INTRODUCTION

Respiratory medicine comprises a large part of everyday clinical practice for two reasons:

- respiratory conditions are common – accounting for more than 13 per cent of all emergency admissions and more than 20 per cent of general practitioner consultations
- respiratory symptoms and signs as elicited by respiratory history and examination are often present in non-respiratory conditions as well as respiratory conditions.

CLINICAL HISTORY

The six key symptoms of respiratory disease are:

- chest pain (that may be extended to chest sensations)
- dyspnoea
- cough
- wheeze
- sputum production
- haemoptysis.

In a respiratory clinic it would be routine as well to ask about snoring and excessive daytime sleepiness, especially in patients who are overweight, because this might lead to a diagnosis of obstructive sleep apnoea syndrome. Other more generic symptoms are also common in respiratory disease and should be covered elsewhere in the history. Enquire about weight loss, anorexia and headache, as these may all be part of common respiratory illnesses. Once the patient has given their account, prompted by open-ended questions, go on to examine the nature of the symptoms in more detail.

Chest pain

Ask about the onset, character, severity, duration, radiation, and any previous history of chest pain.

Dyspnoea

Analysis of dyspnoea should be approached in a similar way to that of chest pain, so ask about sever-
Clinical history

ity, duration, onset, precipitating factors, and previous history. It is absolutely crucial to ask about the onset. Ask the patient what they were doing at the time when the breathlessness started in order to jog their memory and give you some idea as to how sudden the onset was.

- Shortness of breath that has appeared out of the blue with no apparent precipitating factor should make you search for other risk factors for thromboembolic disease and suggest appropriate tests.
- In contrast, a slow onset of gradually increasing shortness of breath over many months may indicate a more chronic condition such as chronic obstructive pulmonary disease (COPD) or interstitial lung disease.
- A previous history of episodes of shortness of breath is often very useful in determining a diagnosis.
- Conditions that have exacerbations of breathlessness such as COPD, asthma or bronchiectasis have often already been diagnosed and may have important clues in the rest of the history.

Relate dyspnoea to other symptoms (see Box 8.2). Patients with asthma and COPD may have associated wheeze, and in bronchiectasis there may be a history of chronic high volume sputum production. Ask about whether the breathlessness is present at rest or whether it is only exercise-related.

- Breathlessness that comes on repeatedly at rest implies different pathophysiology in that there is some sort of disturbance that is causing the sensation of breathlessness and it does not depend on physical activity. This could include pulmonary thromboembolism, cardiac arrhythmias, cardiac ischaemia or spontaneous pneumothorax.
- Breathlessness that is usually precipitated by physical exertion implies that there is some deficiency in the body’s ability to cope with the extra exertion and this might occur in conditions such as asthma, COPD, heart failure and interstitial lung disease. All of the latter conditions can of course, in their most severe forms, produce breathlessness at rest. On its own, it is a relatively non-specific symptom.

**Wheeze**

You must ensure that you understand what your patient means when they answer positively to your direct question: ‘Do you wheeze?’ This question is useful because it will encourage the patient to describe any noises that they make with their breathing. Then, by further questioning, you can clarify exactly what is meant. From a medical perspective, a wheeze is a musical note generated from the lungs that may be a single note (monophonic wheeze) or multiple different notes (polyphonic wheeze). This will be clarified by auscultation. Patients find it difficult to describe the noises that they are making but you should attempt to get them to do so. Prompting them with suggestions that the sound might be musical or a squeaking sort of sound often helps them. If the patient appears to be describing a monophonic wheeze or stridor (see ‘Physical examination’, p. 87), then ask on which side the patient experiences this and whether the noise is worse when lying on the left or right side. This can indicate large airway obstruction, of which lung cancer is the most serious cause.

Ask about the onset, duration and periodicity of wheeze. Wheeze that occurs more at night and first thing in the morning, and that may be exacerbated by exercise, is suggestive of asthma and COPD. A pronounced variation in the severity of wheeze (worse at night and in the morning compared to daytime) is more suggestive of asthma, but by no means excludes COPD. Ask about the relationship between other respiratory symptoms and precipitating factors, specifically asking about exercise and cold or foggy weather. This will give you an idea about whether

**Box 8.2 Chest pain and dyspnoea overlap with other systems**

**Example A:** History of crushing central chest pain radiating to the arm and associated with nausea and vomiting and a feeling of dread.

**Conclusion:** Myocardial ischaemic pain.

**Example B:** Progressive dyspnoea and fatigue over several months. Pallor, but no other symptoms or signs; haemoglobin 6.0 g/dL.

**Conclusion:** Anaemia.

Dyspnoea and chest pain are symptoms that can be present in many conditions – usually related to either the heart or the lungs (example A), but occasionally more generic (example B).
the wheeze is variable rather than fixed and this may be important in relation to the differential diagnosis of asthma or COPD. During this direct questioning, you may identify that the patient experiences a rattling sort of sound that is not a wheeze but nevertheless one that may be important in the differential diagnosis. For example, some patients are aware of a crackling sound that is often generated by the movement of mucus in the lungs and may be accompanied by coarse crackles on physical examination.

Cough

Cough is the commonest symptom that is associated with pure respiratory disease. The function of cough is to expel unwanted elements from the respiratory tract; that includes both foreign elements and substances generated by the host. Thus cough is a prominent feature of upper respiratory infections, inhalation of irritants such as dusts and chemicals, as a result of lower respiratory infections, and the result of accumulation of products within lung (e.g. in pulmonary oedema). In addition to this, cough receptors within the lung can be stimulated as happens in interstitial lung disease or in endobronchial sarcoidosis.

One of the key factors, therefore, is to determine the onset and duration of the cough and its relation to other symptoms. A cough that has been present for a couple of weeks and has been associated with a coryzal illness is easily put down to an upper respiratory tract infection that may be caused by the rhinovirus. Cough of medium duration (3 weeks to 3 months) is more likely to have a non-self-limiting cause – lung cancer fits into this category, as well as the causes of chronic cough shown in Table 8.1. A tickly, irritating cough is often associated with upper respiratory pathologies and is the most common causes of this are upper respiratory tract infections, rhinosinusitis, oesophageal reflux and laryngeal dysfunction syndrome. Ask about whether the cough is productive; this leads to a discussion about sputum production.

Sputum

Establish whether sputum production is a new symptom, whether it is produced most days or intermittently. Sputum produced on a daily basis will be

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<thead>
<tr>
<th>Table 8.1 A quick guide to the causes of chronic cough (more than 6 months)</th>
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<tr>
<td><strong>Asthma</strong></td>
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<td><strong>Chronic obstructive pulmonary disease</strong></td>
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<td><strong>Rhinosinusitis</strong></td>
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<td><strong>Gastro-oesophageal reflux</strong></td>
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<td><strong>Laryngeal hypersensitivity</strong></td>
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<tr>
<td><strong>Other</strong></td>
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due to a condition that is present on a daily basis, i.e. a chronic condition. The two commonest conditions that do this are COPD (chronic bronchitis – ‘a productive cough which occurs for more than 3 months of the year in each of two successive years’ (Medical Research Council (MRC, criteria)) and bronchiectasis. Get an idea of exactly how much sputum is produced by asking if they are coughing up a thimbleful or an egg-cupful or more each day. The higher volume favours bronchiectasis more than COPD, but in practice there are many patients with bronchiectasis who produce very little sputum. It is relatively unusual to find a patient with chronic bronchitis who is coughing up more than an egg-cupful a day. Lastly, the type of sputum that is coughed up is important. Enquire whether the sputum is clear or cloudy, and what colour it is. Purulent sputum is generally coloured yellow or varying shades of green, and importantly it is cloudy denoting its content of inflammatory cells and pus cells. Enquire about
the sputum colour and purulence when the patient is well compared with what it is like when they are poorly with other symptoms. Clear, almost colourless, or white sputum may be a normal phenomenon in small amounts and is the sort of sputum that is produced by patients with chronic bronchitis when they do not have an infective exacerbation. Sputum that becomes yellow and cloudy in relatively small volumes would be consistent with an infective exacerbation of chronic bronchitis or COPD. Some organisms are associated with particular features of the sputum (Table 8.2). Pseudomonas aeruginosa produces brown and green pigments called pyocyanins. When patients with bronchiectasis are colonized with Pseudomonas their lung function may deteriorate much more rapidly.

### Haemoptysis

Haemoptysis is an alarming symptom and if not mentioned in the initial history it is very important that you enquire about this as patients may choose to avoid mentioning it. When a patient says that they have haemoptysis, you need to ask about whether this is in the form of small clots or little lines in the sputum, and you will know by then about the context of the haemoptysis. Larger volumes of bright red blood that persist for many days are more often associated with malignant lesions in the chest, but most studies that have looked at symptoms have shown that any haemoptysis may indicate a diagnosis of malignancy and therefore it should always be treated seriously.

Whether one goes forward to investigate patients for suspected lung cancer will depend not only on the history that you elicit about the type of haemoptysis, but also the risk factors that the patient has for developing lung cancer. The two most important risk factors are:

- the age of the patient
- whether they have ever been a smoker.

### Table 8.2 Characteristics of sputum production in relation to diagnosis

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Likely diagnosis</th>
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<tr>
<td>Acute onset, purulent sputum, clearing after 1–3 weeks</td>
<td>Acute bronchitis</td>
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<td>Pneumonia</td>
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<tr>
<td>Regular sputum production, more than a half egg-cupful, varying in purulence</td>
<td>Bronchiectasis</td>
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<td></td>
<td>Occasionally chronic bronchitis</td>
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<tr>
<td>‘Chronic productive cough for more than 3 months in each of 2 consecutive years...’</td>
<td>Medical Research Council criteria for definition of chronic bronchitis</td>
</tr>
<tr>
<td>Clear or slightly opaque sticky sputum, white yellow or green</td>
<td>Asthma</td>
</tr>
<tr>
<td>Colour of purulent sputum and organism</td>
<td>Lime green – <em>Haemophilus influenzae</em></td>
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<td></td>
<td>‘Rusty’ – <em>Streptococcus pneumoniae</em></td>
</tr>
<tr>
<td></td>
<td>Dark green – <em>Pseudomonas aeruginosa</em></td>
</tr>
<tr>
<td>Foul smell and taste</td>
<td>Chronic pulmonary sepsis with cavities in the lung</td>
</tr>
<tr>
<td></td>
<td>Infection from rotting teeth and associated gum disease</td>
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</tbody>
</table>

**IMPORTANT**

Current recommendations indicate that urgent referral to a hospital clinic should be made when patients have haemoptysis, are over the age of 40, and are current or ex-smokers. However, a young patient who has a small amount of streak (lines in sputum) haemoptysis in the context of an upper respiratory tract infection usually will not require referral. Larger volumes of haemoptysis normally have a cause and even young patients will require referral.

### Snoring and sleep disturbance

These are common symptoms of sleep apnoea syndrome. Snoring is a sign of upper airway obstruction and while it is present in 25 per cent of the normal population, in patients who are overweight the sever-
ity of snoring can be much worse. These patients will often give a history of snoring being audible outside the bedroom or even downstairs (and occasionally outside the house!). These patients also often give a history of excessive daytime sleepiness and will be unable to stay awake at certain times despite wishing to. We now include these symptoms in the respiratory history as they are often not included elsewhere. Sleep apnoea syndrome is a common condition, being present in about 5 per cent of middle-aged men, and is responsible for a good deal of morbidity.

**Past medical history**

In the past medical history always enquire about childhood infections including pneumonia, previous thoracic operations (it is surprising what patients may forget), and any history of tuberculosis. Childhood infections and pneumonia may give a clue as to the cause of bronchiectasis, or may indeed signify the fact that lungs were abnormal from a very early age, which may point to a congenital cause of chronic lung disease. A history of frequent childhood chest infections, or just being chesty as a child, may add supportive evidence towards a diagnosis of asthma if the rest of the history fits with that.

**Family history**

In the family history, it is again important to enquire about tuberculosis. Ask whether any of the family members have had tuberculosis and in particular whether there was any contact with them. Patients will often volunteer that they were screened after contact with tuberculosis and may even tell you about whether they had a Bacille Calmette Guérin (BCG) vaccination or not. Usually patients will have had a Mantoux test or Heaf test to look for any pre-existing immunity to tuberculosis and they will often remember that they did not need a vaccination after this test whereas other peers at the time did.

Note any family history of bronchiectasis, but if there is cystic fibrosis in the family, patients will usually be well aware of this. A strong family history of lung cancer in an elderly patient is important not only because the risk of lung cancer in the individual is increased slightly, but also as they will be very much aware of what a horrible illness lung cancer is and this will give you some insight into their understanding of the condition.

**Social history**

Record an accurate smoking history (as described on p. 7). For ex-smokers, record the time that they have stopped smoking as the chances of smoking-related illness diminish somewhat after cessation of smoking. Enquire about passive smoking as this is known to at least double the risk of lung cancer and ischaemic heart disease.

In the social history, enquire about any relevant exposures. The most important is exposure to asbestos, which overlaps with the occupational history (see below), and also enquire about anything that may cause hypersensitivity pneumonia (extrinsic allergic alveolitis). This includes a variety of moulds (mouldy hay, certain moulds growing on hard woods, and occasionally even in the house). Enquire about exposure to birds and whether any symptoms are related to cleaning the cage. Hypersensitivity pneumonia (Fig. 8.1) results in a type III immune response with symptoms appearing about 6 hours after exposure and continuing for 3 days. Symptoms include shortness of breath and flu-like symptoms.

In the more general social history, try to get an idea of the limitation that the illness is producing in

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**Figure 8.1** High resolution computed tomography (CT) scan of the thorax showing multiple small nodules and patches of dark and lighter lung in the distribution of the secondary pulmonary lobule. The diagnosis was bird fancier’s lung, a form of hypersensitivity pneumonitis.
the patient’s activity and an idea of the level of support that is around in the home. When patients are developing an illness that may result in them becoming dependent, it is important to enquire about how local their children are and about whom they regard as helping them most in the home environment.

**Occupational history**

In the occupational history, record occupations that are known to relate to respiratory disease. Thus electricians, plumbers, power station workers, etc., will have some risk of being exposed to asbestos. These occupations have a markedly raised standardized mortality ratio for mesothelioma and asbestosis. (The standardized mortality ratio is the ratio of observed deaths to expected deaths in a population.) Lesser exposure to asbestos may have occurred in garage mechanics when cleaning out brake-linings (that used to be asbestos lined) and even in teachers who used to conduct science lessons when asbestos sheets were used to insulate desks against the Bunsen burners.

The detail of the occupational history will depend on whether you are considering an occupation-related condition, but it would be important to ask about whether symptoms are worse at work and better away from work if you are considering occupational asthma. There are many agents that have been identified as sensitizers and which may induce asthma, and those present in glues and paints are most prevalent.

**SYSTEMS ENQUIRY**

Respiratory illness may produce symptoms in other systems. Patients with cystic fibrosis may have symptoms of malabsorption which will be revealed on direct questioning about the characteristics of the stool. If you are suspecting collagen vascular disorders, ask about arthropathy or skin rashes and whether the patient has ever had iritis. Occasionally patients will develop blackouts because of hypoxic episodes after a bout of coughing (cough syncope). Remember also that many patients (especially the elderly) with respiratory illness will have co-existent illness in other systems and it is essential that the illnesses are managed together if you are to have any success in alleviating symptoms. Cardiovascular and respiratory illnesses are responsible for the vast majority of deaths in elderly patients and they commonly co-exist.

**GENERAL OBSERVATIONS**

Begin by introducing yourself. Next look from the end of the bed and take in all of the patient’s surroundings, including whether or not they have oxygen, a nebulizer, a sputum pot, and many other observations which may have already been recorded, such as are present on an observation chart. Look closely at the patient and observe whether there are any signs of respiratory distress. These signs include cough, wheeze or stridor, and any signs of laboured breathing. At this point count the respiratory rate and try to characterize any abnormalities noted. Look to see whether the breathing is shallow.

**CLINICAL PEARL**

Rapport is important because it ensures that the patient is cooperative and therefore improves the physical examination and also of course puts the patient at ease in what are often stressful circumstances for them.

Note any abnormality of the voice and look generally to see whether the patient is anaemic, cyanosed or plethoric (as may occur in polycythaemia). Fetor (unpleasant breath) may indicate an anaerobic infection of the lung.

**THE HANDS**

Ask the patient to put their hands out in front of you and cock the wrists back, showing them how to do...
the respiratory system

this yourself, and make sure they put a lot of effort into trying to extend the wrist. A proper examination for a flapping tremor would require about a minute of this, but usually if a flap is going to occur it does so in the first 10 seconds.

Next, scrutinize the hands themselves and look for clubbing of the fingers (Fig. 8.2). The first sign of clubbing is loss of the angle between the nail bed and the nail. It is important that you get an idea of the look of normal nails so as to recognize early clubbing of the fingers. The angle of the nail to the nail bed is lost because of increased swelling beneath the nail. This leads on to the development of fluctuation of the nail bed. To elicit this, you need to fix the finger while wobbling the nail bed from side to side. This is done by placing the middle fingers on the middle phalanx of the finger and the thumbs on the proximal interphalangeal joint from beneath, then using the index fingers to test for fluctuation of the nail bed by wobbling from side to side (Fig. 8.3). It is important that you practise this on normal nails to get an idea of the normal range. Later, in more advanced swelling, there is increased curvature of the nails in short and long axes.

Respiratory causes of finger clubbing

- Bronchial carcinoma (non-small cell)
- Intrathoracic suppuration
  - Bronchiectasis
  - Empyema
  - Cystic fibrosis
  - Pulmonary abscess
- Fibrosing alveolitis (usual interstitial pneumonia).

Rare causes include tuberculosis, sarcoidosis, pleural mesothelioma, pleural fibroma, lipoid pneumonia,
pulmonary artery sarcoma, pulmonary metastases, Castleman's disease, pulmonary lymphoma, idiopathic pulmonary haemosiderosis.

**CLINICAL PEARL**

When checking for clubbing, ask the patient to hold the distal phalanx of one finger ‘back to back’ against the distal phalanx of the same finger on the opposite hand, such that the two fingernails are touching. Normally there is a small ‘window’ separating the two nail beds – loss of this ‘window’ indicates clubbing. This is known as Schamroth’s test.

Occasionally gross clubbing is associated with painful wrists and lower legs. Radiographs may show periosteal new bone formation (Fig. 8.4). This is hypertrophic pulmonary osteoarthropathy (HPOA) and is most commonly associated with non-small cell lung cancer.

The temperature and colour of the hands may give a clue as to whether the patient has features of carbon dioxide retention or central cyanosis. Hands that are abnormally blue but warm indicate that the patient is centrally cyanosed and this can be confirmed by looking centrally at the tongue (Fig. 8.5). Hands that are cool and blue may either indicate that there is peripheral cyanosis or combined central and peripheral cyanosis. Warm, well-perfused hands along with a flapping tremor indicate carbon dioxide retention.

Look for other signs of systemic disease, for example joint abnormalities may suggest a diagnosis of rheumatoid arthritis as a cause of bronchiectasis or telangiectasis a diagnosis of systemic sclerosis (Fig. 8.6).
Head and neck

Next, begin a closer inspection of the head and neck. Look for any evidence of distended veins in the neck before doing a formal examination of the jugular venous pressure. Also look at the upper thorax for any distended veins that may indicate superior vena cava obstruction. This condition occurs when there is lymphadenopathy within the mediastinum that is occluding the superior vena cava. This produces fixed elevation of the jugular venous pressure and distended external jugular veins as well as evidence of collateral venous return on the upper thorax. The face becomes a dusky grey colour in severe cases and there is some swelling of the face. This may include some periorbital (Fig. 8.7) and conjunctival oedema (chemosis). Rarely there is oedema of the hands and forearms.

Look for central cyanosis by asking the patient to put out their tongue and raise it upwards. Minor degrees of central cyanosis are difficult to detect and you need to have good light. Peripheral cyanosis is detected when approximately 1.5 g/dL of deoxyhaemoglobin is present in the blood. Thus, the chance of detecting cyanosis is greater if the haemoglobin concentration is higher. Cyanosis is usually detected when the oxygen saturation is around 90 per cent. This means that 10 per cent of the haemoglobin in the blood is desaturated and if the haemoglobin is 15 g/dL, then there is 1.5 g/dL of deoxyhaemoglobin. If the patient is polycythaemic with a haemoglobin of 20 g/dL, then central cyanosis would be detected at higher levels of saturation (approximately 93 per cent in this case). The converse is that when people are anaemic it will be much more difficult to detect significant hypoxia. Take, for example, a patient with a haemoglobin of only 5 g/dL who has an oxygen saturation of 80 per cent and still only has 1.0 g/dL of deoxyhaemoglobin. This demonstrates why, as part of the routine respiratory examination, an oxygen saturation monitor should be placed on the patient as soon as possible. It is also a very good way of learning how good you are at detecting cyanosis!

Look for signs of anaemia by inspecting the conjunctivae. Anaemia is usually detected when the haemoglobin has fallen to around 8 g/dL. While looking for anaemia, take a general look at the eyes. Look to see if there is any partial ptosis (indicative of Horner’s syndrome). Look at the eyes in general for any signs of previous iritis (a manifestation of sarcoidosis and rarely tuberculosis) and note any evidence of chemosis. Patients with severe and chronic hypocapnia may develop papilloedema so it is important to look in the fundi, especially if the patient is complaining of headaches.

Begin a systematic examination of the neck feeling for supraclavicular and cervical lymph nodes. For this you need to know the position of the lymph nodes and get an idea of what a normal neck feels like. If you find a lymph node, note:

- its size and position
- whether it is fixed or mobile
- whether it is hard or rubbery.

Rubbery lymph nodes are more common in patients who have reactive lymphadenopathy to an infection or in patients with lymphoma. Hard, fixed and craggy lymph nodes are more common in patients with metastases from solid tumours such as carcinoma of the lung or gastro-oesophageal carcinoma. It is best to examine the lymph nodes from behind the patient and to use a gentle probing technique to ensure that the patient stays relaxed. Look for evidence of any thyroid enlargement and note any previous scars including any scars that may indicate a previous tracheotomy or tracheostomy which are present just beneath the thyroid cartilage.

Upper respiratory tract

Look in the mouth, checking for any evidence of swelling at the back of the mouth. In patients with
Physical examination

There is often marked oedema in this area and the posterior wall of the pharynx may be obscured. Patients who present with difficulty breathing and stridor may exhibit swelling of the lips, tongue and other tissues indicating angio-oedema. Look at the nose, asking the patient if they can breathe in through their nostrils while occluding the opposite nostril to get an idea of nasal patency. Use a pen-torch to look up the nose and check whether there is any nasal mucosal inflammation. Further examination of the nose and larynx is usually the domain of an ear, nose and throat specialist (Chapter 20).

Check the position of the trachea (Fig. 8.8). The trachea is deviated when the lungs and mediastinum are pushed over to one or other side of the thorax due to a lesion causing reduction in volume on one side, e.g. collapse of the lung, or a lesion producing expansion on one side of the lung (massive pleural effusion or tension pneumothorax) (Fig. 8.9). Examining the trachea is a real skill and does require a lot of practice. The key is to gently place the tip of your finger in the suprasternal notch and move it from side to side to get an idea of the curvature of the trachea. Once you have a firm idea of where the apex of the curvature is, then that is the centre point of the trachea. You then look to see its relationship to the insertion points of the sternal heads of the sternocleidomastoid muscle.

Once you have perfected the technique you will note that in a proportion of patients, the trachea is very slightly deviated to the right (a matter of 1–2 mm). Remember that it is important to detect deviation just above the very lowest point of the trachea that you can feel in the sternal notch. If you go any higher you will miss tracheal deviation. You also need to notice the distance between the sternal notch and the thyroid cartilage. This will give you an idea whether there is shortening of the palpable trachea, which happens in COPD due to hyperexpansion of the chest. The dist-

Figure 8.8 Checking the position of the trachea. From: Gray D, Toghill P. (eds), An introduction to the symptoms and signs of clinical medicine, with permission. © 2001 London: Hodder Arnold.

Figure 8.9 Movement of the mediastinum (trachea and heart) as the result of various pathologies. The trachea may remain central despite collapse/effusion if fixed by mediastinal cancer. From Gray D, Toghill P (eds), An introduction to the symptoms and signs of clinical medicine, with permission. © 2001 London: Hodder Arnold.
tance should be around 2 cm. You may also note quite a pronounced downward movement of the trachea on inspiration (‘tracheal tug’). Note any thyroid enlargement that may produce deviation of the trachea.

The chest

Skin
Take note of any abnormality of the skin, both on the chest wall and more widely. Although rare, skin abnormalities are often diagnostic. They include evidence of metastatic tumour nodules, manifestations of sarcoidosis that might include erythema nodosum, cutaneous sarcoid, and any systemic features of collagen vascular disorders such as the rash of lupus erythematosus, livedo reticularis, and features of rheumatoid arthritis. If there is unilateral chest pain, then look for any herpetic vesicles that might indicate herpes zoster, or any depigmentated scars in the distribution of a dermatome that may indicate previous herpes zoster.

Abnormalities of chest shape
To observe the chest for any abnormalities of shape you will need full exposure of the thorax. The commonest abnormality of shape occurs in patients with severe COPD, where the term applied is ‘barrel-chested’. This indicates that the lateral and anteroposterior (AP) diameters approximate. It is important, therefore, to look for this and, if you are unsure, to place your hands on either side of the thorax in the lateral plane and then move them in the anteroposterior (AP) plane and see whether there is any evidence of an increase in AP diameter. Chest deformity in COPD is not a reliable measure of functional deficit, as it may not present in patients with moderate disease.

In normal subjects, the ratio between the AP diameter and lateral diameter is 5:7 and may be as low as 1:2 in normal subjects. Pigeon chest deformity (pectus carinatum) is present when there is a localized prominence of the sternum and costal cartilages with indrawing of the ribs producing Harrison’s sulci (symmetrical horizontal grooves above the costal margins). The costal margins themselves may be everted. Pigeon chest deformity is often a result of early respiratory disease producing increased respiratory effort that distorts the development of the chest when it is in a relatively pliable state (Fig. 8.10). Deformity also may be caused by rickets. Funnel chest deformity (pectus excavatum, Fig. 8.11) is present when there is a localized depression of the lower end of the sternum with the attached costal cartilages. It usually produces no respiratory defect but can cause what appears to be displacement of the heart on the chest X-ray, and in very severe abnormalities can cause some compression of the heart between the sternum and the vertebral bodies. On examination the apex beat may be
displaced and lung function may show a reduced total lung capacity.

Look for evidence of thoracic operations (see also Box 8.3). In older patients, there may be evidence of thoracoplasty where the chest has been surgically collapsed to compress the lung beneath, which often results in a reduction in ventilatory capacity and can lead to respiratory failure in later life. Look for any evidence of curvature of the spine, in both the AP and lateral planes. This would indicate kyphoscoliosis (Fig. 8.12). This can not only produce a hunchback deformity but also the twisting of the spine may produce some profound effect on pulmonary function by reducing lung capacity and increasing the work of breathing. Severe kyphoscoliosis may result in early respiratory failure presenting as hypercapnia and hypoxia. Note any protrusion of the ribs on one side of the body – a common feature of kyphoscoliosis and indicative of a more severe defect (Fig. 8.12b).

**Depth and regularity of breathing**

You will have already counted the respiratory rate, the normal being around 14 breaths per minute. Also note the depth and regularity of breathing.

The depth of breathing is increased in states producing metabolic acidosis such as diabetic ketoacidosis or uraemia, and is decreased in patients with type 2 respiratory failure. Periodic, or Cheyne–Stokes, breathing is characterized by a cyclical variation in the depth of respiration with the depth slowly

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**BOX 8.3 LESIONS OF THE CHEST WALL ON INITIAL OBSERVATION**

- Cutaneous lesions, e.g. bruises, scars, sinuses, nodules (e.g. sarcoid), skin eruptions.
- Subcutaneous lesions, e.g. metastatic tumour nodules, lipomas, inflammatory swellings.
- Subcutaneous emphysema (air in the subcutaneous tissues causing diffuse swelling of the chest wall and neck, and recognized by palpating the area for a crackling sensation). Subcutaneous emphysema is usually a result of pneumothorax or the treatment of pneumothorax with intercostal chest drainage. Occasionally it can be due to rupture of alveoli into the mediastinum resulting in mediastinal emphysema. In this circumstance the heart sounds may change considerably reflecting air in the pericardium.
- Abnormal blood vessels, e.g. superior vena caval obstruction.
- Bony prominences, e.g. sternum, ribs, scapula, costochondral junctions.
- Axillary lymphadenopathy.
- Breast lesions (Chapter 16).
- Localized areas of tenderness that may result from tumour invasion or fractures.

![Figure 8.12](https://via.placeholder.com/150)

Figure 8.12 Kyphosis (a) and kyphoscoliosis (b). Note the prominence of the ribs on the right of the chest in (b). Such deformities can produce abnormalities of respiratory function including respiratory failure.
the respiratory system

...decreasing until there is a period of apnoea followed by a sudden increase in the depth of breathing. This occurs in a variety of neurological conditions, especially those involving the medulla oblongata, and occasionally in cardiac failure.

Hyperventilation (see Box 8.4) may occasionally occur in patients with severe brain damage caused by trauma, haemorrhage or infarction. Irregular, gasping or sighing respiration is characteristic of patients who are hyperventilating for non-organic causes. This may be associated with symptoms related to a drop in ionized calcium in the bloodstream due to a reduction in the partial pressure of carbon dioxide. There may be a light-headed feeling and tingling in the fingers and toes. However, it should be noted that hyperventilation is often a co-existing factor in organic disease and it is here that a calm manner in the doctor can improve the symptoms of breathlessness markedly.

Mode of breathing
Note the mode of breathing. In healthy females, more use is made of the intercostal muscles in passive breathing and therefore they appear to be breathing more with their ‘thorax’ rather than their ‘abdomen’. Males use their diaphragms proportionally more and therefore the respiratory movements are mainly abdominal as the diaphragm descends.

Patients with respiratory distress use their accessory muscles for respiration, which include the sternocleidomastoid and intercostal muscles. Patients with severe airflow obstruction will sit forwards, often holding onto something to brace the thorax and improve the mechanical function of the diaphragm and chest wall. This might be accompanied by pursed lip breathing, which works by increasing the air pressure within the airways and preventing them collapsing as the patient exhales.

Note any indrawing of the suprasternal and supraclavicular fossae, intercostal spaces and epigastrium with inspiration. This is a further indicator of respiratory distress. During exhalation you may see some contraction of the abdominal muscles and latissimus dorsi. Normally there is no need for this as the elastic recoil of the lung is adequate to achieve exhalation. However, in airflow obstruction as a result of emphysema there is reduced elastic recoil pressure as well as some airflow obstruction, and hence more active exhalation is required.

Chest wall expansion
Measuring of the expansion of the chest wall is important, especially in examinations as it will give you an immediate clue as to which side the abnormality is on. Even conditions that produce hyperinflation on one side of the chest (pneumothorax) will also produce reduction in expansion on the same side (hyperinflation indicates that the lung is larger than it should be in a static sense, whereas expansion refers to the differences in lung volumes between inhalation and exhalation). Chest expansion in normal individuals varies from 2 cm to more than 5 cm. The majority of the chest wall expansion occurs at the lower chest anteriorly. The ribcage swings upwards and outwards on inspiration; therefore this is the point where most of the expansion will be detected.

The measurement of expansion is often done badly and requires some practice. You need to imagine that your hands are separated from your body and merely glued onto the chest very firmly, but not so firmly that you restrict chest wall expansion. The thumbs project horizontally from your hands and

**Box 8.4 THE SENSATION OF BREATHLESSNESS**
Almost every condition that produces breathlessness can be explained on the basis of one or more of the following three components:

- **Increased work of breathing**: Many patients with airflow obstruction (asthma, COPD) or patients who have stiffer lungs that are therefore less compliant (pulmonary oedema, pulmonary fibrosis) will have an increased work of breathing. This leads directly to a sensation of breathlessness.

- **Chest wall restriction**: Anything that restricts expansion of the chest wall will lead to a sensation of breathlessness.

- **Hyperventilation**: Increased rate and depth of breathing activate the pulmonary stretch receptors and thereby cause a sensation of breathlessness.
almost touch. In practice, your elbows need to be at the sides of your body (not protruding out horizontally). Then, observe the movement of your thumbs while the patient is breathing normally. With good technique you will often see a reduction in expansion on the affected side at this time. Ask the patient to take a breath in and look to see what happens (Fig. 8.13).

In a patient with severe COPD, do not be dismayed if you notice that after a small initial expansion your thumbs then cross over in the midline. This can happen because their diaphragm is so flat that after a little initial chest expansion, they then pull horizontally resulting in inward movement of the lower thorax. This shows you have the correct technique. Placing your hands on the middle of the anterior chest and looking for upwards and outwards movement of the thumbs (towards the respective shoulders) is a very good way of looking for expansion and this can be repeated at the top of the chest just below the clavicles.

Measurement of expansion at the back of the chest is always difficult in clinical situations as you need to get the patient to sit on the end of the bed, facing away from you, and then place your hands in a similar fashion to the anterior chest. In practice it is much more informative to do this from the front of the chest, but it is often done extremely badly from the back of the chest and therefore is likely to be misleading. While palpating the chest, you should also check vocal fremitus (Box 8.5): place the sides of your hands on both sides of the chest simultaneously at the top, mid-zone and lower zone anteriorly and posteriorly, and ask the patient to say ‘one one one’.

**Percussion**

You will need to practise the technique of percussion. Place one hand firmly on the chest wall with the fingers separated and then use the middle finger of your dominant hand to tap the finger with a hammer effect (Fig. 8.14).

When percussing, your aim should be to compare equivalent sites on both sides of the chest for the degree of resonance. It is therefore important that you are clear about the surface anatomy (see below) so that you truly do compare both the sides. Anteriorly this should be done with respect to the mid-clavicular line and mid-axillary line on both sides. It is quite common to see people percussing more laterally over the nearer lung compared with the

**Box 8.5 Vocal Fremitus and Vocal Resonance**

Vocal fremitus and vocal resonance give you the same information:

- when they are increased they indicate better conduction of sound through the chest wall as occurs in consolidation
- when they are decreased they indicate reduced conduction as might occur in lung collapse, pleural effusion or pneumothorax.

With vocal resonance, because of the limits of your hearing, you will only be able to hear sounds down to approximately 20 Hz; with vocal fremitus you will be able to feel lower frequencies than that. However, an advantage of vocal resonance over vocal fremitus is that you can localize areas more effectively via the relatively small area of your stethoscope. In practice it is very quick to do vocal fremitus and then use vocal resonance to further localize an area, although it is rarely of any clinical significance. Occasionally, on palpation you may be able to feel a rhonchus or pleural rub.
Lung furthest away from them, purely because they are having to stretch a little over the patient. This can lead to inaccurate physical signs.

Dullness to percussion occurs in patients with consolidation or pulmonary collapse and a raised hemidiaphragm, stony dullness (akin to percussing on the head) in pleural effusion, and hyperresonance in patients with severe hyperinflation such as marked emphysema and pneumothorax. You will normally be able to detect some dullness to percussion over the third to fifth interspaces anteriorly, indicating cardiac dullness, and loss of this usually indicates hyperinflation.

Figure 8.14 Correct method of percussion. Note the movement at the wrist and the vertical position of the terminal phalanx of the percussing finger as it strikes the other. From: Ogilvie C, Evans CC (eds), Chamberlain’s symptoms and signs in clinical medicine (12th edition), with permission. © 1997 London: Hodder Arnold.

A knowledge of surface anatomy of the lung is always useful. Anteriorly, the lungs come down to the sixth rib, and posteriorly to the eleventh rib (Fig. 8.15). It can be difficult to decide when patients are hyperinflated and more important are the observations of change in the AP diameters and other signs of severe COPD. However, it can be very useful in patients with restrictive lung disease or patients who have bilateral pleural effusions (common in congestive cardiac failure). It is also useful to know the surface markings of the lobes so as to appreciate that the majority of the anterior chest is relevant to the upper lobe (and some middle lobe on the right) and halfway up the thorax at the back is relevant to the lower lobe. The area of dullness to percussion will generally be much smaller than the surface anatomy of any lobe due to some reduction in the size of the affected lobe and also a reduction in the size of its surface representation.

Auscultation

In clinical practice, you will find that auscultation is the most helpful part of the respiratory examination except for the respiratory rate. It is important, therefore, that you familiarise yourself with normal vesicular breath sounds. These have a rustling quality and are louder during inspiration and usually only the first third of expiration is audible. There is no gap between inspiration and expiration. This is in contrast with bronchial breath sounds where the sound is much harsher, there is a pronounced gap between

Figure 8.15 Lung margins in relation to ribs. Note that from the front of the chest the lower lobes have no significant surface reflection.
inspiration and expiration, and you can usually hear the whole of expiration (Fig. 8.16).

Normal breath sounds originate from the larynx so listening to your larynx is a good way of getting used to bronchial breath sounds. To do this you will need to attempt to breathe normally – a difficult thing to do when you are trying to listen to your own breathing. When the sound leaves the larynx it travels down the trachea and then divides when the airway divides. Some sound must be transmitted through the lung parenchyma but most travels down the airway. Eventually the sound travels along airways of different lengths and therefore becomes out of phase. Next it arrives in the respiratory bronchioles and alveoli and then gets transmitted through the chest wall to your stethoscope. The fat layer filters out much of the high frequency sound (above 4 kHz). The resulting sounds are much softer (because the sound has effectively been diluted throughout the whole of the lungs). There is no gap between inspiration and expiration (because all of the sound has become out of phase and therefore ‘filled in’ the gap. Finally, the first third of expiration is now the only part that is audible because the latter two-thirds are much quieter (you will know this from listening to your own laryngeal breath sounds; see Table 8.3).

It is important to understand this because you can then understand why breath sounds differ. For example, if you are listening to an area of consolidation and you have your stethoscope directly over the consolidation, you will hear pure bronchial breath sounds. As you move away from this, you will begin to hear a mixture of vesicular breath sounds and bronchial breath sounds so that the breath sounds will be less harsh, there will be a less pronounced gap, and you may have difficulty hearing all the expiration.

Likewise, in a moderate pleural effusion, breath sounds will be absent directly over the pleural effusion and as you move up towards the top of the effusion, become more distinct. If there is some associated collapse/consolidation at the top of the effusion you may even hear some bronchial breath sounds. As you move up further, the breath sounds will become more vesicular but you may hear a prolonged expiratory phase.

It is important also to understand that this applies to airways that are normal. In patients with COPD, who have fixed narrowing of their airways, a considerable amount of sound is generated from the airways themselves and therefore you can often hear prolonged expiratory breath sounds. In emphysema, the breath sounds will be very much reduced but you may well hear prolonged expiratory breath sounds owing to sound generated in the narrow airways.

<table>
<thead>
<tr>
<th>Table 8.3 Bronchial versus vesicular breath sounds</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vesicular</strong></td>
</tr>
<tr>
<td>Character</td>
</tr>
<tr>
<td>Expiration</td>
</tr>
<tr>
<td>Gap</td>
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</tbody>
</table>
Lastly, it is important to understand the way in which sound is transmitted through various substances. Although sound is transmitted well through fluid (you can hear someone tapping at the other end of a swimming pool when your head is under water), it is not transmitted well from an air to a fluid interface (you cannot hear someone talking to you from the side of the swimming pool when your head is under water). Thus breath sounds that come from the larynx and hit a pleural effusion are reflected and therefore you are unable to hear breath sounds from the chest wall. Consolidated lung transmits sound very well (especially high frequency sounds). This produces the harsh breath sounds that one hears in bronchial breathing. However, there has to be an airway that goes into the consolidated lung tissue so that the sound is transmitted through into the tissue. Consolidation that is associated with bronchial obstruction will not give bronchial breath sounds but instead breath sounds will be reduced or absent. Asking the patient to whisper ‘twenty-two’ demonstrates how well high-pitched breath sounds are transmitted in consolidated lung tissue. This is a phenomenon of aegophony.

The common term of ‘air entry’ is potentially misleading and does not relate to the pathophysiology. For example, over a pneumothorax the breath sounds may be very much reduced but the air entry into the underlying lung is still considerable. In severe emphysema there is still considerable air entry into the lung but the breath sounds may be virtually absent. In consolidated lung, although the breath sounds are increased or even bronchial in nature, there is very little air entry.

**Added pulmonary sounds**

There are three common added pulmonary sounds:

- **Rhonchi or wheezes**, which are continuous
- **Musical type sounds**, crepitations or crackles, which are distinct clicking sounds and discontinuous
- **Pleural sounds**, which essentially consist of a pleural rub – a leathery or creaking sound produced by the movement of the visceral pleura over the parietal pleura when the surfaces are roughened, usually by fibrinous material.

Occasionally a ‘click’ is audible synchronous with cardiac systole and is thought to be due to a pneumothorax between the two layers of pleura overlying the heart.

Wheezes can be divided into those resulting from air travelling through narrowed airways from a single airway (monophonic wheeze) or multiple airways (polyphonic wheeze). Polyphonic wheeze is a characteristic of airway narrowing due to COPD or asthma and monophonic wheezes may be a result of large airway obstruction such as occurs in bronchial carcinoma. Crepitations are divided arbitrarily into fine, medium and coarse varieties (Box 8.6).

**Box 8.6 Types of Crepitations**

- **Fine crepitations**: Very numerous individual clicks of low amplitude and high pitch.
- **Medium crepitations**: Less numerous individual clicks of lower amplitude and lower frequency.
- **Coarse crepitations**: Infrequent individual clicks (few enough to count during an inspiratory cycle) that are individually much louder (higher amplitude) and of lower frequency.

This classification fits very nicely with what we know about the pathophysiology that generates crackles. In pulmonary oedema, alveoli are collapsed due to excess water impeding the role of surfactants keeping them open during expiration. During inspiration, these highly mobile structures snap open very quickly thus producing a very high-pitched sound. They are also very small structures and very numerous hence there is very little sound coming from them (low amplitude) and very many individual clicks. Coarse crackles are commonly due to secretions moving around in airways. Here you can imagine sputum in an airway that, as the patient breathes in, moves or bubbles. There is a comparatively large amount of fluid moving and it moves relatively slowly thus producing a low-pitched sound of considerable volume (amplitude).

Pulmonary fibrosis produces fine to medium crackles depending on the pathophysiology. If there is prominent alveolitis, then numerous alveoli are affected and again these structures snap open, although because they are thickened they snap
Investigation of respiratory disease

open slightly less quickly and with a bit more force, hence the crackles are a bit louder and slightly lower pitched. When there is an established fibrosis, the alveoli are much stiffer and more difficult to snap open, thus even louder and lower pitched because of their increased inertia.

Medium crackles may also be caused by numerous secretions being present as is the case in patients with severe bronchiectasis, especially during an infective exacerbation. These might be termed ‘medium to coarse crackles’ and those resulting from pulmonary fibrosis ‘medium to fine’. Sometimes the crackles of pulmonary fibrosis are akin to the sensation one gets when pulling apart Velcro.

Lastly, decide whether crackles are fixed or not. Crackles that are fixed are those that do not change their pattern during an inspiratory cycle after a period of coughing. It is important, however, to have a good technique here because if you test different depths of inspiration after coughing you will mislead yourself, and likewise if you move your stethoscope from the position that it was in for the first inspiration prior to coughing you will again mislead yourself. So it is important to ensure that the patient takes a full breath in each time and the stethoscope does not move. This is in contrast to crackles that are as a result of sputum where these commonly change. It is very difficult to detect any change in very fine crackles such as those heard in pulmonary oedema because there are simply too many.

Once you have examined the respiratory system, it is important that you pause to think and decide what pathological process would fit the signs. It is important to be clear in your mind about the signs you feel are most prominent and therefore most important, and give these more emphasis than the ones that are perhaps a little less certain. Four common conditions and physical signs are shown in Fig. 8.17.

**INVESTIGATION OF RESPIRATORY DISEASE**

Investigations form an extension of the diagnostic process that has started with the history and physical examination, but they also serve to define the level of respiratory function and give a clue as to the likely treatment. You will usually have a good idea as to the diagnosis and initial investigations are done to confirm the diagnosis or to detect any co-existing...
abnormalities. For example, you may have a history of new-onset haemoptysis in a heavy smoker and on examination a monophonic wheeze is audible. The strong suspicion of bronchial carcinoma is confirmed by a chest X-ray that shows an enlarged right hilum. The rest of the investigations then confirm the diagnosis (obtaining samples for histological examination by bronchoscopy), stage the cancer (computed tomography (CT) scan) and measure fitness for treatment (lung function tests etc.).

As well as targeted investigations, generic tests are very useful.

- The full blood count will detect anaemia, and a differential white cell count may show an increased eosinophil count in asthma and other allergic diseases.
- The lymphocyte count may be depressed in some collagen vascular disorders and sarcoidosis.

Also check the renal, liver and bone profiles.

- Renal function would be important if the patient is to undergo any further tests, especially those relying on a contrast injection such as CT scanning and positron emission tomography (PET) as patients with abnormal renal function may have a deterioration following contrast injection.
- The liver and bone profiles are important mainly in patients who are suspected of having lung cancer as these organs may be affected as a result of metastases, or hypercalcaemia may be present due to ectopic parathyroid hormone (PTH) secretion.

In patients who may be undergoing biopsies, it is important to check the clotting time. The sequence of investigations may vary depending on the prospective diagnosis.

**COMMON DIAGNOSES**

**Asthma and COPD**

These diagnoses are commonly used incorrectly, and interchangeably, in the context of airflow obstruction and this is usually due to the lack of a systematic approach. Table 8.4 shows what to look for when deciding whether a patient has asthma or COPD. Sometimes the differentiation between the two remains difficult and in these circumstances it is best to manage the patient as if they have reversible airflow obstruction as this is the treatment most likely to produce benefit – inhaled or oral steroids and bronchodilators.

Investigation of the peak flow can help in distinguishing the two conditions – marked diurnal variation is much more common in asthma than COPD. A forced expiratory volume in 1 second (FEV1) that increases by more than 15 per cent or 200 mL after a bronchodilator is by definition significant reversibility and much more common in asthma. Similarly, asthma can be defined by inhalation of progressively stronger concentrations of irritants such as methacholine or histamine. An abnormally vigorous response to relatively low concentrations is used to define bronchial hyper-reactivity. This is a much more common finding in asthma than in COPD.

In severe airflow obstruction, a patient may be in danger of going into respiratory failure and so in patients with asthma look for their ability to speak without stopping – an asthmatic person who is unable to speak is in trouble. Measuring the respiratory rate is important as this again suggests the severity of the condition, and record the pulse rate – the greater the pulse rate, the more severe the compromise. COPD patients may be cyanosed in their stable state but it would be unusual for a patient with very good functionality beforehand (who can walk up hills, etc.) to be cyanosed on presentation in an admissions unit. It is important, therefore, to correlate the clinical history with the respiratory examination when assessing severity in COPD. Patients with asthma should not be cyanosed on examination and if they are, this is usually a sign of a very severe acute asthma attack and requires immediate expert intervention with the use of high percentage oxygen, bronchodilators, steroids, and management in a safe environment in the hospital.

**Pneumonia**

Pneumonia is broadly divided into community-acquired pneumonia (CAP) and hospital-acquired pneumonia (HAP). The clinical features are the same – fever, cough, sputum, dyspnoea, pleuritic chest pain and systemic symptoms that may include malaise and confusion. On examination there is a
raised respiratory rate and tachycardia. Signs of consolidation may be present. The commonest finding on auscultation is of localized crackles that are usually coarse to medium. The patient is usually pyrexial but not always. Occasionally elderly patients may present with confusion and immobility (‘off legs’). Apart from a rapid respiratory rate there may be no other specific signs. A chest X-ray is most important for diagnosis. Symptoms and signs are used to assess the severity of CAP (Table 8.5).

Hospital-acquired pneumonia (see Box 8.7) is defined as features of pneumonia set out above with the onset at least 72 hours after a hospital admission. Two further forms of pneumonia exist (ventilator-
The respiratory system

Table 8.5 Severity markers in community-acquired pneumonia

<table>
<thead>
<tr>
<th>Clinical feature</th>
<th>Indicator of severity</th>
</tr>
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<tbody>
<tr>
<td>Confusion (*C)</td>
<td>Mental test score ≤8/10 or new disorientation in person, place or time</td>
</tr>
<tr>
<td>Urea (*U)</td>
<td>&gt;7 mmol/L</td>
</tr>
<tr>
<td>Respiratory rate (*R)</td>
<td>≥30 per minute</td>
</tr>
<tr>
<td>Blood pressure (*B)</td>
<td>Systolic &lt;90 mmHg or diastolic ≤60 mmHg</td>
</tr>
<tr>
<td>Age (*65)</td>
<td>≥65 years</td>
</tr>
<tr>
<td>Co-morbidity</td>
<td>COPD, cardiac disease, diabetes, stroke</td>
</tr>
<tr>
<td>Hypoxaemia</td>
<td>PaO₂ &lt;8kPa</td>
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<tr>
<td>Albumin</td>
<td>&lt;35 g/L</td>
</tr>
<tr>
<td>White cell count</td>
<td>&lt;4 or &gt;20 x 10⁹/L</td>
</tr>
<tr>
<td>Radiology</td>
<td>Bilateral or multilobe involvement</td>
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<tr>
<td>Microbiology</td>
<td>Positive blood culture</td>
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Items marked * are components of the CURB-65 risk score: 1 point is scored for each item present, and a score ≥3 indicates severe pneumonia. CURB-65 score reproduced from Thorax, Lim WS, van der Eerden MM, Laing R, 58: 377–82 © 2003 with permission from BMJ Publishing Group Ltd.

acquired pneumonia and aspiration pneumonia). These conditions arise in different contexts and have the same examination findings.

Lung cancer and mesothelioma

Lung cancer is broadly divided into non-small cell and small cell lung cancer. The clinical features are similar but small cell lung cancer, being of neuroendocrine origin, is more commonly associated with paraneoplastic syndromes. Table 8.6 summarizes the symptoms and signs that may be present. It should be noticed that some patients have remarkably few signs. The commonest presenting symptom is cough, followed by persistent ‘chest infection’ and haemoptysis.

Horner’s syndrome (due to damage to the sympathetic chain due to an apical or Pancoast’s tumour) is important to look for as it can be missed. It is easily remembered by its four features, all of which get smaller:

- myosis (smaller pupil on the affected side)
- ptosis (smaller palpebral fissure). Remember that the ptosis is due to autonomic paralysis – the sympathetic supply to the superior tarsal muscle. It is not, as is commonly reported in error, due to paralysis of the levator palpebrae superioris
- enophthalmos, where the eye is more sunken into the socket (and effectively smaller)
- anhydrosis – where the sweating becomes ‘smaller’. The best way to detect this is to run the back of your index finger gently over the person’s forehead above the eye. Your finger will slip much more easily over the dryer skin than on the unaffected side.

Horner’s syndrome may be caused by Pancoast’s tumour and therefore it is important also to look for any evidence of weakness of the small muscles of the hand and any other motor loss, and also to ask about shoulder pain. About 10 per cent of patients with lung cancer present with metastatic disease, this being more common in non-small cell lung cancer. This may lead to cervical or supraclavicular lymphadenopathy and an enlarged liver, symptoms of bone pain or pathological fracture, evidence of neurological deficit due to cerebral metastases, or symptoms related to hypercalcaemia (confusion, generalized weakness and malaise, nausea, constipation).

Paraneoplastic syndromes (excluding cachexia, wasting and clubbing; Box 8.8) cause symptoms in about 5 per cent of patients with small cell lung cancer, and about 1 per cent in non-small cell lung cancer. Clubbing does not occur in small cell lung cancer and can be a useful distinguishing feature.

Mesothelioma presents with persistent chest wall pain or breathlessness due to the development of a
common diagnoses

In about 50 per cent of people you will find a history of asbestos exposure. Rarely, where there is invasion of the intercostal space, it is possible to palpate filling-in of the space with hard tumour.

**Bronchiectasis and cystic fibrosis**

Bronchiectasis is characterized by cough productive of large volumes of sputum. When you elicit a history of productive cough, quantify it by asking if the patient is coughing a thimbleful, an egg-cupful or a cupful per day. If the patient is coughing an egg-cupful per day, this is strongly suggestive of bronchiectasis. Bronchiectasis may also produce recurrent haemoptysis and as lung function deteriorates the patient may be breathless. Occasionally there is some intermittent polyphonic wheeze but the wheeze is not usually that prominent. There may be focal areas of coarse to medium crackles that may be inspiratory or expiratory. During infections, there may be intermittent pleuritic chest pain and lethargy or malaise. The history and examination may help to suggest an underlying cause of bronchiectasis. High-resolution CT scan confirms the diagnosis (Fig. 8.18).

<table>
<thead>
<tr>
<th>Table 8.6 Tumour symptoms</th>
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<tr>
<td><strong>Symptoms due to endobronchial tumour</strong></td>
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<tr>
<td><strong>Symptoms due to other mass effect of tumour</strong></td>
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**Box 8.8 PARANEOPLASTIC SYNDROMES**

- Syndrome of inappropriate antidiuretic hormone secretion (SIADH)
- Ectopic adrenocorticotropic hormone (ACTH) secretion – features of Cushing’s syndrome but often with weight loss rather than weight gain
- HPOA. This produces painful wrists and shins and is characterized by periosteal new bone formation on X-ray (see Fig. 8.4, p. 89). It is usually associated with marked clubbing of the fingers and toes (more common in squamous cell carcinoma and adenocarcinoma)
- Cerebellar syndrome
- Limbic encephalitis
- Eaton–Lambert syndrome

**Figure 8.18** High-resolution computed tomography (CT) scan of the thorax showing multiple medium-sized cysts. The patient produced a cupful of sputum per day and had bilateral coarse crackles. The diagnosis is bronchiectasis (in this case saccular).
In cystic fibrosis, the history often goes back to childhood (although occasionally patients do present with almost no symptoms in adult life). There may be symptoms of malabsorption.

**Tuberculosis**

Pulmonary tuberculosis presents with a productive cough, haemoptysis and systemic symptoms of weight loss, night sweats and malaise. It may also produce breathlessness and chest pain. Haemoptysis is more common with cavitatory disease. The signs are non-specific and may include coarse crackles and signs of a pleural effusion or signs of consolidation. There may be cervical lymphadenopathy. Symptoms and signs may arise from the complications of tuberculosis that may include bronchiectasis and upper lobe fibrosis. Localized crackles over the affected area may be heard. Occasionally endobronchial disease produces stenosis of the airways and a monoponic wheeze is audible. Recurrent collapse of the right middle lobe may be caused by enlarged hilar lymph nodes compressing the right middle lobe bronchus. Occasionally patients will have had treatment for tuberculosis by thoracoplasty, resulting in marked chest wall deformity (see Respiratory examination, p. 87). Extrapulmonary tuberculosis may produce some specific signs (Table 8.7).

**Sarcoidosis**

Sarcoidosis may present as a mild but acute illness which is usually non-progressive (Löefgren’s syndrome). The symptoms are fever, arthralgia and painful erythema nodosum. The chest X-ray shows bilateral hilar lymphadenopathy. This occurs more commonly in Caucasians. Heerfordt’s syndrome is a similar acute illness with fever, arthralgia, and bilateral parotid enlargement. Again this condition usually responds quickly to treatment with steroids or resolves spontaneously. In other forms of sarcoidosis (and in the minority that present with Löefgren’s syndrome or Heerfordt’s syndrome) there is involvement of the lung parenchyma and airways. Bilateral hilar lymphadenopathy is present. Symptoms are of malaise and arthralgia and respiratory symptoms are usually cough or occasionally chest pain. In a minority of patients (15 per cent) there is progressive interstitial lung involvement causing dyspnoea, continued cough and chest pain. Occasionally lung disease progresses to pulmonary fibrosis. Sarcoidosis is a systemic disease and may affect the heart, central nervous system, spleen, liver and kidneys. The skin and eyes may also be affected. Classic skin rash is lupus pernio, and episodes of iritis may occasionally lead to blindness. Sarcoidosis may also cause hypercalcaemia resulting in the symptoms set out above under lung cancer.

**Interstitial lung disease**

Table 8.8 gives an overview classification of the interstitial lung diseases with particular features on history and examination. Symptoms and signs will depend on the area of the lung that is affected and any associated systemic features. Interstitial lung disease that produces prominent fibrosis will lead to fine to medium crackles that do not change on coughing. Lung diseases that produce some airway narrowing may lead to wheezes or squeaks.

### Table 8.7 Extrapulmonary tuberculosis

<table>
<thead>
<tr>
<th>Spinal tuberculosis</th>
<th>Pott’s disease of the spine with involvement of the thoracic vertebrae which may produce an angular kyphosis or ‘gibbus’. It is important to look for signs of spinal cord compression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central nervous system disease</td>
<td>This may include tuberculous meningitis, which presents with fever, headaches and altered conscious level with or without focal neurological signs. Tuberculomas may act as space-occupying lesions producing a variety of neurological signs</td>
</tr>
<tr>
<td>Pericardial disease</td>
<td>Occasionally large pericardial effusions may lead to cardiac tamponade</td>
</tr>
<tr>
<td>Renal and genitourinary tract tuberculosis</td>
<td>Rarely presents with symptoms, but when it does it may cause prostatitis and epididymitis</td>
</tr>
</tbody>
</table>
### Table 8.8 Features of interstitial lung diseases

<table>
<thead>
<tr>
<th>Interstitial lung disease</th>
<th>Common feature in the history or examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic interstitial pneumonia</td>
<td>Prominent fine to medium basal inspiratory crackles. Clubbing in 50 per cent. Long history onset over years. Cough and shortness of breath progressive</td>
</tr>
<tr>
<td>• Usual interstitial pneumonia (UIP)</td>
<td>Possibly shorter onset over months to years. Clubbing less common. Cough and shortness of breath progressive</td>
</tr>
<tr>
<td>• Non-specific interstitial pneumonia (NSIP)</td>
<td>Onset over months. Breathlessness, dry cough, fever, myalgia, weight loss (may present as persistent chest infection). Clubbing absent. Localized inspiratory crackles</td>
</tr>
<tr>
<td>• Cryptogenic organizing pneumonia (COP)</td>
<td>Occurs in smokers. Mild breathlessness and cough. Scanty crackles on examination</td>
</tr>
<tr>
<td>• Acute interstitial pneumonia (AIP)</td>
<td>Fevers, tiredness, myalgia, followed by rapid onset (over days). Widespread crackles on examination (Velcro). Mortality &gt;50 per cent</td>
</tr>
<tr>
<td>• Respiratory bronchiolitis interstitial lung disease (RBILD)</td>
<td>Occurs in smokers. Onset of breathlessness and cough over weeks to months. Cough, malaise, fever, night sweats, exposure to parasites (foreign travel)</td>
</tr>
<tr>
<td>• Desquamative interstitial pneumonia (DIP)</td>
<td>Occurs in smokers. Onset of breathlessness and cough over weeks to months. Cough, malaise, fever, night sweats, exposure to parasites (foreign travel)</td>
</tr>
<tr>
<td>• Lymphoid interstitial pneumonia (LIP)</td>
<td>Onset of cough and breathlessness over several years. Fever, weight loss, scanty crackles</td>
</tr>
<tr>
<td>Hypersensitivity pneumonitis</td>
<td>History of exposure to appropriate allergen (moulds, animal droppings, birds, etc). Symptoms acute with breathlessness, dry cough, fever, arthralgia, myalgia and headache 6 hours after exposure. Crackle</td>
</tr>
<tr>
<td>Bronchiolitis</td>
<td>Proliferative bronchiolitis may be caused by COP, hypersensitivity pneumonitis, bone marrow, heart and lung transplant, acute infections such as Mycoplasma, Legionella and influenza. Symptoms are of progressive breathlessness and dry cough. Constrictive bronchiolitis rare. Connections with connective tissue disease, particular rheumatoid arthritis. Occasionally triggered by infection – viral adenovirus, respiratory syncytial virus, influenza. Cough and progressive dyspnoea</td>
</tr>
<tr>
<td>Eosinophilic lung disease</td>
<td>Severe asthmatic symptoms</td>
</tr>
<tr>
<td>Asthma and allergic bronchopulmonary aspergillosis (ABPA)</td>
<td>Cough, malaise, rhinitis, fever, night sweats, dyspnoea, wheeze, exposure to parasites (foreign travel)</td>
</tr>
<tr>
<td>Simple pulmonary eosinophilia (Löffler's syndrome)</td>
<td>As per Löffler's syndrome, except symptoms over weeks to months, rather than days to weeks</td>
</tr>
<tr>
<td>Tropical pulmonary eosinophilia</td>
<td>Episodic episodes of cough, malaise, dyspnoea and fever</td>
</tr>
<tr>
<td>Chronic eosinophilic pneumonia</td>
<td>Similar presentation to community-acquired pneumonia</td>
</tr>
<tr>
<td>Acute eosinophilic pneumonia</td>
<td>Symptoms for weeks/months, fever, weight loss, cough, night sweats, pleuritic, Churg–Strauss syndrome, rhinitis, past history of asthma, other organ involvement including vasculitic rash</td>
</tr>
<tr>
<td>Hyper-eosinophilic symptoms</td>
<td>History of recent new drug. Cough, dyspnoea, fever – spectrum of severity</td>
</tr>
<tr>
<td>Drug-induced pulmonary eosinophilia</td>
<td></td>
</tr>
</tbody>
</table>
Pulmonary embolism

In acute pulmonary embolism symptoms depend on the size of the clot. In approximately 60 per cent of patients there is an acute onset of pleuritic chest pain with or without haemoptysis. A pleural rub may be heard on examination. The pathophysiology is pulmonary infarction with obstruction of a peripheral vessel. In 25 per cent of patients there is sudden onset of acute breathlessness. Here the clot tends to be larger and more central. With massive pulmonary embolism there may be sudden circulatory collapse resulting in hypotension, loss of consciousness, or immediate cardiac arrest. In these circumstances there will be evidence of acute right heart failure with elevation of the jugular venous pressure. There will be sinus tachycardia and hypotension with signs of peripheral vasoconstriction. The patient will be cyanosed clinically and there will be reduced oxygen saturation on pulse oximetry. Occasionally in patients with pre-existing lung or heart disease, a relatively small pulmonary embolism will produce symptoms of severe breathlessness. Pulmonary embolism can also be a cause of acute atrial fibrillation. Other examination findings might include a loud pulmonary second sound and splitting of the second heart sound with a gallop rhythm, or a low-grade fever. See Box 8.9 for risk factors of pulmonary embolism.

Pneumothorax

Pneumothorax presents with acute onset of pleuritic chest pain and/or breathlessness, which may be mild or absent in patients with prior normal lung function. The patient may feel bubbles or crackles under the skin and on examination you may feel subcutaneous emphysema. There may be tachycardia and chest signs (as discussed in ‘Physical examination’, p. 87). Hammond’s sign refers to a click on auscultation in time with the heart sounds and is usually in association with a left-sided pneumothorax only.

**Summary**

The six key symptoms of respiratory disease are:
- chest pain (that may be extended to chest sensations)
- dyspnoea
- cough
- wheeze
- sputum production
- haemoptysis.

When examining the respiratory system, assess:
- general observations
  - oxygen, a nebulizer, a sputum pot
  - observation chart
  - signs of respiratory distress
  - respiratory rate and characterize any abnormalities noted
  - any abnormality of the voice
  - anaemic, cyanosed or plethoric
- hands
  - flapping tremor
  - clubbing of the fingers
  - peripheral cyanosis
  - other signs of systemic disease
- head and neck
  - distended veins/jugular venous pressure
  - central cyanosis
  - anaemia
  - partial ptosis (indicative of Horner’s syndrome)

**Box 8.9 Risk Factors for Pulmonary Embolism**

- Recent surgery, especially major surgery
- Late pregnancy
- Malignancy, especially pelvic/abdominal and advanced cancer
- Lower limb fracture
- Reduced mobility (especially hospitalization)
- Previous proven venous thromboembolism
- There are also a variety of minor risk factors, including the contraceptive pill, long-distance air travel, thrombotic disorders, obesity, inflammatory bowel disease and nephritic disease.
- papilloedema
- supraclavicular and cervical lymph nodes
- thyroid enlargement
- scars that may indicate a previous tracheotomy or tracheostomy

- upper respiratory tract
  - look in the mouth
  - look at the nose
  - check the position of the trachea

- the chest
  - skin
  - abnormalities of chest shape
  - evidence of thoracic operations
  - depth and regularity of breathing
  - mode of breathing
  - chest wall expansion
  - vocal fremitus and vocal resonance
  - percussion
  - auscultation
  - rhonchi or wheezes
  - musical type sounds, crepitations or crackles
  - pleural rub.

FURTHER READING