Techniques are outlined and compared with Doppler ultrasound assessment.

[37] Stevenson LW, Perluff JK. The limited reliability of physical signs for estimating hemodynamics in chronic heart failure. *JAMA*. 1989;261:884-888. Physical signs poorly predict haemodynamic changes in heart failure. However, some signs are useful.

[38] Khot U, Jia G, Moliterno DJ, et al. Prognostic value of physical examination for heart failure in non-ST elevation acute coronary syndromes. *JAMA*. 2003;290:2174-2181. This analysis of the Killip classification for patients with acute coronary syndromes expands the relevance of the classification from its original use for patients with ST elevation infarction in the pre-thrombolytic era.

[39] Klompas M. Does this patient have an acute thoracic dissection? *JAMA*. 2002;287(17):2262-2272.

[40] Turnbull JM. Is listening for abdominal bruits useful in the evaluation of hypertension? The rational clinical examination. *JAMA*. 1995;274:16. If an abdominal bruit extends into diastole, this has a high predictive value for a clinically important bruit. The pitch and intensity are not helpful.

[41] Etchells E, Bell C, Robb K. Does this patient have an abnormal systolic murmur? *JAMA*. 1997;277:564-571. The most useful positive predictive features for aortic stenosis appear to be a slow rate of rise of the carotid pulse, a mid-to-late peak intensity of the murmur and a decreased second heart sound; absence of radiation to the right carotid helps rule it out.

[42] Aronow WS. Prevalence and severity of valvular aortic stenosis determined by Doppler echocardiography, and its association with echocardiographic and electrocardiographic left ventricular hypertrophy and physical signs of aortic stenosis in elderly patients. *Am J Cardiol*. 1991;67:776-777. Analysis of the signs of severity of aortic stenosis in elderly patients shows that they are less reliable than in younger patients.

[43] Choudhry NK, Etchells EE. Does this patient have aortic regurgitation? *JAMA*. 1999;281:2231-2238. (Rational Clinical Examination Series)

[44] Babu AN, Kymes SM, Carpenter Fryer SM. Eponyms and the diagnosis of aortic regurgitation: What says the evidence? *Ann Intern Med*. 2003;138:736-745.

Suggested reading

Braunwald E, Zipes DP, Libby P. *Heart disease. A textbook of cardiovascular medicine*, 8th edn. Philadelphia: WB Saunders; 2007.

Crawford MH, DiMarco JP, Paulus WJ. *Cardiology*, 2nd edn. London: Mosby; 2004.

[a] William Heberden's (1710–1801) description of angina (1768) is difficult to improve upon: 'They who are afflicted with it, are seized while they are walking (more especially if it be up hill and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life, if it were to increase or continue; but the moment they stand still, all this uneasiness vanishes.'

[b] Paroxysmal symptoms or signs occur suddenly and intermittently.

[d] Stokes-Adams attacks were probably described first by Gerbezius in 1691 and then by Morgagni in 1761; the latter was a pupil of Valsalva and also described Turner's syndrome 170 years before Turner.

[e] The Roman Emperor Tiberius Claudius Drusus Nero Germanicus (10 BC–54 AD) limped owing to some form of paralysis. 'Claudication' and 'Claudius', however, are etymologically unrelated, which seems rather a cruel coincidence for Claudius. 'Claudicant' first appeared in English in 1624.

[f] Bernard-Jean Antonin Marfan (1858–1942), French physician and first professor of hygiene in Paris.

[g] John Langdon Down (1828–96), Assistant Physician to the London Hospital and founder of the Normansfield Mental Hospital. He described the clinical picture of mongolism in 1866.

[h] Henry Hubert Turner (1892–1970), Clinical Professor of Medicine, Oklahoma University. He described the syndrome in 1938.

[i] The eminent South African cardiologist Leo Schamroth developed clubbing as a result of endocarditis in 1976. As the condition advanced, he observed in his own fingers that the diamond-shaped space formed when the nails of two similar fingers were held facing each other disappeared, only to reappear as he improved.

[j] Sir William Osler (1849–1919), Canadian physician, Professor of Medicine at McGill University at 25, and later famous Regius professor of medicine at Oxford and renowned medical historian. He was made a baronet. His only son was killed at Ypres.

[k] Edward Janeway (1841–1911), American physician.

[l] These signs are now mostly of historical interest. They date from the period when endocarditis could be diagnosed but not treated. Physicians were able to describe and name interesting signs but were unable to

provide treatment (cf the eponymous signs of aortic regurgitation, page 87).

[m] Marel Frederik Wenckebach (1864–1940), Dutch physician who practised in Vienna.

[n] Blood pressure was first measured in a horse in 1708 by Stephen Hales, an English clergyman. Measurement of the blood pressure was the last of the traditional vital signs measurements to come into regular use. It wasn't until early in the 20th century that work by Korotkoff and Janeway led to its routine use.

[o] Nikolai Korotkoff (1874–1920), a St Petersburg surgeon, described the auscultatory method of determining blood pressure in 1905, although his findings were scoffed at.

[p] In pseudohypertension the blood pressure, as measured by the sphygmomanometer, is artificially high because of arterial wall calcification. Osler's manoeuvre traditionally detects this condition: inflate the cuff above systolic pressure and palpate the radial artery, which in pseudohypertension may be palpable despite being pulseless. However, the value of Osler's manoeuvre has been questioned.[14]

[q] The connection of arcus senilis with old age and cardiovascular disease has been made from early in the 19th century. The pathologist Virchow was convinced it was an indicator of vascular disease.

[r] The waves visible in the JVP were named by Sir James Mackenzie (1853–1925), British physician and one of the founders of the specialty of cardiology.

[s] Sir James Mackenzie first applied these labels to the jugular waveforms in the late 19th century.

[t] Adolf Kussmaul (1822–1909), German physician who also described laboured breathing ('air hunger') in diabetic coma (1874) and was the first to use an oesophagoscope. He coined the word 'hemiballismus'.

[u] First described by Louis Pasteur in 1885.

[v] James Hope was the first to demonstrate (in 1830) that the apex beat was caused by ventricular contraction. Jean-Nicholas Corvisant (Napoleon's personal doctor) was the first to associate abnormal palpation of the heart with cardiac chamber enlargements.

[w]

[w] Percussion of the heart and other organs was enthusiastically promoted by Pierre Poiry, a student of Laënnec, in the early 19th century. He performed indirect percussion using an ivory plate instead of his left middle finger.

[x] The term incompetence is synonymous with regurgitation, but the latter better describes the pathophysiology.

[y] Louis Hamman (1877–1946), physician, Johns Hopkins Hospital, Baltimore.

[z] Antonio Valsalva (1666–1723), Professor of Anatomy at Bologna, was noted for his studies of the ear. He described his manoeuvre in 1704. Forced expiration against the closed glottis causes discharge of pus into the external auditory canal in cases of chronic otitis media. Friedrich Weber rediscovered the manoeuvre in 1859 and demonstrated that he could slow his pulse at will. He stopped demonstrating this after he caused himself to faint and have convulsions.

[aa] Achilles, mythical Greek hero, whose body was invulnerable except for his heels, by which he was held when dipped in the River Styx as a baby to make him immortal. He was killed by Paris, who shot an arrow into his heel.

[bb] Leo Buerger (1879–1943), New York physician, born in Vienna, who described thromboangiitis obliterans. He was obsessed with expensive cars.

[cc] John Homans (1877–1954), professor of surgery, Harvard University, Boston. He described his sign in 1941, originally in cases of thrombophlebitis. He later became disenchanted with the sign and is reputed to have asked why if a sign were to be named after him it couldn't be a useful one.

[dd] Rudolph Virchow (1821–1902), brilliant German pathologist, regarded as the founder of modern pathology. professor of pathological anatomy in Berlin. He provided the first description of leukaemia. He died at 81 after fracturing his femur jumping from a moving tram.

[ee] Friedrich Trendelenburg (1844–1924), professor of surgery, Leipzig.

[ff] Georg Clemens Perthes (1869–1927), German surgeon, professor of surgery at Tübingen. He was the first to use radiotherapy for the treatment of cancer (in 1903).

[gg] T. Killip, a New Zealand cardiologist, published his classification in

T. Killip, a New Zealand cardiologist, published his classification in 1967.

[hh] William Dressler (1890–1969), a New York cardiologist, described this syndrome in 1956.

[ii] Moritz von Roth (1839–1914), Swiss physician and pathologist, described these changes in 1872.

[jj] Harvey Cushing (1869–1939), professor of surgery, Harvard University, and 'founder of neurosurgery'. Friend of Osler and prize-winning writer of Osler's biography in 1925.

[kk] This Victorian children's toy consisted of a sealed tube half-filled with fluid, with the other half being a vacuum. Inversion of the tube caused the fluid to fall rapidly without air resistance and strike the other end with a noise like a hammer blow. It is not easy to imagine a child today being entertained by this for very long.

[ll] Claudius Galen (130–200 AD). Born in Pergamum, he worked as a gladiator's surgeon but moved to Rome in 164 AD to become the city's most famous physician. He was the first to describe the cranial nerves. He never performed dissection on human bodies, but his often erroneous anatomical teachings were regarded as infallible for 15 centuries.

[mm] Austin Flint (1812–1886), New York physician and professor of medicine at the New Orleans Medical School, described this murmur in 1862. Author of The principles and practice of medicine. He was very much opposed to the naming of signs after people.

[nn] Doppler echocardiography has shown that trivial tricuspid regurgitation is very common and is then considered physiological. Christian Doppler (1803–1853) was an Austrian physicist and mathematician.

[oo] Wilhelm Ebstein (1836–1912), professor of medicine at Göttingen in Germany, who invented and developed palpation.

[pp] Graham Steell (1851–1942), Manchester physician, described this murmur in 1888.

[qq] Nikolaus Friedreich (1825–82), German physician, described this disease in 1863. He succeeded Virchow as professor of pathological anatomy at Würzburg at the age of 31.

[rr] Victor Eisenmenger (1864–1932), German physician. He described this

syndrome in 1897.

[ss] Etienne-Louis Fallot (1850–1911), professor of hygiene, Marseilles, described this in 1888.

[c] William Stokes (1804–1878) succeeded his father as Regius professor of physic in 1840. He was a member of the 'Dublin School' of medicine along with famous physicians like Graves, Cheyne, Adams and Corrigan. He was an art lover, and insisted his students have an arts degree before studying medicine.

Robert Adams (1791–1875) was Regius professor of surgery in Dublin, and became Queen Victoria's surgeon. He was affected by gout, and wrote a famous paper on it.

Chapter 5

The respiratory system

> A medical chest specialist is long-winded about the short-winded.
> *Kenneth T Bird (b. 1917)*

This chapter deals with common respiratory symptoms, and the examination of the respiratory system.

The respiratory history

Presenting symptoms (Table 5.1)

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.

TABLE 5.1 Respiratory history

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

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).

A differential diagnosis of cough based on its character is shown in Table 5.2 and on its duration is shown in Table 5.3.

TABLE 5.2 Differential diagnosis of cough based on its character

Origin	Character	Causes
Naso-pharynx/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

TABLE 5.3 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 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 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. 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. A chronic cough that is productive of large volumes of purulent sputum may be due to bronchiectasis.

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 inquire 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 (Table 5.4) and must always be investigated. It must be distinguished from haematemesis (vomiting of blood) and from nasopharyngeal bleeding (Table 5.5).

TABLE 5.4 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
(The above four account for about 80% of cases)	
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, multi-system involvement
Rupture of a mucosal blood vessel after vigorous coughing	

Cardiovascular
Mitral stenosis (severe)
Acute left ventricular failure
Bleeding diatheses

Note: 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.

TABLE 5.5 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	

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) (Table 5.6)

Breathlessness (dyspnoea) (Table 5.6)

The awareness that an abnormal amount of effort is required for breathing is called dyspnoea. It can be due to respiratory or cardiac disease, or lack of physical fitness. Careful questioning about the timing of onset, severity and pattern of dyspnoea is helpful in making the diagnosis (*Questions box 5.2* and Table 5.7).[1] The patient may be aware of this

TABLE 5.6 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 hypertonicity

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 Intracardiac shunt
Anaemia
Non-cardiorespiratory
Psychogenic Acidosis (compensatory respiratory alkalosis) Hypothalamic lesions

Questions box 5.2

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)

TABLE 5.7 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 Pleural effusion Anaemia

---

Questions box 5.1

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?—ACE inhibitors

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) (Table 5.8). 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* (Table 5.9). 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* (a not uncommon occurrence).

TABLE 5.8 Characteristics of chronic obstructive pulmonary disease (COPD)

History
History of smoking
Breathlessness and wheeze
Examination
Increased respiratory rate
Pursed-lips breathing
Cyanosis
Leaning forward—arms on knees
Intercostal and supraclavicular indrawing
Hoover’s sign
Tracheal tug

TABLE 5.9 Differential diagnosis of dyspnoea of sudden onset based on other features

Presence of pleuritic chest pain—favours:
Pneumothorax, pleurisy/pneumonia
Pulmonary embolism
Trauma
Absence of chest pain—favours:
Pulmonary oedema

Metabolic acidosis Pulmonary embolism
Presence of central chest pain—favours: Myocardial infarction and cardiac failure Large pulmonary embolism
Presence of cough and wheeze—favours: Asthma Bronchial irritant inhalation Chronic obstructive pulmonary disease (COPD)

Wheeze

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

Chest pain

Chest pain due to respiratory disease is usually different from that associated with myocardial ischaemia (page 35). The pleura and central airways have pain fibres and may be the source of respiratory pain. Pleural pain is characteristically *pleuritic* in nature: sharp and made worse by deep inspiration and coughing. It is typically localised to one area of the chest. It may be of sudden onset in patients with lobar pneumonia, pulmonary embolism and infarction or pneumothorax, and is often associated with dyspnoea. The sudden onset of pleuritic chest pain and dyspnoea is an urgent diagnostic problem, as all three of these conditions may be life-threatening if not treated promptly.

not treated promptly.

Other presenting symptoms

Bacterial pneumonia is an acute illness in which prodromal symptoms (fever, malaise and myalgia) occur for a short period (hours) before pleuritic pain and dyspnoea begin. *Viral pneumonia* is often preceded by a longer (days) prodromal illness. Patients may occasionally present with episodes of *fever at night*. Tuberculosis, pneumonia and lymphoma should always be considered in these cases. Occasionally patients with tuberculosis present with episodes of *drenching sweating* at night.

Hoarseness or *dysphonia* (an abnormality of the voice) may sometimes be considered a respiratory system symptom. It can be due to transient inflammation of the vocal cords (laryngitis), vocal cord tumour or recurrent laryngeal nerve palsy.

Sleep apnoea is an abnormal increase in the periodic cessation of breathing during sleep. Patients with *obstructive sleep apnoea* (OSA) (where airflow stops during sleep for periods of at least 10 seconds and sometimes for over 2 minutes, despite persistent respiratory efforts) typically present with daytime somnolence, chronic fatigue, morning headaches and personality disturbances. Very loud snoring may be reported by anyone within earshot. These patients are often obese and hypertensive. The Epworth sleepiness scale is a way of quantifying the severity of sleep apnoea (Table 5.10).

TABLE 5.10 The Epworth sleepiness scale

'How easily would you fall asleep in the following circumstances?'*
0 = never **1** = slight chance **2** = moderate chance **3** = high chance

- Sitting reading
- Watching television
- At a meeting or at the theatre
- As a passenger in a car on a drive of more than an hour
- Lying down in the afternoon to rest
- Sitting talking to someone
- Sitting quietly after lunch (no alcohol)
- When driving and stopped at traffic lights

* A normal score is between 0 and 9. Severe sleep apnoea scores from 11 to 20.

Patients with *central sleep apnoea* (where there is cessation of inspiratory muscle activity) may also present with somnolence but do not snore excessively (Table 5.11).

TABLE 5.11 Abnormal patterns of breathing

Type of breathing	Cause(s)

Type of breathing	Cause(s)
1 Sleep apnoea—cessation of airflow for more than 10 seconds more than 10 times a night during sleep	Obstructive (e.g. obesity with upper airway narrowing, enlarged tonsils, pharyngeal soft tissue changes in acromegaly or hypothyroidism)
2 Cheyne-Stokes* breathing—periods of apnoea (associated with reduced level of consciousness) alternate with periods of hyperpnoea (lasts 30 s on average and is associated with agitation). This is due to a delay in the medullary chemoreceptor response to blood gas changes	Left ventricular failure Brain damage (e.g. trauma, cerebral haemorrhage) High altitude
3 Kussmaul's breathing (air hunger)—deep, rapid respiration due to stimulation of the respiratory centre	Metabolic acidosis (e.g. diabetes mellitus, chronic renal failure)
4 Hyperventilation, which results in alkalosis and tetany	Anxiety
5 Ataxic (Biot†) breathing—irregular in timing and depth	Brainstem damage
6 Apneustic breathing—a post-inspiratory pause in breathing	Brain (pontine) damage
7 Paradoxical respiration—the abdomen sucks inwards with inspiration (it normally pouches outwards due to diaphragmatic descent)	Diaphragmatic paralysis

* John Cheyne (1777–1836), Scottish physician who worked in Dublin, described this in 1818. William Stokes (1804–1878), Irish physician, described it in 1854.

† Camille Biot (b. 1878), French physician

Camille Blot (b. 1878), French physician.

Some patients respond to anxiety by increasing the rate and depth of their breathing. This is called *hyperventilation*. The result is an increase in CO_2 excretion and the development of alkalosis—a rise in the pH of the blood. These patients may complain of variable dyspnoea; they have more difficulty breathing in than out. The alkalosis results in paraesthesiae of the fingers and around the mouth, light-headedness, chest pain and a feeling of impending collapse.

Treatment

It is important to find out what drugs the patient is using (Table 5.12), how often they are taken and whether they are inhaled or swallowed. The patient's previous and current medications may give a clue to the current diagnosis. Bronchodilators and inhaled steroids are prescribed for COPD and asthma. A patient's increased use of bronchodilators suggests poor control of asthma and the need for review of treatment. Chronic respiratory disease, including sarcoidosis, hypersensitivity pneumonias and asthma, may have been treated with oral steroids. Oral steroid use may predispose to tuberculosis or pneumocystis pneumonia. Patients with chronic lung conditions like cystic fibrosis or bronchiectasis will often be very knowledgeable about their treatment and can describe the various forms of physiotherapy that are essential for keeping their airways clear.

TABLE 5.12 Drugs and the lungs

Cough ACE inhibitors Beta-blockers
Wheeze Beta-blockers Aspirin (non-steroidal anti-inflammatory drugs)

Aspirin (aspirin sensitivity) Other non-steroidal anti-inflammatory drugs (NSAIDs) Tamoxifen, dipyridamole (idiosyncratic) Morphine sulfate Succinylcholine
Interstitial lung disease (pulmonary fibrosis) Amiodarone Hydralazine Gold salts Bleomycin Nitrofurantoin Methotrexate
Pulmonary embolism Oestrogens Tamoxifen Raloxifene
Non-cardiogenic pulmonary oedema Hydrochlorothiazide
Pleural disease/effusion Nitrofurantoin Phenytoin, hydralazine (induction of systemic lupus erythematosus)

erythematosus)
Methotrexate
Methysergide

Almost every class of drug can produce lung toxicity. Examples include pulmonary embolism from use of the oral contraceptive pill, interstitial lung disease from cytotoxic agents (e.g. methotrexate, cyclophosphamide, bleomycin), bronchospasm from beta-blockers or non-steroidal anti-inflammatory drugs (NSAIDs), and cough from ACE inhibitors. Some medications known to cause lung disease may not be mentioned by the patient because they are illegal (e.g. cocaine), are used sporadically (e.g. hydrochlorothiazide), can be obtained over the counter (e.g. tryptophan) or are not taken orally (e.g. timolol; beta-blocker eye drops for glaucoma). The clinician therefore needs to ask about these types of drug specifically.

Past history

One should always ask about previous respiratory illness, including pneumonia, tuberculosis or chronic bronchitis, or abnormalities of the chest X-ray that have previously been reported to the patient. Many previous respiratory investigations may have been memorable, such as bronchoscopy, lung biopsy and video-assisted thoracoscopy. Spirometry, with or without challenge testing for asthma, may have been performed. Many severe asthmatics perform their own regular peak flow testing (page 128). Ask about the results of any of these investigations. Patients with the acquired immunodeficiency syndrome (AIDS) have a high risk of developing *Pneumocystis jiroveci (carinii)* pneumonia and indeed other chest infections, including tuberculosis.

Occupational history

In no system are the patient's present and previous occupations of more importance (Table 5.13).[2] A detailed occupational history is essential. The occupational lung diseases or pneumoconioses cause interstitial lung disease by damaging the alveoli and small airways. Prolonged exposure to substances whose use is now heavily restricted is usually required. Cigarette smoking has an additive effect for these patients. These occupational conditions are now rare, and the most common occupational lung disease is asthma.

TABLE 5.13 Occupational lung disease (pneumoconioses)

Substance	Disease
Coal	Coal worker's pneumoconiosis
Silica	Silicosis
Asbestos	Asbestosis
Talc	Talcosis

One must ask about exposure to dusts in mining industries and factories (e.g. asbestos, coal, silica, iron oxide, tin oxide, cotton, beryllium, titanium oxide, silver, nitrogen dioxide, anhydrides). Heavy exposure to asbestos can lead to asbestosis (Table 5.14), but even trivial exposure can result in pleural plaques or mesothelioma (malignant disease of the pleura). The patient may be unaware that his or her occupation involved exposure to dangerous substances; for example, factories making insulating cables and boards very often used asbestos until 25 years ago. Asbestos exposure can result in the development of asbestosis, mesothelioma or carcinoma of the lung up to 30 years later. Relatives of people working with asbestos may be exposed when handling work clothes.

TABLE 5.14 Possible occupational exposure to asbestos

Asbestos mining, including relatives of miners
Naval dockyard workers and sailors—lagging of pipes
Builders—asbestos in fibreboard (particles are released during cutting or drilling)
Factory workers—manufacture of fibro-sheets, brake linings, some textiles
Building maintenance workers—asbestos insulation
Building demolition workers
Home renovation

Work or household exposure to animals, including birds, is also relevant (e.g. Q fever or psittacosis which are infectious diseases caught from animals).

Exposure to organic dusts can cause a local immune response to organic antigens and result in *allergic alveolitis*. Within a few hours of exposure, patients develop flu-like symptoms. These often include fever, headache, muscle pains, dyspnoea without wheeze and dry cough. The culprit antigens may come from mouldy hay, humidifiers or air conditioners, among others (Table 5.15).

TABLE 5.15 Allergic alveolitis—sources

Bird fancier's lung	Bird feathers and excreta
Farmer's lung	Mouldy hay or straw (*Aspergillus fumigatus*)
Byssinosis	Cotton or hemp dust
Cheese worker's	

Cheese worker's lung	Mouldy cheese (*Aspergillus clavatus*)
Malt worker's lung	Mouldy malt (*Aspergillus clavatus*)
Humidifier fever	Air-conditioning (thermophilic *Actinomycetes*)

It is most important to find out what the patient actually does when at work, the duration of any exposure, use of protective devices and whether other workers have become ill. An improvement in symptoms over the weekend is a valuable clue to the presence of occupational lung disease, particularly occupational asthma. This can occur as a result of exposure to spray paints or plastic or soldering fumes.

Social history

A smoking history must be routine, as it is the major cause of COPD and lung cancer (see Table 1.2, page 6). It also increases the risk of spontaneous pneumothorax and of Goodpasture's syndrome. It is necessary to ask how many packets of cigarettes a day a patient has smoked and how many years the patient has smoked. An estimate should be made of the number of packet-years of smoking. Remember that this is based on 20-cigarette packets and that packets of cigarettes are getting larger; curiously, most manufacturers now make packets of 30 or 35. More recently, giant packets of 50 have appeared. These are too large to fit into pockets and must be carried in the hands as a constant reminder to the patient of his or her addiction. Occupation may further affect cigarette smokers; for example, asbestos workers who smoke are at an especially high risk of lung cancer. Passive smoking is now regarded as a significant risk for lung disease and the patient should be asked about exposure to other people's cigarette smoke at home and at work.

Many respiratory conditions are chronic, and may interfere with the ability to work and exercise and interfere with normal family life. In some cases involving occupational lung disease there may be compensation matters affecting the patient. Ask about these problems and whether the patient has been involved in a pulmonary rehabilitation programme. Housing conditions may be inappropriate for a person with a limited exercise tolerance or an infectious disease. An inquiry about the patient's alcohol consumption is important. The drinking of large amounts of alcohol in binges can sometimes result in aspiration pneumonia, and alcoholics are more likely to develop pneumococcal or *Klebsiella* pneumonia. Intravenous drug users are at risk of lung abscess and drug-related pulmonary oedema. Sexual orientation or

lung abscess and drug-related pulmonary oedema. Sexual orientation of history of intravenous drug use may be related to an increased risk of HIV infection and susceptibility to infection. Such information may influence the decision about whether to advise treatment at home or in hospital.

Family history

A family history of asthma or other atopic diseases, cystic fibrosis, lung cancer or emphysema should be sought. Alpha$_1$-antitrypsin deficiency, for example, is an inherited disease, and those affected are extremely susceptible to the development of emphysema. A family history of infection with tuberculosis is also important. A number of pulmonary diseases may have a familial or genetic association. These include carcinoma of the lung and pulmonary hypertension.

The respiratory examination

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 which 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 with its apex pointing 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 (Figure 5.1).

and oblique (lower) fissures dividing it into three lobes (Figure 5.1).

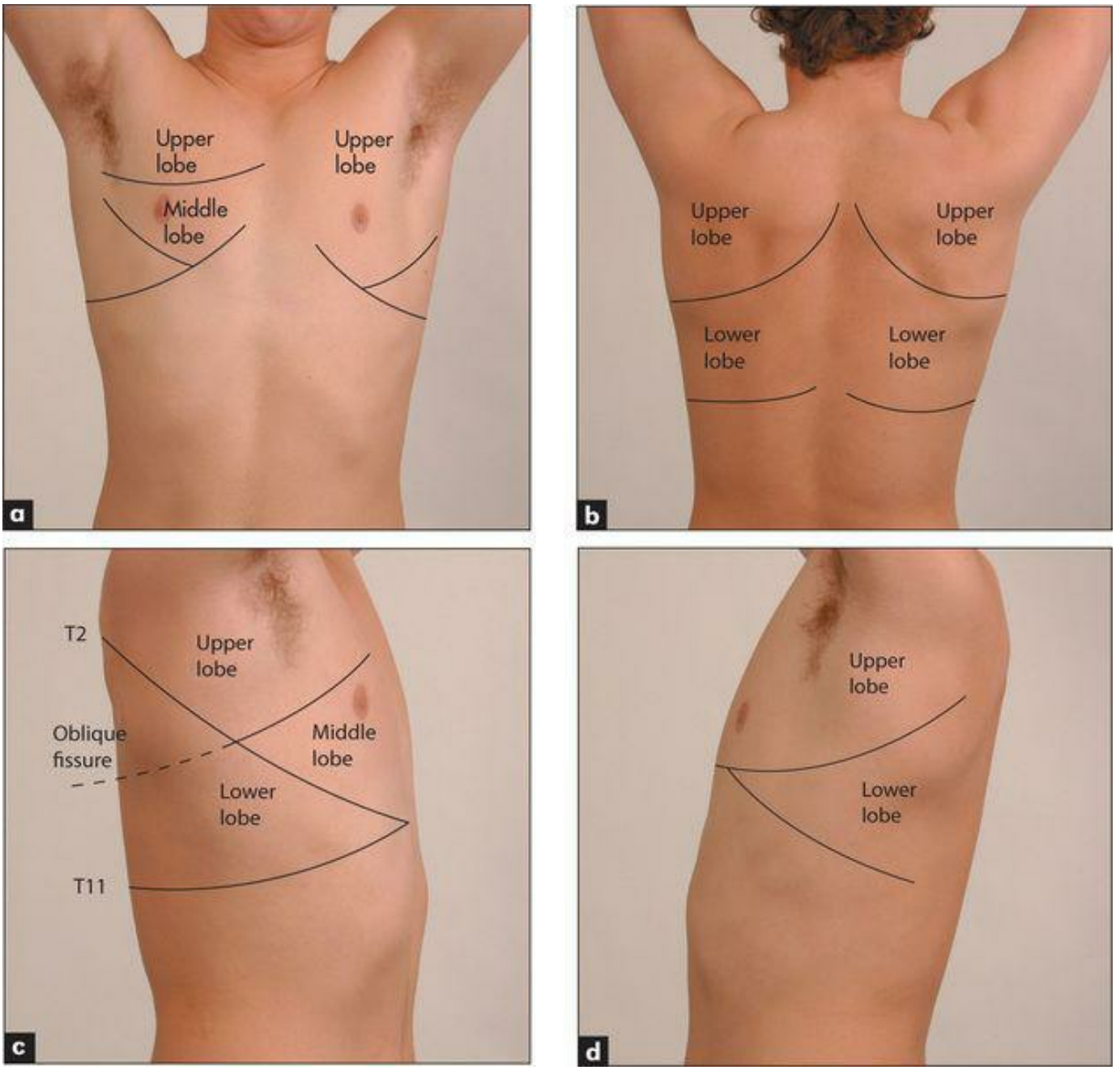


Figure 5.1 Lobes of the lung
(a) Anterior. (b) Posterior. (c) Lobes of the right lung. (d) Lobes of the left lung. Refer to Figure 5.15, page 137, for a list of the segments in each lobe.

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 (COPD, page 133). 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** (Figure 5.1) of the lungs and try to decide which lobes are affected.

Positioning the patient

The patient should be undressed to the waist.[3] 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.

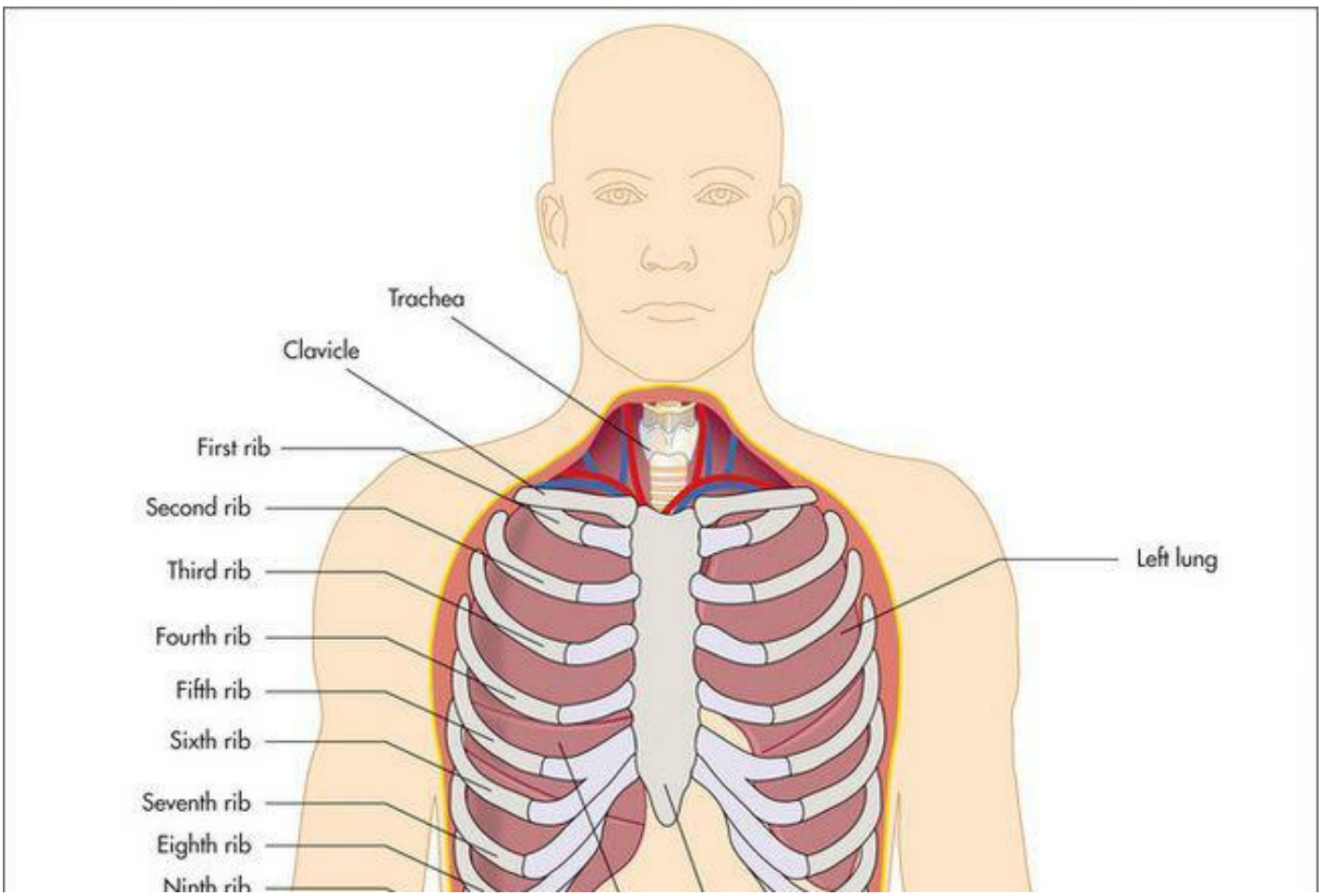
Dyspnoea

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 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 chronic obstructive pulmonary disease (COPD)[a]

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 is an important cause. These muscles include the sternomastoids, 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 may occur in expiration in patients with obstructed airways. Patients with severe COPD often have indrawing 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 (Table 5.11). 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 forward 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.



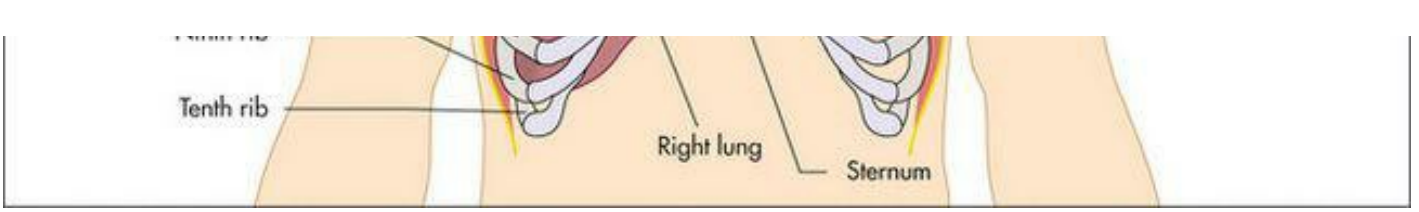


Figure 5.2 Basic anatomy of the lungs

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 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. 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 (Table 5.16). It is a sign that requires urgent attention.

TABLE 5.16 Some 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

Hoarseness

Listen to the 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 commonest cause is laryngitis and the use of inhaled corticosteroids for asthma. Non-respiratory causes include hypothyroidism.

The 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 (Figure 5.3, Table 4.9 on page 51). 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, 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. Remember, chronic bronchitis and emphysema do not cause clubbing. It is important to note that clubbing does not occur as a result of COPD.

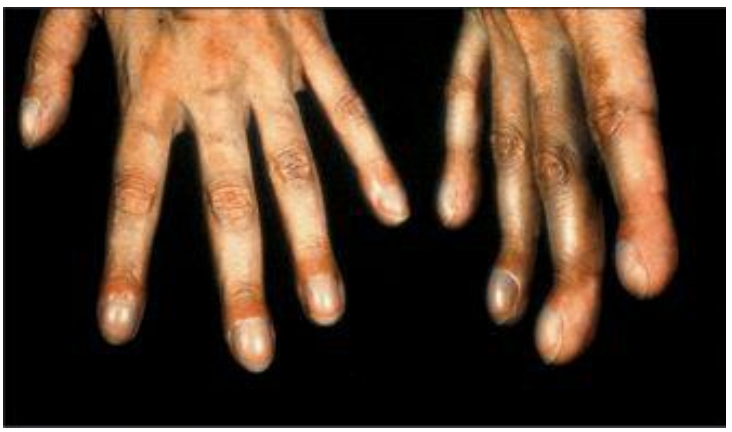


Figure 5.3 Finger clubbing

Staining

Look for staining of the fingers (actually caused by tar, as nicotine is colourless); a sign of cigarette smoking. The density of staining does not indicate the number of cigarettes smoked, but depends rather on the way the cigarette is held in the hand.

Wasting and weakness

Compression and infiltration by a peripheral lung tumour of a lower trunk of the T1 nerve root results in wasting of the small muscles of the hand and weakness of finger abduction.

Pulse rate

Tachycardia and pulsus paradoxus are important signs of severe asthma. Tachycardia is a common side-effect of the treatment of asthma with β-agonist drugs, and accompanies dyspnoea or hypoxia of any cause.

Flapping tremor (asterixis)

Ask the patient to dorsiflex the wrists with the arms outstretched and to spread out the fingers. A flapping tremor with a 2- to 3-second cycle may occur with severe carbon dioxide retention, usually due to severe COPD.[4] The problem is an inability to maintain a posture. Asterixis[b] can also be demonstrated by asking the patient to protrude the tongue or lift the leg and keep the foot dorsiflexed. However, this is a late and unreliable sign and can also occur in patients with liver or renal failure. Patients with severe carbon dioxide retention may be confused, and typically have warm peripheries and a bounding pulse.

The face

The *nose* is sited conveniently in the centre of the face. In this position it may readily be inspected inside and out. Get the patient to tilt the head back. It may be necessary to use a nasal speculum to open the nostrils, and a torch. Look for polyps (associated with asthma), engorged turbinates (various allergic conditions) and a deviated septum (nasal obstruction).

As already discussed, look at the *tongue* for central cyanosis. Look in the *mouth* for evidence of an upper respiratory tract infection (a reddened pharynx and tonsillar enlargement, with or without a coating of pus). A

broken tooth or a rotten tooth stump may predispose to lung abscess or pneumonia. Patients with sleep apnoea may have 'crowding' of the pharynx. This means a reduction in the size of the *velopharyngeal lumen*, which is the space between the soft palate, tonsils and the back of the tongue. Those who use a sleep apnoea mask at night often have marks from the mask on the face and puffiness around the eyes. They tend to be obese and have a short thick neck and a small pharynx; sometimes the maxilla and mandible appear retracted (receding chin).

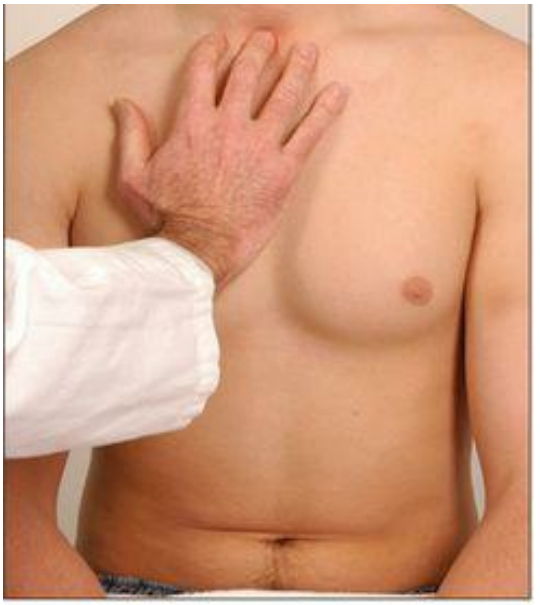
Sinusitis is suggested by tenderness over the *sinuses* on palpation. If acute sinusitis is suspected, a torch can be used to transilluminate the frontal and maxillary sinuses.[5] A torch is placed in the patient's mouth and the sinuses examined in a dark room. Normal transillumination generally excludes sinusitis. Complete opacification suggests sinusitis but partial opacification is less helpful. The torch should then be used to look for purulent discharge in the pharynx.

Look at the patient's *face* for the red, leathery, wrinkled skin of the smoker.[6] There may be facial plethora or cyanosis if superior vena caval obstruction is present. Look for the characteristics of obstructive sleep apnoea (see above).

Inspect the *eyes* for evidence of the rare Horner's[c] syndrome (a constricted pupil, partial ptosis and loss of sweating), which can be due to an apical lung carcinoma (Pancoast[d] tumour) compressing the sympathetic nerves in the neck. There may be skin changes on the face that suggest scleroderma or connective tissue disease.

The trachea

The position of the trachea is most important, and time should be spent establishing it accurately. From in front of the patient the forefinger of the right hand is pushed up and backwards from the suprasternal notch until the trachea is felt (Figure 5.4). If the trachea is displaced to one side, its edge rather than its middle will be felt and a larger space will be present on one side than the other. Slight displacement to the right is fairly common in normal people. This examination is uncomfortable for the patient, so you must be gentle.



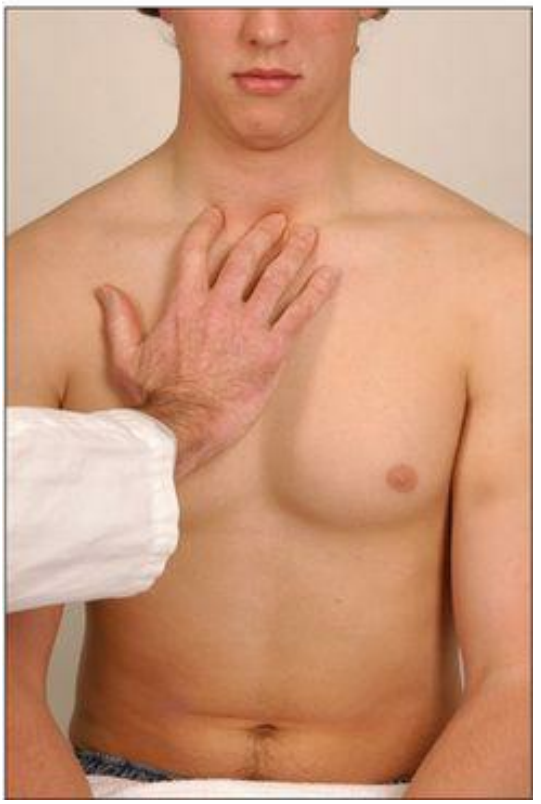


Figure 5.4 Feeling for the position of the trachea—a similar gap should be palpable on each side

Significant displacement of the trachea suggests, but is not specific for, disease of the upper lobes of the lung (Table 5.17).

TABLE 5.17 Causes of tracheal displacement

1 Towards the side of the lung lesion Upper lobe collapse Upper lobe fibrosis Pneumonectomy
2 Away from the side of the lung lesion (uncommon) Massive pleural effusion Tension pneumothorax
3 Upper mediastinal masses, such as retrosternal goitre

A tracheal tug is demonstrated when the finger resting on the trachea feels it move inferiorly with each inspiration. This is a sign of gross overexpansion of the chest because of airflow obstruction. This movement of the trachea may be visible, and it is worth spending time inspecting the trachea when COPD is suspected.

If the patient appears dyspnoeic and use of the accessory muscles of respiration is suspected, the examiner's fingers should be placed in the supraclavicular fossae. When the scalene muscles are recruited, they can be felt to contract under the fingers. Even more severe dyspnoea will result in use of the sternomastoid muscles. Their contraction is also easily felt with inspiration. Use of these muscles for long periods is exhausting and a sign of impending respiratory failure.

The chest

The chest should be examined anteriorly and posteriorly by inspection, palpation, percussion and auscultation.[3] Compare the right and left sides during each part of the examination.

Inspection

Shape and symmetry of chest

When the anteroposterior (AP) diameter is increased compared with the lateral diameter, the chest is described as barrel-shaped (Figure 5.5). An increase in the AP diameter compared with the lateral diameter (the thoracic ratio) beyond 0.9 is abnormal and is seen often in patients with severe asthma or emphysema. It is not always a reliable guide to the severity of the underlying lung disease and may be present in normal elderly people. Severe kyphoscoliosis is a cause of asymmetrical chest deformity.

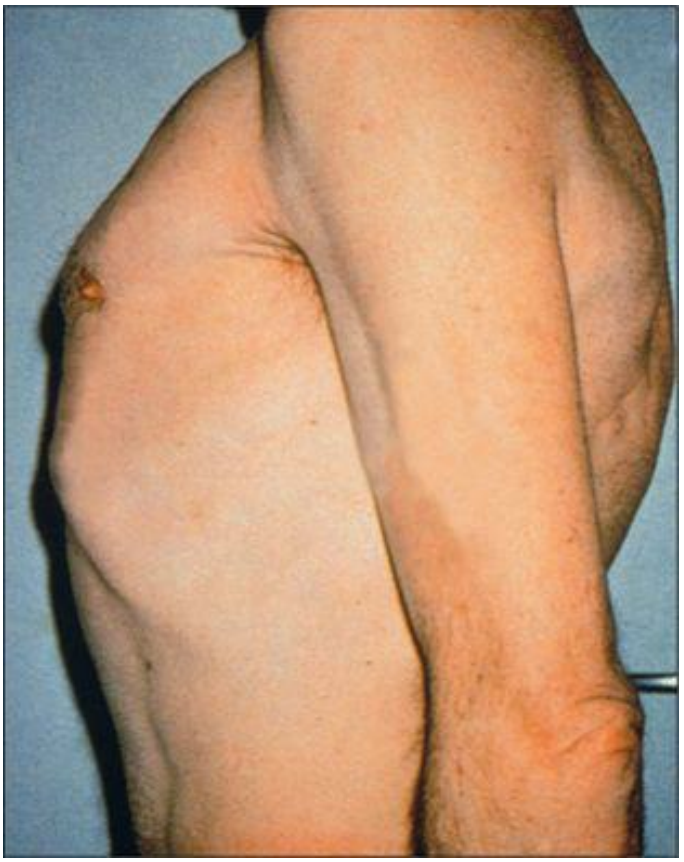


Figure 5.5 Barrel chest

From McDonald FS, ed., Mayo Clinic images in internal medicine, with permission. ©Mayo Clinic Scientific Press and CRC Press.

A **pigeon chest (pectus carinatum)** is a localised prominence (an outward bowing of the sternum and costal cartilages)(Figure 5.6). It may be a manifestation of chronic childhood respiratory illness, in which case it is thought to result from repeated strong contractions of the diaphragm while the thorax is still pliable. It also occurs in rickets.

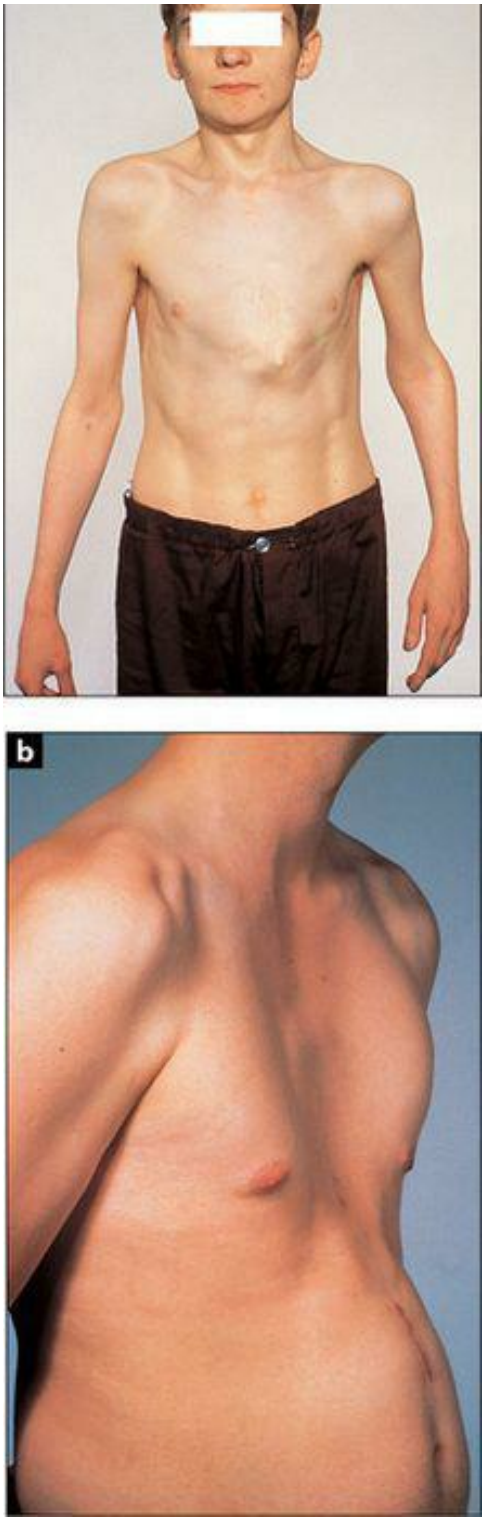


Figure 5.6 (a) Funnel chest (pectus excavatum); (b) Pigeon chest (pectus carinatum)

From Mir MA, Atlas of Clinical Diagnosis, 2nd edn. Edinburgh: Saunders, 2003, with permission.

A **funnel chest (pectus excavatum)** is a developmental defect involving a localised depression of the lower end of the sternum (Figure 5.6). The problem is usually an aesthetic one, but in severe cases lung capacity may be restricted.

Harrison's sulcus[e] is a linear depression of the lower ribs just above the

costal margins at the site of attachment of the diaphragm. It can result from severe asthma in childhood, or rickets.

Kyphosis refers to an exaggerated forward curvature of the spine, while scoliosis is lateral bowing. **Kyphoscoliosis** may be idiopathic (80%), secondary to poliomyelitis, or associated with Marfan's syndrome. Severe thoracic kyphoscoliosis may reduce the lung capacity and increase the work of breathing.

Lesions of the chest wall may be obvious. Look for *scars* from previous thoracic operations, or from chest drains for a previous pneumothorax or pleural effusion. Surgical removal of a lung (pneumonectomy) or of the lobe of a lung (lobectomy) leaves a long diagonal posterior scar on the thorax. The presence of three 2–3 cm scars suggests previous video-assisted thoracoscopic surgery, which can be performed to biopsy lymph nodes or carry out lung reduction surgery or pleurodesis. Thoracoplasty causes severe chest deformity; this operation was performed for tuberculosis and involved removal of a large number of ribs on one side of the chest to achieve permanent collapse of the affected lung. It is no longer performed because of the availability of effective antituberculous chemotherapy.[f] *Radiotherapy* may cause erythema and thickening of the skin over the irradiated area. There is sharp demarcation between abnormal and normal skin. There may be small tattoo marks indicating the limits of the irradiated area. Signs of radiotherapy usually indicate that the patient has been treated for carcinoma of the lung, breast or, less often, for lymphoma.

Subcutaneous emphysema is a crackling sensation felt on palpating the skin of the chest or neck. On inspection, there is often diffuse swelling of the chest wall and neck. It is caused by air tracking from the lungs and is usually due to a pneumothorax; less commonly it can follow rupture of the oesophagus or a pneumomediastinum (air in the mediastinal space).

Prominent veins may be seen in patients with superior vena caval obstruction. It is important to determine the direction of blood flow (page 166).

Movement of the chest wall should be noted. Look for asymmetry of chest wall movement anteriorly and posteriorly. Assessment of expansion of the upper lobes is best achieved by inspection from behind the patient, looking down at the clavicles during moderate respiration (Figure 5.7). Diminished movement indicates underlying lung disease. The affected side will show delayed or decreased movement. For assessment of lower lobe expansion, the chest should be inspected posteriorly.

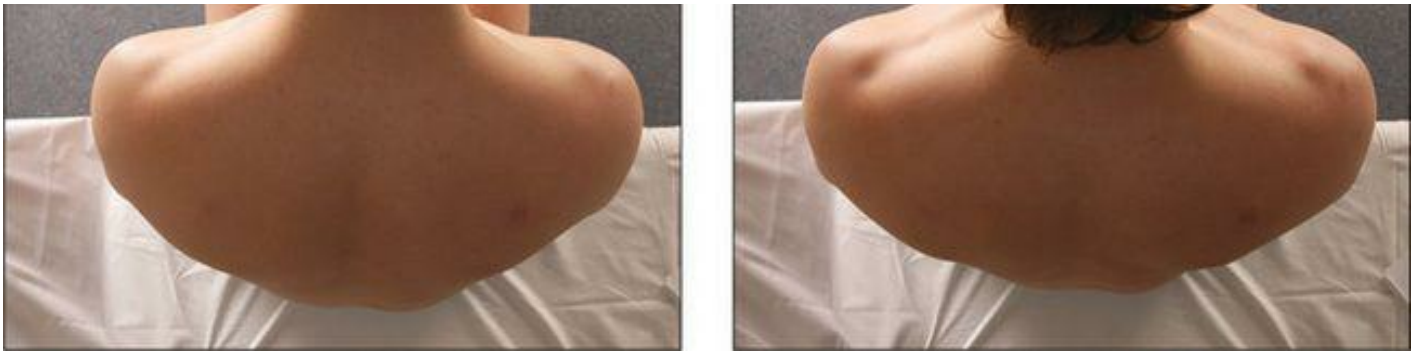


Figure 5.7 Inspecting upper lobe expansion: (a) expiration; (b) inspiration—note symmetrical elevation of the clavicles

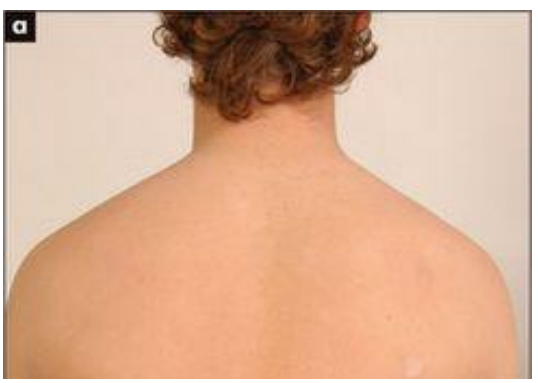
Reduced chest wall movement on one side may be due to localised lung fibrosis, consolidation, collapse, pleural effusion or pneumothorax. Bilateral reduction of chest wall movement indicates a diffuse abnormality such as COPD or diffuse interstitial lung disease. Unilateral reduced chest excursion o r *splinting* may be present when patients have pleuritic chest pain or injuries such as rib fractures.

Look for paradoxical inward motion of the abdomen during inspiration when the patient is supine (indicating diaphragmatic paralysis).

Palpation

Chest expansion

Place the hands firmly on the chest wall with the fingers extending around the sides of the chest. The thumbs should almost meet in the middle line and should be lifted slightly off the chest so that they are free to move with respiration (Figure 5.8). As the patient takes a big breath in, the thumbs should move symmetrically apart at least 5 cm. Reduced expansion on one side indicates a lesion on that side. The causes have been discussed above.



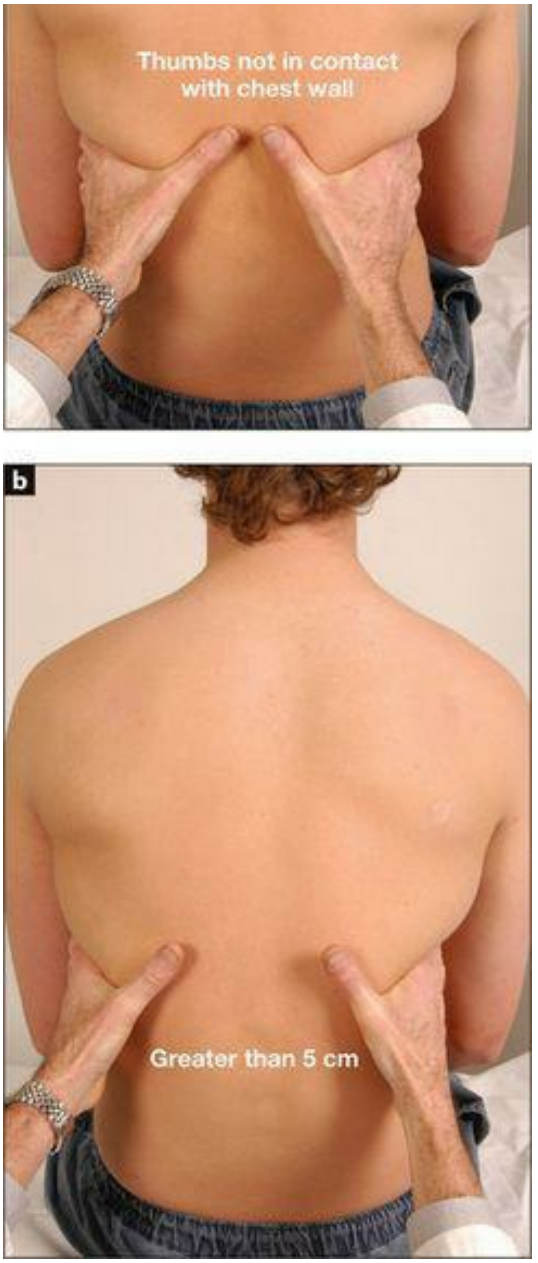
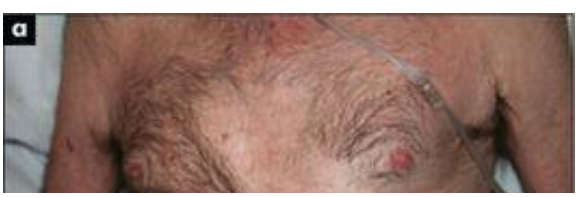


Figure 5.8 Palpation for lower lobe expansion: (a) expiration; (b) inspiration

If COPD is suspected, Hoover's sign[g] may be sought (Figure 5.9). The examiner places the hands along the costal margins with the thumbs close to the xiphisternum. Normally inspiration causes them to separate, but the overinflated chest of the COPD patient cannot expand in this way and the diaphragm pulls the ribs and the examiner's thumbs closer together[7] (positive LR 4.2).[8]



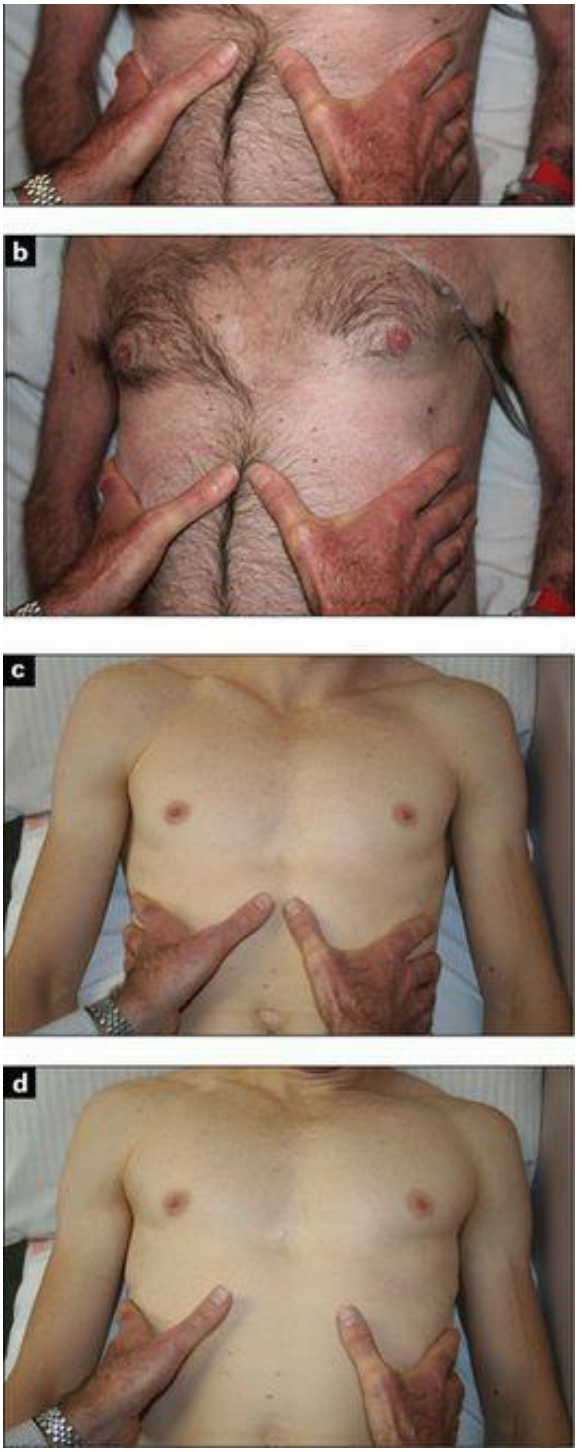


Figure 5.9 Hoover's sign
(a) Expiration; (b) inspiration. (c) Normal expiration; (d) normal inspiration.

Lower lobe expansion is assessed from the back in this way. Some idea of upper and middle lobe expansion is possible when the manoeuvre is repeated on the front of the chest, but this is better gauged by inspection.

Apex beat

When the patient is lying down, establishing the position of the apex beat may be helpful (page 60), as displacement towards the side of the lesion can be caused by collapse of the lower lobe or by localised interstitial lung disease (ILD). Movement of the apex beat away from the side of the lung lesion can be caused by pleural effusion or tension pneumothorax. The apex beat is often impalpable in a chest which is hyperexpanded secondary to chronic obstructive pulmonary disease.

Vocal (tactile) fremitus

Palpate the chest wall with the palm of the hand while the patient repeats 'ninety-nine'. The front and back of the chest are each palpated in two comparable positions, with the palm of one hand on each side of the chest. In this way differences in vibration on the chest wall can be detected. This can be a difficult sign to interpret, with considerable inter-observer variability, and it is no longer a routine part of the examination. It depends on the recognition of changes in vibration conducted to the examiner's hands while the patient speaks. Practice is needed to appreciate the difference between normal and abnormal. Vocal fremitus is more obvious in men because of their lower-pitched voices. It may be absent in normal people (high-pitched voice or thick chest wall). It is only abnormal if different on one side from the other. The causes of change in vocal fremitus are the same as those for vocal resonance (page 127).

Ribs

Gently compress the chest wall anteroposteriorly and laterally. Localised pain suggests a rib fracture, which may be secondary to trauma or may be spontaneous as a result of tumour deposition, bone disease or sometimes the result of severe and prolonged coughing. Tenderness over the costochondral junctions suggests the diagnosis of costochondritis as the cause of chest pain.

Regional lymph nodes

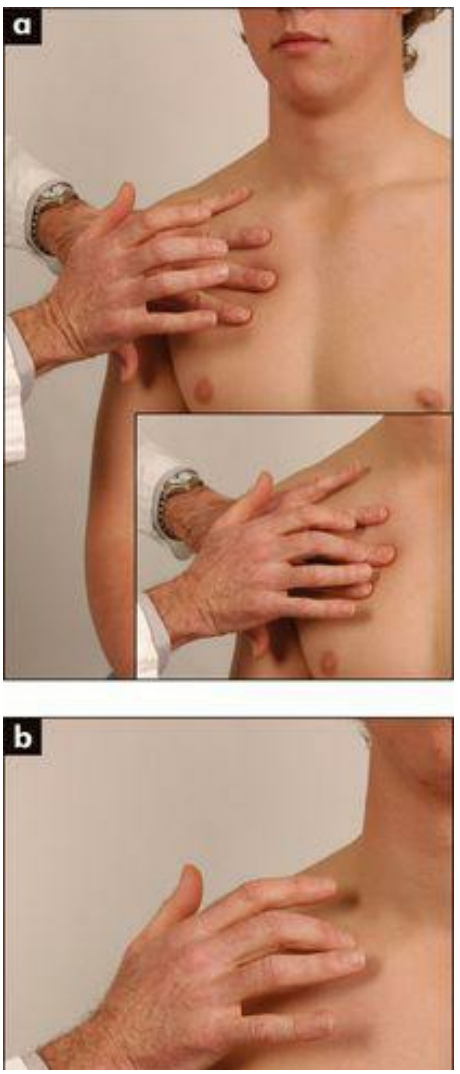
The axillary and cervical and supraclavicular nodes must be examined (pages 227, 228); they may be enlarged in lung malignancies and some infections.

Percussion

With the left hand on the chest wall and the fingers slightly separated and

With the left hand on the chest wall and the fingers slightly separated and aligned with the ribs, the middle finger is pressed firmly against the chest. Then the pad of the right middle finger (the *plexor*) is used to strike firmly the middle phalanx of the middle finger of the left hand (the *pleximeter*); this was often a piece of wood, ivory or a coin in the 19th century but is now always the examiner's finger. The percussing finger (plexor) is quickly removed so that the note generated is not dampened (this may be less important if the pleximeter finger is held firmly on the chest wall, as it should be). The percussing finger must be held partly flexed and a loose swinging movement should come from the wrist and not from the forearm. Medical students will soon learn to keep the right middle fingernail short.

Percussion of symmetrical areas of the anterior, posterior and axillary regions is necessary (Figure 5.10). Percussion in the supraclavicular fossa over the apex of the lung and direct percussion of the clavicle with the percussing finger are a traditional part of the examination. For percussion posteriorly, the scapulae should be moved out of the way by asking the patient to move the elbows forward across the front of the chest; this rotates the scapulae anteriorly.



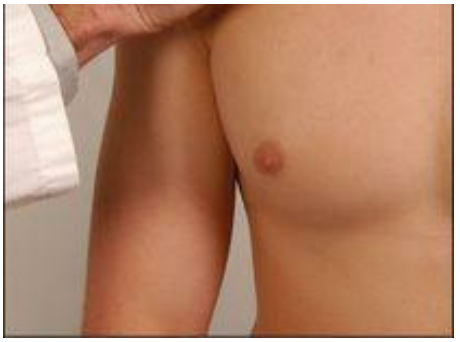


Figure 5.10 Percussion of the chest
(a) Percussing (plexor) finger poised. Inset: plexor finger strikes pleximeter finger. (b) Direct percussion of the clavicle for upper lobe resonance.

The feel of the percussion note is as important as its sound. The note is affected by the thickness of the chest wall, as well as by underlying structures. Percussion over a solid structure, such as the liver or a consolidated or collapsed area of lung, produces a dull note. Percussion over a fluid-filled area, such as a pleural effusion, produces an extremely dull (stony dull) note. Percussion over the normal lung produces a resonant note and percussion over hollow structures, such as the bowel or a pneumothorax, produces a hyperresonant note (*Good signs guide 5.1*).

GOOD SIGNS GUIDE 5.1 Comparative percussion of the chest

Sign	Positive LR	Negative LR
Dullness—pneumonia	3.0	NS
Hyperresonance—COPD	5.1	NS

NS = not significant.