REVIEW ARTICLE

Hoarseness—Causes and Treatments

Rudolf Reiter, Thomas Karl Hoffmann, Anja Pickhard, Sibylle Brosch

SUMMARY

Background: Hoarseness (dysphonia) is the reason for about 1% of all consultations in primary care. It has many causes, ranging from self-limited laryngitis to malignant tumors of the vocal cords.

Methods: This review is based on literature retrieved by a selective search in PubMed employing the terms "hoarseness," "hoarse voice," and "dysphonia," on the relevant guideline of the American Academy of Otolaryngology—Head and Neck Surgery, and on Cochrane reviews.

Results: Hoarseness can be caused by acute (42.1%) and chronic laryngitis (9.7%), functional vocal disturbances (30%), and benign (10.7–31%) and malignant tumors (2.2–3%), as well as by neurogenic disturbances such as vocal cord paresis (2.8–8%), physiologic aging of the voice (2%), and psychogenic factors (2–2.2 %). Hoarseness is very rarely a manifestation of internal medical illness. The treatment of hoarseness has been studied in only a few randomized controlled trials, all of which were on a small scale. Voice therapy is often successful in the treatment of functional and organic vocal disturbances (level 1a evidence). Surgery on the vocal cords is indicated to treat tumors and inadequate vocal cord closure. The only entity causing hoarseness that can be treated pharmacologically is chronic laryngitis associated with gastro-esophageal reflux, which responds to treatment of the reflux disorder. The empirical treatment of hoarseness with antibiotics or corticosteroids is not recommended.

Conclusion: Voice therapy, vocal cord surgery, and drug therapy for appropriate groups of patients with hoarseness are well documented as effective by the available evidence. In patients with risk factors, especially smokers, hoarseness should be immediately evaluated by laryngoscopy.

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Dysphonia, with the cardinal symptom of hoarseness, has a prevalence of around 1% among patients in general (1) and a lifetime prevalence of approximately 30% (e1). The term dysphonia is used to describe any impairment of the voice—alteration in the sound of the voice with hoarseness, restriction of vocal performance, or strained vocalization. The pathophysiology of hoarseness is characterized by muscle tone–related irregularity in the oscillation of the vocal cords owing to hypertonic dysphonia, incomplete closure of the glottis on vocalization, or an increase in vocal cord bulk, perhaps due to a tumor *(Figure 1a, b)*.

The aim of this review is to summarize the current knowledge of hoarseness: the potential causes, the means of diagnosis, the treatment options, and the evidence for their efficacy *(eTable)* (2, e2, e3).

To this end, we carried out a selective survey of the literature using the search terms "hoarseness," "hoarse voice," and "dysphonia," with particular reference to evidence-based guidelines from America (2, e4). Moreover, we included treatment recommendations from Cochrane reviews. Because no evidence-based guidelines have been published in German, we also took account of expert opinion.

The causes of hoarseness are diverse:

- Acute and chronic laryngitis (accounting for 42.1% and 9.7% of cases respectively)
- Functional dysphonia (30%)
- Benign and malignant tumors (10.7 to 31.0% and 2.2 to 3.0% respectively)
- Neurogenic factors such as vocal cord paralysis (2.8 to 8%)
- Physiological aging (2%)
- Psychogenic factors (2.0 to 2.2%) (1, e5).

Very occasionally hoarseness can be attributed to manifestations of laryngeal disease other than tumors *(Table 1)*.

Suspicion of a serious underlying disease *(Box, Figure 2)* or persistence of hoarseness for more than 3 months *(eTable)* (2) should prompt immediate investigation by means of indirect laryngoscopy.

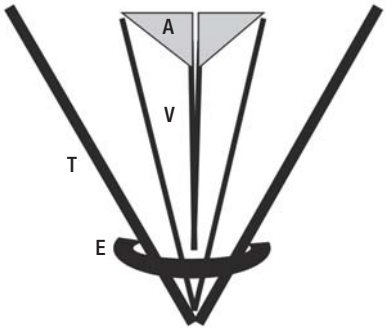
Functional dysphonia

In the absence of a specific anatomic correlate such as a tumor, patients with hyperfunctional dysphonia, i.e., a non-physiological increase in tone of the vocal cords on phonation, in speaking or breathing, develop marked difficulties in speaking, with accompanying hoarseness. Women are more frequently affected than men (e5). The stroboscopic oscillation of the vocal cords is impaired or irregular due to abnormal muscle tone. The

Indirect laryngoscopy during phonation

FIGURE 1a

Phonation



Posterior

Arytenoid tubercle (A)

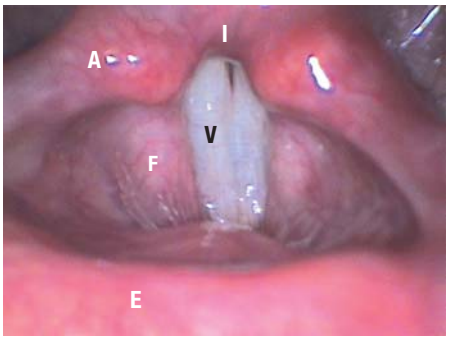
Interarytenoid region (I)

Vocal cords (V)

False vocal cords (F)

Epiglottis (E)

Thyroid cartilage (T)

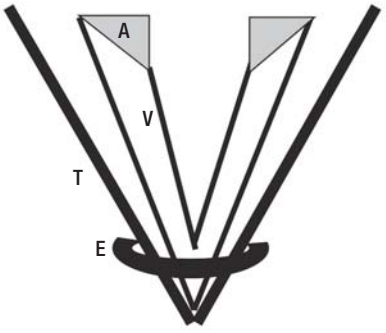


Anterior

Indirect laryngoscopy during respiration

FIGURE 1b

Respiration



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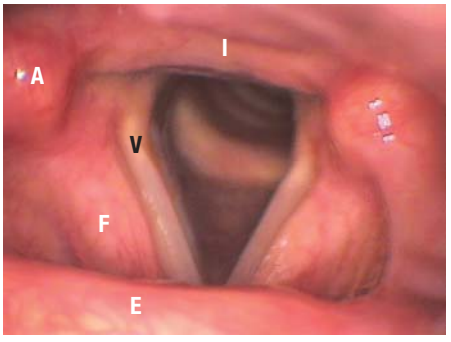


Anterior

measure of choice is conservative treatment to counteract the damaging strain on the voice. Various procedures are available to improve vocal and respiratory technique and vocal hygiene (evidence level 1a, recommendation grade A) (2, e4). The prognosis of speech therapy is favorable (e6–e17); in 46 to 93% of cases (e8, e9) vocal performance is clearly improved. Only a few randomized controlled trials have been published, all of them with small case numbers, and there are no long-term studies *(Table 2)* (3, e16, e17).

Organic secondary manifestation of functional dysphonia

Juvenile and adult forms of vocal cord nodules are distinguished (screamer's nodules and singer's nodules). The vocal cord changes are a secondary manifestation of untreated hyperfunctional dysphonia. Initially, there is reactive phonation hyperplasia of the medial margin of the vocal cord—at the junction of the anterior third and middle third, the site of greatest stress on phonation. The tissue swells reversibly and edema arises. With the passage of time, the soft swellings undergo fibrosis and turn into hard nodules (e18, e19), preventing complete closure of the vocal cords in the affected area. The treatment of choice is voice therapy (evidence level 1a, recommendation grade A) (2, e6, e20). Only infrequently is microsurgical excision necessary in adults (2, e6, e16, e18). In over 80% of patients the voice is restored to normal by voice therapy alone (e21). Recurrence rates of 30% after voice therapy and 13% after phonosurgery have been reported (e22). In children the change of voice is often followed by spontaneous remission (>90% of boys, ca. 50% of girls) (2, e4, e23, e24).

Organic dysphonia

Acute laryngitis

Acute laryngitis is the most common cause of hoarseness, accounting for 40% of cases (1), and is almost always viral in origin. It occurs in infections of the upper respiratory tract and is self-limiting, subsiding after 1 to 2 weeks (2, e25, e26). Patients are counseled

TABLE 1

Causes and characteristics of hoarseness

Pathology			Proportion of all cases	Typical symptoms	Treatment	Evidence
Functional dysphonia	Hyperfunctional dysphonia		30%	Hoarseness with vocal strain	Voice therapy	1a/A
Secondary manifestation of functional dysphonia	Vocal cord nodules		Included in benign tumors (10.7–31%)	Hoarseness with vocal strain	Voice therapy (phonosurgery)	1a/A
Organic dysphonia	Laryngitis	Acute	42.1%	Hoarseness, infection	No medicinal treatment, self-limiting	1a/A
		Chronic	9.7%	Constant hoarseness, dysphonia, throat sensations, compulsion to clear throat	Avoidance of noxae, laryngostroboscopic monitoring	4/X
	Benign tumors	Polyps/cysts	10.7–31%	Hoarseness, reduced volume of voice, vocal fatigue	Phonosurgery, if applicable voice therapy	2a/B
		Reinke edema		Hoarseness, vocal fatigue, deep voice	Avoidance of noxae, phonosurgery, if applicable voice therapy	2a/B
		Recurring papillomatosis		Hoarseness, dyspnea	Phonosurgery	2a/B
	Vocal cord malignancies		2.2–3%	Hoarseness as early symptom	(Laser) surgery, radiotherapy	2a/B
	Vocal cord scarring		n.d.	Constant hoarseness, quiet voice	Voice therapy (phonosurgery)	4/X
	Presbyphonia		2%	Hoarseness, high-pitched voice	Voice therapy, phonosurgery	2a/B
Manifestation of internal disease	Laryngopharyngeal reflux		Included in chronic laryngitis (9.7%)	Only slight hoarseness, throat sensations predominantly at night	With signs of reflux: PPIs	2b/C
					Without signs of reflux: no PPIs	2a/B
	Tuberculosis		n.d.	Dyspnea, cough	Tuberculostatic treatment	3/D
	Rheumatoid diseases	Rheumatoid arthritis	n.d.	Hoarseness, dyspnea, or dysphagia, depending on site	Antirheumatic treatment	3/D
		Collagenoses (systemic lupus erythematosus)	n.d.			
		Vasculitides (Wegener disease)	n.d.			
		Sarcoidosis	n.d.			
	Amyloidosis		n.d.	Hoarseness, dyspnea, or dysphagia, depending on site	Phonosurgery, internal / hematological treatment	3/D
	Lymphoma		n.d.	Dysphonia, dyspnea	Internal / hematological treatment	4/X
Neurological diseases	Vocal cord paresis		2.8–8.0%	Hoarseness, impaired speech breathing	Voice therapy, phonosurgery	1a/A, 2a/B
	Spasmodic dysphonia		n.d.	Variable hoarseness	Administration of botolinumtoxin A	2a/B
Vocal cord dysfunction			n.d.	Hoarseness during an episode of respiratory distress (few seconds)	Discuss with patient, breathing therapy, psychotherapy	4/X
Psychogenic dysphonia			2–2.2%	Sudden hoarseness (hours or days)	Psychological and psychosomatic treatment, psychotherapy	4/X

Etiological classification of dysphonias according to causes, typical symptoms and characteristics of hoarseness, showing each cause's percentage contribution to the total. Phonosurgery is an operative intervention to improve the voice, usually using microinstruments inserted transorally via a laryngoscope, sometimes by means of laser.
n.d., no data or prevalence <1%; PPIs, proton pump inhibitors (1, 2, e2-e5)

BOX

Serious comorbidities of hoarseness

Comorbidities and additional risk factors that require urgent laryngoscopic examination by an otorhinolaryngologist:

- History of nicotine and/or alcohol consumption
- Enlarged cervical lymph nodes
- Hoarseness following trauma
- Association with hemoptysis, dysphagia, odynophagia, otalgia, or dyspnea
- Neurological symptoms
- Unexplained weight loss
- Progression of hoarseness
- Immunosuppression
- Possible bolus aspiration
- Hoarseness after an operative intervention (intubation, neck surgery)

to limit how much they talk, but absolute voice rest is not advised in order to avoid the risk of overcompensation to the point of aphonia (4). Routine antibiotic treatment is discouraged (evidence level 1a, recommendation grade A) (2, 5, e4); antibiotics are prescribed only in exceptional circumstances, e.g., in the presence of bacterial superinfection or laryngeal tuberculosis *(Table 1)*. Indirect or direct laryngoscopy is indicated in such severe cases (evidence level 4, recommendation grade C) *(Figure 2)*. Corticosteroids should not be administered in acute laryngitis (evidence level 3, recommendation grade B) (2).

Chronic laryngitis

Chronic laryngitis has an incidence of 3.5 /1000 in the general population (e27) and is a precursor of vocal cord cancer (6, 7). The following have been proposed as important etiological factors:

- Nicotine abuse
- Inhaled corticoid treatment
- Inhaled environmental noxae
- Gastroesophageal reflux with laryngopharyngeal involvement.

Not infrequently leukoplakia arises *(Figure 3)*. Possible clinical signs of chronic laryngitis are dysphonia, sensations in the throat, and a constant urge to clear the throat (e27). The principal therapeutic measures are avoidance of noxae and regular laryngeal stroboscopy for early detection of malignancy (4, 6). Meta-analyses have shown that laryngeal dysplasia or leukoplakia progresses to cancer in 14 to 16% of patients after a mean interval of 43 months (range 4 to 192 months) (e28, e29).

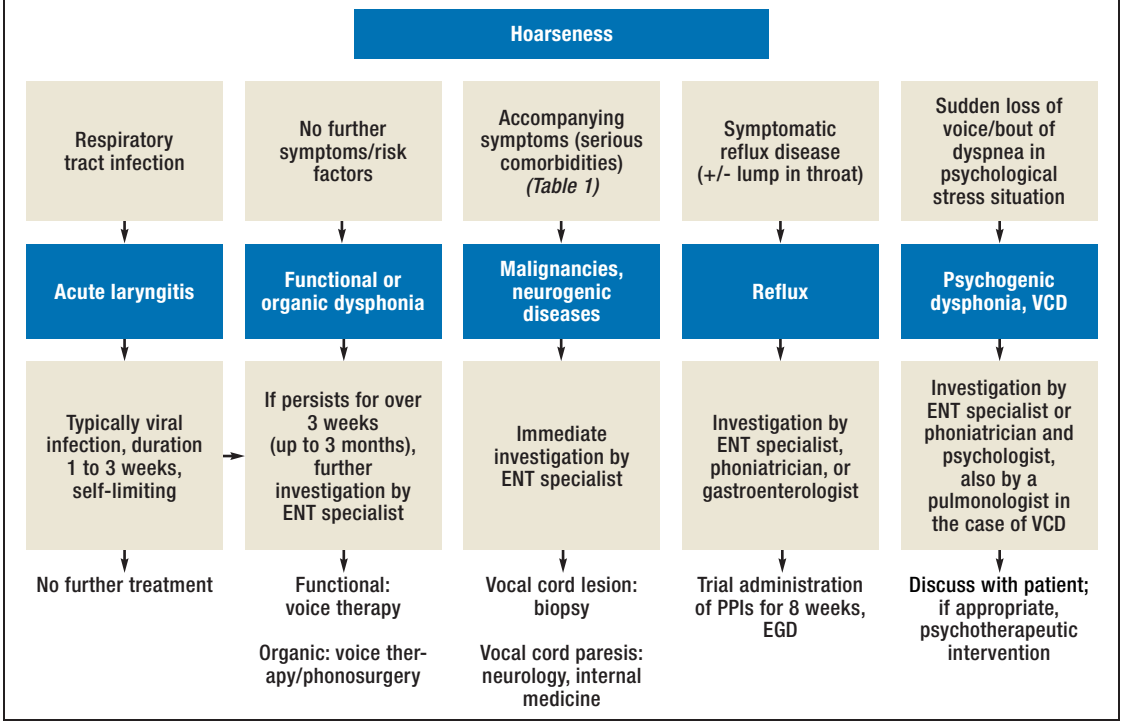
Benign and malignant tumors

Vocal cord polyps/vocal cord cysts – Vocal cord polyps are unilateral tissue proliferations on the free margin of the vocal cord and thus hamper phonation (8). Men are more frequently affected (55%) (e30). The factors promoting the formation of vocal cord polyps include smoking (51 to 90%) (8), chronic laryngitis, and phonation trauma, i.e., microvascular trauma with local edematous remodeling processes and accompanying inflammation as a result of misuse of the voice (9). Retention cysts arise when the excretory ducts of the mucous glands are obstructed. The symptoms are hoarseness together with reduced volume and fatigue of the voice. The treatment of choice for polyps is phonosurgical excision at the base. Cysts must be removed in toto with the capsule (evidence level 2a, recommendation grade B) (2) (9, e24, e31).

Reinke edema – Reinke edema is caused predominantly by tobacco smoke and mainly affects women (80%) between the ages of 40 and 60 years (ca. 47%) (e30). Phonosurgical removal of the edema results in improvement of the pitch, resonance, and also resilience of the voice. Dysplasia is rare (<1%) (e32). It is essential to stop smoking, although this does not always achieve marked amelioration of the edema (e33). As with polyps and cysts, additional voice therapy may be required to correct a reactive vocal dysfunction that has arisen preoperatively (evidence level 2a, recommendation grade B) (2).

Recurring papillomatosis – Juvenile (RJP) and adult (RAP) forms of recurring papillomatosis are distinguished. RJP usually arises between the ages of 2 and 4 years and is a major cause of hoarseness and also dyspnea in childhood (10–12). There are over 100 different types of human papilloma virus (HPV), the most important of which are HPV 6, 11, 16, and 18 (10, 13). In children, infection with HPV 11 has a severe course and may even lead to obstruction of the respiratory tract (12). Hoarseness is the cardinal symptom of RAP, the occurrence of which peaks between the ages of 20 and 40 years. A retrospective cohort study found epithelial dysplasia in 28% of cases (e34). Association of HPV with laryngeal cancer is rare (1.6 to 1.7%) (e35, e36), and association with squamous epithelial carcinoma of the lung has been described only in isolated cases (12, 14, e37). The precise mode of transmission is unknown (10). The primarily benign, cauliflower-like neoplasms are found mostly in the area of the vocal cords or extralaryngeally in the trachea, bronchi, or lungs (11). Papillomas are excised microsurgically (evidence level 2a, recommendation grade B) (2). There is insufficient evidence (15) to support adjuvant antiviral treatment with intralesional administration of cidofovir, which is currently licensed only for the treatment of cytomegalovirus (CMV) retinitis in AIDS patients (e38). Individual cases of successful active immunization against HPV in laryngeal papillomatosis have been reported, albeit with limited follow-up (13). The disease course

FIGURE 2



Algorithm for diagnosis of hoarseness.
ENT, ear, nose, and throat; VCD, vocal cord dysfunction; PPI, proton pump inhibitor; EGD, esophagogastroduodenoscopy; VC, vocal cord

varies from spontaneous remission over a stable stage to aggressive progression necessitating repeated interventions (11).

Vocal cord malignancies – Around two thirds of laryngeal cancers are located in the area of the vocal cords. The incidence in the general population is 7/100 000 (e39). Squamous epithelial carcinoma accounts for more than 90% of cases (7). In contrast to the strong association of HPV with tonsillar carcinoma (OR 15.1, 95% confidence interval [CI] 6.8 to 33.7), only a weak association has been demonstrated for laryngeal cancer (odds ratio [OR] 2.0, 95% CI 1.0 to 4.2) (e40). Dysphonia is considered an early symptom (7). In microlaryngostroboscopy, the term phonatory standstill is used to describe the state in which the fine vibrations of the tumor-infiltrated vocal cords are abolished. The swift occurrence of hoarseness leads to diagnosis of (glottic) laryngeal cancer at an early stage (T1) in 24 to 30% of cases (e39). Correspondingly, the rates of lymph-node and distant metastases at the time of diagnosis are low. The 5-year survival rate is practically 100%. The treatment comprises transoral (laser) resection or primary low-volume radiotherapy (e41). An up-to-date S3 guideline is currently being compiled. Treatment may be followed by hoarseness owing to scarring or, in the case of tissue loss, incomplete glottic closure (16). In this event, voice therapy often has a successful result (evidence level 1b, recommendation grade A) (e42).

Vocal cord scarring – Scarring of the sulcus between the vocal cord epithelium and the vocal muscle may be congenital, but in most cases occurs following severe laryngitis *(Figure 3)* or (phono)surgical interventions. Laryngoscopy of the area around the sulcus shows abolition of the fine vibrations or, in the event of significant tissue loss, incomplete glottic closure (16). The voice is constantly hoarse and may be breathy and poorly audible. Voice therapy usually only corrects the dysfunction, failing to achieve any relevant improvement in resonance, and surgery to release the scars or deal with the incomplete closure of the glottis is also challenging. Reported results mostly describe the authors' own experience; there are no prospective studies from which evidence-based treatment recommendations could be derived (17).

Presbyphonia

Presbyphonia, the physiological hoarseness of old age, is found in around 25% of those over 65 years of age. The frequency is about the same in men and women (e43). The vocal cord musculature atrophies in the course of the physiological aging process, giving rise to a more oval shape of the vocal cord fissure during phonation. Furthermore, the mucus-producing cells of the vocal cord also atrophy with age, so the surface film increases in viscosity, negatively influencing the sound of the voice. The leading symptom is a weak, less intense voice produced at the cost of pronounced strain.

TABLE 2

Characteristics of randomized controlled trials on treatment of hoarseness

Reference	Disease	n	Form of evaluation	Treatment procedure/group	Remarks
(e10)	Functional dysphonia (female teachers with voice problems)	44	1, 3	G1 = vocal hygiene (n = 15) G2 = voice amplifier (n = 15) G3 = no treatment (control) (n = 14)	G1 and G2 benefited compared with G3
(e9)	Functional dysphonia	30	1, 2, 3, 4	G1 = indirect treatment (n = 10) G2 = direct and indirect treatment (n = 10) G3 = no treatment (control) (n = 10)	G1 (60%) and G2 (90%) benefited compared with G3 (10%)
(e8)	Functional dysphonia	45	1, 2, 3, 4	G1 = indirect treatment (n = 10) G2 = direct and indirect treatment (n = 10) G3 = no treatment (control) (n = 10)	G1 (46%) and G2 (93%) benefited most; G3 (14%)
(e11)	Functional dysphonia (female teachers with voice problems)	40	2, 3	G1 = treatment (n = 22) G2 = no treatment (control) (n = 18)	Significant improvement in G1 compared with G2
(e12)	Functional dysphonia	50	1, 2, 5 (electro-glottography)	G1 = classic voice therapy (n = 26) G2 = voice therapy with visual biofeed-back, flexible transnasal (n = 25)	G1 und G2 benefited significantly; G2 was more effective
(e13)	Female teachers with voice problems	20	1, 4	G1 = voice therapy (n = 9) G2 = no treatment (control) (n = 11)	Significant improvement in G1 compared with G2
(e14)	Patients with functional dysphonia but without any other relevant organic pathology such as polyps or vocal cord paresis	133	1, 2, 3, 4	G1 = voice exercise treatment (n = 70) G2 = no treatment (control) (n = 63)	Significant improvement in G1 compared with G2
(e15)	Female student teachers with mild voice problems due to vocal cord edema or functional dysphonia	40	1, 2, 4	G1 = voice exercise treatment (group therapy) (n = 20) G2 = no treatment (control) (n = 20)	Significant improvement in G1 compared with G2
(e42)	Status post laser resection/irradiation of vocal cord cancer	23	1, 2, 4	G1 = voice therapy (n = 12) G2 = no treatment (control) (n = 11)	G1 benefited compared with G2
(e26)	Acute laryngitis	106	2, 4	G1 = erythromycin (n = 56) G2 = no treatment (control) (n = 50)	No differences in voice quality or laryngoscopy findings
(e25)	Acute laryngitis	100	2	G1 = penicillin V (n = 50) G2 = no treatment (control) (n = 50)	No differences in voice quality
(18)	Presbyphonia	16	1	G1 = VFE (n = 6) G2 = PhoRTE (n = 5) G3 = no treatment (control) (n = 5)	Significant improvement in G1 und G2 compared with G3
(36)	Spasmodic dysphonia of adductor type	13	3	G1 = administration of botulinumtoxin A (n = 7) G2 = no treatment (control) (n = 6)	Distinct improvement of voice in G1 compared with control G2

Summary of randomized controlled trials on treatment of hoarseness with evidence level 1a or 1b
Form of evaluation: 1 = assessment of quality of life; 2 = auditory perception; 3 = acoustic/technical analyses; 4 = laryngo(strobo)scopy; 5 = other
G, group; n, number of patients; VFE, vocal function exercise; PhoRTE, phonation resistance training exercise

Presbyphonia must be distinguished from organic disorders of the vocal cords and from other illnesses, e.g., chronic obstructive pulmonary disease (e44). Treatments that can be considered are voice therapy, which may help to regulate the tone and improve the subglottic air pressure, and phonosurgical measures to reinforce glottis closure (evidence level 2a, recommendation grade B) (18, e43).

Manifestation of internal diseases

Laryngopharyngeal reflux

Nine to 26% of the population (e5, e45) suffer from reflux-related mucosal irritation of the larynx and pharynx with chronic laryngitis (e45). Moreover, reflux is an important trigger factor for laryngospasm or vocal cord dysfunction (VCD). Up to 92% of these patients mention hoarseness, chronic urge to cough, throat

clearing, a lump in the throat, and unspecific swallowing difficulties (e45, e46). Laryngoscopy reveals vocal cord edema, mucosal erythema, or gastric laryngitis, i.e., mucosal hyperplasia with plication of the interarytenoid region in the posterior part of the larynx *(Figure 1a, b)* (e45, e46). Trial treatment with proton pump inhibitors is important in establishing the diagnosis (e47). Another treatment option is antireflux therapy, if hoarseness occurs in chronic laryngitis with signs of reflux disease (evidence level 2b, recommendation grade C). In the absence of reflux this treatment is inappropriate (evidence level 2a, recommendation grade B) (2, e4, e48). Overall, the diagnosis of laryngopharyngeal reflux (LPR) is assigned uncritically and too often among patients with dysphonia (e48). Accordingly, the symptomatic measures remain ineffective.

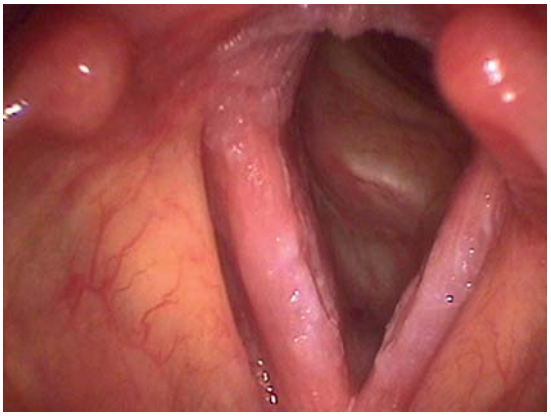


Figure 3
Laryngoscopy in a heavy smoker with chronic leukoplakia and bilateral sulcus vocalis. His voice sounded husky and strained

Internal diseases with occasional laryngeal manifestations

Tuberculosis (19, e49); rheumatoid diseases such as rheumatoid arthritis (20, e50), systemic lupus erythematosus (21, e51), Wegener disease (22, e52), and laryngeal sarcoidosis (23); amyloidosis (24, e53, e54); and manifestations of lymphoma (e55, e56) are among the internal diseases with occasional laryngeal involvement *(Table 1)*. In all these diseases interdisciplinary management is mandatory. The treatment is usually based on experience from case series (evidence level 3, recommendation grade D) (25, 26).

Neurogenic causes

Vocal cord paralysis

Vocal cord paralysis may be partial (reduced mobility) or complete, caused by damage to the recurrent laryngeal nerve; a dysphonia arises from the incomplete glottic closure or irregular vibration of the vocal cords. The majority of vocal cord paralyses (24 to 79%) can be attributed to iatrogenic causes, such as surgery or trauma in the region of the vagus nerve or the recurrent laryngeal nerve (27, 28, e57, e58). A prominent role is played by thyroid gland interventions, 0.5 to 2.3% of which result in permanent vocal cord paresis (29, 30). Other operations that have been described as causing vocal cord paralysis (28, 29) include surgery on the heart or aorta (e59), cervical spine surgery, and thoracic surgery (e58). Vocal cord paresis may be the first symptom of malignancy; this is the case in 0.9 to 1.6% of thyroid cancers (e60) and 1.5 to 43% of bronchial carcinomas (28, e57, e61). In 2 to 41% of cases the paralysis is idiopathic—the cause is never ascertained (27, 28, e57, e58, e62–e65).

The treatment should begin with speech therapy (evidence level 1a, recommendation grade A). If voice quality has shown no decisive improvement after 2 months and incomplete vocal cord closure persists, temporary vocal cord filling (injection glottoplasty/augmentation) is recommended, e.g., with hyaluronic acid (evidence level 2a, recommendation grade B) (32, e66–e70). Once the paresis has persisted for 12 months, recovery is unlikely. In this case autologous fat (less resorbable) should be used for injection glottoplasty (e71), or external thyroplasty can be performed (32, e72).

Spasmodic dysphonia

Spasmodic dysphonia (SD) is one of the focal dystonias. It occurs almost exclusively in adulthood and predominantly in women (33). This severe dysphonia leads to involuntary spasms of the laryngeal musculature with increased adduction or abduction of the vocal cords, depending on subtype. Sensorimotor control of the larynx is affected, probably owing to a neurotransmitter disorder (34). The adductor type of SD, found in 90% of cases, is characterized by the vocal cords pressing harder against each other during phonation. This results in a creaky voice and intermittent voice breaks during speech (so-called vocal stuttering) (e73). The abductor type, in the remaining 10% of patients, leads to voiceless phases with breathy intonation. Treatment comprises injection of the neurotoxic protein botulinum toxin into the affected vocal cord muscles (evidence level 2a, recommendation grade B) (2, 34–36, e74).

Vocal cord dysfunction

VCD, sometimes called "laryngeal asthma," is an intermittent, functional laryngeal obstruction causing dyspnea. It occurs due to laryngeal hyperreactivity on inspiration. A multifactorial etiology is assumed. Proposed trigger mechanisms include repeated exposure of the larynx to irritative inhaled stimuli such as perfumes or allergens and microaspiration in the presence of laryngopharyngeal reflux (37, e75, e76). Patients with VCD subjectively experience the bouts of respiratory distress as life-threatening and often develop secondary anxiety and panic attacks. The episodic dyspnea with stridor is accompanied by further symptoms such as dysphonia or aphonia. Many years often elapse before VCD is diagnosed. Flexible transnasal laryngoscopy is considered the gold standard for

diagnosis and shows the paradoxical vocal cord motion with adduction on inspiration. VCD may occur in isolation but is also found in 3 to 5% of asthmatics (37). In contrast to the classical bronchial asthma, medicinal therapy is ineffective (37, e77, e78). The episodes of respiratory distress can usually be controlled well by means of special breathing techniques (37). Psychotherapy is sometimes also recommended, particularly for patients with secondary anxiety and panic attacks, but the fact that predominantly case series have been published means there is no sufficient evidence of its effect (e79).

Psychogenic dysphonia

Psychogenic dysphonia preferentially affects women between the ages of 20 and 40 years (e80). The patients complain of sudden extreme hoarseness or even acute aphonia. By contrast, loud coughing or throat clearing is still possible, i.e. vocal function is impaired only in communicative contexts. Laryngoscopy shows no inflammation, but sometimes there is muscle tone-related limitation of adduction of the vocal cords during phonation (e81, e82). These findings are often incorrectly diagnosed and treated as acute laryngitis (38, e80). Our investigation of 40 patients showed that psychogenic dysphonia is often an acute manifestation preceded by severe psychological stress (38). Behavioral psychotherapy is helpful (38, e81), whereas voice therapy is completely ineffective (38–40).

Conflict of interest statement
Prof. Reiter has received study support (third-party funding) and reimbursement of travel costs from bess medizintechnik.

The remaining authors declare that no conflict of interest exists.

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KEY MESSAGES

- Hoarseness has many different causes and interdisciplinary management is often required.
- In the presence of risk factors, investigation by laryngoscopy is mandatory.
- Only a small number of randomized controlled trials of treatment for hoarseness have been published.
- Options for treatment are voice therapy and phonosurgery, i.e., an operation to improve the voice.
- Trial use of antibiotics to treat hoarseness is strongly discouraged.

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REVIEW ARTICLE

Hoarseness—Causes and Treatments

Rudolf Reiter, Thomas Karl Hoffmann, Anja Pickhard, Sibylle Brosch

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eTABLE

Definition and classification of evidence levels and recommendation grades

Evidence level	Strength	Definition	Recommendation
1a	A	Meta-analyses, systematic reviews of randomized controlled trials, megatrials	"Should"
1b		Randomized controlled trials	
2a	B	Non-randomized studies, cohort studies with control groups	"Probably should"
2b	C	Observational studies (case–control studies, cohort studies)	"May"
3	D	Cross-sectional studies, ecological studies, cohort studies without control group, case reports	GCP
4	X	Clinical experience	GCP

Classification according to tables of the AAP SCQIM (2, e2) and a DEGAM guideline (e3)
AAP SCQIM, American Academy of Pediatrics Steering Committee on Quality Improvement and Management; DEGAM, German College of General Practitioners and Family Physicians; GCP, good clinical practice