¹ While no differences in hoarseness improvement was seen between three months of esomeprazole vs placebo, one small randomized controlled trial found that findings of erythema, diffuse laryngeal edema, and posterior commissure hypertrophy

showed greater improvement in the treatment arm compared to placebo.¹⁹²

More improvement in signs of laryngitis of the true vocal folds (such as erythema, edema, redundant tissue, and/or surface irregularities), posterior cricoid mucosa, and arytenoid complex were noted in patients whose laryngeal symptoms, including hoarseness, responded to four months of PPI treatment compared to nonresponders.¹⁹³ Additionally, the above abnormalities of the interarytenoid mucosa and true vocal folds were predictive of improvement in laryngeal symptoms, including hoarseness.¹⁹³

Reflux of stomach contents into the laryngopharynx is an important consideration in the management of patients with laryngeal disorders. Reflux of gastric contents into the hypopharynx has been linked with subglottic stenosis.¹⁹⁴ Case-control studies have shown that GERD may be a risk factor for laryngeal cancer,¹⁹⁵ and that anti-reflux therapy may reduce the risk of laryngeal cancer recurrence.¹⁹⁶ Better healing and reduced polyp recurrence after vocal fold surgery in patients taking PPIs compared to no PPIs have also been described.¹⁹⁷

PPI treatment may improve laryngeal lesions and objective measures of voice quality. Observational studies have demonstrated that vocal process granulomas, which may cause hoarseness, have resolved or regressed after treatment with anti-reflux medication with or without voice therapy.¹⁹⁸ Case series also have shown improved acoustic voice measures of voice quality after one to two months of PPI therapy compared to baseline.¹⁹⁹

Nonetheless, there are limitations of the endoscopic laryngeal examination in diagnosing patients who may respond to PPIs. The presence of abnormal findings, such as the interarytenoid bar, has been noted in normal individuals.¹⁷⁷ In addition, in a study of healthy volunteers not routinely using anti-reflux medication and with GERD symptoms no more than three times per month, erythema of the medial arytenoid, posterior commissure hypertrophy, and pseudosulcus were noted.²⁰⁰ Furthermore, the presence of specific findings depended upon the method of laryngoscopy (rigid vs flexible) and the inter-rater reliability ranged from moderate to poor depending on the specific finding.²⁰⁰ In a study of patients with hoarseness from a variety of diagnoses, problems with intra- and inter-rater reliability for findings of edema and erythema of the vocal folds and arytenoids have also been noted.²⁰¹

Further research exploring the sensitivity, specificity, and reliability of laryngoscopic examination findings is necessary to determine which signs are associated with treatment response with respect to hoarseness and which techniques are best to identify them.

Evidence profile for Statement 5A: Anti-reflux Medications and Hoarseness

- Aggregate evidence quality: Grade B, randomized trials with limitations showing lack of benefits for anti-reflux therapy in patients with laryngeal symptoms, including hoarseness; ob-

servational studies with inconsistent or inconclusive results; inconclusive evidence regarding the prevalence of hoarseness as the only manifestation of reflux disease

- Benefit: Avoid adverse events from unproven therapy; reduce cost; limit unnecessary treatment
- Harm: Potential withholding of therapy from patients who may benefit
- Cost: None
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: Acknowledgment by the working group of the controversy surrounding laryngopharyngeal reflux, and the need for further research before definitive conclusions can be drawn; desire to avoid known adverse events from anti-reflux therapy
- Intentional vagueness: None
- Patient preference: Limited
- Exclusions: Patients immediately before or after laryngeal surgery and patients with other diagnosed pathology of the larynx
- Policy level: Recommendation against

Evidence profile for Statement 5B: Anti-reflux Medication and Chronic Laryngitis

- Aggregate evidence quality: Grade C, observational studies with limitations showing benefit with laryngeal symptoms, including hoarseness, and observational studies with limitations showing improvement in signs of laryngeal inflammation
- Benefit: Improved outcomes, promote resolution of laryngitis
- Harm: Adverse events related to anti-reflux medications
- Cost: Direct cost of medications
- Benefits-harm assessment: Relative balance of benefit and harm
- Value judgments: Although the topic is controversial, the working group acknowledges the potential role of anti-reflux therapy in patients with signs of chronic laryngitis and recognizes that these patients may differ from those with an empiric diagnosis of hoarseness (dysphonia) without laryngeal examination
- Patient preference: Substantial role for shared decision making
- Intentional vagueness: None
- Exclusions: None
- Policy level: Option

STATEMENT 6. CORTICOSTEROID THERAPY: Clinicians should not routinely prescribe oral corticosteroids to treat hoarseness. *Recommendation against prescribing based on randomized trials showing adverse events and absence of clinical trials demonstrating benefits with a preponderance of harm over benefit for steroid use.*

Table 8
Documented side effects of short- and long-term steroid therapy²⁰²⁻²⁰⁷

Lipodystrophy
Hypertension
Cardiovascular disease
Cerebrovascular disease
Osteoporosis
Impaired wound healing
Myopathy
Cataracts
Peptic ulcers
Infection
Mood disorder
Ophthalmologic disorders
Skin disorders
Menstrual disorders
Avascular necrosis
Pancreatitis
Diabetogenesis

Supporting Text

Oral steroids are commonly prescribed for hoarseness and acute laryngitis, despite an overwhelming lack of supporting data of efficacy. A systematic search of MEDLINE, CINAHL, EMBASE, and the Cochrane Library revealed no studies supporting the use of corticosteroids as empiric therapy for hoarseness except in special circumstances as discussed below.

Although hoarseness is often attributed to acute inflammation of the larynx, the temptation to prescribe systemic or inhaled steroids for acute or chronic hoarseness or laryngitis should be avoided because of the potential for significant and serious side effects. Side effects from corticosteroids can occur with short- or long-term use, although the frequency increases with longer durations of therapy (Table 8).²⁰² Additionally, there are many reports implicating long-term inhaled steroid use as a cause of hoarseness.²⁰⁸⁻²¹⁹

Despite these side effects, there are some indications for steroid use in specific disease entities and patients. A specific and accurate diagnosis should be achieved, however, before beginning this therapy. The literature does support steroid use for recurrent croup with associated laryngitis in pediatric patients²²⁰ and allergic laryngitis.^{212,221} Patients with chronic laryngitis and dysphonia may have environmental allergy.²²¹ In limited cases, systemic steroids have been reported to provide quick relief from allergic laryngitis for performers.^{212,221} While these are not high-quality trials, they suggest a possible role for steroids in these selected patient populations. Additionally, in patients acutely dependent on their voice, the balance of benefit and harm may be shifted. The length of treatment for allergy-associated dysphonia with steroids has not been well defined in the literature.

Pediatric patients with croup and other associated symptoms such as hoarseness had better outcomes when treated with systemic steroids.²²⁰ Steroids should also be consid-

ered in patients with airway compromise to decrease edema and inflammation. An appropriate evaluation and determination of the cause of the airway compromise is required prior to starting the steroid therapy. Steroids are also helpful in some autoimmune disorders involving the larynx such as systemic lupus erythematosus, sarcoidosis, and Wegener granulomatosis.^{222,223}

Evidence profile for Statement 6: Corticosteroid Therapy

- Aggregate evidence quality: Grade B, randomized trials showing increased incidence of adverse events associated with orally administered steroids; absence of clinical trials demonstrating any benefit of steroid treatment on outcomes
- Benefit: Avoid potential adverse events associated with unproven therapy
- Harm: None
- Cost: None
- Benefits-harm assessment: Preponderance of harm over benefit for steroid use
- Value judgments: Avoid adverse events of ineffective or unproven therapy
- Role of patient preferences: Some; there is a role for shared decision making in weighing the harms of steroids against the potential yet unproven benefit in specific circumstances (ie, professional or avocation voice use and acute laryngitis)
- Intentional vagueness: Use of the word “routine” to acknowledge there may be specific situations, based on laryngoscopy results or other associated conditions, that may justify steroid use on an individualized basis
- Exclusions: None
- Policy level: Recommendation against

STATEMENT 7. ANTIMICROBIAL THERAPY: Clinicians should not routinely prescribe antibiotics to treat hoarseness. *Strong recommendation against prescribing based on systematic reviews and randomized trials showing ineffectiveness of antibiotic therapy and a preponderance of harm over benefit.*

Supporting Text

Hoarseness in most patients is caused by acute laryngitis or a viral upper respiratory infection, neither of which are bacterial infections. Since antimicrobials are only effective for bacterial infections, their routine, empiric use in treating patients with hoarseness is unwarranted.

Upper respiratory infections often produce symptoms of sore throat and hoarseness, which may alter voice quality and function. Acute upper respiratory infections caused by parainfluenza, rhinovirus, influenza, and adenovirus have been linked to laryngitis.^{224,225} Furthermore, acute laryngitis is self-limited, with patients having improvement in 7 to 10 days undergoing placebo treatment.²²⁶ A Cochrane review examining the role of antibiotics in acute laryngitis in

adults found only two studies meeting the inclusion criteria and no benefit of either penicillin or erythromycin.²²⁷ Similar findings of no benefit for antibiotics in acute upper respiratory tract infections in adults and children were noted in another Cochrane review.²²⁸

The potential harm from antibiotics must also be considered. Common adverse effects include rash, abdominal pain, diarrhea, and vomiting and are more common in patients receiving antibiotics compared to placebo.^{228,229} Interactions may also occur between specific antibiotics and other medications.²³⁰

In addition to negative consequences from antibiotic use on an individual level, important societal implications exist. Over-prescribing antibiotics may contribute to bacterial resistance to antibiotics. Compared to the years 2001 to 2003, more methicillin-resistant *Staphylococcus aureus* has been isolated in acute and chronic maxillary sinusitis in the period 2004 to 2006.²³¹ Furthermore, antibiotic treatment costs for infectious diseases, such as community-acquired pneumonia, were 33 percent higher in communities with high antibiotic resistance rates.²³² Thus, overuse of antibiotics for hoarseness has negative potential results for both the individual and the general population.

While uncommon, antibiotics may be appropriate in select rare causes of hoarseness. Laryngeal tuberculosis in renal transplant patients and in patients with human immunodeficiency virus (HIV) have been reported.^{233,234} An atypical mycobacterial laryngeal infection has also been reported in a patient on inhaled steroids.²³⁵ Although immunosuppression may predispose to a bacterial laryngitis, laryngeal tuberculosis has also been documented in patients without HIV and laryngeal actinomycosis has occurred in an immunocompetent patient.²³⁶⁻²³⁸ A laryngeal mass or ulcer is often present in these infectious etiologies, requiring a high index of suspicion for malignancy. For immunocompromised patients with hoarseness, laryngoscopy is warranted and biopsy for diagnosis should be performed, if indicated.

Antibiotics may also be warranted in patients with hoarseness secondary to other bacterial infections. Recently, community outbreaks of pertussis attributed to waning immunity in adolescents and adults have been reported.²³⁹ Among adults with pertussis, multiple symptoms have been reported including hoarseness in 18 percent.²⁴⁰ Among children, bacterial tracheitis, often from *Staphylococcus aureus*, may be associated with crusting and may cause severe upper airway infection and present with multiple symptoms such as cough, stridor, increased work of breathing, and hoarseness.²⁴¹

Evidence profile for Statement 7: Antimicrobial Therapy

- Aggregate evidence quality: Grade A, systematic reviews showing no benefit for antibiotics for acute laryngitis or upper respiratory tract infection; grade A evidence showing potential harms of antibiotic therapy

- Benefit: Avoidance of ineffective therapy with documented adverse events
- Harm: Potential for failing to treat bacterial, fungal, or mycobacterial causes of hoarseness
- Cost: None
- Benefit-harm assessment: Preponderance of harm over benefit if antibiotics are prescribed
- Values: Importance of limiting antimicrobial therapy to treating bacterial infections
- Role of patient preferences: None
- Intentional vagueness: The word “routine” is used in the boldface statement to discourage empiric therapy yet to acknowledge there are occasional circumstances where antibiotic use may be appropriate
- Exclusions: Patients with hoarseness caused by bacterial infection
- Policy level: Strong recommendation against

STATEMENT 8A. LARYNGOSCOPY PRIOR TO VOICE THERAPY: Clinicians should visualize the larynx before prescribing voice therapy and document/communicate the results to the speech-language pathologist. *Recommendation based on observational studies showing benefit and a preponderance of benefit over harm.*

STATEMENT 8B. ADVOCATING FOR VOICE THERAPY: Clinicians should advocate voice therapy for patients diagnosed with hoarseness (dysphonia) that reduces voice-related QOL. *Strong recommendation based on systematic reviews and randomized trials with a preponderance of benefit over harm.*

Laryngoscopy Prior to Voice Therapy

Voice therapy is a well-established treatment modality for some voice disorders, but therapy should not begin until a diagnosis is made. Failure to visualize the larynx and establish a diagnosis can lead to inappropriate therapy or delay in diagnosis of pathology not amenable to voice therapy.^{127,128} Additionally, the information gained by laryngoscopy may help in designing an optimal therapy regimen.

Evidence-based guidelines from the Royal College of Speech and Language Therapists mandate that a patient be evaluated by an ENT surgeon (otolaryngologist) prior to voice therapy, or simultaneously with the speech-language pathologist (SLP).²⁴² While the guideline does not explicitly refer to laryngoscopy, it states that the “evaluation is needed to identify disease, assess structure and contribute to the assessment of function,” and laryngoscopy is the primary tool for this assessment. The American Speech-Language-Hearing Association (ASHA) acknowledges these guidelines and specifies in their own practice policy that the clinical process for voice evaluation entails that “all patients/clients with voice disorders are examined by a phy-

sician, preferably in a discipline appropriate to the presenting complaint.²⁴³

An SLP trained in visual imaging may examine the larynx for the purpose of evaluating vocal function and planning an appropriate therapy program for the voice disorder. In some practices that care for voice disorders, the SLP works with an otolaryngologist in the multidisciplinary treatment of voice disorders and may perform the examination, which is then reviewed by the otolaryngologist.^{50,244} Examination or review by the otolaryngologist will ensure that diagnoses not treatable with voice therapy, such as laryngeal cancer or papilloma, are managed appropriately. This recommendation is consistent with published guidelines of ASHA.²⁴⁵ There are also published guidelines outlining the knowledge, skills, and training necessary for the use of videostroboscopy by the SLP.²⁴⁶ The guideline panel agreed that performance of stroboscopic evaluation by the SLP with diagnosis by the laryngologist may be time saving in certain settings.

There is significant evidence for the usefulness of laryngoscopy, specifically videostroboscopy, in planning voice therapy and in documenting the effectiveness of voice therapy in the remediation of vocal lesions.^{247,248} Accordingly, the results of the laryngeal examination should be documented and communicated to the SLP who will conduct voice therapy prior to the initiation of medical or surgical treatment. The report should include a detailed diagnosis/description of the laryngeal pathology and brief history of the problem. Visual images of the pathology may also help in treatment planning.²⁴⁸

Advocating for Voice Therapy

Clinicians should advocate voice therapy by making patients aware that this is an effective intervention for hoarseness and providing brochures or sources of further information (see Appendix, “Frequently Asked Questions About Voice Therapy”). The clinician can document advocacy in a chart note by documenting a discussion of speech therapy, by recording educational materials dispensed to the patient, by recording that the patient was supplied with a website, or by documenting referral to an SLP.

Clinicians have several choices for managing hoarseness including observation, medical therapy, surgical therapy, voice therapy, or a combination of these approaches. Voice therapy, provided by a certified SLP, attends to the behavioral issues contributing to hoarseness. Voice therapy is effective for hoarseness across the lifespan from children to older adults.^{8,9,245,249-251} Children younger than two years, however, may not be able to participate fully and effectively in many forms of voice therapy. Education and counseling may be of benefit to the family.

Several approaches to voice therapy for treating hoarseness have been identified in the literature.²⁵²⁻²⁵⁶ Hygienic approaches focus on eliminating behaviors considered to be harmful to the vocal mechanism. Symptomatic approaches target the direct modification of aberrant features of pitch, loudness, and quality. Physiologic methods approach treat-

ment holistically, as they work to retrain and rebalance the subsystems of respiration, phonation, and resonance.

A systematic review of the efficacy literature by Thomas and Stemple revealed various levels of support for the three approaches. The efficacy of physiologic approaches was well supported by randomized and other controlled trials. Hygiene approaches showed mixed results in relatively well-designed controlled trials. Furthermore, mostly observational studies were found supporting symptomatic approaches.²⁴⁹

Hoarseness may be recurring or situational. Recurring hoarseness refers to hoarseness that is intermittent, as might be the case with functional voice disorders (characterized by abnormal voice quality not caused by anatomic changes to the larynx). Situational hoarseness refers to hoarseness that occurs only during certain situations, such as lecturing or singing. Voice therapy is often beneficial when combined with other hoarseness treatment approaches, including preoperative and postoperative therapy, or in combination with certain medical treatments (ie, allergy management, asthma therapy, anti-reflux therapy).^{9,249}

Specific voice therapy for treating hoarseness is effective in Parkinson disease²⁵⁷ and paradoxical vocal fold dysfunction/cough.^{258,259} Voice therapy for treating spasmodic dysphonia is useful as an adjunct to botulinum toxin.²⁶⁰ Voice therapy alone for treating spasmodic dysphonia remains controversial and not well supported.²⁶¹

The interdisciplinary treatment of hoarseness may also include contributions from singing teachers, acting voice coaches, and other medical disciplines in conjunction with voice therapy provided by an SLP.²⁴⁵

Evidence profile for Statement 8A: Visualizing the Larynx

- Aggregate evidence quality: Grade C, observational studies of the benefit of laryngoscopy for voice therapy
- Benefit: Avoid delay in diagnosing laryngeal conditions not treatable with voice therapy, optimize voice therapy by allowing targeted therapy
- Harm: Delay in initiation of voice therapy
- Cost: Cost of the laryngoscopy and associated clinician visit
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: To ensure no delay in identifying pathology not treatable with voice therapy. SLPs cannot initiate therapy prior to visualization of the larynx by a clinician
- Intentional vagueness: None
- Role of patient preferences: Minimal
- Exclusions: None
- Policy level: Recommendation

Evidence profile for Statement 8B: Advocating for Voice Therapy

- Aggregate evidence quality: Grade A, randomized controlled trials and systematic reviews

- Benefit: Improve voice-related QOL; prevent relapse; potentially prevent need for more invasive therapy
- Harm: No harm reported in controlled trials
- Cost: Direct cost of treatment
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: Voice therapy is underutilized in managing hoarseness despite efficacy; advocacy is needed
- Role of patient preferences: Adherence to therapy is essential to outcomes
- Intentional vagueness: Deciding which patients will benefit from voice therapy is often determined by the voice therapist. The guideline panel elected to use a symptom-based criterion to determine to which patients the treating clinician should advocate voice therapy
- Exclusions: None
- Policy level: Strong recommendation

STATEMENT 9. SURGERY: Clinicians should advocate for surgery as a therapeutic option in patients with hoarseness with suspected: 1) laryngeal malignancy, 2) benign laryngeal soft tissue lesions, or 3) glottic insufficiency. *Recommendation based on observational studies demonstrating a benefit of surgery in these conditions and a preponderance of benefit over harm.*

Supporting Text

Clinicians should be aware that surgery may be indicated for certain conditions that cause hoarseness. Surgery is not the primary treatment for the majority of hoarse patients and is targeted at specific pathologies. Conditions with surgical options can be categorized into four broad groups: 1) suspected malignancy, 2) benign soft tissue lesions, 3) glottic insufficiency, and 4) laryngeal dystonia.

Suspected malignancy. Characteristics leading to suspicion of malignancy are described above (see laryngoscopy). Hoarseness may be the presenting sign in malignancy of the upper aerodigestive tract. Malignancy was observed to be the cause of hoarseness in 28 percent of patients over age 60 after patients with self-limited disease were excluded.⁹¹ Surgical biopsy with histopathologic evaluation is necessary to confirm the diagnosis of malignancy in upper airway lesions. Highly suspicious lesions with increased vascularity, ulceration, or exophytic growth require prompt biopsy. A trial of conservative therapy with avoidance of irritants may be employed prior to biopsy for superficial white lesions on otherwise mobile vocal folds.²⁶²

Benign soft tissue lesions. The production of normal voice depends, in part, on intact and functional vocal fold mucosal and submucosal layers. Some benign lesions of the vocal fold mucosa and submucosa result in aberrant vibratory patterns.²⁶² Specific benign lesions of the vocal folds include vocal “singer’s” nodules, polypoid degeneration (Reinke’s edema), hemorrhagic or fibrotic polyps, ectatic or

dilated vessels, scar or sulcus vocalis, cysts (epidermal inclusion and mucous retention), and vocal process granulomas. Another benign lesion, laryngeal stenosis, may not affect the vocal folds directly, but may affect the voice.

A trial of conservative management is typically instituted prior to surgical intervention for most pathologies and may obviate the need for surgery. Many benign soft tissue lesions of the vocal folds are self-limited or reversible.²⁶³ The conservative management strategy indicated depends on the likely underlying etiology but may include voice therapy or rest, smoking cessation, and anti-reflux therapy. In a retrospective study of 26 patients with hoarseness secondary to true vocal fold nodules, 80 percent of patients achieved normal or near-normal voice with voice therapy alone.²⁶⁴ Furthermore, failure to address underlying etiologies may lead to frequent postsurgical recurrence of some lesions, especially granulomas.²⁶⁵ Surgery is reserved for benign vocal fold lesions when a satisfactory voice result cannot be achieved with conservative management and the voice may be improved with surgical intervention.²⁶³

Surgery may improve both subjective voice-related QOL and objective vocal parameters in patients with hoarseness secondary to benign vocal fold lesions. A retrospective review of 42 patients with benign vocal fold lesions demonstrated significant improvement in voice-related QOL and acoustic parameters following surgery.²⁶⁶ Multiple studies of surgical treatment of ectatic vessels, polypoid degeneration (Reinke’s edema), nodules, and polyps all showed significant benefit.²⁶⁷⁻²⁶⁹

Surgery is necessary in the management of recurrent respiratory papilloma (RRP), a benign but aggressive neoplasm of the upper airway more commonly seen in children. Human papillomavirus subtypes 6 and 11 are the most common cause. Surgical removal with standard laryngeal instruments, microdebrider, or laser can prevent airway obstruction and is effective in reducing the symptoms of hoarseness, but it is unlikely to be curative since viral particles may be present in adjacent normal-appearing mucosa.²⁷⁰⁻²⁷² Additionally, certain lesions may be amenable to treatment in the office under topical anesthesia using advanced laryngoscopic techniques.²⁶⁷

Type of instrumentation does not seem to affect outcome when comparing laser to cold dissection.²⁷³ The surgical method used is less important than the experience and skill of the operating surgeon in obtaining satisfactory vocal outcomes in the surgical treatment of benign vocal fold lesions.²⁶⁶ While bleeding, scarring, airway compromise, and poor voice outcomes are all possible risks of surgery, no serious surgery-related complications were noted in any case series or trial.^{266,273}

Glottic insufficiency. A normal voice is created by two mobile vocal folds making contact in the midline space of the larynx (glottis), thereby creating the vibratory sound waves perceived as voice. Glottic insufficiency due to vocal fold weakness (eg, paralysis or paresis) or vocal fold soft tissue

defects often results in a weak, breathy hoarseness with poor cough and reduced airway protection during swallow. Details of characteristics leading to suspicion of glottic insufficiency are described above (see laryngoscopy section). Glottic insufficiency is especially common in older adults, in whom up to 30 percent of hoarseness was due to vocal fold changes after self-limited causes were excluded.^{91,92}

Surgical management of glottic insufficiency is primarily through static positioning of the weak vocal fold in the midline glottis (medialization laryngoplasty). Static medialization of the vocal folds can be achieved either by injection of a bulking agent into the vocal fold (injection laryngoplasty) or external medialization with open surgery (laryngeal framework surgery), or a combination of the two. Injection laryngoplasty can be safely performed in the office under local anesthesia or in the operating room under general anesthesia.²⁷⁴ While no randomized trials were found directly comparing injection laryngoplasty to laryngeal framework surgery, observational studies show comparable objective and subjective improvement in voice.²⁷⁵

Resorbable, temporary injectable implants are often used to provide vocal rehabilitation while allowing time for neural recovery or full denervation atrophy of the vocal musculature prior to permanent medialization. In a randomized controlled trial of patients with glottic insufficiency comparing bovine collagen to hyaluronic acid gel, 42 patients with sufficient follow-up demonstrated significantly improved subjective and objective vocal parameters.²⁷⁶ There were no complications noted in this study, but 26 percent of patients required repeat injection over 24 months of observation. Additional retrospective series of temporary injectables demonstrated subjective and objective hoarseness reduction in 80 percent to 95 percent of treated patients.²⁷⁷⁻²⁸⁰ In addition, there are limited data that collagen or lyophilized dermis injections can provide adequate vocal rehabilitation of pediatric patients.²⁸¹

Injection laryngoplasty with stable, semi-permanent implants is used when vocal recovery is unlikely.²⁷⁴ Prospective trials of both silicone and hydroxylapatite paste have demonstrated significant improvement in validated voice QOL measures in 94 percent to 100 percent of patients without significant complications after six-month follow-up.^{282,283} Since there are several suitable alternatives, the use of polytetrafluoroethylene as a permanent injectable implant is not recommended due to its association with foreign body granulomas that can result in voice deterioration and airway compromise.^{284,285}

External medialization laryngoplasty by open laryngeal framework surgery, also known as type I thyroplasty, has demonstrated hoarseness reduction using a variety of implants made of Silastic, titanium, Gore-tex, and hydroxylapatite.²⁸⁶⁻²⁸⁸ When analyzed by trained, blinded listeners, the voices of 15 patients who underwent external laryngoplasty were indistinguishable from normal controls in loudness and pitch but had higher levels of strain and breathiness.²⁸⁹ In a retrospective study of 117 patients with glottic

insufficiency, patients who received external laryngoplasty demonstrated better symptom resolution compared to patients receiving voice therapy alone.²⁹⁰

Arytenoid adduction is an additional laryngeal framework procedure used to rotate the vocal process of the arytenoid medially in patients with large posterior glottic gaps. A meta-analysis of three studies found no clear benefit if arytenoid adduction is added to external laryngoplasty compared to external laryngoplasty alone.²⁹¹ External laryngoplasty has been performed successfully in children but may be technically more challenging due to the variable position of the pediatric vocal fold.^{292,293}

Laryngeal dystonia. Surgical treatment for laryngeal dystonia, or adductor spasmodic dysphonia, is infrequently performed due to the widespread acceptance of botulinum toxin as the first-line treatment for this disorder. Attempts to control the disorder with recurrent laryngeal nerve section resulted in inconsistent, often temporary improvement, with recurrence in up to 80 percent of cases.²⁹⁴⁻²⁹⁷ A single, retrospective study of laryngeal dystonia patients treated with bilateral division of the adductor branch of the recurrent laryngeal nerve followed by ansa cervicalis reinnervation demonstrated resolution of symptoms in 19 of 21 patients followed for at least 12 months.²⁹⁸

Evidence profile for Statement 9: Surgery

- Aggregate evidence quality: Grade B, in support of surgery to reduce hoarseness and improve voice quality in selected patients based on observational studies overwhelmingly demonstrating the benefit of surgery
- Benefit: Potential for improved voice outcomes in carefully selected patients
- Harm: None
- Cost: None
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: Surgical options for treating hoarseness are not always recognized; selected patients with hoarseness may benefit from newer, less invasive technologies
- Role of patient preferences: Limited
- Intentional vagueness: None
- Exclusions: None
- Policy level: Recommendation

STATEMENT 10. BOTULINUM TOXIN: Clinicians should prescribe, or refer the patient to a clinician who can prescribe, botulinum toxin injections for the treatment of hoarseness caused by spasmodic dysphonia. *Recommendation based on randomized controlled trials with minor limitations and preponderance of benefit over harm.*

Supporting Text

Spasmodic dysphonia (SD) is a focal dystonia most commonly characterized by a strained, strangled voice.²⁹⁹ Pa-

tients demonstrate increased tone or tremor of intralaryngeal muscle groups responsible for either opening (abductor SD) or closing (adductor SD) of the vocal folds. Intramuscular injection of botulinum toxin into the affected muscles causes transient, nondestructive flaccid paralysis of these muscles by inhibiting the release of acetylcholine from nerve terminals, thus reducing the spasm.³⁰⁰ SD is a disorder of the central nervous system that cannot be cured by botulinum toxin,³⁰¹ but excellent symptom control is possible for 3 to 6 months with treatment.³⁰² Treatment can be performed on awake, ambulatory patients with minimal discomfort.³⁰³

While not currently FDA approved for SD, a large body of evidence supports the efficacy of botulinum toxin (primarily botulinum toxin A) for treating adductor spasmodic dysphonia. Multiple double-blind, randomized, placebo-controlled trials of botulinum toxin for adductor spasmodic dysphonia using both self-assessment and expert listeners found improved voice in patients treated with botulinum toxin injections.^{304,305} Botulinum toxin treatment has also been shown to improve self-perceived dysphonia, mental health, and social functioning.³⁰⁶ A meta-analysis concluded that botulinum toxin treatment of spasmodic dysphonia results in “moderate overall improvement;” however, it notes concerns of methodological limitations and lack of standardization in assessment of botulinum toxin efficacy and recommends caution when making inferences regarding treatment benefit.²⁶⁰ Despite these limitations, among laryngologists, botulinum toxin is considered the “treatment of choice” for adductor SD.^{301,302,307}

Botulinum toxin has been used for other disorders of excessive or inappropriate muscular contraction.³⁰⁰ There are limited reports addressing the use of botulinum toxin for spastic dysarthria, nerve-section failure, anterior commissure release, adductor breathing dystonia, abductor spasmodic dysphonia, ventricular dysphonia (also called dysphonia plica ventricularis), and voice tremor.^{280,281,289-293}

Botulinum toxin injections have a good safety record. Blitzer et al reported their 13-year experience in 901 patients who underwent 6300 injections; adverse effects included “mild breathiness and coughing on fluids” in the adductor SD patients, and “mild stridor” in abductor SD patients.³⁰⁸ The most common adverse effects of botulinum toxin injection are breathiness and dysphagia, including choking on fluids.³⁰⁹⁻³¹³ Risk of harm may be greater with inexperienced users.³⁰¹ Post-treatment dysphagia appears more common in patients with dysphagia prior to injection.³¹⁴ Exertional wheezing, exercise intolerance, and stridor were reported more commonly in patients with abductor SD.^{308,315}

Adverse events may result from diffusion of drug from the target muscle to adjacent muscles (this has been added as a “boxed warning” by the FDA).³⁰⁰ Adjusting the dose, distribution, and timing of injections may decrease the frequency of adverse events.^{313,316} Bleeding is rare, and vocal fold edema has only been documented in a single patient

receiving saline as a placebo.³⁰⁴ Reports of sensations of burning, tickling, irritation of the larynx or throat, excessive thick secretions, and dryness have also occurred.³¹⁷ Systemic effects are rare, with only two reports of generalized botulism-like syndromes and one report of possible precipitation of biliary colic.³⁰⁰ Acquired resistance to botulinum toxin can occur.^{300,318}

Evidence profile for Statement 10: Botulinum Toxin

- Aggregate evidence quality: Grade B, few controlled trials, diagnostic studies with minor limitations, and overwhelmingly consistent evidence from observational studies
- Benefit: Improved voice quality and voice-related QOL
- Harm: Risk of aspiration and airway obstruction
- Cost: Direct costs of treatment, time off work, and indirect costs of repeated treatments
- Benefit-harm assessment: Preponderance of benefit over harm
- Value judgments: Botulinum toxin is beneficial despite the potential need for repeated treatments considering the lack of other effective interventions for spasmodic dysphonia
- Role of patient preferences: Patient must be comfortable with FDA off-label use of botulinum toxin. While strong evidence supports its use, botulinum toxin injection is an invasive therapy offering only temporarily relief of a non-life-threatening condition. Patients may reasonably elect not to have it performed
- Intentional vagueness: None
- Exclusions: None
- Policy level: Recommendation

STATEMENT 11. PREVENTION: Clinicians may educate/counsel patients with hoarseness about control/preventive measures. *Option based on observational studies and small randomized trials of poor quality.*

Supporting Text

The risk of hoarseness may be diminished by preventive measures such as hydration, avoidance of irritants, voice training, and amplification. Currently available studies evaluating these measures are limited in scope and quality. There is some evidence that adequate hydration may decrease the risk of hoarseness. In a study of 422 teachers, absence of water intake was associated with a 60 percent higher risk of hoarseness.³¹⁹ Objective findings of hoarseness and vocal fold thickness were found in patients with post-dialysis dehydration.³²⁰ An observational study of amateur singers demonstrated less vocal fatigue with hydration and periods of voice rest.³²¹ Phonatory effort may also be decreased by adequate hydration.⁵⁷ There are very limited data suggesting that amplification during heavy voice use may sustain voice quality.³²²

A 2007 Cochrane review evaluated the effectiveness of interventions designed to prevent or reduce voice disorder

ders.³²³ Only two studies were of adequate quality to meet inclusion criteria. Direct voice training, indirect voice training, or a combination of the two approaches were studied in 55 student teachers³²⁴ and 41 kindergarten and primary school teachers.³²⁵ The review did not find sufficient evidence to substantiate the use of voice training as a preventive measure. The two randomized controlled studies included in the review had several methodological problems related to sample size, design, and outcome measures.

Despite limited evidence in the literature, the panel concurred that avoidance of tobacco smoke (primary or secondhand) was beneficial to decrease the risk of hoarseness.³²⁶ There is also observational evidence from a single study of 10 symptomatic rescue workers at the World Trade Center disaster site that irritants such as chemicals, smoke particulates, and pollution can increase the likelihood of developing hoarseness.³²⁷

Evidence profile for Statement 11: Prevention

- Aggregate evidence quality: Grade C, evidence based on several observational studies and a few small randomized trials of poor quality
- Benefit: Possible prevention of hoarseness in high-risk persons
- Harm: None
- Cost: Cost of vocal training sessions
- Benefits-harm assessment: Preponderance of benefit over harm
- Value judgments: Preventive measures may prevent hoarseness
- Role of patient preferences: Patients without symptoms must weigh the benefit of preventive measures based on their risk of developing hoarseness or voice problems
- Intentional vagueness: None
- Exclusions: None
- Policy level: Option

IMPLEMENTATION CONSIDERATIONS

The complete guideline is published as a supplement to *Otolaryngology–Head and Neck Surgery* to facilitate reference and distribution. The guideline will be presented to AAO-HNS members as a mini-seminar at the AAO-HNS annual meeting following publication. Existing brochures and publications by the AAO-HNS will be updated to reflect the guideline recommendations. A full-text version of the guideline will also be accessible free of charge at www.entnet.org.

An anticipated barrier to diagnosis is distinguishing modifying factors for hoarseness in a busy clinical setting. This may be assisted by a laminated teaching card or visual aid summarizing important factors that modify management.

Laryngoscopy is an option at any time for patients with hoarseness, but the guideline also recommends that no pa-

tient should be allowed to wait longer than three months prior to having his or her larynx examined. It is also clearly recommended that if there is a concern of an underlying serious condition, then laryngoscopy should be immediate. Tables in this guideline regarding causes for concern should help to guide clinicians regarding when more prompt laryngoscopy is warranted. The cost of the laryngoscopy and possible wait times to see clinicians trained in the technique may hinder access to care.

While the guideline acknowledges that there may be a significant role for anti-reflux therapy to treat laryngeal inflammation, empiric use of anti-reflux medications for hoarseness has minimal support and a growing list of potential risks. Avoidance of empiric use of anti-reflux therapy represents a significant change in practice for some clinicians. Educational pamphlets about the unfavorable risk-benefit profile of these medications in the absence of GERD symptoms or signs of laryngeal inflammation in the face of newly recognized complications of long-term use of proton pump inhibitors may facilitate acceptance of this shift.

Lack of knowledge about voice therapy by practitioners is a likely barrier to advocacy for its use. This barrier can be overcome by educational materials about voice therapy and its indications.

RESEARCH NEEDS

While there is a body of literature from which these guidelines were drawn, significant gaps in our knowledge about hoarseness and its management remain. The guideline committee identified several areas where further research would improve the ability of clinicians to manage hoarse patients optimally.

Hoarseness is known to be common, but the prevalence of hoarseness in certain populations such as children is not well known. Additionally, the prevalence of specific etiologies of hoarseness is not known. Descriptive statistics would help to shape thinking on distribution of resources, levels of care, and cost mandates.

Although a strong intuitive sense of the natural history of many voice disorders exists among practitioners, data are lacking. This dearth of information makes judgments related to the value of observation vs intervention challenging. Some of the entities that might benefit from study include viral laryngitis, fungal laryngitis, inhaler-related laryngitis, voice abuse, reflux, and benign lesions (ie, nodules, polyps, cysts, etc). A better understanding of the natural history of these disorders could be obtained through prospective observational studies and will have clear implications for the necessity and timing of behavioral, medical, and surgical interventions.

Prospective studies on the value of steroids and antibiotics for infectious laryngitis are also lacking. Given the known potential harms from these medications, prospective studies examining the benefits relative to placebo are warranted.

Reflux laryngitis is a very common diagnosis with much controversy surrounding it. While there are a number of studies looking at the use of anti-reflux therapy for chronic laryngitis, the vast majority have severe limitations. Well-conducted and controlled studies of anti-reflux therapy for patients with hoarseness and for patients with signs of laryngeal inflammation would help to establish the value of these medications. Further clarification of which hoarse patients may benefit from reflux treatment would help to optimize outcomes and minimize costs and potential side effects. Future studies may benefit from strict inclusion criteria and specific investigation of the outcome of hoarseness (dysphonia) control.

Although ancillary testing such as radiographic imaging is often performed to assist in diagnosing the underlying cause of hoarseness, the role of these tests has not been clearly defined. Their usefulness as screening tools is unclear and the cost effectiveness of their use has not been established.

Despite data that strongly demonstrate better survival and local control rates in early-stage laryngeal cancers, the improvement of laryngeal cancer outcomes through early screening has not been shown. Study of the effect of early screening and diagnosis is warranted.

Voice therapy has been shown to provide short-term benefit for hoarse patients, but long-term efficacy has not been shown. Also, the relative harm of voice therapy has not been studied (eg, lost work time, anxiety), making the risk/benefit ratio difficult to evaluate.

As office-based procedures are developed to manage causes of hoarseness previously treated in the operating room, comparative studies on the safety and efficacy of office-based procedures relative to those performed under general anesthesia are needed (eg, injection vs open thyroplasty).

DISCLAIMER

As medical knowledge expands and technology advances, clinical indicators and guidelines are promoted as conditional and provisional proposals of what is recommended under specific conditions, but they are not absolute. Guidelines are not mandates and do not and should not purport to be a legal standard of care. The responsible physician, in light of all the circumstances presented by the individual patient, must determine the appropriate treatment. Adherence to these guidelines will not ensure successful patient outcomes in every situation. The American Academy of Otolaryngology—Head and Neck Surgery (AAO-HNS) emphasizes that these clinical guidelines should not be deemed to include all proper treatment decisions or methods of care, or to exclude other treatment decisions or methods of care reasonably directed to obtaining the same results.

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DISCLOSURES

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42. Sneeuw KC, Sprangers MA, Aaronson NK. The role of health care providers and significant others in evaluating the quality of life of patients with chronic disease. *J Clin Epidemiol* 2002;55:1130–43.
43. Hackett ML, Duncan JR, Anderson CS, et al. Health-related quality of life among long-term survivors of stroke: results from the Auckland Stroke Study, 1991–1992. *Stroke* 2000;31:440–7.
44. Hogikyan ND, Sethuraman G. Validation of an instrument to measure voice-related quality of life (V-RQOL). *J Voice* 1999;13:557–69.
45. Jacobson BH, Johnson A, Grywalski C, et al. The Voice Handicap Index (VHI): development and validation. *Am J Speech Lang Pathol* 1997;6:66–70.
46. Deary IJ, Wilson JA, Carding PN, et al. VoiSS: a patient-derived voice symptom scale. *J Psychosom Res* 2003;54:483–9.
47. Zraick RI, Risner BY, Smith-Olinde L, et al. Patient versus partner perception of voice handicap. *J Voice* 2007;21:485–94.
48. Sataloff RT, Divi V, Heman-Ackah YD, et al. Medical history in voice professionals. *Otolaryngol Clin North Am* 2007;40:931–51.
49. Sataloff RT. Office evaluation of dysphonia. *Otolaryngol Clin North Am* 1992;25:843–55.
50. Rubin JS, Sataloff RT, Korovin GS. Diagnosis and treatment of voice disorders. 3rd ed. San Diego: Plural Publishing, Inc.; 2006. p. 824.
51. Kerr HD, Kwaselew A. Vocal cord hematomas complicating anticoagulant therapy. *Ann Emerg Med* 1984;13:552–3.
52. Laing C, Kelly J, Coman S, et al. Vocal cord haematoma after thrombolysis. *Lancet* 1997;350:1677.
53. Neely JL, Rosen C. Vocal fold hemorrhage associated with coumadin therapy in an opera singer. *J Voice* 2000;14:272–7.
54. Bhutta MF, Rance M, Gillett D, et al. Alendronate-induced chemical laryngitis. *J Laryngol Otol* 2005;119:46–7.
55. Dicipinigitis PV. Angiotensin-converting enzyme inhibitor-induced cough: ACCP evidence-based clinical practice guidelines. *Chest* 2006;129:169S–73S.
56. Abaza MM, Levy S, Hawkshaw MJ, et al. Effects of medications on the voice. *Otolaryngol Clin North Am* 2007;40:1081–90.
57. Verdolini K, Titze IR, Fennell A. Dependence of phonatory effort on hydration level. *J Speech Hear Res* 1994;37:1001–7.
58. Baker J. A report on alterations to the speaking and singing voices of four women following hormonal therapy with virilizing agents. *J Voice* 1999;13:496–507.
59. Pattie MA, Murdoch BE, Theodoros D, et al. Voice changes in women treated for endometriosis and related conditions: the need for comprehensive vocal assessment. *J Voice* 1998;12:366–71.
60. Christodoulou C, Kalaitzi C. Antipsychotic drug-induced acute laryngeal dystonia: two case reports and a mini review. *J Psychopharmacol* 2005;19:307–11.
61. Tsai CS, Lee Y, Chang YY, et al. Ziprasidone-induced tardive laryngeal dystonia: a case report. *Gen Hosp Psychiatry* 2008;30:277–9.
62. Adams NP, Bestall JC, Lasserson TJ, Jones P, Cates CJ. Fluticasone versus placebo for chronic asthma in adults and children. *Cochrane Database of Systematic Reviews* 2008, Issue 4. Art. No.: CD003135. DOI: 10.1002/14651858.CD003135.pub4.
63. Kahraman S, Sirin S, Erdogan E, et al. Is dysphonia permanent or temporary after anterior cervical approach? *Eur Spine J* 2007;16:2092–5.
64. Beutler WJ, Sweeney CA, Connolly PJ. Recurrent laryngeal nerve injury with anterior cervical spine surgery risk with laterality of surgical approach. *Spine* 2001;26:1337–42.
65. Baron EM, Soliman AM, Gaughan JP, et al. Dysphagia, hoarseness, and unilateral true vocal fold motion impairment following anterior cervical discectomy and fusion. *Ann Otol Rhinol Laryngol* 2003;112:921–6.
66. Jung A, Schramm J, Lehnerdt K, et al. Recurrent laryngeal nerve palsy during anterior cervical spine surgery: a prospective study. *J Neurosurg Spine* 2005;2:123–7.
67. Winslow CP, Winslow TJ, Wax MK. Dysphonia and dysphagia following the anterior approach to the cervical spine. *Arch Otolaryngol Head Neck Surg* 2001;127:51–5.
68. Tervonen H, Niemelä M, Lauri ER, et al. Dysphonia and dysphagia after anterior cervical decompression. *J Neurosurg Spine* 2007;7:124–30.
69. Yue WM, Brodner W, Highland TR. Persistent swallowing and voice problems after anterior cervical discectomy and fusion with allograft and plating: a 5- to 11-year follow-up study. *Eur Spine J* 2005;14:677–82.
70. Yeung P, Erskine C, Mathews P, et al. Voice changes and thyroid surgery: is pre-operative indirect laryngoscopy necessary? *Aust N Z J Surg* 1999;69:632–4.
71. Moulton-Barrett R, Crumley R, Jalilie S, et al. Complications of thyroid surgery. *Int Surg* 1997;82:63–6.
72. Bellantone R, Boscherini M, Lombardi CP, et al. Is the identification of the external branch of the superior laryngeal nerve mandatory in thyroid operation? Results of a prospective randomized study. *Surgery* 2001;130:1055–9.
73. Zannetti S, Parente B, De Rango P, et al. Role of surgical techniques and operative findings in cranial and cervical nerve injuries during carotid endarterectomy. *Eur J Vasc Endovasc Surg* 1998;15:528–31.
74. Maniglia AJ, Han DP. Cranial nerve injuries following carotid endarterectomy: an analysis of 336 procedures. *Head Neck* 1991;13:121–4.
75. Espinoza FI, MacGregor FB, Doughty JC, et al. Vocal fold paralysis following carotid endarterectomy. *J Laryngol Otol* 1999;113:439–41.
76. Schindler A, Favero E, Nudo S, et al. Voice after supracricoid laryngectomy: subjective, objective and self-assessment data. *Logoped Phoniatr Vocol* 2005;30:114–9.
77. Holst M, Hertegård S, Persson A. Vocal dysfunction following cricothyroidotomy: a prospective study. *Laryngoscope* 1990;100:749–55.
78. Inada T, Fujise K, Shingu K. Hoarseness after cardiac surgery. *J Cardiovasc Surg (Torino)* 1998;39:455–9.
79. Kamalipour H, Mowla A, Saadi MH, et al. Determination of the incidence and severity of hoarseness after cardiac surgery. *Med Sci Monit* 2006;12:CR206–9.
80. Hamdan AL, Moukarbel RV, Farhat F, et al. Vocal cord paralysis after open-heart surgery. *Eur J Cardiothorac Surg* 2002;21:671–4.
81. Baba M, Natsugoe S, Shimada M, et al. Does hoarseness of voice from recurrent nerve paralysis after esophagectomy for carcinoma influence patient quality of life? *J Am Coll Surg* 1999;188:231–6.
82. Morris GL III, Mueller WM. Long-term treatment with vagus nerve stimulation in patients with refractory epilepsy. The Vagus Nerve Stimulation Study Group E01-E05. *Neurology* 1999;53:1731–5.
83. Colice GL, Stukel TA, Dain B. Laryngeal complications of prolonged intubation. *Chest* 1989;96:877–84.
84. Santos PM, Afrassiabi A, Weymuller EA Jr. Risk factors associated with prolonged intubation and laryngeal injury. *Otolaryngol Head Neck Surg* 1994;111:453–9.
85. Bastian RW, Richardson BE. Postintubation phonatory insufficiency: an elusive diagnosis. *Otolaryngol Head Neck Surg* 2001;124:625–33.
86. Jones MW, Catling S, Evans E, et al. Hoarseness after tracheal intubation. *Anaesthesia* 1992;47:213–6.
87. Zimmert M, Zwirner P, Kruse E, et al. Effects on vocal function and incidence of laryngeal disorder when using a laryngeal mask airway in comparison with an endotracheal tube. *Eur J Anaesthesiol* 1999;16:511–5.
88. Hengerer AS, Strome M, Jaffe BF. Injuries to the neonatal larynx from long-term endotracheal tube intubation and suggested tube modification for prevention. *Ann Otol Rhinol Laryngol* 1975;84:764–70.
89. Hagen P, Lyons GD, Nuss DW. Dysphonia in the elderly: diagnosis and management of age-related voice changes. *South Med J* 1996;89:204–7.
90. Kosztyła-Hojna B, Rogowski M, Pepiński W. The evaluation of voice in elderly patients. *Acta Otorhinolaryngol Belg* 2003;57:107–12.

91. Kandoğan T, Olgun L, Gültekin G. Causes of dysphonia in patients above 60 years of age. *Kulak Burun Bogaz Ihtis Derg* 2003;11:139–43.
92. Lundy DS, Silva C, Casiano RR, et al. Cause of hoarseness in elderly patients. *Otolaryngol Head Neck Surg* 1998;118:481–5.
93. Hartman DE. Neurogenic dysphonia. *Ann Otol Rhinol Laryngol* 1984;93:57–64.
94. Sewall GK, Jiang J, Ford CN. Clinical evaluation of Parkinson's-related dysphonia. *Laryngoscope* 2006;116:1740–4.
95. Feijó AV, Parente MA, Behlau M, et al. Acoustic analysis of voice in multiple sclerosis patients. *J Voice* 2004;18:341–7.
96. Connor NP, Cohen SB, Theis SM, et al. Attitudes of children with dysphonia. *J Voice* 2008;22:197–209.
97. Sederholm E, McAllister A, Dalkvist J, et al. Aetiologic factors associated with hoarseness in ten-year-old children. *Folia Phoniatr Logop* 1995;47:262–78.
98. De Bodd MS, Ketelslagers K, Peeters T, et al. Evolution of vocal fold nodules from childhood to adolescence. *J Voice* 2007;21:151–6.
99. Hocevar-Boltezar I, Jarc A, Kozelj V. Ear, nose and voice problems in children with orofacial clefts. *J Laryngol Otol* 2006;120:276–81.
100. Hirschberg J. Dysphonia in infants. *Int J Pediatr Otorhinolaryngol* 1999;49:S293–6.
101. Shankargouda S, Krishnan U, Murali R, et al. Dysphonia: a frequently encountered symptom in the evaluation of infants with unobstructed supracardiac total anomalous pulmonary venous connection. *Pediatr Cardiol* 2000;21:458–60.
102. Matsuo K, Kamimura M, Hirano M. Polypoid vocal folds. A 10-year review of 191 patients. *Auris Nasus Larynx* 1983;10:S37–45.
103. Tombolini V, Zurlo A, Cavaceppi P, et al. Radiotherapy for T1 carcinoma of the glottis. *Tumori* 1995;81:414–8.
104. Franchin G, Minatel E, Gobitti C, et al. Radiotherapy for patients with early-stage glottic carcinoma: univariate and multivariate analyses in a group of consecutive, unselected patients. *Cancer* 2003;98:765–72.
105. Bernstein IL, Chervinsky P, Falliers CJ. Efficacy and safety of triamcinolone acetonide aerosol in chronic asthma. Results of a multicenter, short-term controlled and long-term open study. *Chest* 1982;81:20–6.
106. Musholt TJ, Musholt PB, Garm J, et al. Changes of the speaking and singing voice after thyroid or parathyroid surgery. *Surgery* 2006;140:978–88.
107. Postma GN, Courey MS, Ossoff RH. Microvascular lesions of the true vocal fold. *Ann Otol Rhinol Laryngol* 1998;107:472–6.
108. Preciado-López J, Pérez-Fernández C, Calzada-Uriondo M, et al. Epidemiological study of voice disorders among teaching professionals of La Rioja, Spain. *J Voice* 2008;22:489–508.
109. Mace SE. Blunt laryngotracheal trauma. *Ann Emerg Med* 1986;15:836–42.
110. Schaefer SD. The acute management of external laryngeal trauma. A 27-year experience. *Arch Otolaryngol Head Neck Surg* 1992;118:598–604.
111. Resouly A, Hope A, Thomas S. A rapid access husky voice clinic: useful in diagnosing laryngeal pathology. *J Laryngol Otol* 2001;115:978–80.
112. Johnson JT, Newman RK, Olson JE. Persistent hoarseness: an aggressive approach for early detection of laryngeal cancer. *Postgrad Med* 1980;67:122–6.
113. Ishizuka T, Hisada T, Aoki H, et al. Gender and age risks for hoarseness and dysphonia with use of a dry powder fluticasone propionate inhaler in asthma. *Allergy Asthma Proc* 2007;28:550–6.
114. Hartl DA, Hans S, Vaissière J, et al. Objective acoustic and aerodynamic measures of breathiness in paralytic dysphonia. *Eur Arch Otorhinolaryngol* 2003;260:175–82.
115. Mao VH, Abaza M, Spiegel JR, et al. Laryngeal myasthenia gravis: report of 40 cases. *J Voice* 2001;15:122–30.
116. Belafsky PC, Rees CJ. Laryngopharyngeal reflux: the value of otolaryngology examination. *Curr Gastroenterol Rep* 2008;10:278–82.
117. Ludlow CL, Adler CH, Berke GS, et al. Research priorities in spasmodic dysphonia. *Otolaryngol Head Neck Surg* 2008;139:495–505.
118. de Jong AL, Kuppersmith RB, Sulek M, et al. Vocal cord paralysis in infants and children. *Otolaryngol Clin North Am* 2000;33:131–49.
119. Nicollas R, Triglia JM. The anterior laryngeal webs. *Otolaryngol Clin North Am* 2008;41:877–88, viii.
120. Thompson DM. Abnormal sensorimotor integrative function of the larynx in congenital laryngomalacia: a new theory of etiology. *Laryngoscope* 2007;117:1–33.
121. Faust RA. Childhood voice disorders: ambulatory evaluation and operative diagnosis. *Clin Pediatr* 2003;42:1–9.
122. Rehberg E, Kleinsasser O. Malignant transformation in non-irradiated juvenile laryngeal papillomatosis. *Eur Arch Otorhinolaryngol* 1999;256:450–4.
123. Portier F, Marianowski R, Morisseau-Durand MP, et al. Respiratory obstruction as a sign of brainstem dysfunction in infants with Chiari malformations. *Int J Pediatr Otorhinolaryngol* 2001;57:195–202.
124. Truong MT, Messner AH, Kerschner JE, et al. Pediatric vocal fold paralysis after cardiac surgery: rate of recovery and sequelae. *Otolaryngol Head Neck Surg* 2007;137:780–4.
125. Dworkin JP. Laryngitis: types, causes, and treatments. *Otolaryngol Clin North Am* 2008;41:419–36, ix.
126. Reveiz L, Cardona Zorrilla AF, Ospina EG. Antibiotics for acute laryngitis in adults. *Cochrane Database of Systematic Reviews* 2007, Issue 2. Art. No.: CD004783. DOI: 10.1002/14651858.CD004783.pub3.
127. Teppo H, Alho OP. Comorbidity and diagnostic delay in cancer of the larynx, tongue and pharynx. *Oral Oncol* 2008 Dec 16. [Epub ahead of print].
128. Carvalho AL, Pintos J, Schlecht NF, et al. Predictive factors for diagnosis of advanced-stage squamous cell carcinoma of the head and neck. *Arch Otolaryngol Head Neck Surg* 2002;128:313–8.
129. Dailey SH, Spanou K, Zeitels SM. The evaluation of benign glottic lesions: rigid telescopic stroboscopy versus suspension microlaryngoscopy. *J Voice* 2007;21:112–8.
130. Patel R, Dailey S, Bless D. Comparison of high-speed digital imaging with stroboscopy for laryngeal imaging of glottal disorders. *Ann Otol Rhinol Laryngol* 2008;117:413–24.
131. Sataloff RT, Spiegel JR, Hawkshaw MJ. Stroboscoped laryngoscopy: results and clinical value. *Ann Otol Rhinol Laryngol* 1991;100:725–7.
132. Shohet JA, Courey MS, Scott MA, et al. Value of videostroboscopic parameters in differentiating true vocal fold cysts from polyps. *Laryngoscope* 1996;106:19–26.
133. Kleinsasser O. *Microlaryngoscopy and endolaryngeal microsurgery*. Philadelphia: W.B. Saunders; 1968. p. 48–62.
134. Lacoste L, Karayan J, Lehuédé MS, et al. A comparison of direct, indirect, and fiberoptic laryngoscopy to evaluate vocal cord paralysis after thyroid surgery. *Thyroid* 1996;6:17–21.
135. Armstrong M, Mark LJ, Snyder DS, et al. Safety of direct laryngoscopy as an outpatient procedure. *Laryngoscope* 1997;107:1060–5.
136. Hill RS, Koltai PJ, Parnes SM. Airway complications from laryngoscopy and panendoscopy. *Ann Otol Rhinol Laryngol* 1987;96:691–4.
137. Rosen CA, Andrade Filho PA, Scheffel L, et al. Oropharyngeal complications of suspension laryngoscopy: a prospective study. *Laryngoscope* 2005;115:1681–4.
138. Bové MJ, Jabbour N, Krishna P, et al. Operating room versus office-based injection laryngoplasty: a comparative analysis of reimbursement. *Laryngoscope* 2007;117:226–30.
139. Andrade Filho PA, Carrau RL, Buckmire RA. Safety and cost-effectiveness of intra-office flexible videolaryngoscopy with transoral vocal fold injection in dysphagic patients. *Am J Otolaryngol* 2006;27:319–22.
140. Rees CJ, Postma GN, Koufman JA. Cost savings of unsedated office-based laser surgery for laryngeal papillomas. *Ann Otol Rhinol Laryngol* 2007;116:45–8.
141. Brenner DJ, Hall EJ. Computed tomography—an increasing source of radiation exposure. *N Engl J Med* 2007;357:2277–84.

142. Brenner D, Elliston C, Hall E, et al. Estimated risks of radiation-induced fatal cancer from pediatric CT. *AJR Am J Roentgenol* 2001;176:289–96.
143. Rice HE, Frush DP, Farmer D, et al. Review of radiation risks from computed tomography: essentials for the pediatric surgeon. *J Pediatr Surg* 2007;42:603–7.
144. Berrington de Gonzalez A, Darby S. Risk of cancer from diagnostic X-rays: estimates for the UK and 14 other countries. *Lancet* 2004;363:345–51.
145. Sources and effects of ionizing radiation: United Nations Scientific Committee on the Effects of Atomic Radiation UNSCEAR 2000 report to the General Assembly. New York: United Nations; 2000.
146. Wang CL, Cohan RH, Ellis JH, et al. Frequency, outcome, and appropriateness of treatment of nonionic iodinated contrast media reactions. *Am J Roentgenol* 2008;191:409–15.
147. Mortelé KJ, Oliva MR, Ondategui S, et al. Universal use of nonionic iodinated contrast medium for CT: evaluation of safety in a large urban teaching hospital. *AJR Am J Roentgenol* 2005;184:31–4.
148. Dillman JR, Ellis JH, Cohan RH, et al. Frequency and severity of acute allergic-like reactions to gadolinium-containing i.v. contrast media in children and adults. *AJR Am J Roentgenol* 2007;189:1533–8.
149. Chung SM. Safety issues in magnetic resonance imaging. *J Neuroophthalmol* 2002;22:35–9.
150. Stecco A, Saponaro A, Carriero A. Patient safety issues in magnetic resonance imaging: state of the art. *Radiol Med* 2007;112:491–508.
151. Quirk ME, Letendre AJ, Ciottone RA, et al. Anxiety in patients undergoing MR imaging. *Radiology* 1989;170:463–6.
152. Prince MR, Arnoldus C, Frisoli JK. Nephrotoxicity of high-dose gadolinium compared with iodinated contrast. *J Magn Reson Imaging* 1996;6:162–6.
153. Tardy B, Guy C, Barral G, et al. Anaphylactic shock induced by intravenous gadopentetate dimeglumine. *Lancet* 1992;22:494.
154. Perazella MA. Gadolinium-contrast toxicity in patients with kidney disease: nephrotoxicity and nephrogenic systemic fibrosis. *Curr Drug Saf* 2008;3:67–75.
155. Brummett RE, Talbot JM, Charuhas P. Potential hearing loss resulting from MR imaging. *Radiology* 1988;169:539–40.
156. Smith-Bindman R, Miglioretti DL, Larson EB. Rising use of diagnostic medical imaging in a large integrated health system. *Health Aff (Millwood)* 2008;27:1491–502.
157. Saini S, Sharma R, Levine LA, et al. Technical cost of CT examinations. *Radiology* 2001;218:172–5.
158. Saini S, Seltzer SE, Bramson RT, et al. Technical cost of radiologic examinations: analysis across imaging modalities. *Radiology* 2000;216:269–72.
159. Pretorius PM, Milford CA. Investigating the hoarse voice. *BMJ* 2008;337:1165–8.
160. Robinson S, Pitkäranta A. Radiology findings in adult patients with vocal fold paralysis. *Clin Radiol* 2006;61:863–7.
161. MacGregor FB, Roberts DN, Howard DJ, et al. Vocal fold palsy: a re-evaluation of investigations. *J Laryngol Otol* 1994;108:193–6.
162. Merati AL, Halum SL, Smith TL. Diagnostic testing for vocal fold paralysis: survey of practice and evidence-based medicine review. *Laryngoscope* 2006;116:1539–52.
163. Mazonakis M, Tzedakis A, Damilakis J, et al. Thyroid dose from common head and neck CT examinations in children: is there an excess risk for thyroid cancer induction? *Eur Radiol* 2007;17:1352–7.
164. Becker M. Neoplastic invasion of laryngeal cartilage: radiologic diagnosis and therapeutic implications. *Eur J Radiol* 2000;33:216–29.
165. Ng SH, Chang TC, Ko SF, et al. Nasopharyngeal carcinoma: MRI and CT assessment. *Neuroradiology* 1997;39:741–6.
166. Ostrower ST, Parikh SR. Hoarseness. In: AAP textbook of pediatric care. McNerny TK, Adam HM, Campbell DE, et al, editors. Elk Grove Village: American Academy of Pediatrics; 2008.
167. Glastonbury CM. Non-oncologic imaging of the larynx. *Otolaryngol Clin North Am* 2008;41:139–56.
168. Blodgett TM, Fukui MB, Snyderman CH, et al. Combined PET-CT in the head and neck: part 1. Physiologic, altered physiologic, and artifactual FDG uptake. *Radiographics* 2005;25:897–912.
169. Hopkins C, Yousaf U, Pedersen M. Acid reflux treatment for hoarseness. *Cochrane Database of Systematic Reviews* 2006, Issue 1. Art. No.: CD005054. DOI: 10.1002/14651858.CD005054.pub2.
170. Belafsky PC, Postma GN, Koufman JA. Laryngopharyngeal reflux symptoms improve before changes in physical findings. *Laryngoscope* 2001;111:979–81.
171. El-Serag HB, Lee P, Buchner A, et al. Lansoprazole treatment of patients with chronic idiopathic laryngitis: a placebo-controlled trial. *Am J Gastroenterol* 2001;96:979–83.
172. Vaezi MF, Richter JE, Stasney CR, et al. Treatment of chronic posterior laryngitis with esomeprazole. *Laryngoscope* 2006;116:254–60.
173. Kahrilas PJ, Shaheen NJ, Vaezi MF, et al. American Gastroenterological Association Institute technical review on the management of gastroesophageal reflux disease. *Gastroenterology* 2008;135:1392–413.
174. Kahrilas PJ, Shaheen NJ, Vaezi MF, et al. American Gastroenterological Association Medical Position Statement on the management of gastroesophageal reflux disease. *Gastroenterology* 2008;135:1383–91.
175. Qua CS, Wong CH, Gopala K, et al. Gastro-oesophageal reflux disease in chronic laryngitis: prevalence and response to acid-suppressive therapy. *Aliment Pharmacol Ther* 2007;25:287–95.
176. Boustani M, Hall KS, Lane KA, et al. The association between cognition and histamine-2 receptor antagonists in African Americans. *J Am Geriatr Soc* 2007;55:1248–53.
177. Hanlon JT, Landerman LR, Artz MB, et al. Histamine2 receptor antagonist use and decline in cognitive function among community dwelling elderly. *Pharmacoepidemiol Drug Saf* 2004;13:781–7.
178. García Rodríguez LA, Ruigómez A, Panés J. Use of acid-suppressing drugs and the risk of bacterial gastroenteritis. *Clin Gastroenterol Hepatol* 2007;5:1418–23.
179. Loo VG, Poirier L, Miller MA, et al. A predominantly clonal multi-institutional outbreak of *Clostridium difficile*-associated diarrhea with high morbidity and mortality. *N Engl J Med* 2005;353:2442–9.
180. Gulmez SE, Holm A, Frederiksen H, et al. Use of proton pump inhibitors and the risk of community-acquired pneumonia: a population-based case-control study. *Arch Intern Med* 2007;167:950–5.
181. Laheij RJ, Sturkenboom MC, Hassing RJ, et al. Risk of community-acquired pneumonia and use of gastric acid-suppressive drugs. *JAMA* 2004;292:1955–60.
182. Gilard M, Arnaud B, Cornily JC, et al. Influence of omeprazole on the antiplatelet action of clopidogrel associated with aspirin: the randomized, double-blind OCLA (Omeprazole CLopidogrel Aspirin) study. *J Am Coll Cardiol* 2008;51:256–60.
183. Sarkar M, Hennessy S, Yang YX. Proton-pump inhibitor use and the risk for community-acquired pneumonia. *Ann Intern Med* 2008;149:391–8.
184. Canani RB, Cirillo P, Roggero P, et al. Therapy with gastric acidity inhibitors increases the risk of acute gastroenteritis and community-acquired pneumonia in children. *Pediatrics* 2006;117:e817–20.
185. Yang YX. Proton pump inhibitor therapy and osteoporosis. *Curr Drug Saf* 2008;3:204–9.
186. Marcuard SP, Albernaz L, Khazanie PG. Omeprazole therapy causes malabsorption of cyanocobalamin (vitamin B12). *Ann Intern Med* 1994;120:211–5.
187. Hirschowitz BI, Worthington J, Mohnen J. Vitamin B12 deficiency in hypersecretors during long-term acid suppression with proton pump inhibitors. *Aliment Pharmacol Ther* 2008;27:1110–21.
188. Khatib MA, Rahim O, Kania R, et al. Iron deficiency anemia: induced by long-term ingestion of omeprazole. *Dig Dis Sci* 2002;47:2596–7.
189. Sundström A, Blomgren K, Alfredsson L, et al. Acid-suppressing drugs and gastroesophageal reflux disease as risk factors for acute pancreatitis—results from a Swedish case-control study. *Pharmacoepidemiol Drug Saf* 2006;15:141–9.

190. Ylitalo R, Ramel S. Extraesophageal reflux in patients with contact granuloma: a prospective controlled study. *Ann Otol Rhinol Laryngol* 2002;111:441–6.
191. Hanson DG, Jiang J, Chi W. Quantitative color analysis of laryngeal erythema in chronic posterior laryngitis. *J Voice* 1998;12:78–83.
192. Reichel O, Dressel H, Wiederanders K, et al. Double-blind, placebo-controlled trial with esomeprazole for symptoms and signs associated with laryngopharyngeal reflux. *Otolaryngol Head Neck Surg* 2008;139:414–20.
193. Park W, Hicks DM, Khandwala F, et al. Laryngopharyngeal reflux: prospective cohort study evaluating optimal dose of proton-pump inhibitor therapy and pretherapy predictors of response. *Laryngoscope* 2005;115:1230–8.
194. Maronian NC, Azadeh H, Waugh P, et al. Association of laryngopharyngeal reflux disease and subglottic stenosis. *Ann Otol Rhinol Laryngol* 2001;110:606–12.
195. Vaezi MF, Qadeer MA, Lopez R, et al. Laryngeal cancer and gastroesophageal reflux disease: a case-control study. *Am J Med* 2006;119:768–76.
196. Qadeer MA, Lopez R, Wood BG, et al. Does acid suppressive therapy reduce the risk of laryngeal cancer recurrence? *Laryngoscope* 2005;115:1877–81.
197. Kantas I, Balatsouras DG, Kamargianis N, et al. The influence of laryngopharyngeal reflux in the healing of laryngeal trauma. *Eur Arch Otorhinolaryngol* 2009;266:253–9.
198. Wani MK, Woodson GE. Laryngeal contact granuloma. *Laryngoscope* 1999;109:1589–93.
199. Jin J, Lee YS, Jeong SW, et al. Change of acoustic parameters before and after treatment in laryngopharyngeal reflux patients. *Laryngoscope* 2008;118:938–41.
200. Milstein CF, Charbel S, Hicks DM, et al. Prevalence of laryngeal irritation signs associated with reflux in asymptomatic volunteers: impact of endoscopic technique (rigid vs. flexible laryngoscope). *Laryngoscope* 2005;115:2256–61.
201. Branski RC, Bhattacharyya N, Shapiro J. The reliability of the assessment of endoscopic laryngeal findings associated with laryngopharyngeal reflux disease. *Laryngoscope* 2002;112:1019–24.
202. Stuck AE, Minder CE, Frey FJ. Risk of infectious complications in patients taking glucocorticosteroids. *Rev Infect Dis* 1989;11:954–63.
203. Fardet L, Kassir A, Cabane J, et al. Corticosteroid-induced adverse events in adults: frequency, screening and prevention. *Drug Saf* 2007;30:861–81.
204. Conn HO, Poynard T. Corticosteroids and peptic ulcer: meta-analysis of adverse events during steroid therapy. *J Intern Med* 1994;236:619–32.
205. Messer J, Reitman D, Sacks HS, et al. Association of adrenocorticosteroid therapy and peptic-ulcer disease. *N Engl J Med* 1983;301:21–4.
206. Warrington TP, Bostwick JM. Psychiatric adverse effects of corticosteroids. *Mayo Clin Proc* 2006;81:1361–7.
207. van Everdingen AA, Jacobs JW, Siewertsz Van Reesema DR, et al. Low-dose prednisone therapy for patients with early active rheumatoid arthritis: clinical efficacy, disease-modifying properties, and side effects: a randomized, double-blind, placebo-controlled clinical trial. *Ann Intern Med* 2002;136:1–12.
208. Williams AJ, Baghat MS, Stableforth DE, et al. Dysphonia caused by inhaled steroids: recognition of a characteristic laryngeal abnormality. *Thorax* 1983;38:813–21.
209. Williamson IJ, Matusiewicz SP, Brown PH, et al. Frequency of voice problems and cough in patients using pressurized aerosol inhaled steroid preparations. *Eur Respir J* 1995;8:590–2.
210. Forrest LA, Weed H. Candida laryngitis appearing as leukoplakia and GERD. *J Voice* 1998;12:91–5.
211. Toogood JH. Inhaled steroid asthma treatment: ‘Primum non nocere’. *Can Respir J* 1998;5(Suppl A):50A–3A.
212. Jackson-Menaldi CA, Dzul AI, Holland RW. Allergies and vocal fold edema: a preliminary report. *J Voice* 1999;13:113–22.
213. Lavy JA, Wood G, Rubin JS, et al. Dysphonia associated with inhaled steroids. *J Voice* 2000;14:581–8.
214. Dubus JC, Mély L, Huiart L, et al. Cough after inhalation of corticosteroids delivered from spacer devices in children with asthma. *Fundam Clin Pharmacol* 2003;17:627–31.
215. DelGaudio JM. Steroid inhaler laryngitis: dysphonia caused by inhaled fluticasone therapy. *Arch Otolaryngol Head Neck Surg* 2002;128:677–81.
216. Sin DD, Man SF. Inhaled corticosteroids in the long-term management of patients with chronic obstructive pulmonary disease. *Drugs Aging* 2003;20:867–80.
217. Mirza N, Kasper Schwartz S, Antin-Ozerkis D. Laryngeal findings in users of combination corticosteroid and bronchodilator therapy. *Laryngoscope* 2004;114:1566–9.
218. Sulica L. Laryngeal thrush. *Ann Otol Rhinol Laryngol* 2005;114:369–75.
219. Gallivan GJ, Gallivan KH, Gallivan HK. Inhaled corticosteroids: hazardous effects on voice—an update. *J Voice* 2007;21:101–11.
220. Leung AK, Kellner JD, Johnson DW. Viral croup: a current perspective. *J Pediatr Health Care* 2004;18:297–301.
221. Jackson-Menaldi CA, Dzul AI, Holland RW. Hidden respiratory allergies in voice users: treatment strategies. *Logoped Phoniatr Vocol* 2002;27:74–9.
222. Dean CM, Sataloff RT, Hawkshaw MJ, et al. Laryngeal sarcoidosis. *J Voice* 2002;16:283–8.
223. Ozcan KM, Bahar S, Ozcan I, et al. Laryngeal involvement in systemic lupus erythematosus: report of two cases. *J Clin Rheumatol* 2007;13:278–9.
224. Higgins PB. Viruses associated with acute respiratory infections 1961–71. *J Hyg (Lond)* 1974;72:425–32.
225. Bove MJ, Kansal S, Rosen CA. Influenza and the vocal performer: Update on prevention and treatment. *J Voice* 2008;22:326–32.
226. Schalén L, Eliasson I, Kamme C, et al. Erythromycin in acute laryngitis in adults. *Ann Otol Rhinol Laryngol* 1993;102:209–14.
227. Reveiz L, Cardona AF, Ospina EG. Antibiotics for acute laryngitis in adults. *Cochrane Database of Systematic Reviews* 2007, Issue 2. Art. No.: CD004783. DOI: 10.1002/14651858.CD004783.pub3.
228. Arroll B, Kenealy T. Antibiotics for the common cold and acute purulent rhinitis. *Cochrane Database of Systematic Reviews* 2005, Issue 3. Art. No.: CD000247. DOI: 10.1002/14651858.CD000247.pub2.
229. Glasziou PP, Del Mar C, Sanders S, et al. Antibiotics for acute otitis media in children. *Cochrane Database of Systematic Reviews* 2004, Issue 1. Art. No.: CD000219. DOI: 10.1002/14651858.CD000219.pub2.
230. Horn JR, Hansten PD. Drug interactions with antibacterial agents. *J Fam Pract* 1995;41:81–90.
231. Brook I, Foote PA, Hausfeld JN. Increase in the frequency of recovery of methicillin-resistant *Staphylococcus aureus* in acute and chronic maxillary sinusitis. *J Med Microbiol* 2008;57:1015–7.
232. Asche C, McAdam-Marx C, Seal B, et al. Treatment costs associated with community-acquired pneumonia by community level of antimicrobial resistance. *J Antimicrob Chemother* 2008;61:1162–8.
233. Singh B, Balwally AN, Nash M, et al. Laryngeal tuberculosis in HIV-infected patients: a difficult diagnosis. *Laryngoscope* 1996;106:1238–40.
234. Tato AM, Pascual J, Orofino L, et al. Laryngeal tuberculosis in renal allograft patients. *Am J Kidney Dis* 1998;31:701–5.
235. Wang BY, Amolat MJ, Woo P, et al. Atypical mycobacteriosis of the larynx: an unusual clinical presentation secondary to steroids inhalation. *Ann Diagn Pathol* 2008;12:426–9.
236. Lightfoot SA. Laryngeal tuberculosis masquerading as carcinoma. *J Am Board Fam Pract* 1997;10:374–6.
237. Silva L, Damrose E, Bairão F, et al. Infectious granulomatous laryngitis: a retrospective study of 24 cases. *Eur Arch Otorhinolaryngol* 2008;265:675–80.
238. Sari M, Yazici M, Bağlam T, et al. Actinomycosis of the larynx. *Acta Otolaryngol* 2007;127:550–2.

239. Sotir MJ, Cappozzo DL, Warshauer DM, et al. A countywide outbreak of pertussis: initial transmission in a high school weight room with subsequent substantial impact on adolescents and adults. *Arch Pediatr Adolesc Med* 2008;162:79–85.
240. Postels-Multani S, Schmitt HJ, Wirsing von König CH, et al. Symptoms and complications of pertussis in adults. *Infection* 1995;23:139–42.
241. Hopkins A, Lahiri T, Salerno R, et al. Changing epidemiology of life-threatening upper airway infections: the reemergence of bacterial tracheitis. *Pediatrics* 2006;118:1418–21.
242. Royal College of Speech & Language Therapists. Clinical voice disorders. Royal College of Speech & Language Therapists; 2005. http://www.rcslt.org/resources/RCSLT_Clinical_Guidelines.pdf (accessed June 10, 2009).
243. American Speech-Language-Hearing Association. Preferred practice patterns for the profession of speech-language pathology. 2004. <http://www.asha.org/docs/html/PP2004-00191.html>.
244. Bastian RW, Levine LA. Visual methods of office diagnosis of voice disorders. *Ear Nose Throat J* 1988;67:363–79.
245. American Speech-Language-Hearing Association. The use of voice therapy in the treatment of dysphonia. 2005. <http://www.asha.org/docs/html/TR2005-00158.html>.
246. American Speech-Language-Hearing Association. Training guidelines for laryngeal videoendoscopy/stroboscopy. 1998. <http://www.asha.org/docs/html/GL1998-00064.html>.
247. Thomas G, Mathews SS, Chrysolite SB, et al. Outcome analysis of benign vocal cord lesions by videostroboscopy, acoustic analysis and voice handicap index. *Indian J Otolaryngol* 2007;59:336–40.
248. Woo P, Colton R, Casper J, et al. Diagnostic value of stroboscopic examination in hoarse patients. *J Voice* 1991;5:231–8.
249. Thomas LB, Stemple JC. Voice therapy: Does science support the art? *Communicative Disorders Review* 2007;1:49–77.
250. Anderson T, Sataloff RT. The power of voice therapy. *Ear Nose Throat J* 2002;81:433–4.
251. Speyer R, Weineke G, Hosseini EG, et al. Effects of voice therapy as objectively evaluated by digitized laryngeal stroboscopic imaging. *Ann Otol Rhinol Laryngol* 2002;111:902–8.
252. Pedersen M, Beranova A, Møller S. Dysphonia: medical treatment and a medical voice hygiene advice approach. A prospective randomised pilot study. *Eur Arch Otorhinolaryngol* 2004;261:312–5.
253. Boone DR, McFarlane SC, Von Berg SL. The voice and voice therapy. 7th ed. Boston: Allyn and Bacon; 2005.
254. Stemple JC, Glaze LE, Klaben BG. Clinical voice pathology: Theory and management. 3rd ed. San Diego: Singular; 2000.
255. Roy N, Gray SD, Simon M, et al. An evaluation of the effects of two treatment approaches for teachers with voice disorders: a prospective randomized clinical trial. *J Speech Lang Hear Res* 2001;44:286–96.
256. Verdolini-Marston K, Burke MK, Lessac A, et al. Preliminary study of two methods of treatment for laryngeal nodules. *J Voice* 1995;9:74–85.
257. Fox CM, Ramig LO, Ciucci MR, et al. The science and practice of LSVT/LOUD: neural plasticity-principled approach to treating individuals with Parkinson disease and other neurological disorders. *Semin Speech Lang* 2006;27:283–99.
258. Kim J, Davenport P, Sapienza C. Effect of expiratory muscle strength training on elderly cough function. *Arch Gerontol Geriatr* 2009;48:361–6.
259. Sullivan MD, Heywood BM, Beukelman DR. A treatment for vocal cord dysfunction in female athletes: an outcome study. *Laryngoscope* 2001;111:1751–5.
260. Boutsen F, Cannito MP, Taylor M, et al. Botox treatment in adductor spasmodic dysphonia: a meta-analysis. *J Speech Lang Hear Res* 2002;45:469–81.
261. Pearson EJ, Sapienza CM. Historical approaches to the treatment of Adductor-Type Spasmodic Dysphonia (ADSD): review and tutorial. *NeuroRehabilitation* 2003;18:325–38.
262. Zeitels SM, Casiano RR, Gardner GM, et al. Management of common voice problems: committee report. *Otolaryngol Head Neck Surg* 2002;126:333–48.
263. Johns MM. Update on the etiology, diagnosis, and treatment of vocal fold nodules, polyps, and cysts. *Curr Opin Otolaryngol Head Neck Surg* 2003;11:456–61.
264. McCrory E. Voice therapy outcomes in vocal fold nodules: a retrospective audit. *Int J Lang Commun Disord* 2001;36(Suppl):19–24.
265. Havas TE, Priestley J, Lowinger DS. A management strategy for vocal process granulomas. *Laryngoscope* 1999;109:301–6.
266. Johns MM, Garrett CG, Hwang J, et al. Quality-of-life outcomes following laryngeal endoscopic surgery for non-neoplastic vocal fold lesions. *Ann Otol Rhinol Laryngol* 2004;113:597–601.
267. Zeitels SM, Akst LM, Bums JA, et al. Pulsed angiolytic laser treatment of ectasias and varices in singers. *Ann Otol Rhinol Laryngol* 2006;115:571–80.
268. Bennett S, Bishop SG, Lumpkin SM. Phonatory characteristics following surgical treatment of severe polypoid degeneration. *Laryngoscope* 1989;99:525–32.
269. Ragab SM, Elsheikh MN, Saafan ME, et al. Radiophonosurgery of benign superficial vocal fold lesions. *J Laryngol Otol* 2005;119:961–6.
270. Dedo HH, Yu KC. CO₂ laser treatment in 244 patients with respiratory papillomas. *Laryngoscope* 2001;111:1639–44.
271. Pasquale K, Wiatrak B, Woolley A, et al. Microdebrider versus CO₂ laser removal of recurrent respiratory papillomas: a prospective analysis. *Laryngoscope* 2003;113:139–43.
272. Steinberg B, Topp W, Schneider P. Laryngeal papilloma virus infection during clinical remission. *N Engl J Med* 1983;308:1261–4.
273. Benninger MS. Microdissection or microspot CO₂ laser for limited benign vocal fold lesions: a prospective, randomized trial. *Laryngoscope* 2000;110:1–37.
274. O'Leary MA, Grillone GA. Injection laryngoplasty. *Otolaryngol Clin North Am* 2006;39:43–54.
275. Morgan JE, Zraick RI, Griffin AW, et al. Injection versus medialization laryngoplasty for the treatment of unilateral vocal fold paralysis. *Laryngoscope* 2007;117:2068–74.
276. Hertegård S, Hallén L, Laurent C, et al. Cross-linked hyaluronan versus collagen for injection treatment of glottal insufficiency: 2-year follow-up. *Acta Otolaryngol* 2004;124:1208–14.
277. Kimura M, Nito T, Sakakibara K, et al. Clinical experience with collagen injection of the vocal fold: a study of 155 patients. *Auris Nasus Larynx* 2008;35:67–75.
278. Cantarella G, Mazzola RF, Domenichini E, et al. Vocal fold augmentation by autologous fat injection with liposuction procedure. *Otolaryngol Head Neck Surg* 2005;132.
279. Karpenko AN, Dworkin JP, Meleca RJ, et al. Cymetra injection for unilateral vocal fold paralysis. *Ann Otol Rhinol Laryngol* 2003;112:927–34.
280. Lee SW, Son YI, Kim CH, et al. Voice outcomes of polyacrylamide hydrogel injection laryngoplasty. *Laryngoscope* 2007;117:1871–5.
281. Patel NJ, Kerschner JE, Merati AL. The use of injectable collagen in the management of pediatric vocal unilateral fold paralysis. *Int J Pediatr Otorhinolaryngol* 2003;67:1355–60.
282. Sittel C, Echtermach M, Federspil PA, et al. Polydimethylsiloxane particles for permanent injection laryngoplasty. *Ann Otol Rhinol Laryngol* 2006;115:103–9.
283. Rosen CA, Gartner-Schmidt J, Casiano R, et al. Vocal fold augmentation with calcium hydroxylapatite (CaHA). *Otolaryngol Head Neck Surg* 2007;136:198–204.
284. Kasperbauer JL, Slavik DH, Maragos NE. Teflon granulomas and overinjection of Teflon: a therapeutic challenge for the otorhinolaryngologist. *Ann Otol Rhinol Laryngol* 1993;102:748–51.
285. Varvares MA, Montgomery WW, Hillman RE. Teflon granuloma of the larynx: etiology, pathophysiology, and management. *Ann Otol Rhinol Laryngol* 1995;104:511–5.

286. Schneider B, Bigenzahn W, End A, et al. External vocal fold medialization in patients with recurrent nerve paralysis following cardiothoracic surgery. *Eur J Cardiothorac Surg* 2003;23:477–83.
287. Zeitels SM, Mauri M, Dailey SH. Medialization laryngoplasty with Gore-Tex for voice restoration secondary to glottal incompetence: indications and observations. *Ann Otol Rhinol Laryngol* 2003;112:180–4.
288. Cummings CW, Purcell LL, Flint PW. Hydroxylapatite laryngeal implants for medialization. Preliminary report. *Ann Otol Rhinol Laryngol* 1993;102:843–51.
289. Gray SD, Barkmeier J, Jones D, et al. Vocal evaluation of thyroplastic surgery in the treatment of unilateral vocal fold paralysis. *Laryngoscope* 1992;102:415–21.
290. Kelchner LN, Stemple JC, Gerdeman E, et al. Etiology, pathophysiology, treatment choices, and voice results for unilateral adductor vocal fold paralysis: a 3-year retrospective. *J Voice* 1999;13:592–601.
291. Chester MW, Stewart MG. Arytenoid adduction combined with medialization thyroplasty: An evidence-based review. *Otolaryngol Head Neck Surg* 2003;129:305–10.
292. Gardner GM, Altman JS, Balakrishnan G. Pediatric vocal fold medialization with silastic implant: intraoperative airway management. *Int J Pediatr Otorhinolaryngol* 2000;52:37–44.
293. Link DT, Rutter MJ, Liu JH, et al. Pediatric type I thyroplasty: an evolving procedure. *Ann Otol Rhinol Laryngol* 1999;108:1105–10.
294. Schiratzki H, Fritzell B. Treatment of spasmodic dysphonia by means of resection of the recurrent laryngeal nerve. *Acta Otolaryngol Suppl* 1988;449:115–7.
295. Sapis S, Aronson AE. Clinical reliability in rating voice improvement after laryngeal nerve section for spastic dysphonia. *Laryngoscope* 1985;95:200–2.
296. Biller HF, Som ML, Lawson W. Laryngeal nerve crush for spastic dysphonia. *Ann Otol Rhinol Laryngol* 1983;92:469.
297. Dedo HH, Izdebski K. Evaluation and treatment of recurrent spasticity after recurrent laryngeal nerve section. A preliminary report. *Ann Otol Rhinol Laryngol* 1984;93:343–5.
298. Berke GS, Blackwell KE, Gerratt BR, et al. Selective laryngeal adductor denervation-reinnervation: a new surgical treatment for adductor spasmodic dysphonia. *Ann Otol Rhinol Laryngol* 1999;108:227–31.
299. Truong DD, Bhidayasiri R. Botulinum toxin therapy of laryngeal muscle hyperactivity syndromes: comparing different botulinum toxin preparations. *Eur J Neurol* 2006;13(Suppl 1):36–41.
300. Blitzer A, Sulica L. Botulinum toxin: basic science and clinical uses in otolaryngology. *Laryngoscope* 2001;111:218–26.
301. Sulica L. Contemporary management of spasmodic dysphonia. *Curr Opin Otolaryngol Head Neck Surg* 2004;12:543–8.
302. Stong BC, DelGaudio JM, Hapner ER, et al. Safety of simultaneous bilateral botulinum toxin injections for abductor spasmodic dysphonia. *Arch Otolaryngol Head Neck Surg* 2005;131:793–5.
303. Blitzer A, Brin MF, Fahn S, et al. Localized injections of botulinum toxin for the treatment of focal laryngeal dystonia (spastic dysphonia). *Laryngoscope* 1988;98:193–7.
304. Truong DD, Rontal M, Rolnick M, et al. Double-blind controlled study of botulinum toxin in adductor spasmodic dysphonia. *Laryngoscope* 1991;101:630–4.
305. Cannito MP, Woodson GE, Murry T, et al. Perceptual analyses of spasmodic dysphonia before and after treatment. *Arch Otolaryngol Head Neck Surg* 2004;130:1393–9.
306. Courey MS, Garrett CG, Billante CR, et al. Outcomes assessment following treatment of spasmodic dysphonia with botulinum toxin. *Ann Otol Rhinol Laryngol* 2000;109:819–22.
307. Watts C, Whurr R, Nye C. Botulinum toxin injections for the treatment of spasmodic dysphonia. *Cochrane Database of Systematic Reviews* 2004, Issue 3. Art. No.: CD004327. DOI: 10.1002/14651858.CD004327.pub2.
308. Blitzer A, Brin MF, Stewart CF. Botulinum toxin management of spasmodic dysphonia (laryngeal dystonia): a 12-year experience in more than 900 patients. *Laryngoscope* 1998;108:1435–41.
309. Adler CH, Bansberg SF, Krein-Jones K, et al. Safety and efficacy of botulinum toxin type B (Myobloc) in adductor spasmodic dysphonia. *Mov Disord* 2004;19:1075–9.
310. Thomas JP, Siupsinskiene N. Frozen versus fresh reconstituted botox for laryngeal dystonia. *Otolaryngol Head Neck Surg* 2006;135:204–8.
311. Blitzer A, Brin MF. Laryngeal dystonia: a series with botulinum toxin therapy. *Ann Otol Rhinol Laryngol* 1991;100:85–9.
312. Inagi K, Ford CN, Bless DM, et al. Analysis of factors affecting botulinum toxin results in spasmodic dysphonia. *J Voice* 1996;10:306–13.
313. Koriwchak MJ, Netterville JL, Snowden T, et al. Alternating unilateral botulinum toxin type A (BOTOX) injections for spasmodic dysphonia. *Laryngoscope* 1996;106:1476–81.
314. Holzer SE, Ludlow CL. The swallowing side effects of botulinum toxin type A injection in spasmodic dysphonia. *Laryngoscope* 1996;106:86–92.
315. Woodson G, Hochstetler H, Murry T. Botulinum toxin therapy for abductor spasmodic dysphonia. *J Voice* 2006;20:137–43.
316. Lundy DS, Lu FL, Casiano RR, et al. The effect of patient factors on response outcomes to Botox treatment of spasmodic dysphonia. *J Voice* 1998;12:460–6.
317. Fisher KV, Giddens CL, Gray SD. Does botulinum toxin alter laryngeal secretions and mucociliary transport? *J Voice* 1998;12:389–98.
318. Park JB, Simpson LL, Anderson TD, et al. Immunologic characterization of spasmodic dysphonia patients who develop resistance to botulinum toxin. *J Voice* 2003;17(2):255–64.
319. Ferreira LP, de Oliveira Latorre MD, Pinto Giannini SP, et al. Influence of abusive vocal habits, hydration, mastication, and sleep in the occurrence of vocal symptoms in teachers. *J Voice* 2009 Jan 8. [Epub ahead of print].
320. Ori Y, Sabo R, Binder Y, et al. Effect of hemodialysis on the thickness of vocal folds: a possible explanation for postdialysis hoarseness. *Nephron Clin Pract* 2006;103:c144–8.
321. Yiu EM, Chan RM. Effect of hydration and vocal rest on the vocal fatigue in amateur karaoke singers. *J Voice* 2003;17:216–27.
322. Jónsdóttir V, Laukkanen AM, Siikki I. Changes in teachers' voice quality during a working day with and without electric sound amplification. *Folia Phoniatr Logop* 2003;55:267–80.
323. Ruotsalainen JH, Sellman J, Lehto L, et al. Interventions for preventing voice disorders in adults. *Cochrane Database of Systematic Reviews* 2007, Issue 4. Art. No.: CD006372. DOI: 10.1002/14651858.CD006372.pub2.
324. Duffy OM, Hazlett DE. The impact of preventive voice care programs for training teachers: a longitudinal study. *J Voice* 2004;18:63–70.
325. Bovo R, Galceran M, Petruccioli J, et al. Vocal problems among teachers: evaluation of a preventive voice program. *J Voice* 2007;21:705–22.
326. Landes BA, McCabe BF. Dysphonia as a reaction to cigaret smoke. *Laryngoscope* 1957;67:155–6.
327. de la Hoz RE, Shohet MR, Bienenfeld LA, et al. Vocal cord dysfunction in former World Trade Center (WTC) rescue and recovery workers and volunteers. *Am J Ind Med* 2008;51:161–5.

APPENDIX

Frequently Asked Questions About Voice Therapy

Why is voice therapy recommended for hoarseness? Voice therapy has been demonstrated to be effective for hoarseness across the lifespan from children to older adults.^{A1,A2} Voice therapy is the first line of treatment for vocal fold lesions like vocal nodules, polyps, or cysts.^{A3,A4} These lesions often occur in people with vocally intense occupations, like teachers, attorneys, or clergymen.^{A5} Another pos-

sible cause of these lesions is vocal overdoing, often seen in sports enthusiasts; in socially active, aggressive, or loud children; or in high-energy adults who often speak loudly.^{A6-A9}

Voice therapy, specifically the Lee Silverman Voice Therapy method, has been demonstrated to be the most effective method of treating the lower volume, lower energy, and rapid-rate voice/speech of individuals with Parkinson disease.^{A10,A11}

Voice therapy has been used to treat hoarseness concurrently with other medical therapies like botulinum toxin injections for spasmodic dysphonia and/or tremor.^{A12,A13} Voice therapy has been used alone in the treatment of unilateral vocal fold paralysis^{A14,A15} and has been used to improve the outcome of surgical procedures as in vocal fold augmentation^{A16} or thyroplasty.^{A17} Voice therapy is an important component of any comprehensive surgical treatment for hoarseness.^{A18}

What happens in voice therapy? Voice therapy is a program designed to reduce hoarseness through guided change in vocal behaviors and lifestyle changes. Voice therapy consists of a variety of tasks designed to eliminate harmful vocal behavior, shape healthy vocal behavior, and assist in vocal fold wound healing after surgery or injury. Voice therapy for hoarseness generally consists of 1 to 2 therapy sessions each week for 4 to 8 weeks.^{A19} The duration of therapy is determined by the origin of the hoarseness and severity of the problem, co-occurring medical therapy, and importantly, patient commitment to the practice and generalization of new vocal behaviors outside the therapy session.^{A20}

Who provides voice therapy? Certified and licensed speech-language pathologists are healthcare professionals with the expertise needed to provide effective behavioral treatment for hoarseness.^{A21}

How do I find a qualified speech-language pathologist who has experience in voice? The American Speech-Language-Hearing Association (ASHA) is an excellent resource for finding a certified speech-language pathologist by going to the ASHA website (www.asha.org) or by accessing ASHA's online search engine, called ProSearch, at: <http://www.asha.org/proserv>. You may also contact ASHA's Action Center, Monday through Friday (8:30 am-5:30 pm), at: 1-800-638-8255; fax: 301-296-8580; TTY (Text Telephone Communication Device): 301-296-5650; e-mail: actioncenter@asha.org.

Does insurance cover voice therapy? Generally, Medicare, under the guidelines for coverage of speech therapy, will cover voice therapy if provided by a certified and licensed speech-language pathologist, ordered by a physician, and deemed medically necessary for the diagnosis. Medicaid varies from state to state but generally covers voice therapy, under the rules for speech therapy, up to the age of 18 years. It is best to contact your local Medicaid office, as there are state differences and program differences. Private insurance companies

vary and the consumer is guided to contact his or her insurance company for specific guidelines for their purchased policies.

Are speech therapy and voice therapy the same? Speech therapy is a term that encompasses a variety of therapies, including voice therapy. Most insurance companies refer to voice therapy as speech therapy, but they are the same thing if provided by a certified and licensed speech-language pathologist.

REFERENCES

- A1. Thomas LB, Stemple JC. Voice therapy: Does science support the art? *Communicative Disorders Review* 2007;1:49–77.
- A2. Ramig LO, Verdolini K. Treatment efficacy: voice disorders. *J Speech Lang Hear Res* 1998;41:S101–16.
- A3. Johns MM. Update on the etiology, diagnosis, and treatment of vocal fold nodules, polyps, and cysts. *Curr Opin Otolaryngol Head Neck Surg* 2003;11:456–61.
- A4. Anderson T, Sataloff RT. The power of voice therapy. *Ear Nose Throat J* 2002;81:433–4.
- A5. Roy N, Gray SD, Simon M, et al. An evaluation of the effects of two treatment approaches for teachers with voice disorders: a prospective randomized clinical trial. *J Speech Lang Hear Res* 2001;44:286–96.
- A6. Trani M, Ghidini A, Bergamini G, et al. Voice therapy in pediatric functional dysphonia: a prospective study. *Int J Pediatr Otorhinolaryngol* 2007;71:379–84.
- A7. Rubin JS, Sataloff RT, Korovin GW. *Diagnosis and treatment of voice disorders*. 3rd ed. San Diego: Plural Publishing Group; 2006.
- A8. Stemple J, Glaze L, Klaben B. *Clinical voice pathology: Theory and management*. 3rd ed. San Diego: Singular; 2000.
- A9. Boone DR, McFarlane SC, Von Berg S. *The voice and voice therapy*. 7th ed. Boston: Allyn and Bacon; 2005.
- A10. Fox CM, Ramig LO, Ciucci MR, et al. The science and practice of LSVT/LOUD: neural plasticity-principled approach to treating individuals with Parkinson disease and other neurological disorders. *Semin Speech Lang* 2006;27:283–99.
- A11. Dromey C, Ramig LO, Johnson AB. Phonatory and articulatory changes associated with increased vocal intensity in Parkinson disease: a case study. *J Speech Hear Res* 1995;38:751–64.
- A12. Pearson EJ, Sapienza CM. Historical approaches to the treatment of Adductor-Type Spasmodic Dysphonia (ADSD): review and tutorial. *NeuroRehabilitation* 2003;18:325–38.
- A13. Murry T, Woodson GE. Combined-modality treatment of adductor spasmodic dysphonia with botulinum toxin and voice therapy. *J Voice* 1995;9:460–5.
- A14. Schindler A, Bottero A, Capaccio P, et al. Vocal improvement after voice therapy in unilateral vocal fold paralysis. *J Voice* 2008;22:113–8.
- A15. Miller S. Voice therapy for vocal fold paralysis. *Otolaryngol Clin North Am* 2004;37:105–19.
- A16. Rosen CA. Phonosurgical vocal fold injection: procedures and materials. *Otolaryngol Clin North Am* 2000;33:1087–96.
- A17. Billiante CR, Clary J, Sullivan C, et al. Voice therapy following thyroplasty with long standing vocal fold immobility. *Auris Nasus Larynx* 2002;29:341–5.
- A18. Branski, RC, Murray T. Voice therapy. 2008. Available at <http://emedicine.medscape.com/article/866712-overview>. Accessed May 18, 2009.
- A19. Hapner E, Portone-Maira C, Johns MM. A study of voice therapy dropout. *J Voice* 2009;23:337–40.
- A20. Behrman A. Facilitating behavioral change in voice therapy: the relevance of motivational interviewing. *Am J Speech Lang Pathol* 2006;15:215–25.
- A21. American Speech-Language-Hearing Association. The use of voice therapy in the treatment of dysphonia. 2005. <http://www.asha.org/docs/html/TR2005-00158.html>. Accessed May 18, 2009.