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EDITION



# clinical examination

A systematic guide to physical diagnosis

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**Nicholas J Talley and  
Simon O'Connor**

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A systematic guide to physical diagnosis

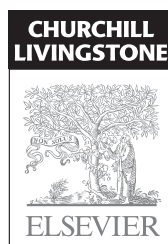
**Seventh edition**

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# Foreword

The authors deserve warm congratulations and much thanks in preparing this seventh edition of their highly-acclaimed *Clinical examination: a systematic guide to physical diagnosis*. Moving with the times the format and content provide for more flexible use in print and online and confront the educational challenge of tuition and assessment in the development of clinical skills. The central mission of the original edition—to enhance clinical skills—remains soundly intact.

The quality and outcome of an encounter between a sick or injured person and a health professional depends upon the integrity of the exchange of information. That exchange may constitute the entire clinical encounter, as in psychotherapy. The psychotherapist may spend months or years understanding his or her patient and leading them to insights and altered behaviour as a result. Alternatively, the encounter may be fleeting and nonverbal, the prelude to an urgent, extensive technical intervention, as occurs when a trauma surgeon treats a severely injured patient or a physician manages a patient suffering an unheralded life-threatening heart attack. The skills for interviewing and examining a patient physically required of the doctor vary widely among the range of specialities that now offer care. The appropriate mix of information derived from speaking with the patient, examining him or her physically and interpreting test results varies just as widely. This variation is recognised well in this book in that its chapters relate separately to the clinical encounter in fields as diverse as neurology and gastroenterology.

In the haste and pressure characteristic of the practice of medicine and surgery in a large hospital, corners are easily cut, the patient history is stripped to its bare essentials and physical examination can be forgone in favour of scans. On-call medical attendants have been known to resist travelling to

review a patient in the emergency department until investigations have been done. These abbreviations to the clinical encounter, while understandable, come at a high cost. Junior doctors in the emergency room adopt the behaviours endorsed by their seniors: stories abound of how patients died while awaiting a CT scan and whose clinical condition would have been detected and emergency treatment instituted if they had been examined properly and their story heard.

All of us engaged in clinical care, whether as front-line clinicians, research workers or managers, should remind ourselves that the most fundamental unit of our concern should be the patient. As such, wherever possible, a human (and humane) relationship with that individual is the platform on which subsequent therapy is then built. The development of those skills is the core business of this book. In its earlier incarnation, it has proved to be immensely popular and the authors have now responded by expanding, clarifying and adding new sections of assessment to the original edition. This book will prove to be valuable and valued by medical students, young doctors and older practitioners in different ways. However used, it can enhance the quality of care we offer our patients—and that is a truly splendid compliment to the authors.

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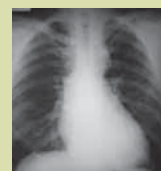
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# Preface

*The four points of a medical student's compass are: Inspection, Palpation, Percussion, and Auscultation.*

Sir William Osler

It is now more than 25 years since we set out to write the first edition of *Clinical examination* in our spare time as registrars at the Royal North Shore Hospital, Sydney. Much has changed since then with regard to both the management of patients and medical education. At the time we began the first edition we were both strongly influenced by our recent experiences as basic physician trainees. The Royal Australasian College of Physicians training scheme had and still has a major emphasis on excellence in history taking and physical examination as the basis of management. We both feel strongly that a thorough grounding in these skills and arts is essential for everyone beginning a career in medicine.

A diagnosis is the foundation of all medical practice, and history taking and physical examination are key in the diagnostic process despite the ever-increasing sophistication of diagnostic testing. Tests ordered indiscriminately or inappropriately can seriously mislead and be dangerous: test ordering must be based on the findings of a proper clinical assessment of the patient. Making the wrong diagnosis can cause harm and distress that lasts a lifetime. It is distressingly common for tests to be ordered and

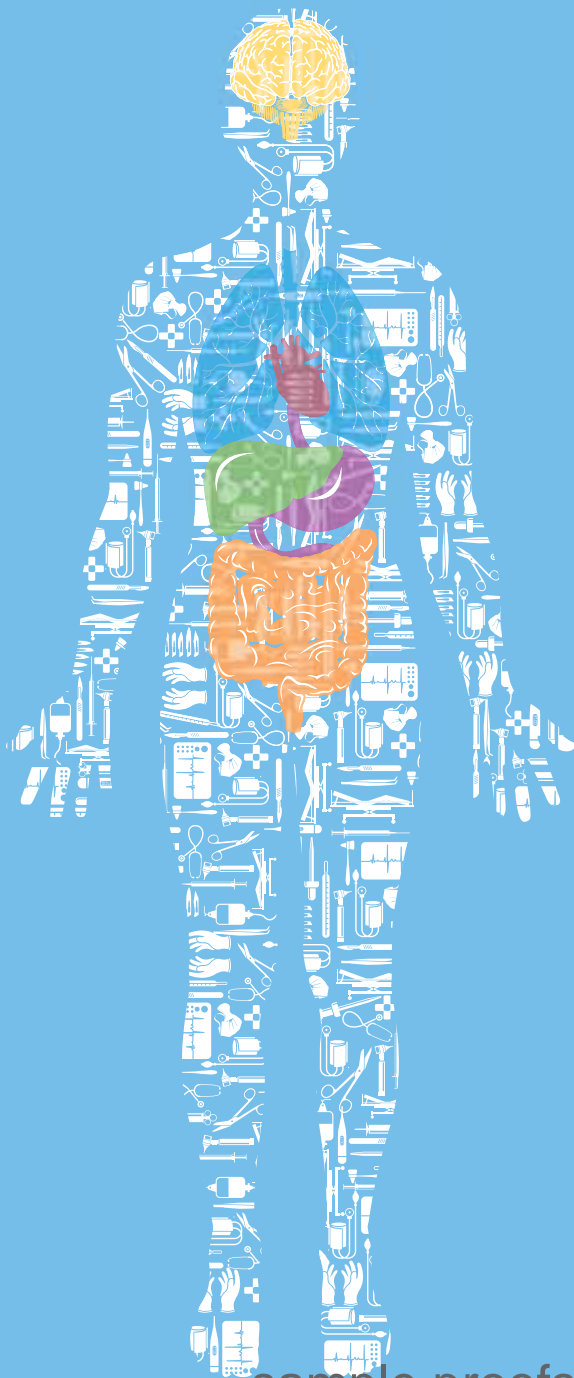
referrals made without an adequate history or even a cursory examination of the patient.

In this new edition of *Clinical examination* we cover the core clinical skills from the basics to an advanced level. We have taken an approach that is patient-centric and evidence-based; the patient must always come first. This edition has been brought right up to date with the latest clinical data, including new research specially commissioned for this edition. Learning should also be fun and the book is deliberately laced with humour and historical anecdotes that generations of students have told us enhance the learning experience.

Not only have there been changes to medical practice over the last 25 years but the format of teaching and use of books has changed irrevocably. The flexibility now offered by computer and tablet versions of books has altered the way students study and perhaps think. This edition offers the book in three formats: a paper book, an eBook and an enhanced eBook. Each version offers new features to help students to study.

Again we are grateful to all those who have helped us with the increasingly complicated job of producing a book like this: our publishers, the colleagues who have contributed pictures and advice, and the many students who have written to us to correct errors (real or perceived).

Nicholas Talley & Simon O'Connor  
Newcastle and Canberra, August 2013



## The respiratory system

- Chapter 9** The respiratory history
- Chapter 10** The respiratory examination
- Chapter 11** Correlation of physical signs and respiratory disease
- Chapter 12** A summary of the respiratory examination and extending the respiratory examination

# The respiratory history

*A medical chest specialist is long winded about the short winded.*

Kenneth T Bird (b.1917)

## Presenting symptoms

(see List 9.1)

### LIST 9.1 Presenting symptoms

#### Major symptoms

Cough  
Sputum  
Haemoptysis  
Dyspnoea (acute, progressive or paroxysmal)  
Wheeze  
Chest pain  
Fever  
Hoarseness  
Night sweats

## COUGH AND SPUTUM

Cough is a common presenting respiratory symptom. It occurs when deep inspiration is followed by explosive expiration. Flow rates of air in the trachea approach the speed of sound during a forceful cough. Coughing enables the airways to be cleared of secretions and foreign bodies.

The duration of a cough is important (see Questions box 9.1). Find out when the cough first became a problem. A cough of recent origin, particularly if associated with fever and other symptoms of respiratory tract infection, may be due to acute bronchitis or pneumonia. A chronic cough (of more than 8 weeks duration) associated with wheezing may be due to asthma; sometimes asthma can present with just cough alone. A change in the character of a chronic cough may indicate the development of a new and serious underlying problem (e.g. infection or lung cancer).

### Questions box 9.1

#### Questions to ask the patient with a cough

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. How long have you had the cough?
2. Do you cough up anything? What? How much?
3. Have you had sinus problems?
- ! 4. Is the sputum clear or discoloured? Is there any blood in the sputum?
5. Have you had high temperatures?
6. Does coughing occur particularly at night (acid reflux)?
7. Have you become short of breath?
8. Have you had lung problems in the past?
9. Have you been a smoker? Do you still smoke?
10. Have you noticed wheezing? (Asthma, chronic obstructive pulmonary disease [COPD])?
11. Do you take any tablets? (e.g. ACE inhibitors)

A differential diagnosis of cough based on its character is shown in Table 9.1 and on its duration is shown in List 9.2.

A cough associated with a postnasal drip or sinus congestion or headaches may be due to the upper airway cough syndrome, which is the single most common cause of chronic cough. Although patients with this problem often complain of a cough, when asked to demonstrate their cough they do not cough but clear the throat. This annoys chest doctors (who feel their time is valuable) intensely.

An irritating, chronic dry cough can result from oesophageal reflux and acid irritation of the lungs. There is some controversy about these as causes of true cough. A similar dry cough may be a feature of late interstitial lung disease or associated with the use of the angiotensin-converting enzyme (ACE) inhibitors—drugs used in the treatment of hypertension and cardiac failure. Cough that wakes a patient from sleep may be a symptom of cardiac failure or of the reflux of acid from the oesophagus into the lungs that can occur when a person lies down.

**TABLE 9.1** Differential diagnosis of cough based on its character

Origin	Character	Causes
Nasopharynx/larynx	Throat clearing, chronic	Postnasal drip, acid reflux
Larynx	Barking, painful, acute or persistent	Laryngitis, pertussis (whooping cough), croup
Trachea	Acute, painful	Tracheitis
Bronchi	Intermittent, sometimes productive, worse at night	Asthma
	Worse in morning	Chronic obstructive pulmonary disease (COPD)
	With blood	Bronchial malignancy
Lung parenchyma	Dry then productive	Pneumonia
	Chronic, very productive	Bronchiectasis
	Productive, with blood	Tuberculosis
	Irritating and dry, persistent	Interstitial lung disease
	Worse on lying down, sometimes with frothy sputum	Pulmonary oedema
ACE inhibitors	Dry, scratchy, persistent	Medication-induced

**LIST 9.2** Differential diagnosis of cough based on its duration**Acute cough (<3 weeks duration): differential diagnosis**

Upper respiratory tract infection

- Common cold, sinusitis

Lower respiratory tract infection

- Pneumonia, bronchitis, exacerbation of COPD
- Irritation—inhalation of bronchial irritant (e.g. smoke or fumes)

**Chronic cough: differential diagnosis and clues**

COPD—smoking history

Asthma—wheeze, relief with bronchodilators

Gastro-oesophageal reflux—occurs when lying down, burning central chest pain

Upper airway cough syndrome—history of rhinitis, postnasal drip, sinus headache and congestion

Bronchiectasis—chronic, very productive

ACE inhibitor medication—drug history

Carcinoma of the lung—smoking, haemoptysis

Cardiac failure—dyspnoea, PND

Psychogenic—variable, prolonged symptoms, usually mild

ACE = angiotensin-converting enzyme; COPD = chronic obstructive pulmonary disease; PND = paroxysmal nocturnal dyspnoea.

A chronic cough that is productive of large volumes of purulent sputum may be due to bronchiectasis.

Some patients feel the need to cough after an ectopic heartbeat. There may be an associated sensation of a missed heartbeat.

Patients' descriptions of their cough may be helpful. In children, a cough associated with inflammation

of the epiglottis may have a muffled quality and cough related to viral croup is often described as 'barking'. Cough caused by tracheal compression by a tumour may be loud and brassy. Cough associated with recurrent laryngeal nerve palsy has a hollow sound because the vocal cords are unable to close completely; this has been described as a bovine cough. A cough that is worse at night is suggestive of asthma or heart failure, while coughing that comes on immediately after eating or drinking may be due to incoordinate swallowing or oesophageal reflux or, rarely, a tracheo-oesophageal fistula.

It is an important (though perhaps a somewhat unpleasant task) to enquire about the type of sputum produced and then to look at it, if it is available. Be warned that some patients have more interest in their sputum than others and may go into more detail than you really want. A large volume of purulent (yellow or green) sputum suggests the diagnosis of bronchiectasis or lobar pneumonia. Foul-smelling dark-coloured sputum may indicate the presence of a lung abscess with anaerobic organisms. Pink frothy secretions from the trachea, which occur in pulmonary oedema, should not be confused with sputum. It is best to rely on the patient's assessment of the taste of the sputum, which, not unexpectedly, is foul in conditions like bronchiectasis or lung abscess.

**HAEMOPTYSIS**

Haemoptysis (coughing up of blood) can be a sinister sign of lung disease (see Table 9.2) and must always be investigated. It must be distinguished

**TABLE 9.2** Causes (differential diagnosis) of haemoptysis and typical histories

Respiratory	
Bronchitis	Small amounts of blood with sputum
Bronchial carcinoma	Frank blood, history of smoking, hoarseness
Bronchiectasis	Large amounts of sputum with blood
Pneumonia	Fever, recent onset of symptoms, dyspnoea
<i>(The above four account for about 80% of cases)</i>	
Pulmonary infarction	Pleuritic chest pain, dyspnoea
Cystic fibrosis	Recurrent infections
Lung abscess	Fever, purulent sputum
Tuberculosis (TB)	Previous TB, contact with TB, HIV-positive status
Foreign body	History of inhalation, cough, stridor
Goodpasture's* syndrome	Pulmonary haemorrhage, glomerulonephritis, antibody to basement membrane antigens
Wegener's granulomatosis	History of sinusitis, saddle-nose deformity
Systemic lupus erythematosus	Pulmonary haemorrhage, multisystem involvement
Rupture of a mucosal blood vessel after vigorous coughing	History of severe cough preceding haemoptysis
Cardiovascular	
Mitral stenosis (severe)	
Acute left ventricular failure	
Bleeding diatheses	
<i>Note:</i> Exclude spurious causes, such as nasal bleeding or haematemesis.	
*Ernest W Goodpasture (1886–1960), pathologist at Johns Hopkins, Baltimore. He described this syndrome in 1919.	

from haematemesis (vomiting of blood) and from nasopharyngeal bleeding (see Table 9.3).

Ask how much blood has been produced. Mild haemoptysis usually means less than 20 mL in 24 hours. It appears as streaks of blood discolouring sputum. Massive haemoptysis is more than 250 mL of blood in 24 hours and represents a medical emergency. Its most common causes are carcinoma, cystic fibrosis, bronchiectasis and tuberculosis.

## BREATHLESSNESS (DYSPNOEA)

The awareness that an abnormal amount of effort is required for breathing is called dyspnoea. It can

**TABLE 9.3** Features distinguishing haemoptysis from haematemesis and nasopharyngeal bleeding

Favours haemoptysis	Favours haematemesis	Favours nasopharyngeal bleeding
Mixed with sputum	Follows nausea	Blood appears in mouth
Occurs immediately after coughing	Mixed with vomitus; follows dry retching	

be due to respiratory or cardiac disease, or lack of physical fitness or sometimes to anxiety (see List 9.3). Careful questioning about the timing of onset, severity and pattern of dyspnoea is helpful in making the diagnosis (see Questions box 9.2 and List 9.4).<sup>1</sup> The patient may be aware of this only on heavy exertion or have much more limited exercise tolerance. Dyspnoea can be graded from I to IV based on the New York Heart Association classification:

*Class I* Disease present but no dyspnoea or dyspnoea only on heavy exertion.

*Class II* Dyspnoea on moderate exertion.

*Class III* Dyspnoea on minimal exertion.

*Class IV* Dyspnoea at rest.

It is more useful, however, to determine the amount of exertion that actually causes dyspnoea—that is, the distance walked or the number of steps climbed.

The association of dyspnoea with wheeze suggests *airways disease*, which may be due to asthma or chronic obstructive pulmonary disease (COPD; see List 9.5). The duration and variability of the dyspnoea are important. Dyspnoea that worsens progressively over a period of weeks, months or years may be due to *interstitial lung disease* (ILD). Dyspnoea of more rapid onset may be due to an *acute respiratory infection* (including bronchopneumonia or lobar pneumonia) or to *pneumonitis* (which may be infective or secondary to a hypersensitivity reaction). Dyspnoea that varies from day to day or even from hour to hour suggests a diagnosis of *asthma*. Dyspnoea of very rapid onset associated with sharp chest pain suggests a *pneumothorax* (see List 9.6). Dyspnoea that is described by the patient as inability to take a breath big enough to fill the lungs and associated with sighing suggests *anxiety*. Dyspnoea that occurs on moderate exertion may be due to the combination of *obesity* and a *lack of physical fitness* ('deconditioning'; a not uncommon occurrence).

## WHEEZE

A number of conditions can cause a continuous whistling noise that comes from the chest (rather

**LIST 9.3 Causes of dyspnoea****Respiratory****1. Airways disease**

Chronic bronchitis and emphysema (chronic obstructive pulmonary disease [COPD])

Asthma

Bronchiectasis

Cystic fibrosis

Laryngeal or pharyngeal tumour

Bilateral cord palsy

Tracheal obstruction or stenosis

Tracheomalacia

Cricoarytenoid rheumatoid arthritis

**2. Parenchymal disease**

Interstitial lung diseases (diffuse parenchymal lung diseases, e.g. idiopathic pulmonary fibrosis, sarcoidosis, connective tissue disease, inorganic or organic dusts)

Diffuse infections

Acute respiratory distress syndrome (ARDS)

Infiltrative and metastatic tumour

Pneumothorax

Pneumoconiosis

**3. Pulmonary circulation**

Pulmonary embolism

Chronic thromboembolic pulmonary hypertension

Pulmonary arteriovenous malformation

Pulmonary arteritis

**4. Chest wall and pleura**

Effusion or massive ascites

Pleural tumour

Fractured ribs

Ankylosing spondylitis

Kyphoscoliosis

Neuromuscular diseases

Bilateral diaphragmatic paralysis

**Cardiac**

Left ventricular failure

Mitral valve disease

Cardiomyopathy

Pericardial effusion or constrictive pericarditis

Intra-cardiac shunt

**Anaemia****Non-cardiorespiratory**

Psychogenic

Acidosis (compensatory respiratory alkalosis)

Hypothalamic lesions

than the throat) during breathing. These include asthma or COPD, infections such as bronchiolitis and airways obstruction by a foreign body or tumour. Wheeze is usually maximal during expiration and is accompanied by prolonged expiration. This must be differentiated from *stridor* (see below), which can have a similar sound, but is loudest over the trachea and occurs during inspiration.

**Questions box 9.2****Questions to ask the breathless patient**

! denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. How long have you been short of breath? Has it come on quickly?
2. How much exercise can you do before your shortness of breath stops you or slows you down? Can you walk up a flight of stairs?
- ! 3. Have you been woken at night by breathlessness or had to sleep sitting up? (Paroxysmal nocturnal dyspnoea [PND], orthopnoea)
4. Have you had heart or lung problems in the past?
- ! 5. Have you had a temperature?
6. Do you smoke?
- ! 7. Is there a feeling of tightness in the chest when you feel breathless? (Angina)
8. Do you get wheezy in the chest? Cough?
9. Is the feeling really one of difficulty getting a satisfying breath? (Anxiety)
10. Is it painful to take a big breath? (Pleurisy or pericarditis)
- ! 11. Did the shortness of breath come on very quickly or instantaneously? (Pulmonary embolus [very quick onset] or pneumothorax [instantaneous onset])
12. Are you often short of breath when you are anxious? Do you feel numbness and tingling around your lips when you are breathless? (Hyperventilation associated with anxiety)

**LIST 9.4 Differential diagnosis of dyspnoea based on time course of onset****Seconds to minutes—favours:**

Asthma  
Pulmonary embolism  
Pneumothorax  
Pulmonary oedema  
Anaphylaxis  
Foreign body causing airway obstruction

**Hours or days—favours:**

Exacerbation of chronic obstructive pulmonary disease (COPD)  
Cardiac failure  
Asthma  
Respiratory infection  
Pleural effusion  
Metabolic acidosis

**Weeks or longer—favours:**

Pulmonary fibrosis  
COPD  
Interstitial lung disease  
Pleural effusion  
Anaemia

# The respiratory examination

*More would I, but my lungs are wasted so,  
That strength of speech is utterly  
denied me.*

William Shakespeare, *Henry IV, Part 2*

## Examination anatomy

The **lungs** are paired asymmetrical organs protected by the cylinder composed of the ribs, vertebrae and diaphragm. The surface of the lungs is covered by the visceral **pleura**, a thin membrane, and a similar outer layer (the parietal pleura) lines the rib cage. These membranes are separated by a thin layer of fluid and enable the lungs to move freely during breathing. Various diseases of the lungs and of the pleura themselves, including infection and malignancy, can cause accumulation of fluid within the pleural cavity (a pleural effusion).

The heart, trachea, oesophagus and the great blood vessels and nerves sit between the lungs and make up the structure called the **mediastinum**. The left and right pulmonary arteries supply their respective lung. Gas exchange occurs in the pulmonary capillaries that surround the alveoli, the tiny air sacs which lie beyond the terminal bronchioles. Oxygenated blood is returned via the pulmonary veins to the left atrium. Abnormalities of the pulmonary circulation such as raised pulmonary venous pressure resulting from heart failure or pulmonary hypertension can interfere with gas exchange.

The position of the heart, whose apex points to the left, means that the **left lung** is smaller than the right and has only two lobes, which are separated by the oblique fissure. The **right lung** has both horizontal (upper) and oblique (lower) fissures dividing it into three lobes (see Figure 10.1).

The muscles of respiration are the **diaphragm** upon which the bases of the lungs rest and the **intercostal muscles**. During inspiration, the diaphragm flattens and the intercostal muscles contract

to elevate the ribs. Intrathoracic pressure falls as air is forced under atmospheric pressure into the lungs. Expiration is a passive process resulting from elastic recoil of the muscles. Abnormalities of lung function or structure may change the normal anatomy and physiology of respiration, for example as a result of over-inflation of the lungs (chronic obstructive pulmonary disease [COPD]). Muscle and neurological diseases can also affect muscle function adversely, and abnormalities of the control of breathing in the respiratory centres of the brain in the pons and medulla can interfere with normal breathing patterns.

During the respiratory examination, keep in mind the **surface anatomy** (see Figure 10.2) of the lungs and try to decide which lobes are affected.

## Positioning the patient

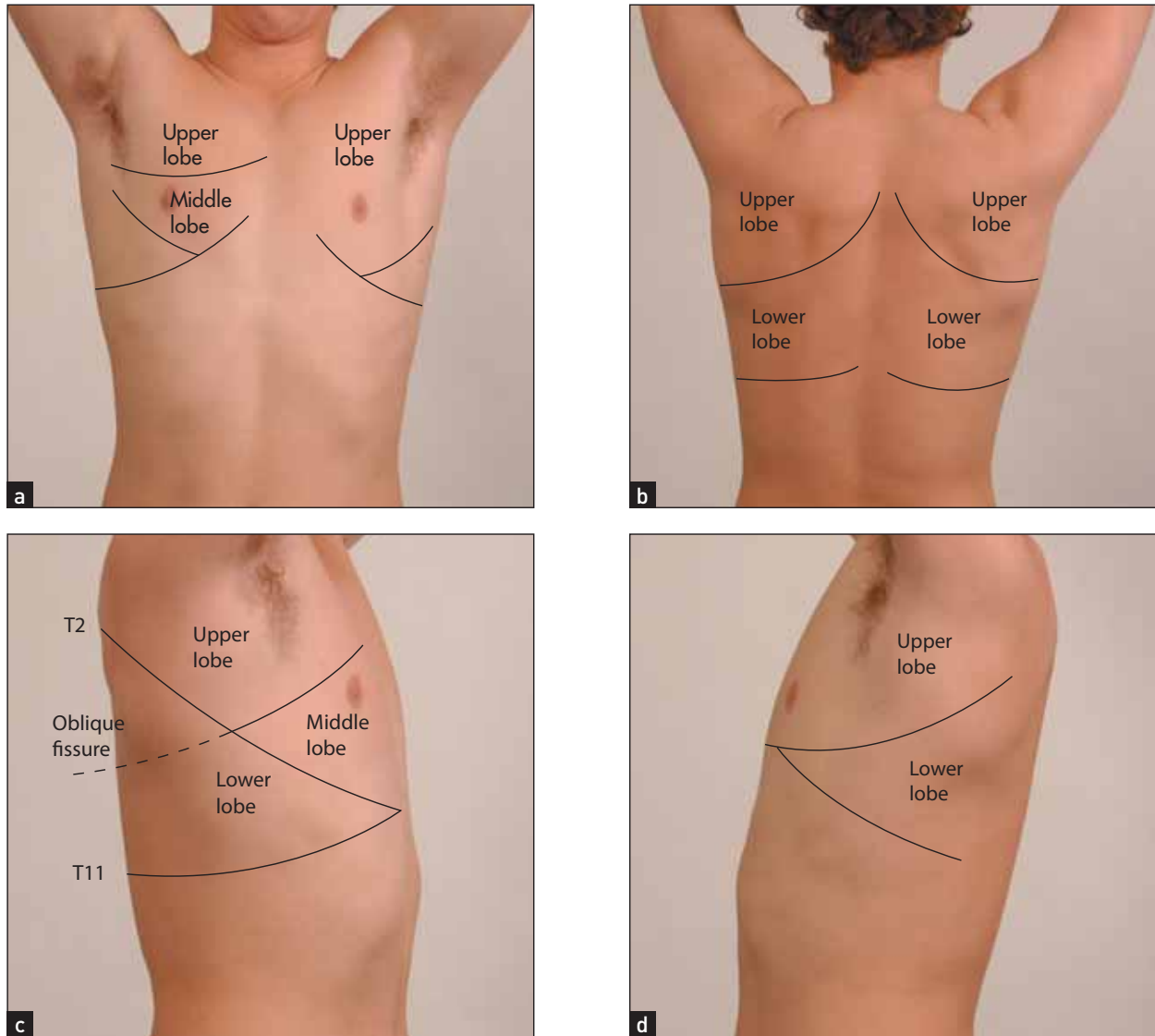
The patient should be undressed to the waist.<sup>1</sup> Women should wear a gown or have a towel or some clothing to cover their breasts when the front of the chest is not being examined. If the patient is not acutely ill, the examination is easiest to perform with him or her sitting over the edge of the bed or on a chair.

## General appearance

If the patient is an inpatient in hospital, look around the bed for oxygen masks, metered dose inhalers (puffers) and other medications, and the presence of a sputum mug. Then make a deliberate point of looking for the following signs before beginning the detailed examination.

## DYSPNOEA

Watch the patient for signs of dyspnoea at rest. Count the respiratory rate; the normal rate at rest should not exceed 25 breaths per minute (range 16–25). The frequently quoted normal value of 14 breaths per minute is probably too low; normal people can



**Figure 10.1** Lobes of the lung

(a) Anterior; (b) posterior; (c) lobes of the right lung; (d) lobes of the left lung. Refer to Figure 12.4, page 165, for a list of the segments in each lobe.

(Courtesy of Glenn McCulloch)

have a respiratory rate of up to 25, and the average is 20 breaths per minute. It is traditional to count the respiratory rate surreptitiously while affecting to count the pulse. The respiratory rate is the only vital sign that is under direct voluntary control. *Tachypnoea* refers to a rapid respiratory rate of greater than 25. *Bradypnoea* is defined as a rate below 8, a level associated with sedation and adverse prognosis. In normal relaxed breathing, the diaphragm is the only active muscle and is active only in inspiration; expiration is a passive process.

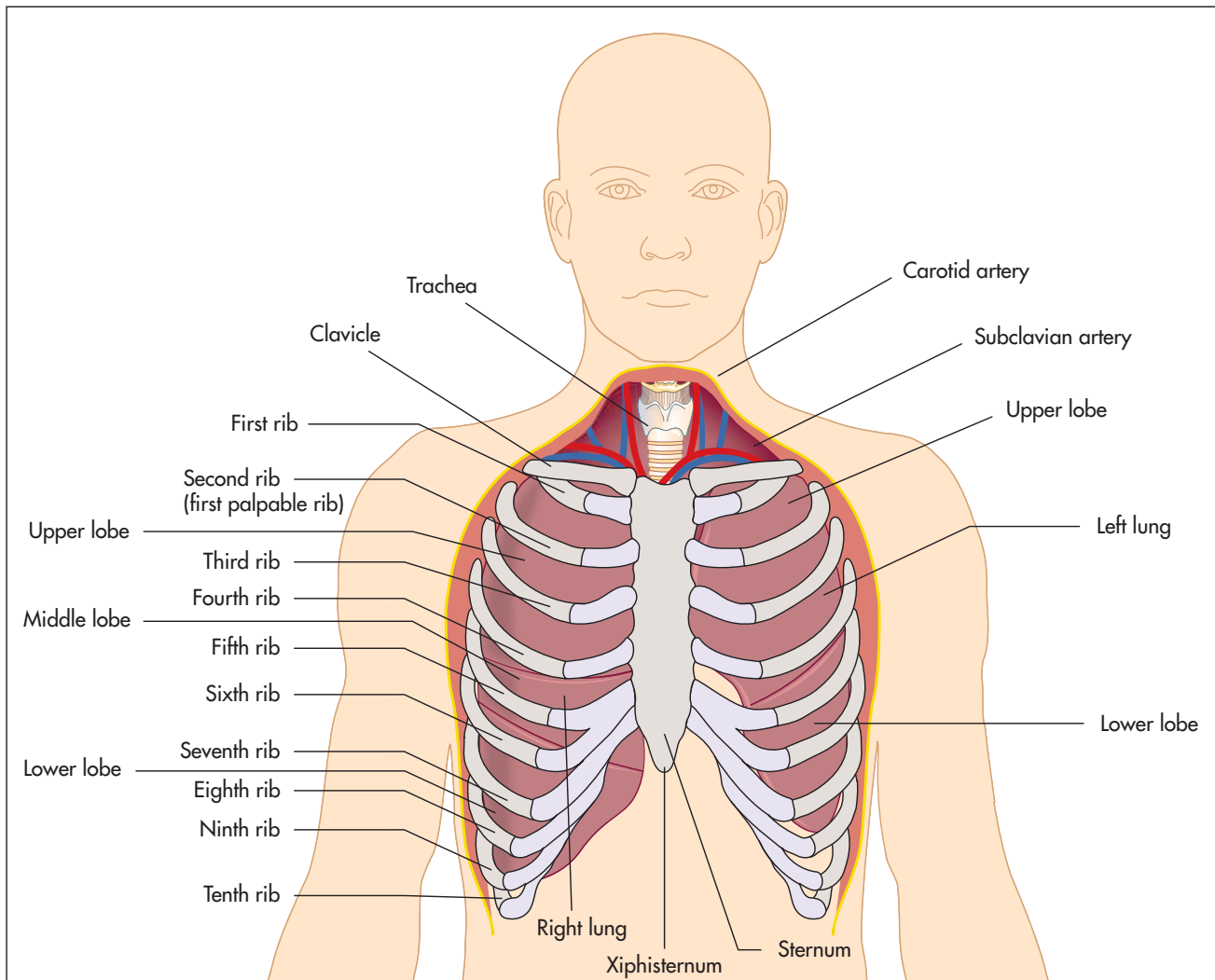
### CHARACTERISTIC SIGNS OF COPD

Look to see whether the accessory muscles of respiration are being used. This is a sign of an

increase in the work of breathing, and COPD<sup>a</sup> is an important cause. These muscles include the sternocleidomastoids, the platysma and the strap muscles of the neck. Characteristically, the accessory muscles cause elevation of the shoulders with inspiration, and aid respiration by increasing chest expansion. Contraction of the abdominal muscles

<sup>a</sup> This condition has undergone many changes in nomenclature, and it is pleasing to think that chest doctors have something to keep them occupied. The term COPD encompasses emphysema, chronic bronchitis, chronic obstructive lung disease (COLD) and chronic airflow limitation (CAL). This term seems quite firmly established (at least for now). The diagnosis of COPD depends on clinical, radiographic and lung function assessment. There may be components of what used to be called chronic bronchitis and emphysema.





**Figure 10.2** Basic anatomy of the lungs

may occur in expiration in patients with obstructed airways. Patients with severe COPD often have in-drawing of the intercostal and supraclavicular spaces during inspiration. This is due to a delayed increase in lung volume despite the generation of large negative pleural pressures.

In some cases, the pattern of breathing is diagnostically helpful (see Table 9.4, page 134). Look for pursed-lips breathing, which is characteristic of patients with severe COPD. This manoeuvre reduces the patient's breathlessness, possibly by providing continuous positive airways pressure and helping to prevent airways collapse during expiration. Patients with severe COPD may feel more comfortable leaning forwards with their arms on their knees. This position compresses the abdomen and pushes the diaphragm upwards. This partly restores its normal domed shape and improves its effectiveness during inspiration. Increased diaphragmatic movements

may cause downward displacement of the trachea during inspiration—tracheal tug (this is also a sign of severe asthma, especially in children).<sup>b</sup>

## CYANOSIS

Central cyanosis is best detected by inspecting the tongue. Examination of the tongue differentiates central from peripheral cyanosis. Lung disease severe enough to result in significant ventilation-perfusion imbalance (such as pneumonia, COPD and pulmonary embolism), may cause reduced arterial oxygen saturation and central cyanosis. Cyanosis becomes evident when the absolute concentration of deoxygenated haemoglobin is

<sup>b</sup> When the trachea moves in time with the pulse, the sign suggests an aneurysm of the thoracic aorta—this is the original meaning of *tracheal tug*. Its appropriation by the chest doctors annoys those aware of its original meaning.

50 g/L of capillary blood. Cyanosis is usually obvious when the arterial oxygen saturation falls below 90% in a person with a normal haemoglobin level. Central cyanosis is therefore a sign of severe hypoxaemia. In patients with anaemia, cyanosis does not occur until even greater levels of arterial desaturation are reached. The absence of obvious cyanosis does not exclude hypoxia. The detection of cyanosis is much easier in good (especially fluorescent) lighting conditions and is said to be more difficult if the patient's bed is surrounded by cheerful pink curtains.

## CHARACTER OF THE COUGH

Coughing is a protective response to irritation of sensory receptors in the submucosa of the upper airways or bronchi. Ask the patient to cough several times. *Lack of the usual explosive beginning* may indicate vocal cord paralysis (the 'bovine' cough). *A muffled, wheezy, ineffective cough* suggests obstructive pulmonary disease. *A very loose productive cough* suggests excessive bronchial secretions due to chronic bronchitis, pneumonia or bronchiectasis. *A dry, irritating cough* may occur with chest infection, asthma or carcinoma of the bronchus and sometimes with left ventricular failure or interstitial lung disease (ILD). It is also typical of the cough produced by ACE inhibitor drugs. *A barking or croupy cough* may suggest a problem with the upper airway—the pharynx and larynx, or pertussis infection.

## SPUTUM

Sputum should be inspected. Careful study of the sputum is an essential part of the physical examination. The colour, volume and type (purulent, mucoid or mucopurulent), and the presence or absence of blood, should be recorded.

## STRIDOR

Obstruction of the larynx or trachea (the extra-thoracic airways) may cause stridor, a rasping or croaking noise loudest on inspiration. This can be due to a foreign body, a tumour, infection (e.g. epiglottitis) or inflammation (see List 10.1). It is a sign that requires urgent attention.

## HOARSENESS

Listen to the patient's voice for hoarseness (dysphonia), as this may indicate recurrent laryngeal nerve palsy associated with carcinoma of the lung (usually left-sided), or laryngeal carcinoma. However, the most common cause is laryngitis and the use of inhaled corticosteroids for asthma. Non-respiratory causes include hypothyroidism.

### LIST 10.1 Important causes of stridor in adults

#### Sudden onset (minutes)

Anaphylaxis  
Toxic gas inhalation  
Acute epiglottitis  
Inhaled foreign body

#### Gradual onset (days, weeks)

Laryngeal or pharyngeal tumours  
Cricoarytenoid rheumatoid arthritis  
Bilateral vocal cord palsy  
Tracheal carcinoma  
Paratracheal compression by lymph nodes  
Post-tracheostomy or intubation granulomata

## Hands

As usual, examination in detail begins with the hands.

### CLUBBING

Look for clubbing, which is due to respiratory disease in up to 80% of cases (see Figure 10.3 and List 5.1 on page 62). An uncommon but important association with clubbing is *hypertrophic pulmonary osteoarthropathy* (HPO). HPO is characterised by the presence of periosteal inflammation at the distal ends of long bones, the wrists, the ankles and the metacarpal and the metatarsal bones. There is swelling and tenderness over the wrists and other involved areas. Rarely HPO may occur without clubbing. The causes of HPO include primary lung carcinoma and pleural fibromas. It is important to note that clubbing does not occur as a result of COPD.

### STAINING

Look for staining of the fingers (actually caused by tar, as nicotine is colourless); a sign of cigarette smoking (see Figure 3.12 on page 36). The density of staining does not indicate the number of cigarettes



Figure 10.3 Finger clubbing

# Correlation of physical signs and respiratory disease

*Life cannot be maintained without respiration, neither can respiration be performed without motion.*

Crooke, *Body of Man* (1615)

## Respiratory distress: respiratory failure

A severe respiratory illness may be a medical emergency. Many of the respiratory illnesses discussed below and some non-respiratory illnesses (see List 11.1) can result in acute respiratory problems and it is important to recognise signs that suggest there is an urgent problem. These signs include:

- cyanosis or low SpO<sub>2</sub> on oximetry
- use of accessory muscles of respiration
- inability to speak
- greatly increased or reduced respiratory rate
- signs of exhaustion
- silent lung fields
- stridor (airway obstruction)
- drowsiness
- chest injury
- tachycardia
- pulsus paradoxus.

There may be signs of an underlying respiratory illness (see Table 11.1).

## Consolidation (lobar pneumonia)

Pneumonia is defined as inflammation of the lung that is characterised by exudation into the alveoli (see Good signs guide 11.1). X-ray changes of new shadowing in one or more lung segments (lobes) are present. Pneumonia is now classified as:

1. community-acquired (CAP)
2. hospital-acquired

### LIST 11.1 Causes of acute respiratory distress or failure

#### Lung disease

COPD or asthma  
 Very large pleural effusion  
 Pneumonia  
 Non-cardiogenic pulmonary oedema (e.g. from toxic gas inhalation)  
 Pulmonary embolism  
 Chest injury or pneumothorax

#### Airways obstruction

Inhaled foreign body  
 Facial or neck injury  
 Angio-oedema  
 Epiglottitis or quinsy  
 Unconsciousness and aspiration—loss of airway protecting reflexes

#### Non-respiratory causes

Anaemia  
 Diabetic ketoacidosis  
 Anxiety and hyperventilation

3. occurring in a damaged lung (e.g. as a result of aspiration)
  4. occurring in an immunocompromised host.
- This classification allows prediction of the likely pathogens and assists in the choice of antibiotics for treatment. The signs of lobar pneumonia are characteristic and are referred to clinically as *consolidation*.<sup>1</sup>

There may be a history of the sudden onset of malaise, chest pain, dyspnoea and fever. Patients may appear very ill and the vital signs—including the temperature, respiratory rate and blood pressure—must be recorded. There may be signs of cyanosis and exhaustion in sick patients. The term *broncho-pneumonia* refers to lung infection characterised by more patchy X-ray changes that often affect both lower lobes. The clinical signs of consolidation may be absent.

**TABLE 11.1** Comparison of the chest signs in common respiratory disorders

Disorder	Mediastinal displacement	Chest wall movement	Percussion note	Breath sounds	Added sounds
Consolidation	None	Reduced over affected area	Dull	Bronchial	Crackles
Collapse	Ipsilateral shift	Decreased over affected area	Dull	Absent or reduced	Absent
Pleural effusion	Heart displaced to opposite side (trachea displaced only if massive)	Reduced over affected area	Stony dull	Absent over fluid; may be bronchial at upper border	Absent; pleural rub may be found above effusion
Pneumothorax	Tracheal deviation to opposite side if under tension	Decreased over affected area	Resonant	Absent or greatly reduced	Absent
Bronchial asthma	None	Decreased symmetrically	Normal or decreased	Normal or reduced	Wheeze
Interstitial pulmonary fibrosis	None	Decreased symmetrically (minimal)	Normal unaffected by cough or posture	Normal	Fine, late or pan-inspiratory crackles over affected lobes

## SYMPTOMS

- Cough (painful and dry at first).
- Fever and rigors (shivers).
- Pleuritic chest pain.
- Dyspnoea.
- Tachycardia.
- Confusion.

### GOOD SIGNS GUIDE 11.1 Pneumonia

Sign	LR+	LR-
<b>General appearance</b>		
Dementia	4.0	NS
Vital signs	3.4	0.94
Temperature >37.8°C	2.4	0.58
Respiratory rate >25/minute	1.5	0.8
<b>Heart rate</b>		
>100 beats/minute	2.3	0.49
<b>Lung findings</b>		
Percussion dullness	4.3	0.79
Reduced breath sounds	2.5	0.6
Bronchial breath sounds	3.5	0.9
Aegophony	5.3	0.76
Crackles	3.5	0.62
Wheezes	1.4	0.76

NS = not significant.  
(Heckerling PS, Tape TG et al. Clinical prediction rule for pulmonary infiltrates. *Ann Intern Med* 1990; 113(9):664–670.)

## SIGNS

- **Expansion:** reduced on the affected side.
- **Vocal fremitus:** increased on the affected side (in other chest disease this sign is of very little use!).
- **Percussion:** dull, but not stony dull.
- **Breath sounds:** bronchial.
- **Additional sounds:** medium, late or pan-inspiratory crackles as the pneumonia resolves.
- **Vocal resonance:** increased.
- **Pleural rub:** may be present.

## CAUSES OF COMMUNITY-ACQUIRED PNEUMONIA

- *Streptococcus pneumoniae* (>30%).
- *Chlamydia pneumoniae* (10%).
- *Mycoplasma pneumoniae* (10%).
- *Legionella pneumoniae* (5%).

## Atelectasis (collapse)

If a bronchus is obstructed by a tumour mass, retained secretions or a prolonged presence of a foreign body, the air in the part of the lung supplied by the bronchus is absorbed and the affected part of the lung collapses.

## SIGNS

- **Trachea:** displaced towards the collapsed side.
- **Expansion:** reduced on the affected side with flattening of the chest wall on the same side.
- **Percussion:** dull over the collapsed area.

- **Breath sounds:** reduced, often without bronchial breathing above the area of atelectasis when a tumour is the cause, because the airways are not patent.

*Note:* (1) There may be no signs with complete lobar collapse. (2) The early changes after the inhalation of a foreign body may be over-inflation of the affected side.

## CAUSES

- **Intraluminal:** mucus (e.g. postoperative, asthma, cystic fibrosis), foreign body, aspiration.
- **Mural:** bronchial carcinoma.
- **Extramural:** peribronchial lymphadenopathy, aortic aneurysm.

## Pleural effusion

This is a collection of fluid in the pleural space. Note that pleural collections consisting of blood (haemothorax), chyle (chylothorax) or pus (empyema) have specific names and are not called pleural effusions, although the physical signs are similar.

## SIGNS

- **Trachea and apex beat:** displaced away from a massive effusion.
- **Expansion:** reduced on the affected side.
- **Percussion:** stony dullness over the fluid.
- **Breath sounds:** reduced or absent. There may be an area of bronchial breathing audible above the effusion due to compression of overlying lung.
- **Vocal resonance:** reduced.

## CAUSES

- **Transudate** (Light's criteria): (1) cardiac failure; (2) hypoalbuminaemia from the nephrotic syndrome or chronic liver disease; (3) hypothyroidism.
- **Exudate** (Light's criteria<sup>a</sup>): (1) pneumonia; (2) neoplasm—bronchial carcinoma, metastatic carcinoma, mesothelioma; (3) tuberculosis; (4) pulmonary infarction; (5) subphrenic abscess; (6) acute pancreatitis; (7) connective tissue disease such as rheumatoid arthritis, systemic lupus erythematosus; (8) drugs such as methysergide, cytotoxics; (9) irradiation; (10) trauma; (11) Meigs' syndrome<sup>b</sup> (ovarian fibroma causing pleural effusion and ascites).

<sup>a</sup> The formal definition of an exudate is that the fluid has at least one of the following (Light's) criteria; (1) fluid protein/serum protein >0.5; (2) pleural fluid LDH/serum LDH >0.6; (3) pleural fluid LDH >2/3 normal upper limit of LDH in serum. The fluid is otherwise a transudate.

<sup>b</sup> Joe Vincent Meigs (1892–1963), Professor of Gynaecology at Harvard, described this in 1937.

- **Haemothorax** (blood in the pleural space): (1) severe trauma to the chest; (2) rupture of a pleural adhesion containing a blood vessel.
- **Chylothorax** (milky-appearing pleural fluid due to leakage of lymph): (1) trauma or surgery to the thoracic duct; (2) carcinoma or lymphoma involving the thoracic duct.
- **Empyema** (pus in the pleural space): (1) pneumonia; (2) lung abscess; (3) bronchiectasis; (4) tuberculosis; (5) penetrating chest wound.

## YELLOW NAIL SYNDROME

This is a rare condition that is caused by hypoplasia of the lymphatic system. The nails are thickened and yellow (see Figure 11.1) and there is separation of the distal nail plate from the nail bed (onycholysis). It may be associated with a pleural effusion and bronchiectasis, and usually with lymphoedema of the legs.



**Figure 11.1** Yellow nail syndrome: (a) hands; (b) feet (From McDonald FS, ed. *Mayo Clinic images in internal medicine*, with permission. © Mayo Clinic Scientific Press and CRC Press.)

## Pneumothorax

Leakage of air from the lung or a chest wall puncture into the pleural space causes a pneumothorax.

### SIGNS

- **Expansion:** reduced on the affected side.
- **Percussion:** hyperresonance if the pneumothorax is large.
- **Breath sounds:** greatly reduced or absent.
- There may be subcutaneous emphysema.
- There may be no signs if the pneumothorax is small (less than 30%).

### CAUSES

#### Primary

- **'Spontaneous':** subpleural bullae rupture, usually in tall, healthy young males.

#### Secondary

- **Traumatic:** rib fracture, penetrating chest wall injury, or during pleural or pericardial aspiration.
- **Iatrogenic<sup>c</sup>** (caused by medical intervention): following the insertion of a central venous catheter.
- Emphysema with rupture of bullae, usually in middle-aged or elderly patients with generalised emphysema.
- Rarer causes include asthma, lung abscess, bronchial carcinoma, eosinophilic granuloma, lymphangioliomyomatosis (LAM—pre-menopausal women), end-stage fibrosis or Marfan's syndrome.

## Tension pneumothorax

This occurs when there is a communication between the lung and the pleural space, with a flap of tissue acting as a valve, allowing air to enter the pleural space during inspiration and preventing it from leaving during expiration. A tension pneumothorax results from air accumulating under increasing pressure in the pleural space; it causes considerable displacement of the mediastinum with obstruction and kinking of the great vessels, and represents a medical emergency.

### SIGNS

- The patient is often tachypnoeic and cyanosed, and may be hypotensive.
- **Trachea and apex beat:** displaced away from the affected side.
- **Expansion:** reduced or absent on affected side.

- **Percussion:** hyperresonant over the affected side.
- **Breath sounds:** absent.
- **Vocal resonance:** absent.

### CAUSES

- Trauma.
- Mechanical ventilation at high pressure.
- Spontaneous (rare cause of tension pneumothorax).

## Bronchiectasis

This is a pathological dilation of the bronchi, resulting in impaired clearance of mucus and chronic infection. A history of chronic cough and purulent sputum since childhood is virtually diagnostic.

### SIGNS

Most likely during an exacerbation of the condition.

- **Systemic signs:** fever, cachexia; sinusitis (70%).
- **Sputum:** voluminous, purulent, foul-smelling, sometimes bloodstained.
- **Breath sounds:** Coarse pan-inspiratory or late inspiratory crackles over the affected lobe.
- **Signs of severe bronchiectasis:** very copious amounts of sputum and haemoptysis, clubbing, cyanosis, widespread crackles, signs of airways obstruction, signs of respiratory failure and cor pulmonale, signs of secondary amyloidosis (e.g. oedema from proteinuria, cardiac failure, enlarged liver and spleen, carpal tunnel syndrome).

### CAUSES

- **Congenital:** (1) primary ciliary dyskinesia (including the immotile cilia syndrome); (2) cystic fibrosis; (3) congenital hypogammaglobulinaemia.
- **Acquired:** (1) infections in childhood, such as whooping cough, pneumonia or measles; (2) localised disease such as a foreign body, a bronchial adenoma or tuberculosis; (3) allergic bronchopulmonary aspergillosis—this causes proximal bronchiectasis.

## Bronchial asthma

This may be defined as paroxysmal recurrent attacks of wheezing (or in childhood of cough) due to airways narrowing, which changes in severity over short periods of time.

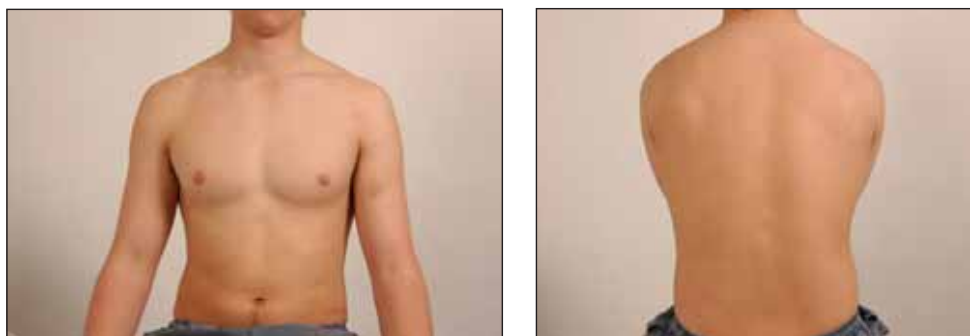
<sup>c</sup> *Iatros* means physician in Greek.

# A summary of the respiratory examination and extending the respiratory examination

*Investigation; the act of the mind by which unknown truths are discovered.*

Samuel Johnson, *A Dictionary of the English Language* (1775)

## TEXT BOX 12.1 The respiratory examination: a suggested method



**Figure 12.1** Respiratory system  
(Courtesy of Glenn McCulloch)

Sitting up (if not acutely ill)

### 1. General inspection

- Sputum mug contents (blood, pus etc)
- Type of cough
- Rate and depth of respiration, and breathing pattern at rest
- Accessory muscles of respiration

### 2. Hands

- Clubbing
- Cyanosis (peripheral)
- Nicotine staining
- Wasting, weakness—finger abduction and adduction (lung cancer involving the brachial plexus)
- Wrist tenderness (hypertrophic pulmonary osteoarthropathy)
- Pulse (tachycardia, pulsus paradoxus)
- Flapping tremor (CO<sub>2</sub> narcosis)

### 3. Face

- Eyes—Horner's syndrome (apical lung cancer), anaemia
- Mouth—central cyanosis

- Voice—hoarseness (recurrent laryngeal nerve palsy)
- Facial plethora—smoker, SVC obstruction

### 4. Trachea

### 5. Chest posteriorly

- Inspect*
- Shape of chest and spine
- Scars
- Prominent veins (determine direction of flow)
- Palpate*
- Cervical lymph nodes
- Expansion
- Vocal fremitus
- Percuss*
- Supraclavicular region
- Back
- Axillae
- Tidal percussion (diaphragm paralysis)
- Auscultate*
- Breath sounds
- Adventitious sounds
- Vocal resonance

*continues*

TEXT BOX 12.1 The respiratory examination: a suggested method *continued***6. Chest anteriorly***Inspect*

Radiotherapy marks, other signs as noted above

*Palpate*

Supraclavicular nodes

Expansion

Vocal fremitus

Apex beat

*Percuss**Auscultate*

Pemberton's sign (SVC obstruction)

**7. Cardiovascular system (lying at 45°)**

Jugular venous pressure (SVC obstruction etc)

Cor pulmonale

**8. Forced expiratory time****9. Other**

Lower limbs—oedema, cyanosis

Breasts

Temperature chart (infection)

Evidence of malignancy or pleural effusion:

examine the breasts, abdomen, rectum, lymph

nodes etc

Respiratory rate after exercise

Ask the patient to undress to the waist (provide women with a gown) and to sit over the side of the bed. In the clinic or surgery the examination can often be performed with the patient sitting on a chair. While standing back to make your usual **inspection** (does the patient appear breathless while walking into the room or undressing?), ask if sputum is available for inspection. Purulent sputum always indicates respiratory infection, and a large volume of purulent sputum is an important clue to bronchiectasis. Haemoptysis is also an important sign. Look for dyspnoea at rest and count the respiratory rate. Note any paradoxical inward motion of the abdomen during inspiration (diaphragmatic paralysis). Look for use of the accessory muscles of respiration, and any intercostal in-drawing of the lower ribs anteriorly (a sign of emphysema). General cachexia should also be noted.

Pick up the patient's **hands**. Look for clubbing, peripheral cyanosis, tar staining and anaemia. Note any wasting of the small muscles of the hands and weakness of finger abduction (lung cancer involving the brachial plexus). Palpate the wrists for tenderness (hypertrophic pulmonary osteoarthropathy). While holding the patient's hand, palpate the radial pulse for obvious pulsus paradoxus. Take the blood pressure if indicated.

Go on to the **face**. Look closely at the eyes for constriction of one of the pupils and for ptosis (Horner's syndrome from an apical lung cancer). Inspect the tongue for central cyanosis.

Palpate the position of the **trachea**. This is an important sign, so spend time on it. If the trachea is displaced, you must concentrate on the upper lobes for physical signs. Also look and feel for a tracheal tug, which indicates severe airflow obstruction, and feel for the use of the accessory muscles. Now ask the patient to speak (hoarseness) and then cough, and note whether this is a loose cough, a dry cough or a bovine cough. Next measure the forced expiratory time (FET).<sup>a</sup> Tell the patient to take a maximal inspiration and blow out as rapidly and forcefully as possible while you

listen. Note audible wheeze and prolongation of the time beyond 3 seconds as evidence of chronic obstructive pulmonary disease.<sup>1</sup>

The next step is to examine the **chest**. You may wish to examine the front first, or go to the back to start. The advantage of the latter is that there are often more signs there, unless the trachea is obviously displaced.

Inspect the **back**. Look for kyphoscoliosis. Do not miss ankylosing spondylitis, which causes decreased chest expansion and upper lobe fibrosis. Look for thoracotomy scars and prominent veins. Also note any skin changes from radiotherapy.

**Palpate** first from behind for the cervical nodes. Then examine for expansion—first upper lobe expansion, which is best seen by looking over the patient's shoulders at clavicular movement during moderate respiration. The affected side will show a delay or decreased movement. Then examine lower lobe expansion by palpation. Note asymmetry and reduction of movement.

Now ask the patient to bring his or her elbows together in the front to move the scapulae out of the way. Examine for vocal fremitus and then **percuss** the back of the chest.

**Auscultate** the chest. Note breath sounds (whether normal or bronchial) and their intensity (normal or reduced). Listen for adventitious sounds (crackles and wheezes). Finally examine for vocal resonance. If a localised abnormality is found, try to determine the abnormal lobe and segment.

Return to the **front of the chest**. Inspect again for chest deformity, distended veins, radiotherapy changes and scars. Palpate the supraclavicular nodes carefully. Then proceed with percussion and auscultation as before. Listen high up in the axillae too. Before leaving the chest feel the axillary nodes and examine the breasts (see Chapter 36).

**Lay the patient down at 45°** and measure the jugular venous pressure. Then examine the praecordium and lower limbs for signs of cor pulmonale. Finally examine the **liver** and take the **temperature**.

Remember that most respiratory examinations are 'targeted'. Not every part of the examination is necessary for every patient.

<sup>a</sup> There is good correlation between clinicians for the results of this test: κ value 0.7. It is most accurate if performed with a stopwatch.



## Extending the respiratory physical examination

### BEDSIDE ASSESSMENT OF LUNG FUNCTION

#### Forced expiratory time

Physical examination can be complemented with an estimate of the forced expiratory time (FET).<sup>1</sup> Measure the time taken by the patient to exhale forcefully and completely through the open mouth after taking a maximum inspiration. It may be necessary to demonstrate this to the patient. The normal forced expiratory time is 3 seconds or less. Note any audible wheeze or cough. An increased FET indicates airways obstruction. The combination of a significant smoking history and an FET of 9 seconds or more is predictive of COPD (positive LR 9.6).<sup>2</sup> A peak flow meter or spirometer, however, will provide a more accurate measurement of lung function.

#### Peak flow meter

A peak flow meter is a simple gauge that is used to measure the maximum flow rate of expired air. Again the patient is asked to take a full breath in, but rather than a prolonged expiration, a rapid forced maximal expiratory puff is made through the mouth.<sup>b</sup> The value obtained (the peak expiratory flow [PEF]) depends largely on airways diameter. Normal values are approximately 600 litres per minute for young men and 400 litres per minute for young women. The value depends on age, sex and height, so tables of normal values should be consulted. Airways obstruction, such as that caused by asthma or COPD, results in a reduced and variable PEF. It is a simple way of assessing and following patients with airways obstruction, but is rather effort-dependent. The PEF

is most useful when used for serial estimates of lung function.

#### Spirometry

The spirometer records graphically or numerically the forced expiratory volume and the forced vital capacity (see Figure 12.2). The *forced expiratory volume* (FEV) is the volume of air expelled from the lungs after maximum inspiration using maximum forced effort, and is measured in a given time.<sup>c</sup> Usually this is 1 second (FEV<sub>1</sub>). The *forced vital capacity* (FVC) is the total volume of air expelled from the lungs after maximum inspiratory effort followed by maximum forced expiration. The FVC is often nearly the same as the vital capacity, but in airways obstruction it may be less because of premature airways closure. It is usual to record the best of three attempts and to calculate the FEV<sub>1</sub>/FVC ratio as a percentage. In healthy youth, the normal value is 80%, but this may decline to as little as 60% in old age. Normal values also vary with sex, age, height and race.

Reversibility of a reduced FEV<sub>1</sub>/FVC after the use of bronchodilators is an important test for distinguishing asthma from COPD.

#### Obstructive ventilatory defect

When the FEV<sub>1</sub>/FVC ratio is reduced (<0.7) this is referred to as an obstructive defect. Both values tend to be reduced, but the FEV<sub>1</sub> is disproportionately low. The causes are loss of elastic recoil or airways narrowing, as in asthma or COPD.

#### Restrictive ventilatory defect

When the FEV<sub>1</sub>/FVC ratio is normal or higher than normal, but both values are reduced, the pattern is described as a restrictive defect. This occurs in parenchymal lung disease, such as ILD, sarcoidosis or when lung expansion is reduced by pneumonia or chest wall abnormalities.

<sup>b</sup> Students are advised to practise this so as to be able to demonstrate it without embarrassment.

<sup>c</sup> Ask the patient to breathe in as far as possible and then to breathe out as hard and fast as possible until the lungs are apparently empty.

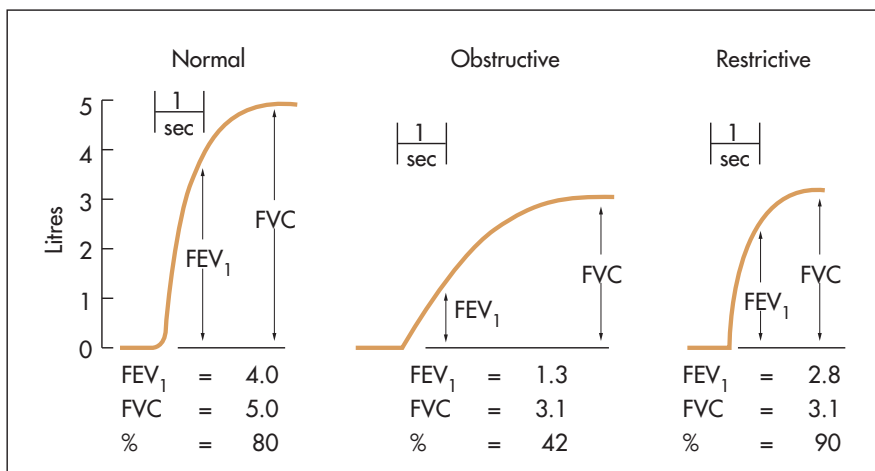


Figure 12.2 Spirometry tracings

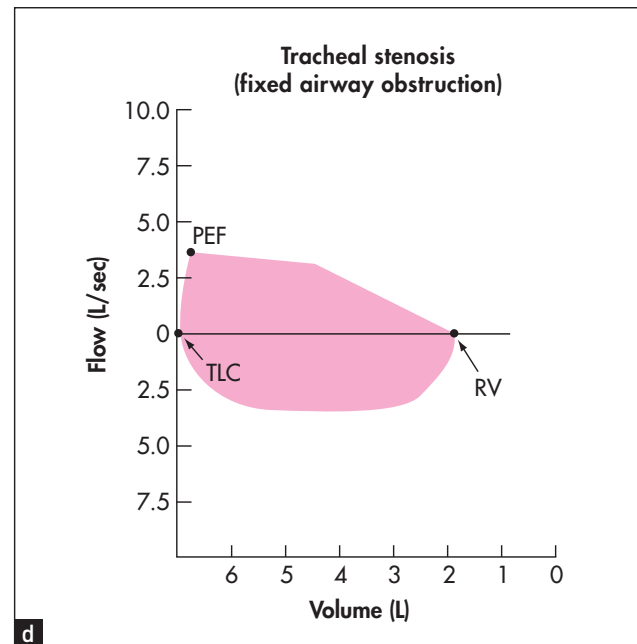
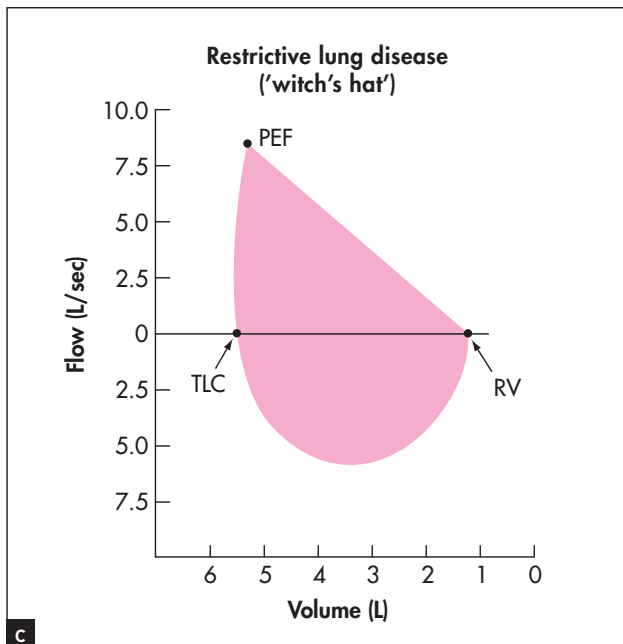
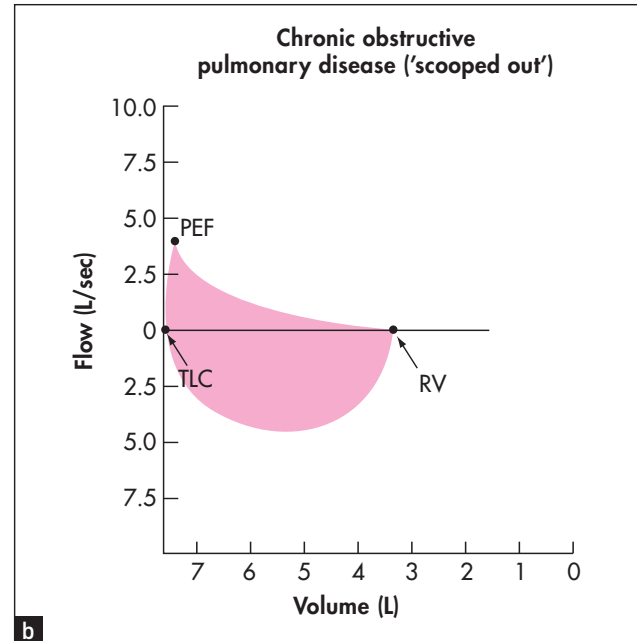
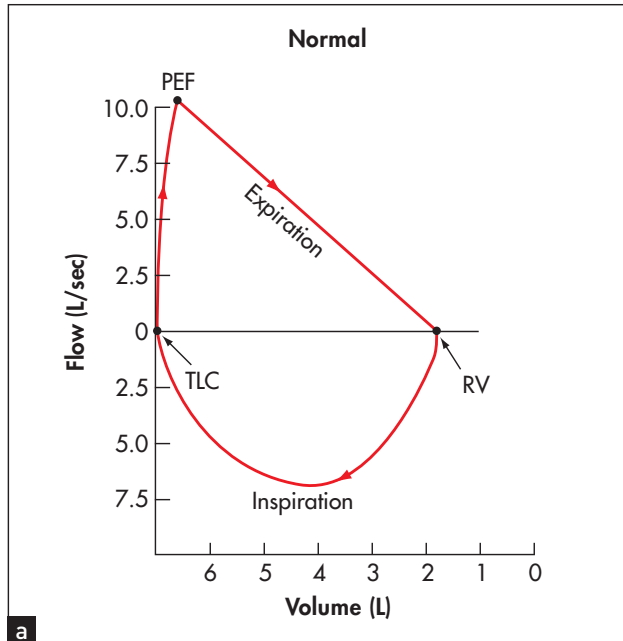
### Flow volume curve

As a part of spirometric assessment, the flow volume curve may be measured using a portable electronic device. This measures expiratory and inspiratory flow as a function of exhaled volume rather than against time. It is a simple and reproducible test easily performed in the respiratory laboratory or at the bedside. The FVC, FEV<sub>1</sub> and various flow

measurements (e.g. peak flow) can be calculated from the curve (see Figure 12.3).

### Pulse oximetry

Continuous measurement of a patient's arterial blood oxygen saturation (SpO<sub>2</sub>) is now possible with readily available oximetry devices. These simple devices can be used as an extension of the physical examination,



**Figure 12.3** Flow volume curves

Look at the shape of the loop in each case ((a) to (d)). A normal flow volume curve is convex and symmetrical. In chronic obstructive lung disease (COPD), all flow routes are reduced and there is prolonged expiration (creating a 'scooped out' shape). In restrictive lung disease (e.g. pulmonary fibrosis), the loop is narrow but the shape normal (like a witch's hat). In fixed airway obstruction (e.g. tracheal stenosis), the loops look flattened as both expiration and inspiration are limited.

PEF = peak expiratory flow; TLC = total lung capacity; RV = residual volume.