COPD = chronic obstructive pulmonary disease.

From McGee S, Evidence-based physical diagnosis, 2nd edn. St Louis: Saunders, 2007.

Considerable practice is required before expert percussion can be performed, particularly in front of an audience. The ability to percuss well is usually obvious in clinical examinations and counts in a student's favour, as it indicates a reasonable amount of experience in the wards.

Liver dullness

The upper level of liver dullness is determined by percussing down the anterior chest in the midclavicular line. Normally, the upper level of the liver dullness is the fifth rib in the right midclavicular line. If the chest is resonant below this level, it is a sign of hyperinflation, usually due to emphysema or asthma. This is a sign with considerable inter-observer variability.

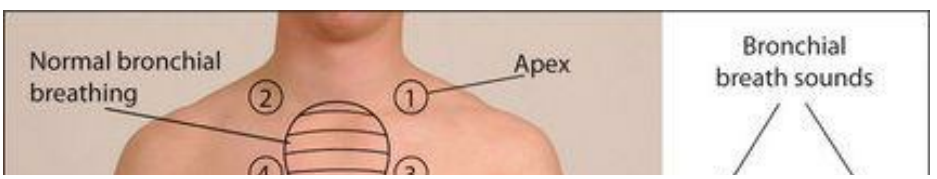
Cardiac dullness

The area of cardiac dullness usually present on the left side of the chest may be decreased in emphysema or asthma.

Auscultation

Breath sounds

Using the diaphragm of the stethoscope, one should listen to the breath sounds in the areas shown in Figure 5.11.[9-11] It is important to compare each side with the other. Remember to listen high up into the axillae and, using the bell of the stethoscope applied above the clavicles, to listen to the lung apices. A number of observations must be made while auscultating and, as with auscultation of the heart, different parts of the cycle must be considered. Listen for the quality of the breath sounds, the intensity of the breath sounds, and the presence of additional (adventitious) sounds.



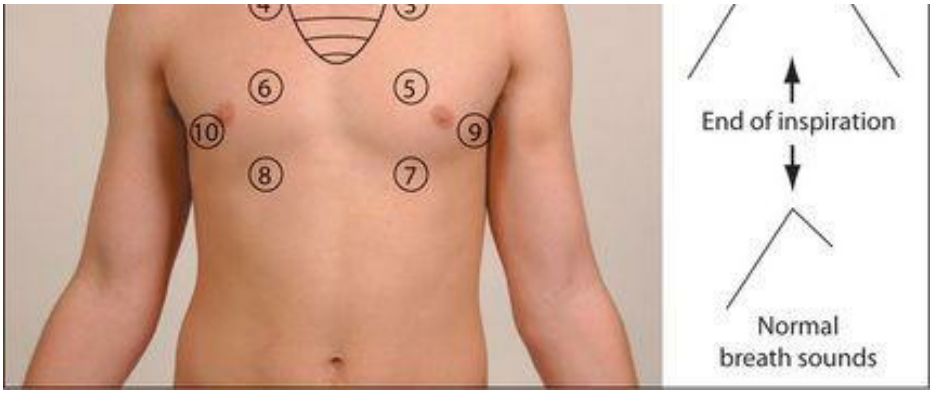


Figure 5.11 Normal and bronchial breath sounds
Auscultate in each area shown (the numbers represent a suggested order). Distinguish normal breath sounds from bronchial breathing.

Quality of breath sounds

Normal breath sounds are heard with the stethoscope over nearly all parts of the chest. The patient should be asked to breathe through the mouth so that added sounds from the nasopharynx do not interfere. These sounds are produced in the airways rather than the alveoli. They had once been thought to arise in the alveoli (vesicles) of the lungs and are therefore called vesicular sounds. They have rather fancifully been compared by Laënnec to the sound of wind rustling in leaves. Their intensity is related to total airflow at the mouth and to regional airflow. Normal (vesicular) breath sounds are louder and longer on inspiration than on expiration and there is no gap between the inspiratory and expiratory sounds. They are due to the transmission of air turbulence in the large airways filtered through the normal lung to the chest wall.

Bronchial breath sounds are present when turbulence in the large airways is heard without being filtered by the alveoli, producing a different quality. Bronchial breath sounds have a hollow, blowing quality. They are audible throughout expiration and there is often a gap between inspiration and expiration. The expiratory sound has a higher intensity and pitch than the inspiratory sound. Bronchial breath sounds are more easily remembered than described. They are audible in normal people, posteriorly over the right upper chest where the trachea is contiguous with the right upper bronchus. They are heard over areas of consolidation, as solid lung conducts the sound of turbulence in main airways to peripheral areas without filtering. Causes of bronchial breath sounds are shown in Table 5.18.

TABLE 5.18 Causes of bronchial breath sounds

Common
Lung consolidation (lobar pneumonia)
Uncommon
Localised pulmonary fibrosis
Pleural effusion (above the fluid)
Collapsed lung (e.g. adjacent to a pleural effusion)

Note: The large airways must be patent.

Occasionally breath sounds over a large cavity have an exaggerated bronchial quality. This very hollow or *amphoric* sound has been likened to that heard when air passes over the top of a hollow jar (Greek *amphoreus*).

Intensity of the breath sounds

It is better to describe breath sounds as being of normal or reduced intensity than to speak about air entry. The entry of air into parts of the lung cannot be directly gauged from the breath sounds. Asymmetrical reduction of breath sounds is a sign of bronchial obstruction, for example by carcinoma or a foreign body on the side where breath sounds are reduced.

Causes of reduced breath sounds include COPD (especially emphysema), pleural effusion, pneumothorax, pneumonia, a large neoplasm and pulmonary collapse.

Added (adventitious) sounds

There are two types of added sounds—continuous (wheezes) and interrupted (crackles).

Continuous sounds are called *wheezes*. They are abnormal findings and have a musical quality. The wheezes must be timed in relation to the respiratory cycle. They may be heard in expiration or inspiration, or both. Wheezes are due to continuous oscillation of opposing airway walls and

imply significant airway narrowing. Wheezes tend to be louder on expiration. This is because the airways normally dilate during inspiration and are narrower during expiration. An inspiratory wheeze implies severe airway narrowing.

The pitch (frequency) of wheezes varies. It is determined only by the velocity of the air jet and is not related to the length of the airway. High-pitched wheezes are produced in the smaller bronchi and have a whistling quality, whereas low-pitched wheezes (sometimes called rhonchi) arise from the larger bronchi.

Wheezes are usually the result of acute or chronic airflow obstruction due to asthma (often high-pitched) or COPD (often low-pitched), secondary to a combination of bronchial muscle spasm, mucosal oedema and excessive secretions. Wheezes are a poor guide to the severity of airflow obstruction. In severe airways obstruction, wheeze can be absent because ventilation is so reduced that the velocity of the air jet is reduced below a critical level necessary to produce the sound.

A fixed bronchial obstruction, usually due to a carcinoma of the lung, tends to cause a localised wheeze, which has a single musical note (monophonic) and does not clear with coughing.

Wheezes must be distinguished from *stridor* (page 118), which sounds very similar to wheeze but is louder over the trachea and is always inspiratory (wheezes usually occur in expiration—the majority—but can occur in both inspiration and expiration).

Interrupted non-musical sounds are best called *crackles*.[12,13] There is a lot of confusion about the naming of these sounds, perhaps as a result of mistranslations of Laënnec. Some authors describe low-pitched crackles as rales and high-pitched ones as crepitations, but others do not make this distinction. The simplest approach is to call all these sounds crackles, but also to describe their timing and pitch. Crackles are sometimes present in normal people but these crackles will always clear with coughing.

Crackles are probably the result of loss of stability of peripheral airways that collapse on expiration. With high inspiratory pressures, there is rapid air entry into the distal airways. This causes the abrupt opening of alveoli and of small- or medium-sized bronchi containing secretions in regions of the lung deflated to residual volume. More compliant (distensible) areas open up first, followed by the increasingly stiff areas. Fine- and medium-pitched crackles are not caused by air moving through secretions as was once thought, but by the opening and closing of small airways.

The timing of crackles is of great importance. *Early inspiratory crackles* (cease before the middle of inspiration) suggest disease of the small airways, and are characteristic of COPD.[12] The crackles are heard only in early inspiration and are of medium coarseness. They are different from

those heard in left ventricular failure, which occur later in the respiratory cycle.

Late or *pan-inspiratory crackles* suggest disease confined to the alveoli. They may be fine, medium or coarse in quality. *Fine crackles* have been likened to the sound of hair rubbed between the fingers, or to the sound Velcro makes when pulled apart—they are typically caused by interstitial lung disease (pulmonary fibrosis). Characteristically, more crackles are heard in each inspiration when they are due to fibrosis—up to 14 compared with 1 to 4 for COPD and 4 to 9 for cardiac failure. As ILD becomes more severe the crackles extend earlier into inspiration and are heard further up the chest.[h] *Medium crackles* are usually due to left ventricular failure. Here the presence of alveolar fluid disrupts the function of the normally secreted surfactant. *Coarse crackles* are characteristic of pools of retained secretions and have an unpleasant gurgling quality. They tend to change with coughing, which also has an unpleasant gurgling quality. Bronchiectasis is a common cause, but any disease that leads to retention of secretions may produce these features. (See *Good signs guide 5.2*.)

GOOD SIGNS GUIDE 5.2 Crackles and wheezes

Sign	Positive LR	Negative LR
Crackles		
Pulmonary fibrosis in asbestos workers	5.9	0.2
Pneumonia patients with cough and fever	2.0	0.8
Early inspiratory crackles		
Detecting COPD	14.6	NS
Severe COPD	20.8	0.1
Unforced (audible during breathing at rest) **wheezing**		
Detecting COPD	6.0	NS

NS = not significant. COPD = chronic obstructive pulmonary disease.

From McGee S, Evidence-based physical diagnosis, 2nd edn. St Louis: Saunders, 2007.

Pleural friction rub: when thickened, roughened pleural surfaces rub together as the lungs expand and contract, a continuous or intermittent grating sound may be audible. A pleural rub indicates pleurisy, which may be secondary to pulmonary infarction or pneumonia. Rarely, malignant involvement of the pleura, a spontaneous pneumothorax or pleurodynia may cause a rub.

Vocal resonance

Auscultation over the chest while a patient speaks gives further information about the lungs' ability to transmit sounds. Over normal lung, the low-pitched components of speech are heard with a booming quality and high-pitched components are attenuated. Consolidated lung, however, tends to transmit high frequencies so that speech heard through the stethoscope takes on a bleating quality (called *aegophony* by Laënnec[14]—from Greek *aix* 'goat', *phone* 'voice'). When a patient with aegophony says 'e' as in 'bee' it sounds like 'a' as in 'bay'.

Increased vocal resonance is a helpful sign in confirming consolidation but may not be necessary as a routine. Ask the patient to say 'ninety-nine' while you listen over each part of the chest. Over consolidated lung the numbers will become clearly audible, while over normal lung the sound is muffled. If vocal resonance is present, bronchial breathing is likely to be heard (Table 5.18). Sometimes vocal resonance is increased to such an extent that whispered speech is distinctly heard; this is called whispering pectoriloquy.

If a very localised abnormality is found at auscultation, try to determine the lobe and approximately which segment or segments are involved (Figure 5.1, page 116).

The heart

Cardiac examination is an essential part of the respiratory assessment and vice versa. These two systems are intimately related.

Lay the patient down at 45 degrees and measure the jugular venous pressure for evidence of right heart failure (page 58). Next examine the praecordium. It is important to pay close attention to the pulmonary component of the second heart sound (P2). This is best heard at the second intercostal space on the left. It should not be louder than the aortic component, best heard at the right second inter-costal space. If the P2 is louder (and especially if it is palpable), pulmonary hypertension should be strongly suspected. There may be signs of right ventricular failure or hypertension. Pulmonary hypertensive heart disease (cor pulmonale) may be due to COPD, ILD, pulmonary thromboembolism, marked obesity, sleep apnoea or severe kyphoscoliosis.

The abdomen

Palpate the liver for ptosis,[i] due to emphysema, or for enlargement from secondary deposits of tumour in cases of lung carcinoma.

Other

Pemberton's[i] sign

Ask the patient to lift the arms over the head and wait for one minute.[15] Note

the development of facial plethora, cyanosis, inspiratory stridor and non-pulsatile elevation of the jugular venous pressure. This occurs in superior vena caval obstruction.

Legs

Inspect for swelling (oedema) or cyanosis, which may be clues to cor pulmonale, and look for evidence of deep venous thrombosis.

Respiratory rate on exercise

Patients complaining of dyspnoea should have their respiratory rate measured at rest, at maximal tolerated exertion (e.g. after climbing one or two flights of stairs or during a treadmill exercise test), and supine. If dyspnoea is not accompanied by tachypnoea when a patient climbs stairs, one should consider the possibility of anxiety or malingering.

Temperature

Fever may occur with any acute or chronic chest infection.

Bedside assessment of lung function

Forced expiratory time

Physical examination can be complemented with an estimate of the forced expiratory time (FET).[16] Measure the time taken by a patient to exhale forcefully and completely through the open mouth after taking a maximum inspiration. 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).[8] 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. The value obtained (the peak expiratory flow, PEF) depends largely on airways diameter. Normal values for young men are approximately 600 litres a minute and for women 400 litres a minute. 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 (Figure 5.12)

The spirometer records graphically or numerically the forced expiratory volume and the forced vital capacity. 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. Usually this is 1 second (FEV_1). 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 calculate the FEV_1/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.

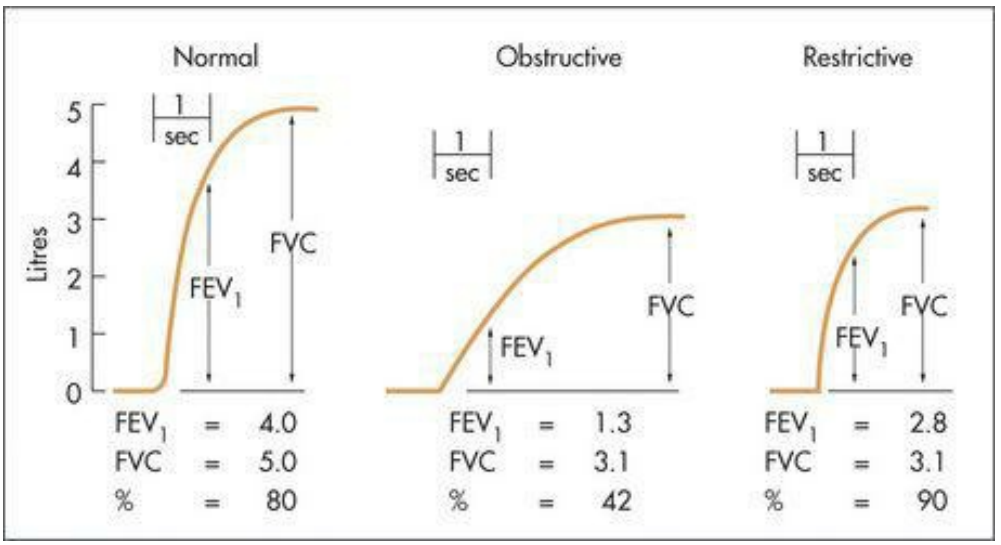


Figure 5.12 Spirometry tracings

Reversibility of a reduced FEV_1/FVC after the use of bronchodilators is an important test for distinguishing asthma from COPD.

Obstructive ventilatory defect

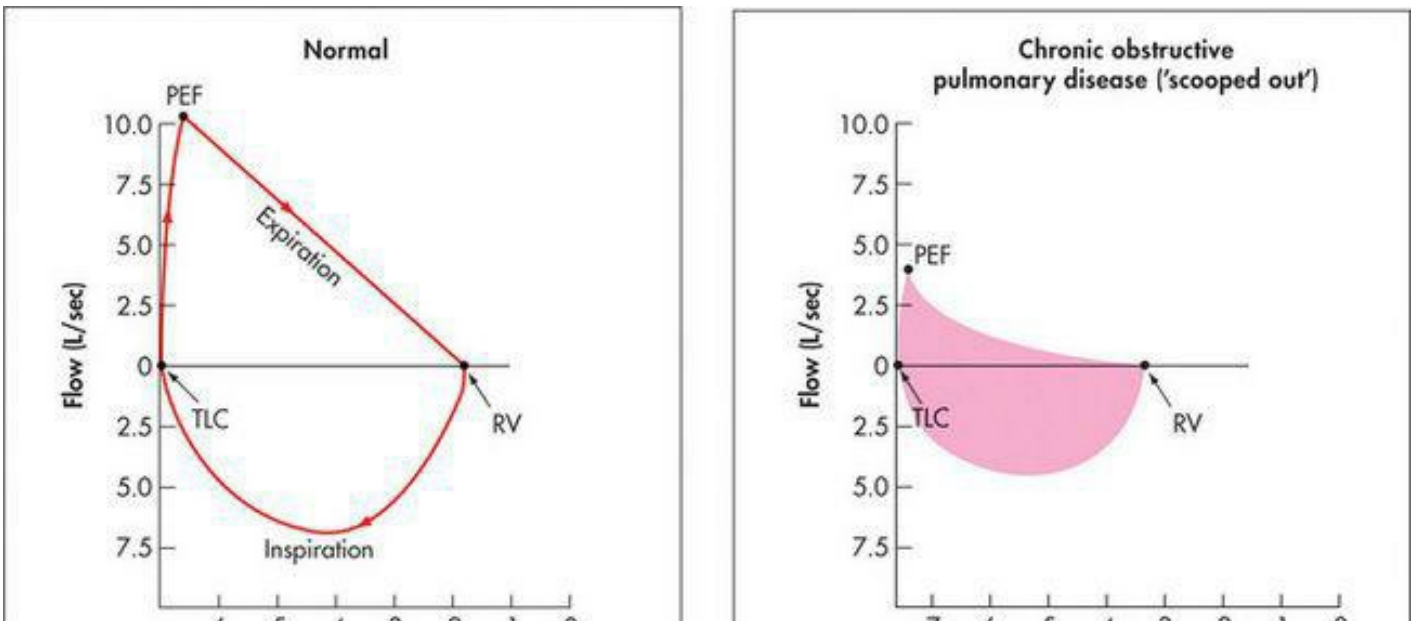
When the FEV_1/FVC ratio is reduced (<0.7) this is referred to as an obstructive defect. Both values tend to be reduced, but the FEV_1 is disproportionately low. The causes are loss of elastic recoil or airways narrowing, as in asthma or COPD.

Restrictive ventilatory defect

When the FEV_1/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.

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_1 and various flow measurements (e.g. peak flow) can be calculated from the curve (Figure 5.13).



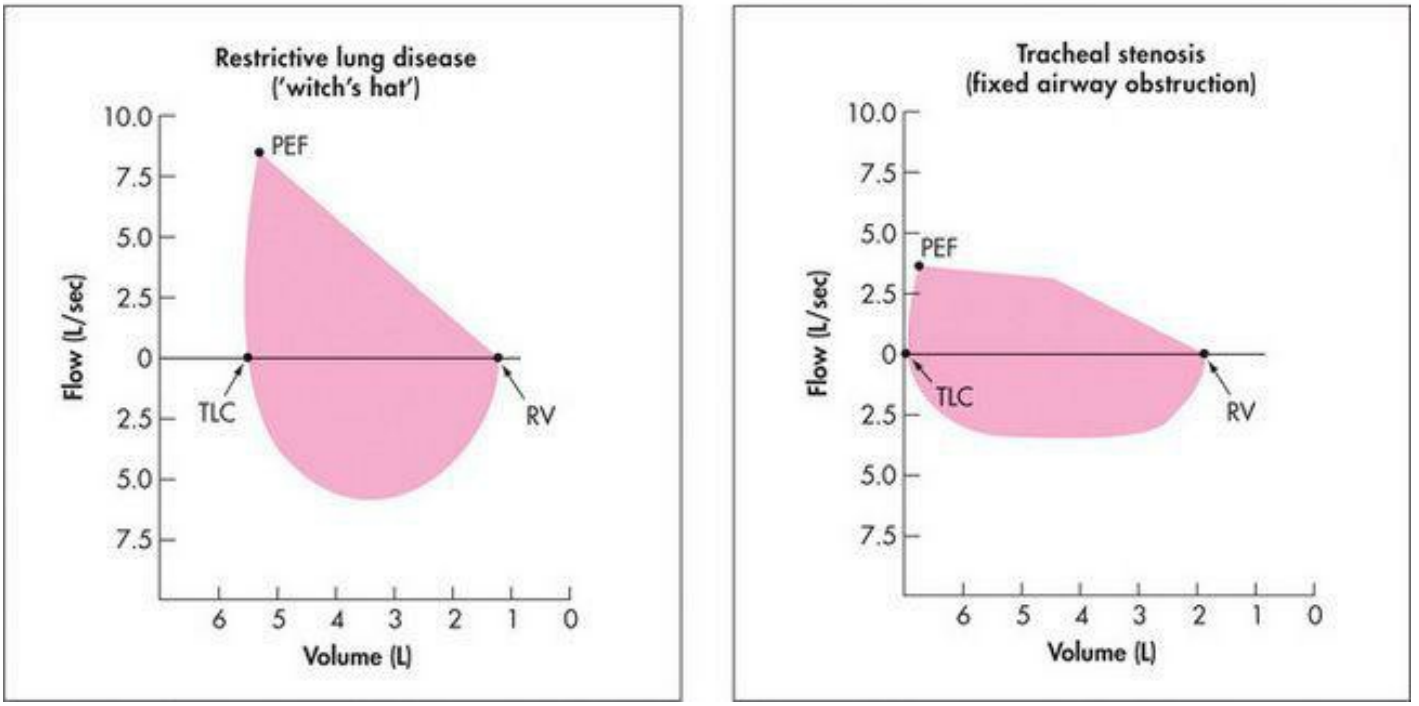


Figure 5.13 Flow volume curves
Look at the shape of the loop in each case. A normal flow volume curve is convex and symmetrical. In chronic obstructive lung disease (COPD), all flow routes are reduced and there is prolonged expiration (creating a 'scooped out' shape). In restrictive lung disease (e.g. 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

Correlation of physical signs and respiratory disease (Table 5.19)

Consolidation (lobar pneumonia)

Pneumonia is defined as inflammation of the lung which is characterised by exudation into the alveoli. 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
3. occurring in a damaged lung, e.g. as a result of aspiration; or
4. occurring in an immuno-compromised host.

TABLE 5.19 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

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*.[17]

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 *bronchopneumonia* refers to lung infection characterised by more patchy X-ray changes which often affect both lower lobes. The clinical signs of consolidation may be absent.

Symptoms

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

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.

See also *Good signs guide 5.3*.

GOOD SIGNS GUIDE 5.3 Pneumonia

Sign	Positive LR	Negative LR
General appearance		
Cachexia	4.0	NS
Abnormal mental state	2.2	NS
Vital signs		
Temperature >37.8°C	2.2	0.7

Respiratory rate >28/minute	2.2	0.8
Heart rate >100 beats/minute	1.6	0.7
Lung findings		
Percussion dullness	3.0	NS
Reduced breath sounds	2.3	0.8
Bronchial breath sounds	3.3	NS
Aegophony	4.1	NS
Crackles	2.0	0.8
Wheezes	NS	NS

NS = not significant.

From McGee S, Evidence-based physical diagnosis, 2nd edn. St Louis: Saunders, 2007.

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

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: (i) There may be no signs with complete lobar collapse. (ii) 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): (i) cardiac failure; (ii) hypoalbuminaemia from the nephrotic syndrome or chronic liver disease; (iii) hypothyroidism; (iv) Meigs syndrome[k] (ovarian fibroma causing pleural effusion and ascites).

• **Exudate** (Light's criteria[l]): (i) pneumonia; (ii) neoplasm—bronchial carcinoma, metastatic carcinoma, mesothelioma; (iii) tuberculosis; (iv) pulmonary infarction; (v) subphrenic abscess; (vi) acute pancreatitis; (vii) connective tissue disease such as rheumatoid arthritis, systemic lupus erythematosus; (viii) drugs such as methysergide, cytotoxics; (ix) irradiation; (x) trauma.

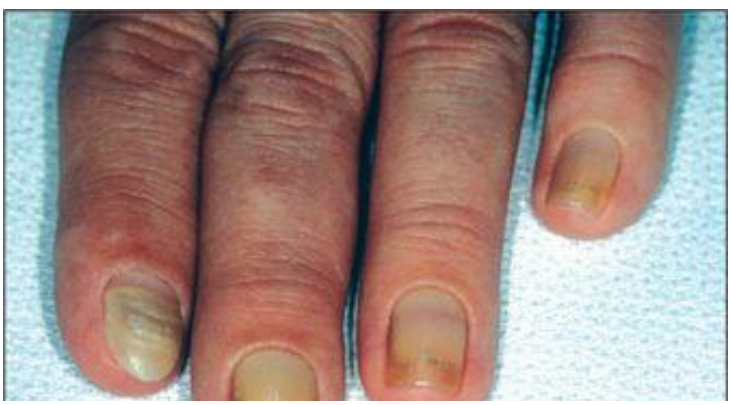
• **Haemothorax** (blood in the pleural space): (i) severe trauma to the chest; (ii) rupture of a pleural adhesion containing a blood vessel.

• **Chylothorax** (milky-appearing pleural fluid due to leakage of lymph): (i) trauma or surgery to the thoracic duct; (ii) carcinoma or lymphoma involving the thoracic duct.

• **Empyema** (pus in the pleural space): (i) pneumonia; (ii) lung abscess; (iii) bronchiectasis; (iv) tuberculosis; (v) penetrating chest wound.

Yellow nail syndrome

This is a rare condition which is caused by hypoplasia of the lymphatic system. The nails are thickened and yellow (Figure 5.14) 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.



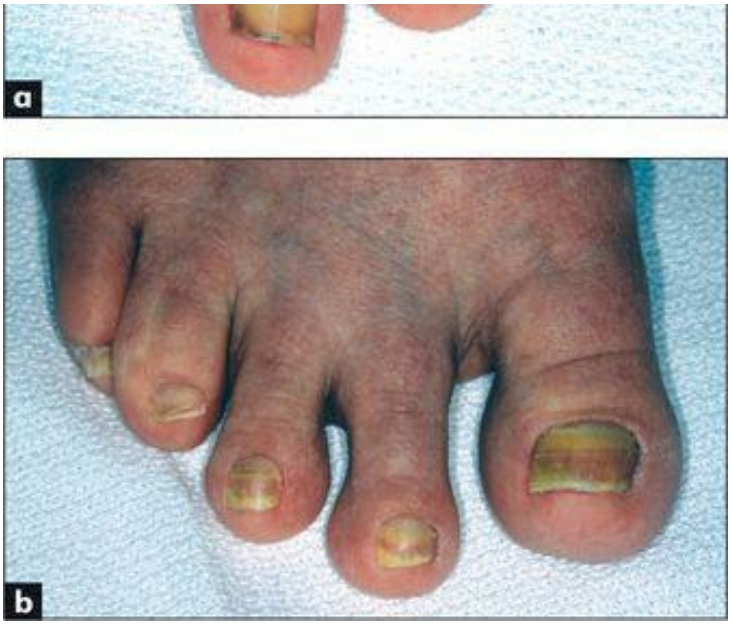


Figure 5.14 Yellow nail syndrome: (a) hands; (b) feet

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 (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 lymphangioleiomyomatosis (LAM—premenopausal 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

Causes

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

Bronchiectasis

This is a pathological dilatation 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%).
- Clubbing and cyanosis (if disease is severe).
- **Sputum:** voluminous, purulent, foul-smelling, sometimes bloodstained.
- Coarse pan-inspiratory or late inspiratory crackles over the affected lobe.
- **Signs of severe bronchiectasis:** very copious sputum and haemoptysis, clubbing, 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:** (i) primary ciliary dyskinesia (including the immotile cilia syndrome); (ii) cystic fibrosis; (iii) congenital hypogammaglobulinaemia.
- **Acquired:** (i) infections in childhood, such as whooping cough, pneumonia or measles; (ii) localised disease such as a foreign body, a bronchial adenoma or tuberculosis; (iii) 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.

Signs

- Wheezing.
- Dry or productive cough.
- Tachypnoea.
- Tachycardia.
- Prolonged expiration.
- Prolonged forced expiratory time (decreased peak flow, decreased FEV_1).
- Use of accessory muscles of respiration.
- Hyperinflated chest (increased anteroposterior diameter with high shoulders and, on percussion, decreased liver dullness).
- Inspiratory and expiratory wheezes.
- **Signs of severe asthma:** appearance of exhaustion and fear, inability to speak because of breathlessness, drowsiness due to hypercapnia (preterminal), cyanosis (a very sinister sign), tachycardia (pulse above 130/minute correlates with significant hypoxaemia), pulsus paradoxus (more than 20 mmHg), reduced breath sounds or a 'silent' chest.

Chronic obstructive pulmonary disease (COPD, chronic airflow limitation)

This represents a spectrum of abnormalities: from predominantly emphysema, where there is pathologically an increase beyond normal in the size of the air spaces distal to the terminal bronchioles, to chronic bronchitis

size of the air spaces distal to the terminal bronchioles, to chronic bronchitis, where there is mucous gland hypertrophy, increased numbers of goblet cells and hypersecretion of mucus in the bronchial tree resulting in a chronic cough and sputum. Chronic obstructive pulmonary disease limitation does not cause clubbing or haemoptysis. Fifty per cent of patients with chronic bronchitis have emphysema, so there is often considerable overlapping of signs.[18]

The diagnosis can often be made on the basis of three findings:

1. A history of heavy smoking (more than 70 packet-years).
2. Reduced breath sounds.
3. Previous diagnosis of emphysema or COPD.
   If two or three of these are present, the positive LR of COPD is 25.7.

Signs

The patients are usually not cyanosed but are dyspnoeic, and used to be called ‘pink puffers’. The signs result from hyperinflation.

- Barrel-shaped chest with increased anteroposterior diameter.
- Pursed-lip breathing (this occurs in emphysema and not in chronic bronchitis): expiration through partly closed lips increases the end-expiratory pressure and keeps airways open, helping to minimise air trapping.
- Use of accessory muscles of respiration and drawing in of the lower intercostal muscles with inspiration.
- **Palpation:** reduced expansion and a hyperinflated chest, Hoover’s sign, tracheal tug.

GOOD SIGNS GUIDE 5.4 Chronic obstructive pulmonary disease

Sign	Positive LR	Negative LR
Hoover's sign	4.2	0.5
Absent cardiac dullness, left sternal border	11.8	NS
Early inspiratory crackles	14.6	NS
Unforced wheeze	2.8	0.8
Greatly reduced breath sounds	10.2	—
Forced expiratory time:		
<3s	0.2	—
3–9s	NS	—
>9s	4.1	—

From McGee, S, Evidence-based physical diagnosis, 2nd edn. St Louis: Saunders, 2007.

- **Percussion:** hyperresonant with decreased liver dullness.
- **Breath sounds:** decreased, early inspiratory crackles

• **Breath sounds:** decreased, early inspiratory crackles.

• Wheeze is often absent.

• Signs of right heart failure may occur, but only late in the course of the disease.

Causes of generalised emphysema

• Usually, smoking.

• Occasionally, alpha$_1$-antitrypsin deficiency.

Chronic bronchitis

This is defined clinically as the daily production of sputum for three months a year for at least two consecutive years. It is not now diagnosed as a separate entity from COPD and is probably now of mostly historical interest.

Signs

The signs are the result of bronchial hypersecretion and airways obstruction.

• Loose cough and sputum (mucoid or mucopurulent), particularly in the morning shortly after wakening, and lessening as the day progresses.

• **Cyanosis:** these patients were sometimes called ‘blue bloaters’ because of cyanosis present in the latter stages, and because of associated oedema from right ventricular failure.

• **Palpation:** hyperinflated chest with reduced expansion.

• **Percussion:** increased resonance.

• **Breath sounds:** reduced with end-expiratory high or low-pitched wheezes and early inspiratory crackles.

• Signs of right ventricular failure.

Causes

Smoking is the major cause, but recurrent bronchial infection may cause progression of the disease.

Interstitial lung disease (ILD)

Diffuse fibrosis of the lung parenchyma impairs gas transfer and causes ventilation–perfusion mismatching. This fibrosis may be the result of inflammation (alveolitis and interstitial inflammation) or granulomatous disease (Table 5.20). It has often no known cause (idiopathic interstitial fibrosis) or is secondary to a disease of unknown aetiology (e.g. sarcoidosis, connective tissue disease). It can result from inhalation of mineral dusts (focal fibrosis), replacement of lung tissue following disease which damages the lungs (e.g. aspiration pneumonia, tuberculosis). Collagen diseases and vasculitis are important causes.

TABLE 5.20 Interstitial lung disease

Secondary to alveolitis (previously called fibrosing alveolitis)
Unknown cause Idiopathic pulmonary fibrosis Connective tissue disease (e.g. SLE, rheumatoid arthritis, ankylosing spondylitis, systemic sclerosis) Pulmonary haemorrhage syndromes (e.g. Goodpasture's syndrome) Graft versus host disease Gastrointestinal or liver diseases (Crohn's disease, primary biliary cirrhosis, chronic active hepatitis)
Known cause

Asbestosis Radiation injury Aspiration pneumonia Drugs (e.g. amiodarone) Exposure to gases or fumes
Secondary to granulomatous disease
Unknown cause Sarcoidosis Wegener's disease, Churg-Strauss disease
Known cause Hypersensitivity pneumonitis to organic or inorganic dusts (silica, beryllium)

SLE = systemic lupus erythematosus.

Remember the three Cs:

Cough (dry)

Clubbing

Crackles.

Signs

- **General:** dyspnoea, cyanosis and clubbing may be present.
- **Palpation:** expansion is slightly reduced.
- **Auscultation:** fine (Velcro-like) late inspiratory or pan-inspiratory

crackles heard over the affected lobes.

• **Signs of associated connective tissue disease:** rheumatoid arthritis, systemic lupus erythematosus, scleroderma, Sjögren's[m] syndrome, polymyositis and dermatomyositis.

Causes

• **Upper lobe predominant:** SCART. S = silicosis (progressive massive fibrosis), sarcoidosis; C = coal workers' pneumoconiosis (progressive massive fibrosis); A = ankylosing spondylitis, allergic bronchopulmonary aspergillosis; R = radiation; T = tuberculosis. Also cystic fibrosis, alveolar haemorrhage syndromes, chronic allergic alveolitis, chronic eosinophilic pneumonitis.

• **Lower lobe predominant:** RASIO. R = rheumatoid arthritis; A = asbestosis; S = scleroderma (systemic sclerosis); I = idiopathic interstitial fibrosis; O = other (drugs, e.g. busulfan, bleomycin, nitrofurantoin, hydralazine, methotrexate, amiodarone). Also other collagen vascular diseases, acute allergic alveolitis, acute eosinophilic pneumonitis.

Tuberculosis

Primary tuberculosis

A Ghon[n] focus with hilar lymphadenopathy occurs usually in children.

Usually no abnormal chest signs are found, but segmental collapse, due to bronchial obstruction by the hilar lymph nodes, occasionally occurs. Erythema nodosum (page 191) is an important associated sign, but is rare.

Post-primary tuberculosis

Reactivation of a primary lesion or occasionally reinfection are the causes of post-primary or adult tuberculosis. Immune suppression and malnutrition predispose to reactivation of tuberculosis.

There are often no chest signs. The clues to the diagnosis are the classical symptoms of cough, haemoptysis, weight loss, night sweats and malaise.

Miliary tuberculosis

Widespread haematogenous dissemination of tubercle bacilli causes multiple millet-seed tuberculous nodules in various organs—spleen, liver, lymph nodes, kidneys, brain or joints. Miliary tuberculosis may complicate both childhood and adult tuberculosis.

Fever, anaemia and cachexia are the general signs. The patient may also be dyspnoeic, and pleural effusions, lymphadenopathy, hepatosplenomegaly or signs of meningitis may be present.

Mediastinal compression

Mediastinal structures may be compressed by a variety of pathological masses, including carcinoma of the lung (90%), other tumours (lymphoma, thymoma, dermoid cyst), a large retrosternal goitre or rarely an aortic aneurysm.

Signs

• **Superior vena caval obstruction:** the face is plethoric and cyanosed with periorbital oedema; the eyes may show exophthalmos, conjunctival injection, and venous dilatation in the fundi; in the neck the jugular venous pressure is raised but not pulsatile, the thyroid may be enlarged, there may be supraclavicular lymphadenopathy and a positive Pemberton's sign; the chest may show dilated collateral vessels, or signs of lung carcinoma.

• **Tracheal compression:** stridor, usually accompanied by respiratory distress.

• **Recurrent laryngeal nerve involvement:** hoarseness of the voice.

• **Horner's syndrome**.

• **Paralysis of the phrenic nerve:** dullness to percussion at the affected base, which does not change with deep inspiration (abnormal tidal percussion), and absent breath sounds suggest a paralysed diaphragm due to phrenic nerve involvement.

Carcinoma of the lung

Many patients have no signs.

Respiratory and chest signs

- Haemoptysis.
- Clubbing, sometimes with hypertrophic pulmonary osteoarthropathy (usually not small cell carcinoma).
- Lobar collapse or volume loss.
- Pneumonia.
- Pleural effusion.
- Fixed inspiratory wheeze.
- Tender ribs (secondary deposits of tumour in the ribs).
- Mediastinal compression, including signs of nerve involvement.
- Supraclavicular or axillary lymphadenopathy.

Apical (Pancoast) tumour

Horner's syndrome, recurrent laryngeal nerve palsy (hoarseness), C8/T1 nerve root lesion.

Distant metastases

Brain, liver and bone are the most commonly affected organs.

Non-metastatic extrapulmonary manifestations

- Anorexia, weight loss, cachexia, fever.
- **Endocrine changes:** (i) hypercalcaemia, due to secretion of parathyroid hormone-like substances, occurs in squamous cell carcinoma; (ii)

hyponatraemia—antidiuretic hormone is released by small (oat) cell carcinomas; (iii) ectopic adrenocorticotrophic hormone (ACTH) syndrome (small cell carcinoma); (iv) carcinoid syndrome (small cell carcinoma); (v) gynaecomastia (gonadotrophins—rare; more often squamous cell); (vi) hypoglycaemia (insulin-like peptide from squamous cell carcinoma).

• **Neurological manifestations:** Eaton-Lambert[p] syndrome (progressive muscle weakness) and retinal blindness (small cell carcinoma), peripheral neuropathy, subacute cerebellar degeneration, polymyositis, cortical degeneration.

• **Haematological features:** migrating venous thrombophlebitis, disseminated intravascular coagulation, anaemia.

• **Skin:** acanthosis nigricans, dermatomyositis (rare).

• **Renal:** nephrotic syndrome due to membranous glomerulonephritis (rare).

Sarcoidosis

This is a systemic disease, characterised by the presence of non-caseating granulomas which commonly affect the lungs, skin, eyes, lymph nodes, liver and spleen, and the nervous system. The aetiology is unknown. There may be no pulmonary signs.

Pulmonary signs

• **Lungs:** no signs usually, although 80% of patients have lung involvement. In severe disease there may be signs of ILD.

Extrapulmonary signs

• **Skin:** lupus pernio (violaceous patches on the face, especially the nose, fingers or toes), pink nodules and plaques (granulomata) in old scars, erythema nodosum on the shins.

• **Eyes:** ciliary injection, anterior uveitis.

• **Lymph nodes:** generalised lymphadenopathy.

• **Liver and spleen:** enlarged (uncommon).

• **Parotids:** gland enlargement (uncommon) (page 161).

• **Central nervous system:** cranial nerve lesions, peripheral neuropathy (uncommon).

• **Musculoskeletal system:** arthralgia, swollen fingers, bone cysts (rare).

• **Heart:** heart block presenting as syncope, cor pulmonale (both rare).

• Signs of hypercalcaemia.

Pulmonary embolism (PE)

Embolism to the lungs often occurs without symptoms or signs. One should always entertain this diagnosis if there has been sudden and unexplained dyspnoea when a patient has risk factors for embolism (Table 5.21). Pleuritic chest pain and haemoptysis occur only when there is infarction. Syncope or the sudden onset of severe substernal pain can occur with massive embolism.

• **General signs:** tachycardia, tachypnoea, fever (with infarction).

• **Lungs:** pleural friction rub if infarction has occurred.

• **Massive embolism:** elevated jugular venous pressure, right ventricular gallop, right ventricular heave, tricuspid regurgitation murmur, palpable pulmonary component of the second heart sound (P2), gallop (S3 and/or S4).

• **Signs of deep venous thrombosis:** fewer than 50% of patients have clinical evidence of a source.

TABLE 5.21 Risk factors for pulmonary embolism (PE)

1 Previous PE
2 Immobilisation (long aeroplane flight or especially after surgery)

3 Known clotting-factor abnormalities
4 Known malignancy

Note: A firm diagnosis cannot be made on the symptoms and signs alone.

The chest X-ray

The radiological appearance of a normal lung, with the lung segments labelled, is shown in Figure 5.15.

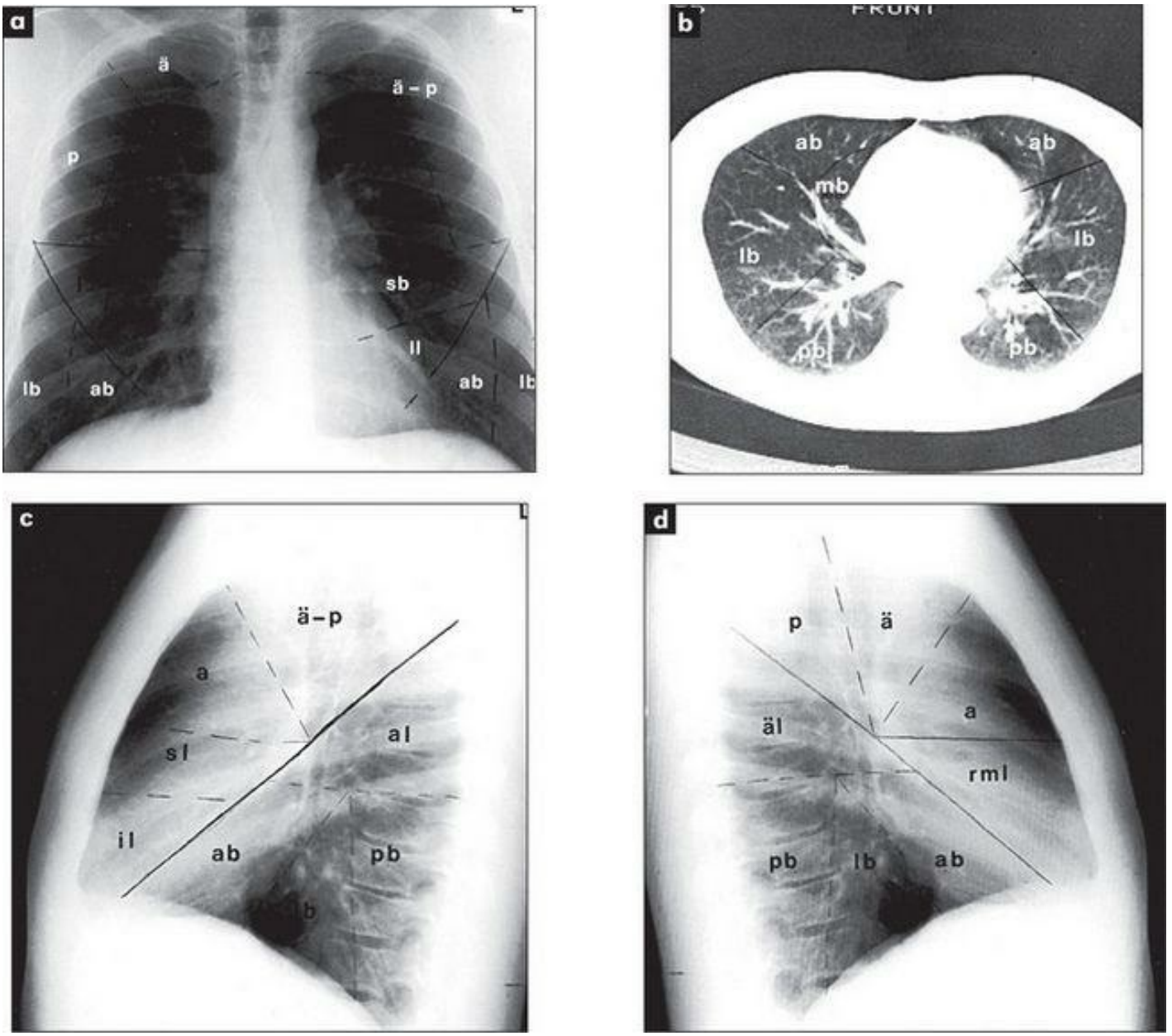


Figure 5.15 Lung segments

(a) Posteroanterior view. (b) CT scan through lung bases. (c) Left lateral view. (d) Right lateral view.
Right upper lobe: ä = apical segment; a = anterior segment, p = posterior segment.
Left upper lobe: ä-p = apico-posterior segment, s = anterior segment, sl = superior lingular segment, il = inferior lingular segment.
Right middle lobe (rml): m = medial segment, l = lateral segment.
Right lower lobe: äl = apical segment, mb = medial basal segment, lb = lateral basal segment, ab = anterior basal segment, pb = posterior basal segment.
Left lower lobe: äl = apical segment, lb = lateral basal segment, ab = anterior basal segment, pb = posterior basal segment.

The radiological changes of consolidation, pleural effusion, pneumothorax and hydropneumothorax are shown in Figures 5.16 to 5.19.

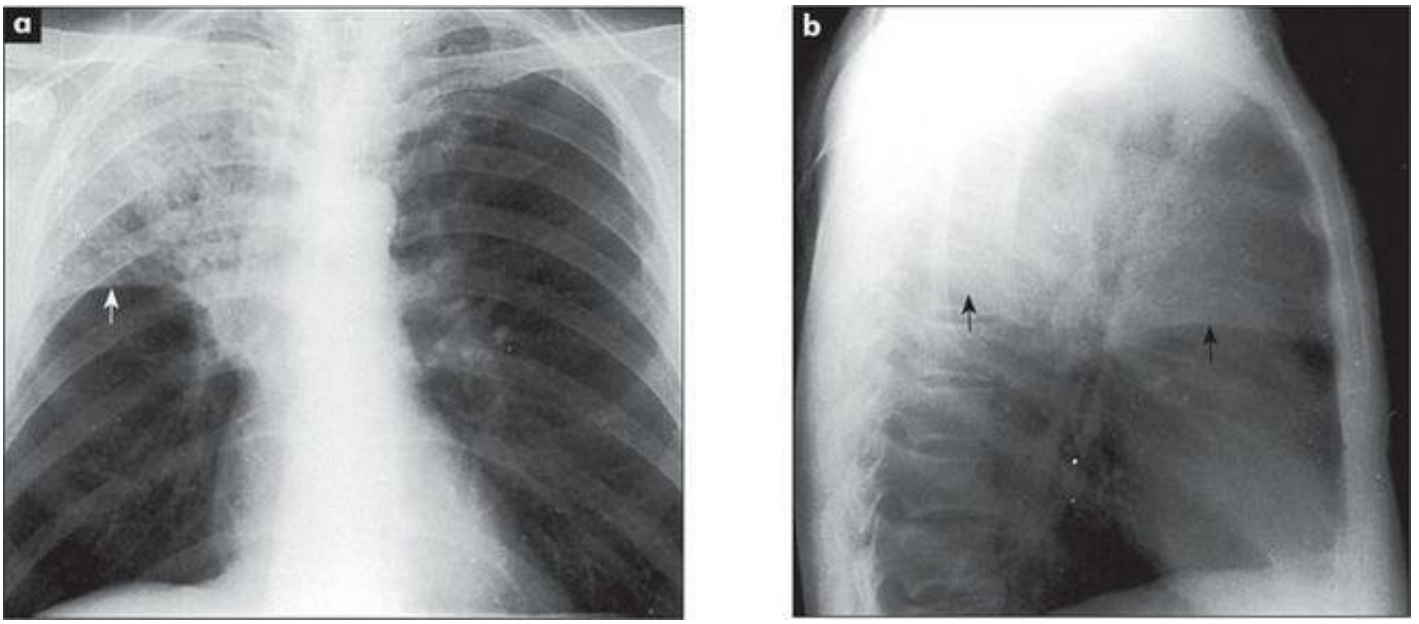
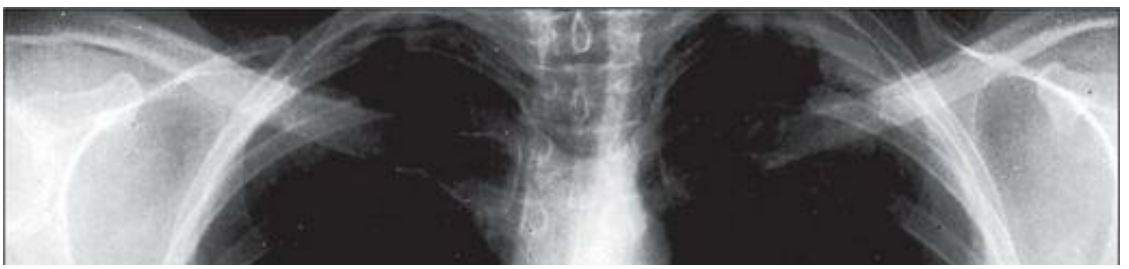


Figure 5.16 Right upper lobe consolidation
The right upper lobe is opacified and is limited inferiorly by the horizontal fissure (arrows). There must be some collapse as well, as the fissure shows some elevation. These changes could be due to a bacterial lobar pneumonia per se, but a central bronchostenotic lesion should be considered. If the pneumonia persists, a bronchoscopy is indicated to search for a central carcinoma.



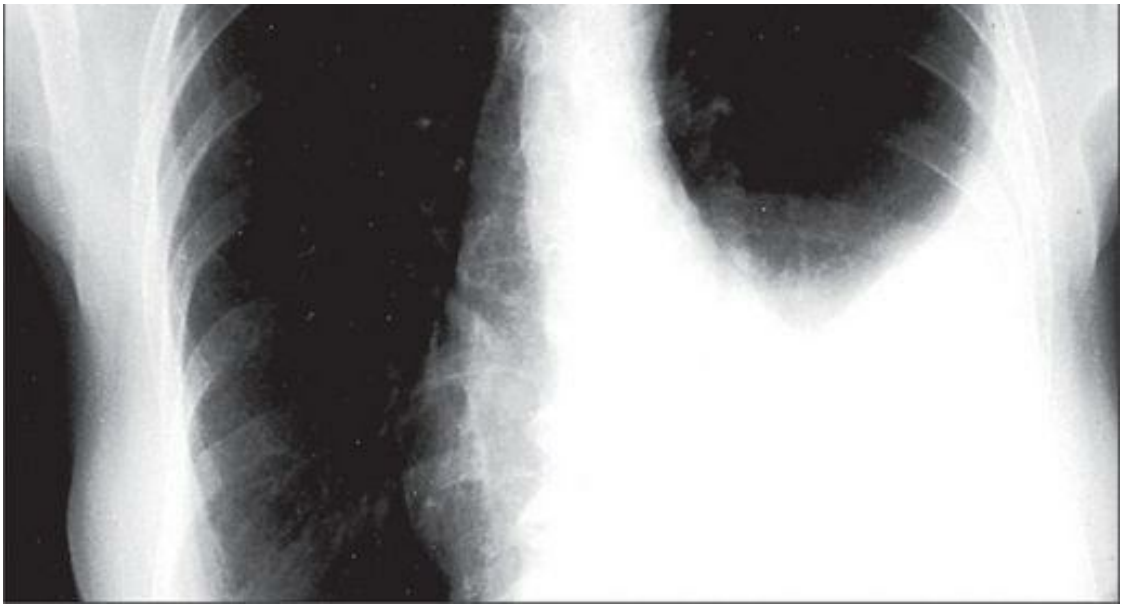


Figure 5.17 Pleural effusion
The upper margin of the effusion is curved ('meniscus sign'). The left hemidiaphragm is not seen because there is no adjacent aerated lung for contrast. The heart shows some deviation to the right. It is unlikely that this is caused by an effusion of this size. It is probably related to the lower thoracic scoliosis.

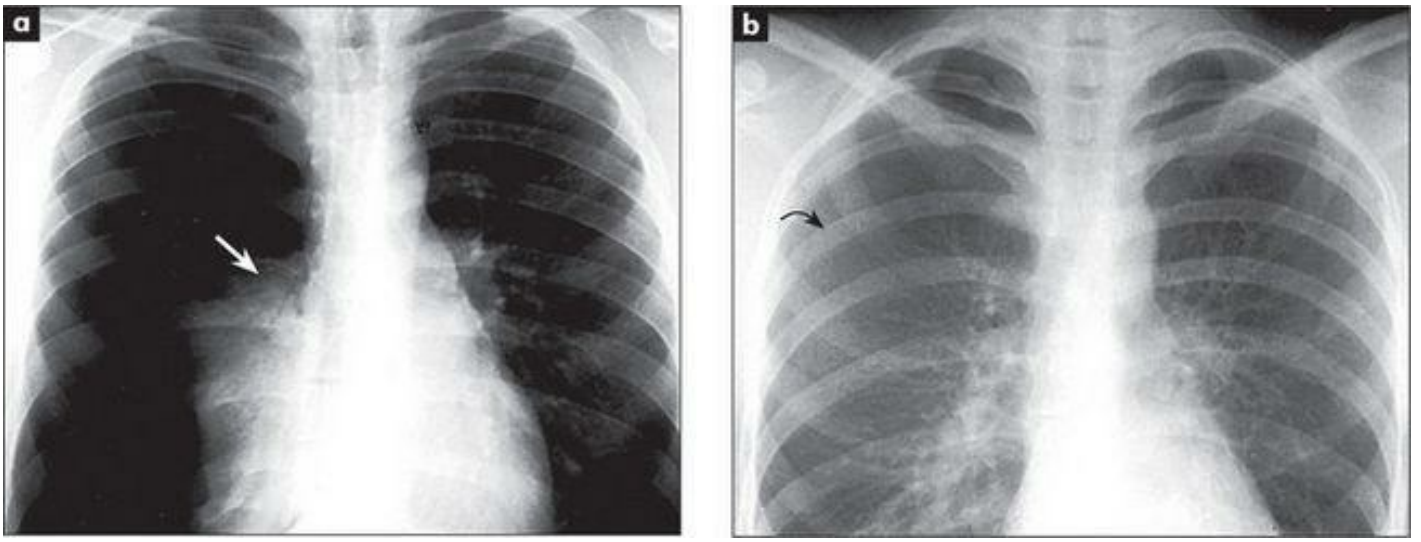


Figure 5.18 Pneumothorax
(a) There is a massive right pneumothorax with collapsed lung seen against the hilum (arrow). There is increased translucency because of the absence of vascular shadows. (b) Different patient with a smaller pneumothorax. Small pneumothoraces are easier to see on an expiratory film as the pneumothorax volume remains constant, surrounding the partly deflated lung. The visceral pleural surface is marked (arrow).

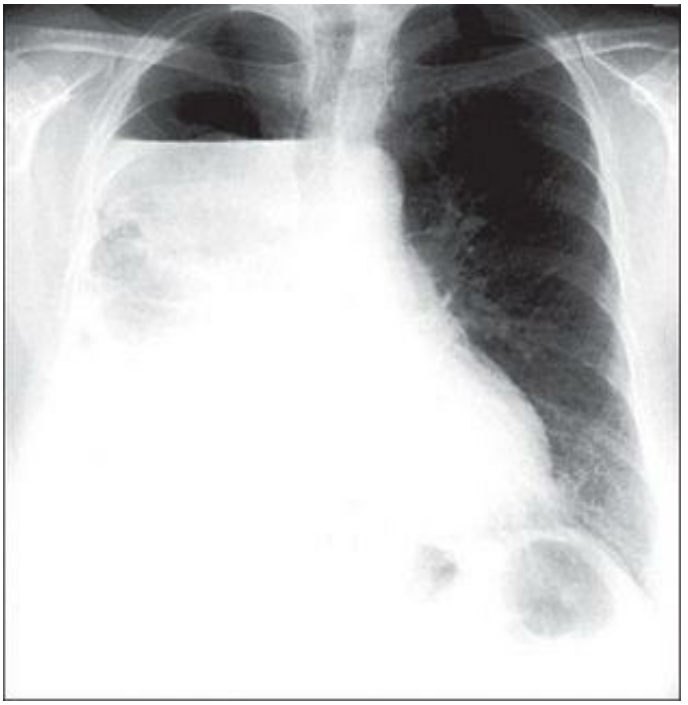


Figure 5.19 Hydropneumothorax
An air–fluid level is seen in the upper portion of the right hemithorax. When air and fluid are present in the pleural space, the fluid no longer forms a meniscus at its upper margin. Some aerated lung is seen deep to the fluid.

A pulmonary mass is obvious in Figure 5.20, while multiple metastases are seen in Figure 5.21. Primary tuberculosis is shown in Figure 5.22, and Figure 5.23 illustrates the features of emphysema.

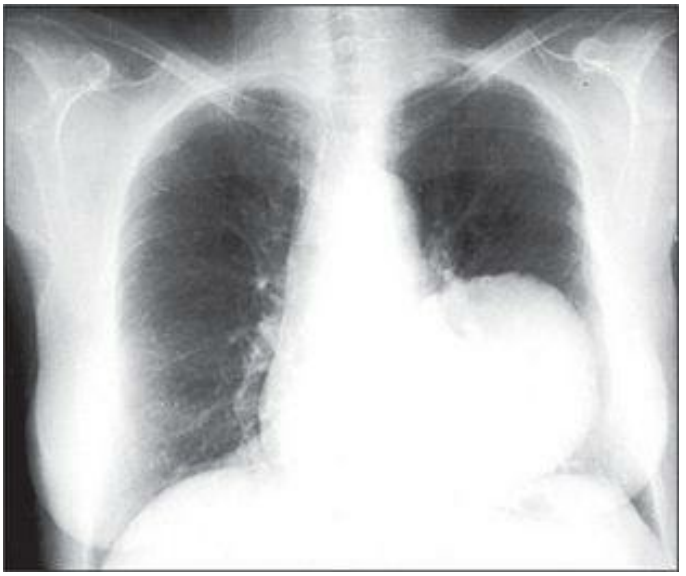


Figure 5.20 A pulmonary mass
There is a large solitary mass lesion in the left lower zone. The differential diagnosis is primary or secondary neoplasm, hydatid cyst or large abscess. No air–fluid level is seen within it to indicate cavitation.

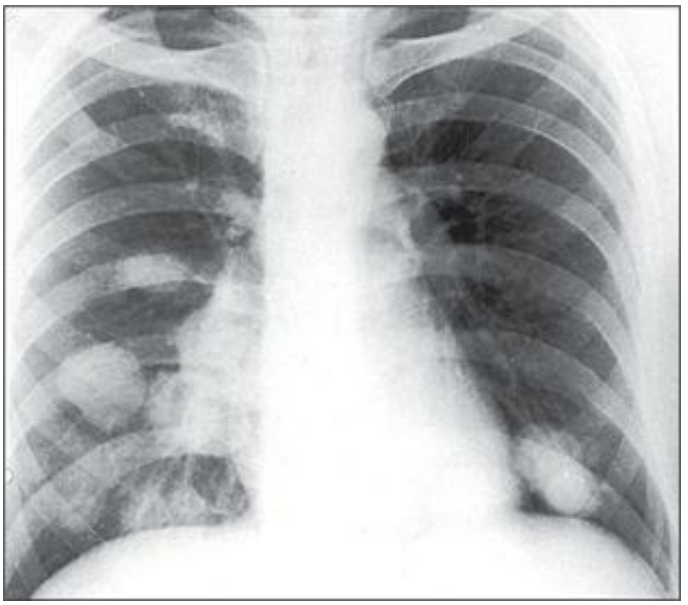


Figure 5.21 Pulmonary metastases
Multiple rounded opacities are seen in both lung fields, mainly at the left base and around the right hilum. The most likely cause is multiple pulmonary metastases. Other rare possibilities are hydatid cysts, large sarcoid nodules or large rheumatoid nodules. Multiple abscesses are extremely unlikely in the absence of cavitation.

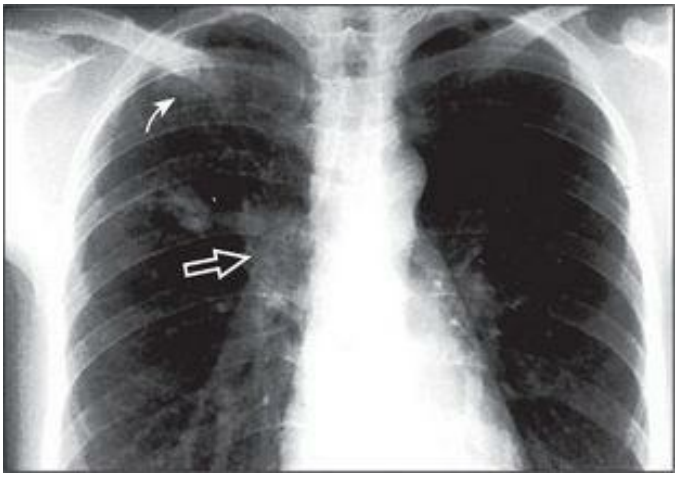


Figure 5.22 Primary tuberculosis
Two small rounded areas of shadowing are seen in the right upper zone (solid arrow). The right hilum is enlarged by the enlarged draining lymph nodes (open arrow). This combination of focal shadowing and enlarged lymph nodes is the primary (Ghon) complex of tuberculosis. With healing, calcification may occur in the parenchymal and nodal lesions. In contrast, in tuberculosis reactivation or reinfection, cavitation may occur and there is no lymphadenopathy.

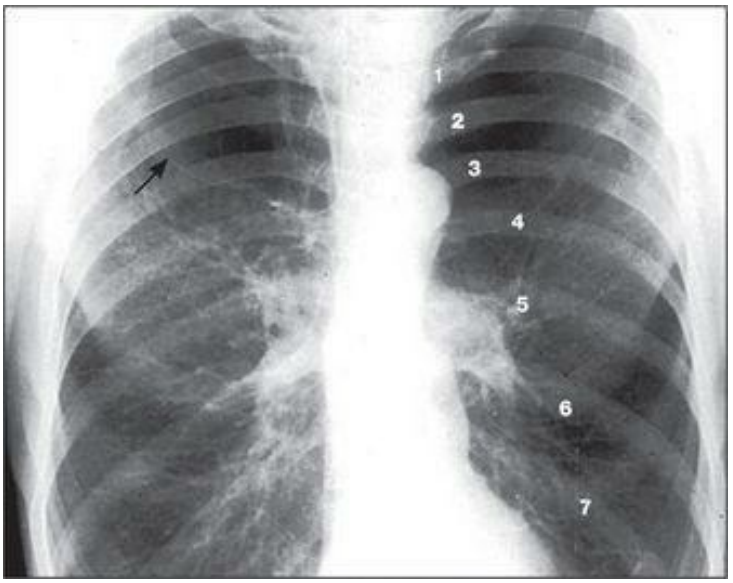


Figure 5.23 Emphysema
The lungs are overinflated with low, flat hemidiaphragms. The level of the hemidiaphragms is well below the anterior aspects of the sixth ribs. The diaphragm normally projects over the sixth rib anteriorly and the tenth intercostal space posteriorly. Count the ribs anteriorly (1–6). There is increased translucency of both upper zones with loss of the vascular markings due to bulla formation (arrow). This increased translucency is not due to overexposure. The hila are prominent because of the enlarged central pulmonary arteries. In contrast, the smaller peripheral pulmonary arteries (the lung markings) are decreased in size and number. This is due to actual destruction, displacement around bullae, and decreased perfusion through emphysematous areas.

Chest X-ray checklist

A—Airway (midline, no obvious deformities, no paratracheal masses).

B—Bones and soft tissue (no fractures, subcutaneous emphysema).

C—Cardiac size, silhouette and retrocardiac density normal.

D—Diaphragms (right above left by 1–3 cm, costophrenic angles sharp, diaphragmatic contrast with lung sharp).

E—Equal volume (count ribs, look for mediastinal shift).

F—Fine detail (pleura and lung parenchyma).

G—Gastric bubble (above the air bubble one shouldn't see an opacity of

any more than 0.5 cm width).

H—Hilum (left normally above right by up to 3 cm, no larger than a thumb), hardware (especially in the intensive care unit: endotracheal tube, central venous catheters, pacemaker).

Summary

The respiratory examination: a suggested method (Figure 5.24)

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 indrawing of the lower ribs anteriorly (a sign of emphysema). General cachexia should also be noted.

Figure 5.24 Respiratory system
SVC = superior vena cava.
Sitting up (if not acutely ill)

1. **General inspection**
   Sputum mug contents (blood, pus etc)
   Type of cough

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_2 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

6. **Chest anteriorly**
Inspect
- Radiotherapy marks, other signs as noted above

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

Pick up the **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 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). 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.

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, 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 (Chapter 14).

Lay the patient down at 45 degrees 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.

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additional information to these predictors

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[a] This condition has undergone many changes in nomenclature, and it is pleasing to think that chest physicians have something to keep them occupied. The term COPD encompasses emphysema, chronic bronchitis, chronic obstructive lung disease (COLD) and chronic airflow limitation (CAL). It now seems quite firmly established. 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.

[b] The word is derived from the Greek word *sterigma* which means *to support*, and refers to a flapping tremor.

[c] Johann Horner (1831–1886), professor of ophthalmology in Zurich, described this syndrome in 1869.

[d] Henry Khunrath Pancoast (1875–1939), professor of roentgenology, University of Pennsylvania, described this in 1932.

[e] Edward Harrison (1766–1838), British general practitioner in Lincolnshire, described this deformity in rickets in 1798. The sign has also been ascribed to Edwin Harrison (1779–1847), a London physician.

[f] It also probably didn't help!

[g] Charles Hoover (1865–1927), professor of medicine in Cleveland from

1907. He also described Hoover's test for non-organic limb weakness.

[h] Expiratory crackles may also occur with lung fibrosis.[13]

[i] From the Greek word for falling, this was once mostly applied to the eyelid but now seems accepted as a description of the displacement of any organ.

[j] Hugh Pemberton (1891–1956), physician, Liverpool, UK.

[k] Joe Vincent Meigs (1892–1963), Professor of Gynaecology at Harvard, described this in 1937.

[l] 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.

[m] Henrik Samuel Conrad Sjögren (1899–1986), Stockholm ophthalmologist. He described the syndrome in 1933.

[n] Anton Ghon (1866–1936), Austrian pathologist and Professor of Anatomical Pathology in Prague. He described the lesion in 1912.

[o] First described by William Hunter (1718–83; brother of John Hunter) in a patient with a syphilitic aortic aneurysm.

[p] ML Eaton, 20th century American physician, and EH Lambert (b. 1915), American neurologist.

Chapter 6

The gastrointestinal system

> To study the phenomena of disease without books is to sail an uncharted sea, while to study books without patients is not to go to sea at all.
> *Sir William Osler (1849–1919)*

> This lord wears his wit in his belly, and his guts in his head.
> *William Shakespeare*, Troilus and Cressida

Gastroenterologists and gastrointestinal surgeons concern themselves with the entire length of the gut, the liver, the exocrine pancreas and the

peripheral effects of alimentary disease.

The gastrointestinal history

Presenting symptoms (Table 6.1)

Abdominal pain

There are many causes of abdominal pain, and careful history taking will often lead to the correct diagnosis. The following should be considered.

TABLE 6.1 Gastrointestinal history

Major symptoms
Abdominal pain
Appetite and/or weight change
Postprandial fullness or early satiation, or both
Nausea and/or vomiting
Heartburn and/or acid regurgitation
Waterbrash
Dysphagia
Disturbed defecation (diarrhoea, constipation, faecal incontinence)
Bloating or visible distension, or both

Bleeding (haematemesis, melaena, rectal bleeding)
Jaundice
Dark urine, pale stools
Pruritus
Lethargy
Fever

Frequency and duration

Try to determine whether the pain is acute or chronic, when it began and how often it occurs.

Site and radiation

The site of pain is important. Ask the patient to point to the area affected by pain and to the point of maximum intensity. Parietal peritoneal inflammation that causes pain usually does so in a localised area. Ask about radiation of pain. Pain often radiates through to the back with pancreatic disease or a penetrating peptic ulcer. It may radiate to the shoulder with diaphragmatic irritation or to the neck with oesophageal reflux.

Character and pattern

The pain may be colicky (coming and going in waves and related to peristaltic movements) or steady. Colicky pain comes from obstruction of the bowel or the ureters. If the pain is chronic, ask about the daily pattern of pain.

Aggravating and relieving factors

Pain due to peptic ulceration may or may not be related to meals. Eating may

precipitate ischaemic pain in the gut. Antacids or vomiting may relieve peptic ulcer pain or that of gastro-oesophageal reflux. Defaecation or passage of flatus may relieve the pain of colonic disease temporarily. Patients who get some relief by rolling around vigorously are more likely to have a colicky pain, while those who lie perfectly still are more likely to have peritonitis.

Patterns of pain

Peptic ulcer disease

This is classically a dull or burning pain in the epigastrium that is relieved to a degree by food or antacids. It is typically episodic and may occur at night, waking the patient from sleep. This combination of symptoms is suggestive of the diagnosis. The pain is often unrelated to meals, despite classical teaching to the contrary. It is not possible to distinguish duodenal ulceration from gastric ulceration clinically.

Pancreatic pain

This is a steady epigastric pain that may be partly relieved by sitting up and leaning forwards. There is often radiation of the pain to the back, and vomiting is common.

Biliary pain

Although usually called ‘biliary colic’, this pain is rarely colicky. With cystic duct obstruction there is often epigastric pain. It is usually a severe, constant pain that can last for hours. There may be a history of episodes of similar pain in the past. If cholecystitis develops, the pain typically shifts to the right upper quadrant and becomes more severe.

Renal colic

This is a colicky pain superimposed on a background of constant pain in the renal angle, often with radiation towards the groin. It can be very severe indeed.

Bowel obstruction

This is colicky pain. Periumbilical pain suggests a small bowel origin but colonic pain can occur anywhere in the abdomen. Small bowel obstruction tends to cause more frequent colicky pain (with a cycle every 2–3 minutes) than large bowel obstruction (every 10–15 minutes). Obstruction is often associated with vomiting, constipation and abdominal distension.

Appetite and weight change

Loss of appetite (anorexia) and weight loss are important gastrointestinal symptoms. The presence of both anorexia and weight loss should make one suspicious of an underlying malignancy, but may also occur with depression and in other diseases. The combination of weight loss with an increased appetite suggests malabsorption of nutrients or a hypermetabolic state (e.g. thyrotoxicosis). It is important to document when the symptoms began and how much weight loss has occurred over this period. Liver disease can sometimes cause disturbance of taste. This may cause smokers with acute hepatitis and jaundice to give up smoking.

Early satiation and postprandial fullness

Inability to finish a normal meal (early satiation) may be a symptom of gastric diseases, including gastric cancer and peptic ulcer. A feeling of inappropriate fullness after eating can also be a symptom of functional (unexplained) gastrointestinal disease.

Nausea and vomiting

Nausea is the sensation of wanting to vomit. Heaving and retching may occur but there is no expulsion of gastric contents. There are many possible causes for these complaints. Gastrointestinal tract infections (e.g. from food poisoning by *Staphylococcus aureus*) or small bowel obstruction can cause acute symptoms. In patients with chronic symptoms, pregnancy and drugs (e.g. digoxin, opiates, dopamine agonists, chemotherapy) should always be ruled out. In the gastrointestinal tract, peptic ulcer disease with gastric outlet obstruction, motor disorders (e.g. gastroparesis from diabetes mellitus, or after gastric surgery), acute hepatobiliary disease and alcoholism are important causes. Finally, psychogenic vomiting, eating disorders (e.g. bulimia) and, rarely, increased intracranial pressure should be considered in patients with chronic unexplained nausea and vomiting.

The timing of the vomiting can be helpful; vomiting delayed more than 1 hour after the meal is typical of gastric outlet obstruction or gastroparesis, while early morning vomiting before eating is characteristic of pregnancy, alcoholism and raised intracranial pressure. Also ask about the contents of the vomitus (e.g. bile indicates an open connection between the duodenum and stomach, old food suggests gastric outlet obstruction, while blood suggests ulceration).

Heartburn and acid regurgitation

Heartburn refers to the presence of a burning pain or discomfort in the retrosternal area. Typically, this sensation travels up towards the throat and occurs after meals or is aggravated by bending, stooping or lying supine. Antacids usually relieve the pain, at least transiently. This symptom is due to regurgitation of stomach contents into the oesophagus. Usually these contents are acidic, although occasionally alkaline reflux can induce similar problems. Associated with gastro-oesophageal reflux may be *acid regurgitation,* in which the patient experiences a sour or bitter-tasting fluid coming up into the mouth. This symptom strongly suggests that reflux is occurring. Some patients complain of a cough that troubles them when they lie down. In patients with gastro

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Questions box 6.1

Questions to ask a patient presenting with recurrent vomiting denotes symptoms for the possible diagnosis of an urgent or dangerous problem.

1. How long have you been having attacks of vomiting (distinguish acute from chronic)?

2. Does the vomiting occur with nausea preceding it, or does it occur without any warning?

3. Is the vomiting usually immediately after a meal or hours after a meal?

4. Do you have vomiting early in the morning or late in the evening?

5. What does the vomit look like? Is it bloodstained, bile-stained or faeculent?—Gastro-intestinal bleeding or bowel obstruction

6. Do you have specific vomiting episodes followed by feeling completely well for long periods before the vomiting episode occurs again?—Cyclical vomiting syndrome

7. Is there any abdominal pain associated with the vomiting?

7. Is there any abdominal pain associated with the vomiting?

8. Have you been losing weight?

9. What medications are you taking?

10. Do you have worsening headaches?—Neurological symptoms suggest a central cause

oesophageal reflux disease, the lower oesophageal sphincter muscle relaxes inappropriately. Reflux symptoms may be aggravated by alcohol, chocolate, caffeine, a fatty meal, theophylline, calcium channel blockers and anticholinergic drugs, as these lower the oesophageal sphincter pressure.

Waterbrash refers to excessive secretion of saliva into the mouth and should not be confused with regurgitation; it may occur, uncommonly, in patients with peptic ulcer disease or oesophagitis.

Dysphagia

Dysphagia is difficulty in swallowing. Such difficulty may occur with solids or liquids. The causes of dysphagia are listed in Table 6.2. If a patient complains of difficulty swallowing, it is important to differentiate painful swallowing from actual difficulty.[1] Painful swallowing is termed *odynophagia* and occurs with any severe inflammatory process involving the oesophagus. Causes include infectious oesophagitis (e.g. *Candida*, herpes simplex), peptic ulceration of the

TABLE 6.2 Causes of dysphagia

Mechanical obstruction
Intrinsic (within oesophagus)
Reflux oesophagitis with stricture formation
Carcinoma of oesophagus or gastric cardia
Pharyngeal or oesophageal web
Pharyngeal pouch