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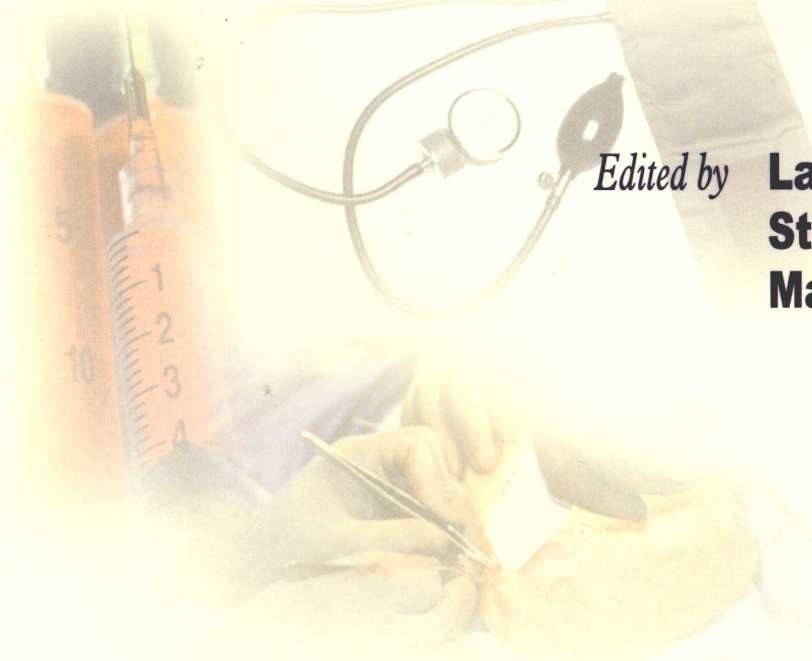
现代内科学英语精要

Current Medical Diagnosis & Treatment



Edited by **Lawrence M. Tierney, Jr.**
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人民卫生出版社
Education

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图书在版编目（CIP）数据

现代内科学英语精要/王吉耀等摘编. -北京：人民卫生出版社，2001

ISBN 7-117-04459-4

Ⅰ.现… Ⅱ.王… Ⅲ.内科学-英文 Ⅳ.H31

中国版本图书馆 CIP 数据核字(2001)第 076626 号

现代内科学英语精要

Current Medical Diagnosis & Treatment

摘　　编：王吉耀　刘文忠
出版发行：人民卫生出版社(中继线 67616688)
地　　址：(100078)北京市丰台区方庄芳群园 3 区 3 号楼
网　　址：http://www.pmph.com
E - mail：pmph@pmph.com
印　　刷：北京人卫印刷厂(天运)
经　　销：新华书店
开　　本：850×1168　1/16　　**印张：**33.5
字　　数：1115 千字
版　　次：2002 年 12 月第 1 版　2004 年 7 月第 1 版第 2 次印刷
标准书号：ISBN 7-117-04459-4/R·4460
定　　价：42.00 元

序

英语是世界医学领域不可由其他语言替代的通用语言。作为医学科技工作者，只有掌握英语，才能顺利阅读日益增多的医学文献和原著或熟练地从因特网上获取医学专业发展的前沿信息，从而充实提高自己的医学专业知识；也只有掌握英语才能将自己研究的成果或经验体会报道和发表出去。

对于医学生和青年医生来说，有了一定的公共英语基础知识以后，尽快接触医学专业英语，掌握一定量的医学专业单词，有目的地训练自己在英语方面的听说读写能力，对从事专业上的对外交流和对内传播有着极其重要的意义。正是由于这个原因，经全国高等医药教材建设研究会研究决定，由人民卫生出版社邀请内、外、妇、儿四大专业具有良好英语功底和丰富临床经验的专家选摘编写了这套“现代医学英语精要”系列丛书。我相信这套丛书在引导医学生和青年医生获得医学知识的同时，还可以使他们学习到规范的医学专业英语单词和语句，对提高他们阅读英语文献和原著的能力将有很大的帮助。希望读者认真利用这套丛书，体会医学专业英语的精髓、特点和使用习惯，举一反三，触类旁通，不断提高自己的医学英语水平。

裘法祖

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2002年10月1日

前　言

本书为全国高等医药院校五年制和七年制规划教材《内科学》的英文配套教材，是为了体现教材“新、精、深”的原则，强化学生专业英语训练，帮助学生了解国际上最新的学术动态而编写。教材内容主要摘编自 McGraw-Hill 公司出版的 Current Medical Diagnosis & Treatment。在内容编排上基本按照中文版《内科学》的编排顺序，以便学生对照阅读。本教材增加了较为详细的索引，以便于查阅；为体现实用性，其侧重面与中文版有所不同，着重介绍诊断和治疗方面内容。由于中英文教材编写时间起点上的差异，本教材的少部分内容可能比中文版《内科学》更新。本教材治疗方面内容非常详尽，考虑到学生负担，教学时主要让学生了解要点，详细内容可留在临床实习时参考。

本教材的原版书在学术上有高度权威性，充分体现了循证医学的原则；我们节录的内容尽可能地忠于英文原著。因此本教材可能对临床工作的各级医师也有一定参考价值。

需强调指出的是，为使学生了解英文原版教材的写法，本教材尽量保留了原文药物剂量、计量单位及有关写法。本教材中的部分药物剂量可能与目前国内所用剂量不同，需注意调整；以英制表示的单位未作改动，如有必要，需进行换算；疾病的流行病学资料、药物或治疗方法选择、病因排序等内容也与我国情况有所不同，应以中文版教材为准。

这本内科学英文教材是我们的首次尝试，希望各院校的广大教师和学生在使用过程中，不断提出宝贵意见，供今后修订时参考。

编　者

2001 年 6 月

Contents

Diseases of Respiratory System

1

Mark S. Chesnutt, MD, & Thomas J. Prendergast, MD 摘编 康健

Chapter 1. APPROACH TO THE PATIENT WITH RESPIRATORY DISEASES

COMMON MANIFESTATIONS

DYSPNEA

Dyspnea is a common symptom. It is analogous to pain in that sensory input from multiple sites in the respiratory system is integrated in the cerebral cortex. In general, dyspnea increases with the level of functional impairment as measured by spirometry. However, there is only a weak correlation between airflow limitation or exercise tolerance and the severity of dyspnea.

Sensations arising from several pathophysiologic processes contribute to dyspnea. The most important is the increased sense of respiratory effort that accompanies many different diseases: airflow obstruction (asthma; chronic obstructive pulmonary disease [COPD]), changes in pulmonary compliance (interstitial fibrosis, congestive heart failure) or chest wall compliance (obesity, pleural disease), intrinsic respiratory muscle weakness (inanition, neuromuscular disease, chronic respiratory failure), or the weakness conveyed by the mechanical disadvantage of hyperinflation (asthma or emphysema). Dyspnea is magnified by increased respiratory drive. Acute hypercapnia is therefore a potent stimulus to dyspnea, hypoxia a weak one. In mechanically ventilated patients, failure to provide adequate inspiratory flow rates to patients with heightened respiratory drive commonly results in dyspnea that may present as agitation. Stimulation of irritant receptors in the airways intensifies dyspnea, while stimulation of pulmonary stretch receptors decreases it.

Clinical Findings

The history should focus on timing of symptoms, the patient's position at onset of symptoms, the relationship of symptoms to activity, and any factors that may improve or exacerbate symptoms. The clinician can assess dyspnea and response to treatment with a ten-point numeric rating scale by asking the patient, "On a scale of zero to ten, with zero being no shortness of breath and ten being the worst shortness of breath you can imagine, how short of breath are you?" Exertional dyspnea should be quantified,but the absolute level of exertion that precipitates dyspnea is less important than acute changes in the threshold level of activity. A complete allergic, occupational, and smoking history is essential.

Acute dyspnea has a short list of causes,

most of which are readily identified: asthma, pulmonary infection, pulmonary edema, pneumothorax, pulmonary embolus, or acute respiratory distress syndrome (ARDS). Panic attacks may present as a respiratory complaint. Orthopnea (dyspnea on recumbency) and nocturnal dyspnea suggest asthma, gastroesophageal reflux disease (GERD), left ventricular dysfunction, or obstructive sleep apnea. Rapid onset of severe dyspnea when supine suggests phrenic nerve impairment and diaphragmatic paralysis. Platypnea (dyspnea that worsens in the upright position) is commonly associated with arteriovenous malformations at the lung bases, resulting in increased shunting and hypoxia in the upright position (orthodeoxia).

Chronic dyspnea is invariably progressive. Symptoms first appear during exertion; patients learn to limit their activity to accommodate their diminished pulmonary reserve until dyspnea occurs with minimal activity or at rest. Episodic dyspnea suggests congestive heart failure, asthma, or recurrent pulmonary emboli. Constant dyspnea is most commonly due to COPD but may indicate interstitial lung disease (eg, pulmonary fibrosis), pulmonary vascular disease, or fixed airflow obstruction from severe asthma.

Dyspnea is increasingly being recognized as a major issue in the care of dying patients, and physicians typically undertreat this symptom in patients dying from pulmonary diseases.

Evaluation should include a blood count, renal function tests, chest radiograph, spirometry, and noninvasive oximetry. Patients over 40 or with a family history of early coronary disease should have an electrocardiogram. Arterial blood gases, measurement of lung volumes, ventilation/perfusion scanning, echocardiography, and cardiopulmonary exercise testing are reserved for cases that elude diagnosis on initial evaluation.

Treatment

In patients with advanced lung disease, the responsible condition may be easily identified but treatment only partially effective. Oxygen improves survival in those who are hypoxemic and can improve the exercise training of all patients. Its effect on dyspnea is variable. Anxiety can play an important role in the distress caused by dyspnea and may be relieved by judicious use of benzodiazepines such as lorazepam, 0.5-1 mg orally every 4-6 hours. Pulmonary rehabilitation can improve respiratory function and train patients in energy conservation and breathing techniques that help moderate their sense of respiratory effort. Opioids reduce respiratory drive and blunt dyspnea. They can usually be titrated safely even in patients with advanced lung disease. Finally, fresh air or a fan may offer additional relief. Patients with progressive exertional dyspnea should know that they can limit future loss of function through smoking cessation.

COUGH

Cough is one of the most common symptoms for which patients seek medical care. It is an important physiologic mechanism that helps to defend against pathogens and to clear the tracheobronchial tree of mucus, foreign particles, and noxious aerosols. Impairment of oxygenation may result from a reduced or absent cough, as in some postoperative patients or those with neuromuscular disorders, or from excessive cough, which disrupts not only respiration but also sleep and social functioning. Bronchospasm, syncope, rib fractures, and urinary incontinence are all potential complications of severe cough. Referrals for chronic cough may constitute up to one-third of a pulmonologist's outpatient practice.

Cough may be voluntary or involuntary. Involuntary cough is stimulated by vagal afferent receptors in the trachea, especially at the carina,and the larynx but also from

others throughout the head and neck. Stimulation of cough receptors may be mechanical, as in cases of aspiration, or irritative.

Clinical Findings

It is important to distinguish acute (<3 weeks) from chronic cough. Acute cough most commonly follows viral or bacterial upper respiratory tract infection. Within 2 days after onset of the common cold, 85% of untreated patients cough; 26% are still coughing 14 days later, and in a few it will persist for 6–8 weeks. Many patients with persistent cough following upper respiratory tract infection have underlying asthma. Other causes of acute cough include aspiration, pneumonia, pulmonary embolism, and pulmonary edema.

The most common cause of chronic cough is a low-grade chronic bronchitis secondary to exposure to tobacco smoke, though smokers do not commonly seek medical attention for this problem. Over 90% of cases of chronic cough in nonsmokers presenting for evaluation of cough are due to postnasal drip, gastroesophageal reflux disease, and asthma. Angiotensin-converting enzyme (ACE) inhibitors have become another common cause. In primary care settings, single causes predominate.

The character and timing of chronic cough and the presence or absence of sputum production do not permit an etiologic diagnosis and should not be used as the sole basis for empirical therapy. The history and physical examination should attempt to identify anatomic locations of the afferent limb of the cough reflex in light of the common causes listed above. A nasal discharge, frequent need to clear the throat, and mucoid or mucopurulent secretions in the posterior pharynx suggest postnasal drip. Sinus radiographs may be diagnostic of acute or chronic sinusitis. Wheezing on chest auscultation or airway obstruction on pulmonary function tests suggest asthma. In cough-variant asthma, methacholine bronchoprovocation testing may be positive in the absence of clinical findings of asthma. Gastroesophageal reflux disease is an important cause of chronic cough but is associated with the fewest clinical clues. Patients may complain of heartburn or regurgitation, but cough may be the only symptom. Barium swallow is specific but insensitive, and esophageal pH monitoring may be necessary. Chest radiographs are best reserved for cough in smokers and patients with hemoptysis or constitutional symptoms such as fever and weight loss.

Treatment

The first step is to eliminate irritant exposures such as tobacco smoke (primary or secondary) and occupational agents and to discontinue medications such as ACE inhibitors or beta-blockers, including eyedrops. Cough due to ACE inhibitors should subside within 1–4 days after discontinuing the medication, though it may take weeks to months. Angiotensin II receptor antagonists do not cause cough. Patients whose cough began after an upper respiratory tract infection usually respond to treatment with an antihistamine-decongestant combination or treatment for asthma, with inhaled bronchodilators and corticosteroids. Postnasal drip syndrome due to allergic rhinitis that does not respond to antihistamines should be treated with intranasal steroids. Chronic sinusitis may require prolonged antibiotics directed against *Haemophilus influenzae*. Cough caused by asthma that does not respond after 2 weeks of bronchodilators and corticosteroids suggests that another condition is contributing. Gastroesophageal reflux disease is difficult to treat, since H_2 blockers may not be adequate. Most practitioners now initiate antitussive therapy for gastroesophageal reflux disease with proton pump inhibitors.

HEMOPTYSIS

Hemoptysis is the expectoration of blood that originates below the vocal cords. It is commonly classified as trivial, mild, or massive, the last defined as more than 200–600 mL in 24 hours. The dividing lines are arbitrary and difficult to draw, since the amount of blood is rarely quantified with precision. Massive hemoptysis can be usefully defined as any amount that is hemodynamically significant or threatens ventilation, in which case the initial management goal is not diagnostic but therapeutic.

The lungs are supplied with a dual circulation. The pulmonary arteries arise from the right ventricle to supply the pulmonary parenchyma in a low-pressure circuit. The bronchial arteries arise from the aorta or intercostal arteries and carry blood under systemic pressure to the airways, blood vessels, hila, and visceral pleura. The bronchial arterial circulation represents only 1–2% of total pulmonary blood flow but is frequently the source of hemoptysis: It is a high-pressure circuit; it provides the blood supply to the airways and lesions within those airways; and it can increase dramatically under conditions of chronic inflammation–eg, chronic bronchiectasis.

The causes of hemoptysis can be classified anatomically. Blood may arise from the airways in chronic bronchitis, bronchiectasis, and bronchogenic carcinoma; from the pulmonary vasculature in left ventricular failure, mitral stenosis, pulmonary emboli, and arteriovenous malformations; or from the pulmonary parenchyma in pneumonia, inhalation of crack cocaine, or autoimmune diseases such as Goodpasture's disease or Wegener's granulomatosis. Iatrogenic hemorrhage may follow transbronchial lung biopsies, anticoagulation, or pulmonary artery rupture due to distal placement of a balloon-tipped catheter.

Clinical Findings

Blood-tinged sputum in the setting of acute bronchitis in an otherwise healthy nonsmoker does not warrant an extensive diagnostic evaluation if the hemoptysis subsides with resolution of the infection. However, hemoptysis is frequently a sign of serious disease, especially in patients with a high prior probability of an underlying pulmonary condition. The goal of the history is to identify patients at risk for one of the disorders listed above. Pertinent features are tobacco use, duration of symptoms, and the presence of respiratory infection. Nonpulmonary sources of hemorrhage–from the nose or the gastrointestinal tract–should also be ruled out.

Laboratory evaluation should include a chest radiograph and complete blood count, including platelet count. Renal function tests, urinalysis, and coagulation studies are appropriate in specific circumstances. Flexible bronchoscopy will reveal endobronchial cancer in approximately 3–6% of patients with hemoptysis who have a normal (nonlateralizing) chest radiograph. Nearly all of these patients will be smokers over the age of 40, and most will have had symptoms for more than a week. Bronchoscopy is indicated in such patients; observation and follow-up is appropriate in patients without risk factors for cancer. High-resolution CT of the chest is complementary to bronchoscopy. It can diagnose unsuspected bronchiectasis and arteriovenous malformations and will show central endobronchial lesions in many cases. It is the test of choice for suspected small peripheral malignancies.

Treatment

The management of mild hemoptysis consists of identifying and treating the specific cause. Massive hemoptysis is life-threatening. The airway must be protected, ventilation ensured, and effective circulation maintained. If the locations of the bleeding sites are known, the patient should be

placed in the decubitus position with the involved lung dependent. Uncontrollable hemorrhage warrants rigid bronchoscopy and surgical consultation. In stable patients, flexible bronchoscopy may localize the site of bleeding, and angiography can embolize the source. Embolization is effective initially in 85% of cases, though rebleeding may occur in up to 20% of patients over the following year. The anterior spinal artery arises from the bronchial artery in up to 5% of people, and paraplegia may result if it is inadvertently cannulated.

PHYSICAL EXAMINATION

Examination of the patient with suspected pulmonary disease includes inspection, palpation, percussion, and auscultation of the chest. An efficient approach begins with observing the pattern of breathing, auscultation of the chest, and inspection for extrapulmonary signs of pulmonary disease. More detailed examination follows from initial findings.

The pattern of breathing refers to the respiratory rate and rhythm, the tidal volume, and the relative amount of time spent in inspiration and expiration. Normal values are a rate of 12-14 breaths per minute, tidal volumes of 5 mL/kg, and a ratio of inspiratory to expiratory time of 2 : 3.Tachypnea is an increased rate of breathing and is commonly associated with a decrease in tidal volume. The rhythm is normally regular, with a sigh (1.5-2 times normal tidal volume) every 90 breaths or so to recruit surfactant to maintain patency of alveoli. Alterations in the rhythm of breathing include rapid, shallow breathing, seen in restrictive lung disease and as a precursor to respiratory failure; Kussmaul breathing, rapid large-volume breathing indicating intense stimulation of the respiratory center, seen in metabolic acidosis; and Cheyne-Stokes respirations, a rhythmic waxing and waning of both rate and tidal volumes that includes regular periods of apnea. This pattern is seen in patients with end-stage left ventricular failure or neurologic disease and in many normal subjects at high altitude, especially during sleep.

During normal quiet breathing, the primary muscle of respiration is the diaphragm. Movement of the chest wall is minimal. The use of accessory muscles of respiration, the intercostal and sternocleidomastoid muscles, indicates high work of breathing. At rest, this is a sign of significant pulmonary impairment. As the diaphragm contracts, it pushes the abdominal contents down. Hence, the chest and abdominal wall normally expand simultaneously. Expansion of the chest but collapse of the abdomen on inspiration indicates weakness of the diaphragm. The thorax is normally symmetric. Asymmetric expansion suggests unilateral volume loss, as in atelectasis or pleural effusion, unilateral airway obstruction, asymmetric pulmonary or pleural fibrosis, or splinting from chest pain.

The examiner may palpate as follows: at the suprasternal notch, to detect shifts in the mediastinum; on the posterior chest wall, to gauge fremitus and the transmission through the lungs of vibrations of spoken words and to assess the cardiac impulse. All are characterized by low interobserver agreement.

Chest percussion identifies dull areas that correspond to lung consolidation or pleural effusion or hyperresonant areas of emphysema or pneumothorax. Percussion has a low sensitivity (10-20% in several studies) compared with chest radiographs to detect abnormalities. Specificity is high (85-99%), however, so percussion remains useful. Since an insensitive test is a poor screening examination, percussion and palpation are not necessary in every patient. These techniques do serve as important confirmatory tests in specific patients when the prior probability of a finding is increased. For example, in a patient with

a suspected tension pneumothorax, the finding of tracheal shift and hyperresonance can be lifesaving, permitting immediate decompression of the affected side.

Auscultation of the chest depends on a reliable and consistent classification of auditory findings. Normal lung sounds heard over the periphery of the lung are called vesicular. They have a gentle, rustling quality heard throughout inspiration that fades during expiration.Normal sounds heard over the suprasternal notch are called tracheal or bronchial lung sounds. They are louder, higher-pitched, and have a hollow quality that tends to be louder on expiration. Bronchial lung sounds heard over the periphery of the lung are abnormal and imply consolidation. Globally diminished lung sounds are an important finding predictive of significant airflow obstruction.

Abnormal lung sounds ("adventitious") may be continuous (> 80 ms in duration) or discontinuous (< 20 ms). Continuous lung sounds are divided into wheezes, which are high-pitched, musical, and have a distinct whistling quality; and rhonchi, which are lower-pitched, sonorous, and may have a gurgling quality. Wheezes occur in the setting of bronchospasm, mucosal edema, or excessive secretions. In each, the airway is narrowed to the point where adjacent airway walls flutter as airflow is limited. Rhonchi originate in the larger airways when excessive secretions and abnormal airway collapsibility cause repetitive rupture of fluid films. Rhonchi frequently clear after cough.

Discontinuous lung sounds are called crackles-brief, discrete, nonmusical sounds with a popping quality. Fine crackles are soft, high-pitched, and crisp (<10 ms in duration). They are formed by the explosive opening of small airways previously held closed by surface forces and are heard in interstitial diseases or early pulmonary edema. Coarse crackles are louder, lower-pitched, and slightly longer in duration (< 20 ms) and probably result from gas bubbling through fluid). Coarse crackles are heard in pneumonia, obstructive lung disease, and late pulmonary edema.

Interobserver agreement regarding auditory findings is good. The clinical usefulness of these findings is also well established. The presence of wheezes on physical examination is a powerful predictor of obstructive lung disease. The absence of wheezes is not helpful since patients may have significant airflow limitation without wheezing. Such patients will have globally diminished lung sounds as the clinical clue to their obstructive lung disease. Normal lung sounds exclude obstruction. The timing and character of crackles can reliably distinguish different pulmonary disorders. Fine, late inspiratory crackles suggest pulmonary fibrosis, while early coarse crackles suggest pneumonia or heart failure.

Extrapulmonary signs of intrinsic pulmonary disease include digital clubbing, cyanosis, elevation of central venous pressures, and lower extremity edema.

Digital clubbing refers to structural changes at the base of the nails that include softening of the nail bed and loss of the normal 150-degree angle between the nail and the cuticle. The distal phalanx is convex and enlarged: its thickness is equal to or greater than the thickness of the distal interphalangeal joint. Symmetric clubbing may be a normal variant but more commonly is a sign of underlying disease. Clubbing is seen in chronic infections of the lungs and pleura (lung abscess, empyema, bronchiec-tasis, cystic fibrosis), malignancies of the lungs and pleura, chronic interstitial lung disease (idiopathic pulmonary fibrosis), and arteriovenous malformations. It does not normally accompany asthma or COPD; when seen in the latter, one should suspect concomitant lung cancer. It is observed less often in small-cell cancer than in other histologic types. Clubbing is not specific to pulmonary disorders; it is also seen in cyanotic congenital heart disease, infective