



Original Article

Laryngeal Lesions in Patients with Chronic Cough and Normal Chest Radiographs and Auscultation

Jiann-Jy Chen^{1,2,3*}

¹Department of Otorhinolaryngology, Taoyuan Hospital, Department of Health, Executive Yuan, Taoyuan, Taiwan

²Department of Otorhinolaryngology, Chu-Tung Hospital, Department of Health, Executive Yuan, Hsinchu, Taiwan

³Faculty of Medicine, School of Medicine, Fu Jen Catholic University, Taipei, Taiwan

Article info

Article history:

Received: January 2, 2007

Revised: February 8, 2007

Accepted: February 28, 2007

Keywords:

Chronic cough
Flexible fiberoptic
nasopharyngoscope
Laryngeal lesion

Abstract

Objectives: Chronic cough, defined as a cough persisting for more than 8 weeks, is a common chief complaint in the outpatient department. The role of laryngeal lesions in these patients is discussed in this paper.

Materials and Methods: Seventy-seven patients (29 men, 48 women) were enrolled retrospectively at my clinics in one regional hospital and two local hospitals located in Taoyuan county and in Hsinchu county, from January to June, 2006. Their ages ranged from 19 to 83 years (mean, 52.7 years). The duration of symptoms was from 8 weeks to over 2 decades (70.1% of the patients: ≤ 3 years). Before examination with a flexible fiberoptic nasopharyngoscope, obvious bronchopulmonary disease had been ruled out using chest radiography and chest auscultation.

Results: Of the patients examined, 61.0% had laryngeal lesions, including vocal fold atrophy (61.7%), vocal fold sulcus (19.1%), vocal nodules (14.9%), vocal polyps (8.5%), vocal corditis (4.3%), unilateral vocal cord palsy (2.1%), and laryngeal neoplasm (2.1%). Of the patients with laryngeal lesions, 48.9% complained of hoarseness. Among patients with hoarseness, 88.5% had laryngeal lesions. There was a statistically significant difference in the presence of hoarseness between patients with and without laryngeal lesions ($p < 0.05$, χ^2 test). Patients without laryngeal lesions had a higher cure rate for chronic cough than patients with these lesions ($p < 0.05$, χ^2 test) after 10 patients who were lost to follow-up were excluded.

Conclusion: Both the larynx and its dynamics should be evaluated together when a patient presents with chronic cough because laryngeal lesions are comitant. In addition, laryngeal malignancy needs early diagnosis and early treatment. (*Tzu Chi Med J* 2007;19(3):145–151)

*Corresponding author. Department of Otorhinolaryngology, Taoyuan Hospital, Department of Health, Executive Yuan, 1492, Jhongshan Road, Taoyuan, Taiwan.
E-mail address: jiannjy@yahoo.com.tw

1. Introduction

Chronic cough, defined as a cough of at least 8 weeks duration (1,2), is a common chief complaint in the outpatient department (OPD). Chronic cough is a very complicated condition, and the key to treatment is to find the exact cause. A complete medical history, physical examination, chest radiograph (1), methacholine inhalation challenge test (2), and therapeutic trials (2 weeks of therapy with antihistamines, bronchodilators/inhaled steroids, or antireflux therapy) (3) are recommended. Other diagnostic modalities include pulmonary function tests, continuous 24-hour dual-probe esophageal pH monitoring, esophageal manometry, sinus imaging (4), cough challenges with a tussigenic agent, sputum analysis, exhaled nitric oxide test, and bronchoscopy (5). Asthma, acid reflux, and rhinosinusitis are three common diagnoses that arise from three different anatomic areas (1–3). Others include cigarette smoking, angiotensin-converting enzyme inhibitor treatment, cough variant asthma, eosinophilic bronchitis, irritation of the external auditory canal, infection (pertussis, tuberculosis, viruses), foreign body aspiration, aspiration pneumonia, immunodeficiency, primary ciliary dyskinesia, tracheobronchomalacia, tracheoesophageal fistula, congenital cardiovascular anomaly, and psychogenic cough.

The larynx is the gate to the airway. Laryngeal lesions may cause hoarseness and airway symptoms (6), such as cough, dyspnea, and choking. It is difficult to find laryngeal lesions on a chest radiograph and routine physical examination, but a flexible fiberoptic nasopharyngoscope is a useful tool to look for laryngeal lesions in patients with chronic cough (7).

2. Materials and methods

2.1. Patients

This retrospective study investigated 84 patients with chronic cough treated at my clinics in one regional hospital and two local hospitals located in Taoyuan county and in Hsinchu county from January to June, 2006. Chest auscultation and routine ear, nose and throat (ENT) examinations were performed, and chest radiographs were obtained. One patient with cerumen impaction and six patients with obvious bronchopulmonary disease were excluded. A total of 77 patients, including 48 women and 29 men (Table 1), were examined with flexible fiberoptic nasopharyngoscopy. The average age of the 77 patients was 52.7 years (range, 19–83 years). Of these, 59.8% were between 40 and 70 years old. The duration of symptoms was 8 weeks to over 20 years (≤ 3 years: 70.1%). The common coincident symptoms were hoarseness

Table 1 — Basic data of the 77 candidates

	n (%)
Gender	
Male	29 (37.7)
Female	48 (62.3)
Duration of cough	
2 mo – 1 yr	41 (53.2)
1 yr – 3 yr	13 (16.9)
> 3yr	23 (29.9)
Age (yr)	
< 10	0 (0)
≥ 10 , <20	1 (1.3)
≥ 20 , <30	10 (13.0)
≥ 30 , <40	8 (5.5)
≥ 40 , <50	13 (16.9)
≥ 50 , <60	19 (27.3)
≥ 60 , <70	12 (15.6)
≥ 70 , <80	12 (15.6)
≥ 80	2 (2.6)
Mean age	52.7
Coincident symptom	
Hoarseness	26 (33.8)
Globus pharyngitis	22 (28.6)
Tinnitus/hearing-block	12 (15.6)
PND/rhinorrhea	8 (10.4)
Sore throat	3 (3.9)
Choke	3 (3.9)
Headache	3 (3.9)
Thirst	2 (2.6)
Dysphagia	2 (2.6)
Chest pain/tightness	2 (2.6)
Hemoptysis	2 (2.6)
Dizziness	2 (2.6)
Insomnia	2 (2.6)
Itchy ear	1 (1.3)
Stuffy nose	1 (1.3)
Pitting edema	1 (1.3)
Sore palate	1 (1.3)
Facial swelling	1 (1.3)
Hyposmia	1 (1.3)
None	17 (22.1)
Past history	
Yes	24 (31.2)
Hypertension/hypertensive heart disease (without taking ACEI)	12
Diabetes mellitus	4
Chronic otitis media	3
Gastric ulcer	3
Benign prostatic hyperplasia	2
Gout	1
Rheumatoid arthritis	1
Hepatic disease	1
Arrhythmia	1
Mitral valve prolapse	1
Psychosis	1
VBI	1
State of post-thyroidectomy	1
No	53 (68.8)

PND = postnasal drip; ACEI = angiotensin-converting enzyme inhibitors; VBI = vertebral-basilar artery insufficiency.

(33.8%), globus pharyngis (28.6%), tinnitus/hearing-block (15.6%), and postnasal drip/rhinorrhea (10.4%). Fifty-three patients (68.8%) had no significant history of disease. Twelve patients had hypertension/hypertensive

heart disease, but did not take angiotensin-converting enzyme inhibitors.

2.2. Fiberoptic nasopharyngoscopic examination

Patients were examined in the ENT-OPD. The patients were seated comfortably in an ENT chair. Four ENT cotton sticks impregnated with a solution of 2% lidocaine + epinephrine (1:200,000) were placed in the common nasal meati and middle nasal meati for 5 minutes. Then a flexible fiberoptic nasopharyngoscope was carefully inserted into one side of the nasal cavity. A solution of 70% alcohol was applied to the surface of the scope to prevent moisture accumulation. A large high-resolution view was obtained using a charge-coupled device camera and monitor. The ipsilateral nasal cavity, nasopharynx, and oropharynx were inspected with the patient's mouth closed and breathing through the nose. The hypopharynx and larynx were inspected with the patient breathing through the mouth, and phonating a long "e" sound. The dynamics of the larynx were inspected during speech, or when breathing through the nose with two or three quick breaths. The ipsilateral middle nasal meatus was inspected while the scope was being pulled out. The same method was performed on the other side of the nasal cavity and the middle nasal meatus. The view was shown on a monitor and explained to the patient during the procedure.

2.3. Specific therapy

After specific therapy was given, the results of the examination and the progress of disease were recorded. Specific therapy included antihistamines, antitussives, mucolytics, antibiotics, decongestants, steroids, antireflux therapy or surgery. Among the 77 patients studied, one had laryngeal neoplasm, one had vocal polyp, and one had vocal fold atrophy. These patients received surgery initially. The others received conservative treatment. All patients were followed in the 1st or 2nd week, then in the 1st month, and then once every month until they were symptom-free. It was expected that the cough could be controlled or cured within 3 months after a specific therapy was given. Therefore, after the last patient visit on June 30, 2006, data collection continued until the end of September 2006.

2.4. Data collection

Data were collected and communicated to patients by telephone from September 29 to October 1, 2006 (3 days). Patients were considered cured if (1)

there were no complaints of cough in either the OPD or during the telephone call, and (2) there were no complaints of cough in the OPD of any other department which was again confirmed by a telephone call, and no treatment related to coughing had been given. Patients were considered being treated if (1) the cough was not cured, and the patient was still being followed up in the OPD every month at the end of September or (2) the cough was not cured, and the patient was transferred to and followed by other departments every month until the end of September. Patients were considered lost to follow-up if (1) they had visited the OPD only once and could not be contacted as of the end of September or (2) they had visited the OPD several times and the cough was being treated, but they had not returned to the OPD as of the end of September.

2.5. Statistical analysis

χ^2 test or Fisher's exact test was used for statistical analysis with an alpha of 0.05.

3. Results

There was no iatrogenic trauma in this study. The results of fiberoptic nasopharyngoscopy are listed in Table 2. The four categories of diagnosis of chronic cough were postnasal drip (rhinosinusitis and allergic rhinitis), oropharyngeal masses (chronic hypertrophic tonsillitis, epiglottic cyst, and lingual tonsil hypertrophy), suspected laryngopharyngeal reflux, and laryngeal lesions (vocal corditis, vocal polyp, vocal nodule, vocal fold atrophy, vocal fold sulcus,

Table 2 — Flexible fiberoptic nasopharyngoscope findings

Site	Finding	n
Nasal cavity	Postnasal drip	46
	Nasal septal deviation	6
	Nasal septal perforation	1
Larynx	Vocal fold atrophy	29
	Vocal fold sulcus	9
	Vocal nodules	7
	Vocal polyp	4
	Vocal corditis	2
	Unilateral vocal cord palsy	1
	Laryngeal neoplasm (malignancy)	1
Nasopharynx	Nasopharyngeal mass/ adenoid hypertrophy	5
Oropharynx	Lingual tonsil hypertrophy	4
	Epiglottic cyst	2
	Chronic hypertrophic tonsillitis	1
	Ω shape epiglottis	1
Hypopharynx	Suspected laryngopharyngeal reflux	13

Table 3 — Lesions related with chronic cough under flexible fiberoptic nasopharyngoscope

A. Only 1 category group	n
Laryngeal lesion	21
VFA	10
VN + VFA	3
VFS + VFA	2
VN	2
VP	1
VFS	1
VP + VFA	1
Laryngeal neoplasm (malignancy)	1
PND	16
R/O LPR	5
Total	42
B. Combined 2 category group	n
Laryngeal lesion + PND	20
VFA + PND	10
VFS + PND	6
VC + PND	2
UVCP + PND	1
VP + PND	1
PND + oropharyngeal mass	2
PND + chronic hypertrophic tonsillitis	1
PND + EC	1
PND + R/O LPR	5
Laryngeal lesion + oropharyngeal mass	2
VFA + LTH	1
VP + oropharyngeal mass	1
Laryngeal lesion + R/O LPR	1
VFA + R/O LPR	1
Oropharyngeal mass + R/O LPR	1
EC + R/O LPR	1
Total	31
C. Combined 3 category group	n
Laryngeal lesion + PND + oropharyngeal mass	2
VFA + PND + LTH	1
VN + PND + LTH	1
Laryngeal lesion + PND + R/O LPR	1
VN + PND + R/O LPR	1
Total	3

VFA = vocal fold atrophy; VN = vocal nodule; VFS = vocal fold sulcus; VP = vocal polyp; PND = postnasal drip; R/O LPR = suspected laryngopharyngeal reflux; VC = vocal corditis; UVCP = unilateral vocal cord palsy; EC = epiglottic cyst; LTH = lingual tonsil hypertrophy.

unilateral vocal cord palsy, and laryngeal neoplasm). In the literature, studies have not proven a direct relationship between nasal septal deviation, nasal septal perforation, nasopharyngeal mass/adenoid hypertrophy, or Ω shape epiglottitis and chronic cough. Of all patients, 54.5% had nasal conditions in only one category (Table 3A, $n=42$); 40.3% had conditions in two categories (Table 3B, $n=31$); and 3.9% had

Table 4 — Correlation between laryngeal lesions and hoarseness in chronic cough*

	Hoarseness ($n=26$)	No hoarseness ($n=51$)
With laryngeal lesions ($n=47$)	23	24
Without laryngeal lesions ($n=30$)	3	27

*Statistical analysis using χ^2 test with an alpha of 0.05.

Table 5 — Results of specific therapy*

A. Within the first 2 weeks after an initial treatment			
	Cough relieved	Cough persisted	Lost to follow-up
With laryngeal lesions ($n=47$)	35	10 ^{†‡}	2
Without laryngeal lesions ($n=30$)	24	3	3
Total ($n=77$)	59	13 ^{†‡}	5
B. Final summarizing period — from September 29 to October 1, 2006 (3 days)			
	Cured	Being treated	Lost to follow-up
With laryngeal lesions ($n=47$)	22 [†]	18 [‡]	7
Without laryngeal lesions ($n=30$)	22	5	3
Total ($n=77$)	44 [†]	23 [‡]	10

* χ^2 test or Fisher's exact test used for statistical analysis with an alpha of 0.05; [†]2 cases received autogenous fat injection laryngoplasty and microlaryngeal surgery respectively; [‡]2 cases were transferred to other departments—a patient with vocal fold atrophy and gastroesophageal reflux disease was transferred to the department of internal medicine, and a patient with laryngeal neoplasm found by me and laryngeal malignancy proven at another hospital was finally transferred back to my hospital and radiotherapy was then implemented.

problems in three categories (Table 3C, $n=3$). One patient (1.3%) had negative findings.

Sixty-one percent of the 77 patients had laryngeal lesions. Most of them had vocal fold atrophy (29/47, 61.7%), and the others had vocal fold sulcus (9/47, 19.1%), vocal nodules (7/47, 14.9%), vocal polyps (4/47, 8.5%), vocal corditis (2/47, 4.3%), unilateral vocal cord palsy (1/47, 2.1%), and laryngeal neoplasm (1/47, 2.1%). Twenty-three of the 47 patients (48.9%) with laryngeal lesions complained of hoarseness, and 23 of 26 patients (88.5%) with hoarseness had laryngeal lesions. There was a statistically significant difference in hoarseness between patients with and without laryngeal lesions ($p=0.0004$, χ^2 test; Table 4).

After the specific therapy was given, patients were divided into two groups — those with laryngeal lesions (group 1) and those without laryngeal lesions (group 2) (Table 5). Within the first 2 weeks after initial

treatment, 74.5% of group 1 and 80.0% of group 2 patients had relief of cough; 21.3% of group 1 and 80.0% of group 2 had a persistent cough. Five patients were lost to follow-up. After these five patients were excluded, there was no statistically significant difference in therapeutic effect between the two groups ($p=0.132$, Fisher's exact test). At the end of September, 46.8% of group 1 and 73.3% of group 2 had relief of their cough; 38.3% of group 1 and 16.7% of group 2 had been treated but were not cured. Ten patients were lost to follow-up. After these 10 patients were excluded, there was a significant difference in the cure rate between the two groups ($p=0.025$, χ^2 test). But the number of patients with each of the seven types of laryngeal lesions (vocal corditis, vocal polyp, vocal nodule, vocal fold atrophy, vocal fold sulcus, unilateral vocal cord palsy, and laryngeal neoplasm) was too small to obtain statistical analysis of therapeutic effect.

4. Discussion

Chest auscultation and chest radiography are mandatory in patients with chronic cough to look for obvious bronchopulmonary lesions. However, normal chest auscultation and normal chest radiography do not necessarily mean that the chronic cough is not caused by a bronchopulmonary lesion. For example, in the early stages of chronic bronchitis, tuberculosis, bronchiectasis, and other pulmonary diseases, chest radiography and auscultation alone may fail to detect the problem. However, these basic and easy diagnostic methods are used first by primary-care physicians in rural regional hospitals with only basic equipment and low staffing. It is possible that one or more of these 77 patients had bronchopulmonary disease.

The dynamics of the larynx cannot be established by a medical history or physical examination, but can be determined with nasopharyngoscopy (8). Laryngeal lesions, such as superior laryngeal nerve palsy (9), vocal hyperfunction (10), and paradoxical vocal cord movement (11) can be diagnosed. However, allergic disease affecting the pharyngolarynx, and lesions outside the nasal cavity, pharynx, and larynx cannot be diagnosed by flexible fiberoptic nasopharyngoscopy. For example, one patient with an impression of vocal fold atrophy had a cough that persisted after 2 months of conservative treatment. He had gastroesophageal reflux disease that was diagnosed using upper gastrointestinal endoscopy. After antireflux therapy, his cough was cured.

It is controversial to refer to laryngopharyngeal reflux as a type of laryngeal lesion because laryngopharyngeal reflux arises from gastric acid reflux, and then affects the upper esophagus, hypopharynx,

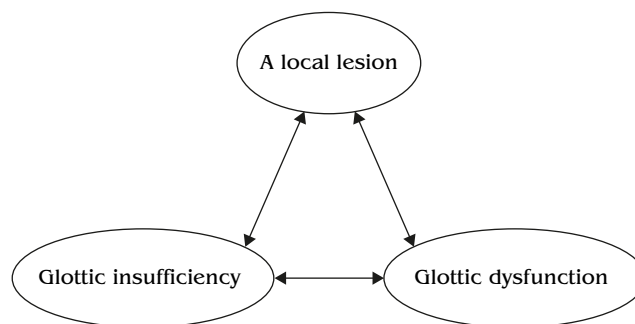


Fig. 1 — Three types of laryngeal lesions may coexist, interact, and be comitant.

larynx, oropharynx, or even the nasopharynx. It must be diagnosed using continuous 24-hour dual-probe esophageal pH monitoring (3), and then treated with antireflux therapy. However, no patient complained of heartburn in this study. Without esophageal pH monitoring, I was forced to inspect injection of the arytenoid regions, interarytenoid region, post-cricoid region, false vocal cords, or vocal cords. In addition, allergic or infected pharyngolaryngitis has an appearance similar to laryngopharyngeal reflux. A therapeutic trial of antireflux therapy can be used when laryngopharyngeal reflux is suspected. Laryngopharyngeal reflux may be independent of laryngeal lesions, so it may be more appropriate to use the term *suspected laryngopharyngeal reflux*. The results of this study show that laryngeal lesions play an important role in chronic cough. Not all patients with laryngeal lesions complained of hoarseness, but most patients with hoarseness had laryngeal lesions. Therefore, there appears to be a relationship between laryngeal lesions and hoarseness ($p=0.0004$, χ^2 test; Table 4). A literature review indicated that these laryngeal lesions can be classified into three patterns: a local lesion, glottic insufficiency, and glottic dysfunction. The three patterns may exist together, interact, and be comitant (Fig. 1).

A local lesion of the larynx is defined as an inflammatory or anatomic problem in the larynx, such as a vocal nodule, a vocal polyp, vocal corditis, vocal fold sulcus, laryngeal neoplasm (5), laryngeal cleft (1), and eversion of the laryngeal ventricle (12). It may disturb the exit of airway mucus. When enough mucus accumulates on a laryngeal lesion, a cough reflex or voluntary phonation may be generated to expel it out with a fast airflow or vibration of the cords. The patient continually coughs and clears the throat. On fiberoptic nasopharyngoscopy, the physician may see a little phlegm coating the vocal cords. If a laryngeal local lesion becomes more serious, it may block glottic closure, and then lead to glottic insufficiency and glottic dysfunction.

Glottic insufficiency is defined as incomplete glottic closure. It is seen in vocal fold atrophy and unilateral vocal cord palsy. Patient with glottic insufficiency have hoarseness, cough, choking, or aspiration, especially when eating, drinking, swallowing saliva, and hiccupping, and when they have nasal congestion or an upper respiratory infection. Because food debris, water, gastric acid, saliva, or nasal secretions are aspirated incidentally, a protective cough reflex occurs. But the subglottic pressure is not high enough to cause a cough or phonation. The patient often complains of hoarseness, vocal fatigue, thirst, and difficulty coughing up phlegm. In order to improve glottic closure, false vocal cords and extrinsic laryngeal muscles may compensate (13), and gradually generate glottic dysfunction. A vocal nodule, a vocal polyp, or vocal corditis may then develop. In this study, most cases of glottic insufficiency (29/47, 61.7%) resulted from vocal fold atrophy.

Glottic dysfunction is defined as uncoordinated or hyperfunctional movement of the larynx disturbing the exit of phlegm and airflow, such as in superior laryngeal nerve palsy (9), laryngospasm (14), vocal hyperfunction (10), or paradoxical vocal cord movement (11). When vocal cords always vibrate unequally and closure is uncoordinated, the laryngeal mucosa is easily injured. Therefore, a globus sensation, or a subsequent local lesion, such as a vocal nodule, a vocal polyp, and vocal corditis, may develop.

It is mandatory to treat chronic cough with therapy specific to the etiology. Most laryngeal lesions are not easy to cure, so surgery is suggested if necessary. In this study, fewer patients with chronic cough with laryngeal lesions were cured than those without laryngeal lesions, and there was a statistically significant difference in the cure rate ($p=0.025$, χ^2 test; Table 5B). Early diagnosis can give patients a sense of security, and appropriate therapy decreases the possibility of unnecessary treatment or examinations. Laryngeal malignancy requires early diagnosis and early treatment. Before treatment, laryngeal lesions should be differentiated into primary and secondary laryngeal lesions.

A primary laryngeal lesion is a lesion that is located on the larynx, such as a vocal nodule, vocal fold atrophy, vocal fold sulcus, superior laryngeal nerve palsy, unilateral vocal cord palsy, or laryngeal neoplasm. Other non-laryngeal lesions seldom cause a primary laryngeal lesion. There may be no symptoms in the early stage of disease or when there is good compensation. When the disease progresses to a stage too serious to benefit from compensation, it becomes clinically symptomatic. However, a vicious cycle of chronic cough starts if a silent laryngeal lesion meets a non-laryngeal lesion and interacts (Fig. 2). Therefore, in order to break the vicious cycle, specific therapies for multiple lesions should

be given simultaneously. Then, symptoms of laryngeal lesion will abate, or they can be controlled until further treatment is implemented.

Speech therapy and mucolytics are recommended for a local lesion or glottic insufficiency. Microlaryngeal surgery, laryngoplasty or thyroplasty may be suggested if conservative treatment fails to cure the problem. The use of mucolytics can decrease the viscosity of phlegm, prevent it from accumulating in laryngeal lesions, and relieve cough. However, the use of antitussives alone is controversial because they may elevate the threshold of the cough reflex, cause more phlegm accumulation in laryngeal lesions, and lead to a more serious cough or further pulmonary problems.

Speech therapy and mucolytics are recommended for glottic dysfunction. Antitussives or sedatives are useful to decrease abnormal movement of the larynx and possible injury to the laryngeal mucosa, to prevent a secondary laryngeal lesion. Furthermore, botulinum toxin injection is suggested if conservative treatments fail. Surgical biopsy is mandatory for laryngeal neoplasm. If malignancy is proven pathologically, aggressive treatment should be implemented after staging of the lesion.

A secondary laryngeal lesion is induced after a non-laryngeal lesion or a primary laryngeal lesion. For example, a vocal polyp or vocal corditis develops from inflammation; a vocal nodule develops physically; some vocal dysfunction develops physiologically. It causes a globus sensation, and disturbs the exit of airway mucus, so a vicious cycle of chronic cough forms (Fig. 2). For example, acid reflux irritates the esophagus or trachea and causes cough. It also possibly irritates the larynx directly, and causes vocal leukoplakia, vocal granuloma or vocal corditis. Under these circumstances, the cough becomes severe. Lingual tonsil hypertrophy and postnasal drip cause a cough directly. After repeated glottic closures, a vocal polyp or vocal corditis develops from inflammation and the cough becomes severe. A secondary laryngeal lesion may interact with the original lesions and induce further laryngeal lesions. Therefore, specific therapies should be given simultaneously for multiple lesions to break the vicious cycle.

Chronic cough has complicated causes. A complete medical history, physical examination, chest radiography, and other tests should be done. It is possible that there were potential bronchopulmonary lesions, allergic diseases affecting the pharyngolarynx, and other lesions beyond the realm of the flexible fiberoptic nasopharyngoscope in this retrospective study. Patients without laryngeal lesions had a higher cure rate for chronic cough than patients with laryngeal lesions ($p < 0.05$, χ^2 test). But the number of patients with each of the seven types of laryngeal lesions (vocal corditis, vocal polyp, vocal nodule, vocal fold atrophy, vocal fold sulcus, unilateral vocal cord palsy,

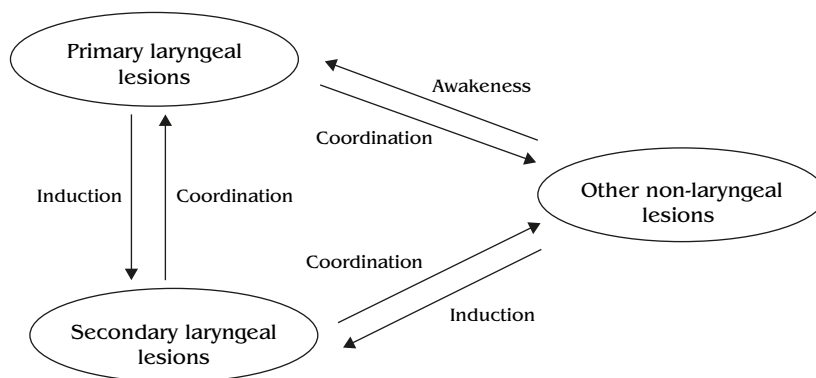


Fig. 2 — Laryngeal lesions play a role in the vicious cycle of chronic cough.

and laryngeal neoplasm) was too small to obtain statistical analysis of therapeutic effect. Therefore, both the larynx and its dynamics should be evaluated together because laryngeal lesions are comitant.

Acknowledgments

I would like to thank both Kuen-Wang Tai, a senior college English instructor, and John K. Lin, a senior otorhinolaryngologist, for proofreading the paper.

References

1. Morice AH, Fontana GA, Sovijarvi AR, et al. The diagnosis and management of chronic cough. *Eur Respir J* 2004; 24:481-92.
2. Irwin RS, Pratter MR, Holland PS, Corwin RW, Hughes JP. Postnasal drip causes cough and is associated with reversible upper airway obstruction. *Chest* 1984;85:346-52.
3. Lawler WR. An office approach to the diagnosis of chronic cough. *Am Fam Physician* 1998;58:2015-22.
4. Pratter MR, Bartter T, Lotano R. The role of sinus imaging in the treatment of chronic cough in adults. *Chest* 1999; 116:1287-91.
5. Sen RP, Walsh TE. Fiberoptic bronchoscopy for refractory cough. *Chest* 1991;99:35-5.
6. Woodson GE. Upper airway anatomy and function. In: Bailey BJ. *Head & Neck Surgery-Otolaryngology*, 3rd edition. Philadelphia: Lippincott Williams & Wilkins, 2001: 479-87.
7. Guo YC, Chu PY, Tai SK, Chang SY. Removal of laryngopharyngeal foreign bodies under flexible videolaryngoscopy. *J Taiwan Otolaryngol Head Neck Surg* 2001;36: 277-81.
8. Selkin SG. Kinolaryngoscopy for documentation of laryngeal pathophysiology. *Laryngoscope* 1984;94:58-62.
9. Adour KK, Schneider GD, Hilsinger RL Jr. Acute superior laryngeal nerve palsy: analysis of 78 cases. *Otolaryngol Head Neck Surg* 1980;88:418-24.
10. Zwitman DH, Calcaterra TC. The "silent cough" method for vocal hyperfunction. *J Speech Hear Disord* 1973;38: 119-25.
11. Murry T. Chronic cough: in search of the etiology. *Semin Speech Lang* 1998;19:85-91.
12. Templer JW, Baker BB, Hemenway WG. Eversion of the laryngeal ventricle. *Arch Otolaryngol* 1975;101:37-8.
13. Belafsky PC, Postma GN, Reulbach TR, Holland BW, Koufman JA. Muscle tension dysphonia as a sign of underlying glottal insufficiency. *Otolaryngol Head Neck Surg* 2002;127:448-51.
14. Morrison M, Rammage L, Emami AJ. The irritable larynx syndrome. *J Voice* 1999;13:447-55.