CONTENTS

Preface 6

Introduction 7

Chapter I. THE RESPIRATORY SYSTEM 8

Chapter II. THE CARDIOVASCULAR SYSTEM 42

Chapter III. THE ALIMENTARY SYSTEM 82

Chapter IV. SYMPTOMS AND SIGNS IN LIVER DISEASES 108

Chapter V. INVESTIGATION OF PATIENTS
WITH GALLBLADDER AND PANCREATIC DISEASES 122

Chapter VI. INVESTIGATION OF PATIENTS
WITH DISEASES OF THE GENITOURINARY SYSTEM 128

Chapter VII. INVESTIGATION OF PATIENTS
WITH DISEASES OF ENDOCRINE SYSTEM 142

Chapter VIII. NUTRITIONAL AND INHERITED
METABOLIC DISORDERS 152

Chapter IX. INVESTIGATION OF PATIENTS
WITH DISEASES OF BLOOD. ANAEMIAS 158

Chapter X. INVESTIGATION OF PATIENTS
WITH MYELOPROLIFERATIVE DISORDERS 166

Chapter XI. DISORDERS OF BLOOD COAGULATION.
PARAPROTEINAEMIAS 171

CHAPTER 1
THE RESPIRATORY SYSTEM

History

As with any other system a comprehensive history, comprising a characterization of each system, is essential for synthesizing a diagnosis when faced with respiratory problem. A detailed **occupational**, **social** and **drug history** are of special importance in evaluating respiratory symptoms. There are six main presenting symptoms which point to an underlying respiratory disorder (Table 1).

Table 1. Respiratory symptoms

• Cough
• Sputum
• Breathlessness
• Chest pain
• Haemoptysis
• Wheeze

Cough

Cough is a protective reflex and is the result of an effort to expel any accumulated secretions or a foreign substance in the respiratory passages, by means of a release of increased intrathoracic pressure through an open glottis. As such, cough is highly specific to respiratory disorders but it has a low diagnostic sensitivity. However, it should not be dismissed as a «normal winter cough» or a «smoker's usual cough» in anyone. Patient should be asked about its onset, duration, the time of occurrence, any change in frequency and severity, and about the associated expectoration.

As the production of cough depends on an increase in intrathoracic pressure with the glottis closed and then its release through the open glottis, inflammation of the larynx gives the cough a harsh, barking quality. If one of the vocal cords is paralysed the cough loses its explosive character and becomes like a uniform low of cattle (bovine cough).

A bout of coughing may be precipitated by inhalation of cold air, dust or toxic fumes. Recurrent nocturnal cough may suggest either nasopharyngeal dripping or gastroesophageal regurgitation. It is commoner in older people and may be associated with oesophageal stricture, neurological swallowing problems and cardiac failure.

Cough originating in the upper respiratory passages (pharynx, larynx, trachea) tends to be harsh, painful and loose whereas that due to secretions in the small airways comes in paroxysms and culminates in expectoration. In bronchiectasia, cough is characteristically loose and long bouts occur in the mornings when patients bring up large quantities of purulent sputum. Persistent cough is an important feature of a variety of pulmonary disorders including cystic fibrosis, bronchiectasia, pulmonary oedema, sarcoidosis, pulmonary tuberculosis, and pulmonary fibrosis.

Sputum

Information should be obtained about its quantity (an eggcupful, teacupful, etc.), colour (white, grey, black, pink, yellow or green), viscosity (serous or tacky), taste and odour (Table 2).

Table 2. Characteristics of sputum

Sputum	Condition
Mucoid, excessive quantities	Chronic bronchitis
Mucopurulent or purulent (yellow or green)	Infection — acute or chronic bronchitis
Excessive in early mornings, or at change of posture, purulent	Bronchiectasia
Black	Cigarette or atmospheric smoke, coal-miner's sputum
Pink, frothy	Acute pulmonary oedema
Rusty	Lobar pneumonia
Blood-stained	Acute bronchitis, tuberculosis, neoplasia
Viscous with plugs	Asthmatic pulmonary eosinophilia

A change of colour from white to green or yellow suggests the onset of infections in patients with chronic bronchitis. A pink and frothy sputum associated with breathlessness is commonly encountered in pulmonary oedema.

Breathlessness

Breathlessness, or dyspnoea, may be simply a subjective uncomfortable awareness of breathing (from any cause), but in cardiorespiratory disorders it is key symptom of the cause, an index of severity, and an indicator of progression of the underlying disease. Some important respiratory causes of dyspnoea are given in Table 3.

Bearing these causes in mind, ask the patient about the onset (when he really became aware of breathlessness), its severity (how much he could do before becoming breathless), the present state (what activities cause breathlessness now), and about the associated symptoms (e.g. cough, haemoptysis, chest pain, etc.).

Table 3. Some respiratory causes of breathlessness

Site	Lesion
• Upper respiratory passages	• Pharyngeal / laryngeal / tracheal obstruction
• Major bronchi	• Chronic bronchitis, bronchiectasia
• Lung parenchyma	• Asthma, pneumonia, allergic alveolitis, sarcoidosis, fibrosis, respiratory distress syndrome, malignant disorders
• Pleura and chest wall	• Pneumothorax, pleural effusion, tumours, kyphoscoliosis, ankylosing spondylitis, neuromuscular disorders

Dyspnoea may be severe and present at rest as in pulmonary oedema and asthma; in both cases it is episodic with normal intervals in between the paroxysms. In chronic obstructive airways disease, many patients get used to slowly progressive respiratory disability and only become aware of breathlessness when disease is advanced with considerable structural damage.

Chest pain

Retrosternal pain, worse on coughing but unaffected by exercise, may be caused by acute tracheitis and by inflammation, emphysema or tumours involving the mediastinum. A pleuritic pain (a sharp pain arising in the parietal pleura or chest wall and aggravated by inspiration) is of particular importance in pointing to those respiratory disorders which involve the pleura (e.g. pneumonia, pulmonary infarction, pneumothorax, primary inflammation, infection or malignant infiltration of the pleura).

Haemoptysis

A history of haemoptysis should not be ignored without a proper clinical assessment and may require further investigations. Among the diseases producing blood-stained sputum are acute and chronic bronchitis, pulmonary oedema, mitral stenosis, pneumonia, pulmonary infarction, pulmonary tuberculosis and carcinoma. Frank haemoptysis may be seen in any of those conditions. Recurrent haemoptysis may be seen in pulmonary tuberculosis, adenoma, mitral stenosis and apical fibrosis with aspergillosis.

Wheeze

Wheeze is a high-pitched, musical noise and it can be readily demonstrated to the patient by increasing the intrathoracic pressure and then forcing the air through voluntarily narrowed upper air passages. In disease it can be caused by the high velocity of expiration through the narrowed, small airways (bronchitis, asthma).

In addition to a standard systems review, enquiries should be made about some common disorders which also involve the respiratory system. Some of these together with their pulmonary complications are given in Table 4.

Table 4. Some systemic disorders with pulmonary complications

Disease	Pulmonary complication
• Rheumatoid arthritis	• Pleural effusion, pulmonary nodules and fibrosis
• Systemic lupus erythematosus	• Pleurisy and effusion, pulmonary infarction, pulmonary hypertension
• Systemic sclerosis	• Pulmonary fibrosis, aspiration pneumonia
• Sjögren's syndrome	• Pulmonary fibrosis, pneumonia
• Ankylosing spondylitis	• Upper lobe fibrosis
• Behcet's syndrome	• Pulmonary arteritis, infarcts
• Coeliac disease	• Interstitial lung disease
• Neuromuscular disorders	• Chronic respiratory failure

Family history

Ask if any of the close relatives has had asthma, hay fever, eczema or rhinitis. A family history of these atopic conditions is found in a significant proportion of asthmatic patients. A variety of genetically determined conditions have a significant pulmonary component. For example, pulmonary fibrosis occurs in association with some autosomal recessive (cystic fibrosis, α_1-antitrypsin deficiency) and autosomal dominant disorders (Marfan's syndrome, neurofibromatosis, tuberculous sclerosis). Patients with sickle-cell disease and Ehlers-Danlos syndrome are prone to respiratory infections.

A detailed **occupational** and **environmental** history should be obtained and enquiries should be made about present and past employment (miner, stonemason, farmworker, soldier, baker, etc.), and about exposure to chemicals, inorganic and organic dusts and toxic fumes. It is important to record the degree and duration of exposure, and its temporal relationship to the onset of symptoms. Patients should be asked whether they have pets and about any other factors in the environment which they think may cause symptoms (e.g. hay, dust, feathers, pollen, etc.)

Social history including the details of home circumstances, both structural (cold, damp or dusty) and personal (stress, conflicts) should be recorded. People living downwards from an asbestos-polluted environment may develop clinical asbestosis or even mesothelioma. Patients should be asked about their past and present smoking habits, duration of smoking, and about the number of cigarettes used per day, of the quantity of tobacco. Chronic bronchitis and emphysema are uncommon among non-smokers.

A full **drug history** should be obtained including the information about those drugs that are available without a prescription (e.g. aspirin-containing drugs). Aspirin may cause asthma and a variety of non-steroid anti-inflammatory drugs may cause or worsen bronchospasm. Beta-blockers may precipitate an acute attack of asthma. Many drugs, such as nitrofurantoin and amiodarone, and cytotoxic drugs cause pulmonary fibrosis.

General inspection

On general examination, there may be clues to the underlying disease:

Cachexia may occur in malignant disease, and in severe chronic lung disease, including fibrosis, infection and emphysema. Cachexia may occur in a number of severe disorders, including chronic lung disease such as pulmonary fibrosis, tuberculosis and emphysema, malignant disease, including bronchial carcinoma, and systemic infection, especially with HIV («slim disease»). Note the obvious signs of weight loss, fever (Fig. 1), with widespread muscle and soft-tissues wasting.

Cyanosis — best seen in the lips, tongue, buccal mucosa and fingers — indicates significant desaturation of circulating haemoglobin. Cyanosis is a fundamental sign of cardiorespiratory disorders and suggests capillary oxygen desaturation of 85% or lower.

A plethoric appearance may result from polycythaemia most commonly secondary to chronic hypoxia in lung disease.

A herpetic eruption on and around the lips is sometimes seen in a patient with a respiratory infection.

Coal dust tattoos may be seen on the face, though these are more often seen on the arms, as an occupational legacy in a patient with pulmonary fibrosis.

You may see small reddish papules of sarcoid infiltration which sometimes coalesce to form a dense induration called *lupus pernio*. *Lupus vulgaris* is a cutaneous manifestation of tuberculosis. In both cases there is reddish induration of the skin but the lesion of *lupus vulgaris* has a transparent appearance and there may be associated scarring which does not occur in *lupus pernio*.

Nasal polyps frequently occur in patients with an atopic background and in those with cystic fibrosis.

Eczema is often found in conjugation with hay fever and asthma.

The oscillations of the jugular venous pulse are difficult to interpret in patients with chronic airways obstruction who generate a high intrathoracic pressure to drive the air out through the narrowed bronchi. However, static engorgement of the neck veins is an important sign of obstruction of the superior vena cava, usually caused by mediastinal malignancy.

«Nicotine» stained fingers occur in heavy smokers, and typical pigmented scars may occur in coal miners; in association with finger clubbing both signs have an ominous significance, suggesting underlying bronchial carcinoma, pulmonary fibrosis, bronchiectasia or chronic sepsis.

Finger clubbing is frequently present in a number of conditions (Table 5), especially bronchial carcino-

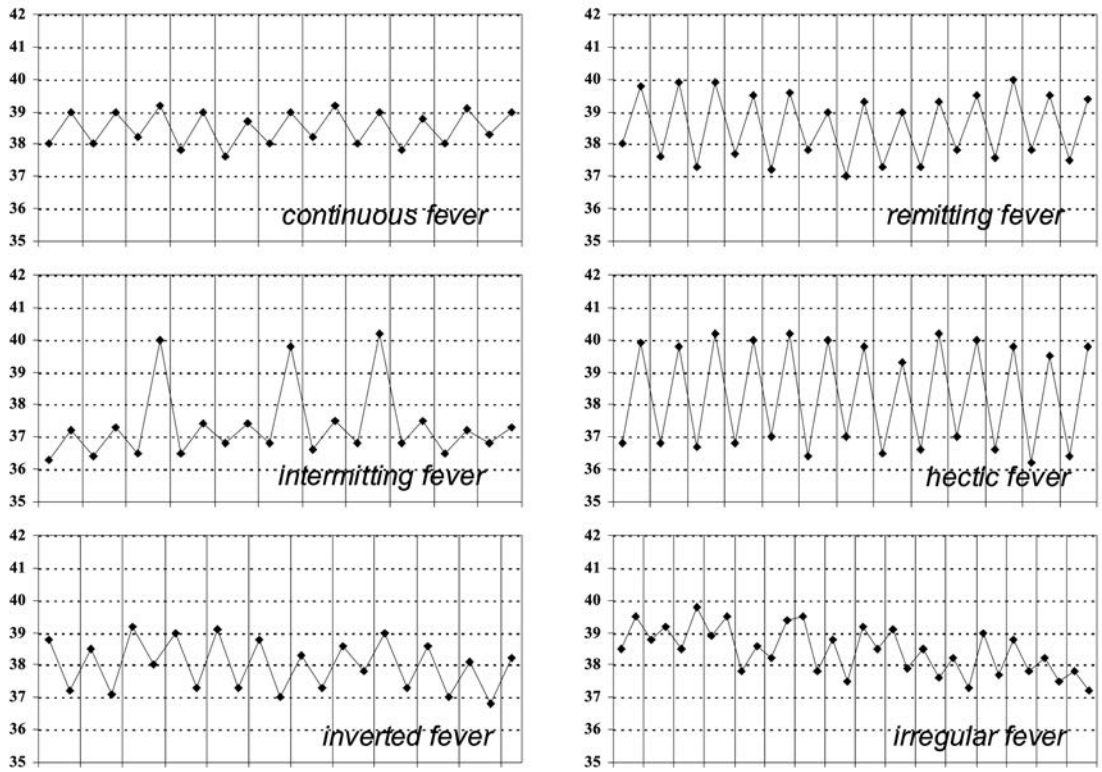

Fig. 1. Basic types of fever.

Table 5. Some causes of finger clubbing

• Hereditary	
• Pulmonary and thoracic	Bronchogenic carcinoma, metastatic carcinoma, fibrosing alveolitis, bronchiectasia, cystic fibrosis, pulmonary arteriovenous fistula, mesothelioma
• Cardiac	Congenital right-to-left shunt, infective endocarditis
• Gastrointestinal	Hepatic cirrhosis, inflammatory bowel disease

ma (occasionally with a pleural fibrinoma), and in those with chronic purulent conditions such as bronchiectasia, lung abscess and empyema (Fig. 2).

The short and stunted, excessively curved, and greenish yellow nails of yellow nail syndrome may be seen in association with a pleural effusion or pulmonary neoplasm. Because of the rarity of this syndrome, the abnormal nails are easily overlooked.

Fig. 2. Finger clubbing in a patient with bronchiectasia.

Hands should also be examined for the sweating, tremor or twitching associated with hypercapnia. In severe **hypercapnoic respiratory failure**, the patient may be confused and there may be a **flapping tremor** (inability to sustain extension at the wrist), which is often seen in hepatic failure. The survey should be completed by looking at the chest, abdomen and legs for any swellings or discoloration.

Examination of the chest

Inspection

After the general inspection of the patient, attention should be directed next to the chest and spine, looking for the shape of the ribcage and the presence of any deformities. Deformities of the ribcage may be congenital or may be caused by recurrent infections, cardiac enlargement, or deficiency states during early childhood.

The effects of vitamin D deficiency on a growing skeleton may leave a deep groove passing outwards from the xiphisternum, also known as Harrison's sulcus.

Cystic fibrosis is a common cause of recurrent respiratory infections leading to chest wall deformity.

You should bear these in mind when interpreting your findings, because a chest wall deformity may alter the normal anatomical position of intrathoracic organs (e.g. location of the apex beat).

The commonest deformities of the chest and spine are **scoliosis**, **kyphosis**, and a combination of the two, **kyphoscoliosis** (Fig. 3). **Kyphosis** results in anterior concavity of the thoracic spine and thereby leads to shortening of the chest. Kyphosis is frequently seen in elderly people with osteoporosis, **chronic obstructive airways disease**, and sometimes in younger men with **ankylosing spondylitis**.

These deformities make the ribcage unyielding and in severe cases lead to respiratory failure.

In the so-called barrel-shaped chest the anteroposterior diameter of the chest is increased and the ribs are more flatly set than usual, the sternum becomes prominent anteriorly and the manubrium extends upwards in the neck (Fig. 4).

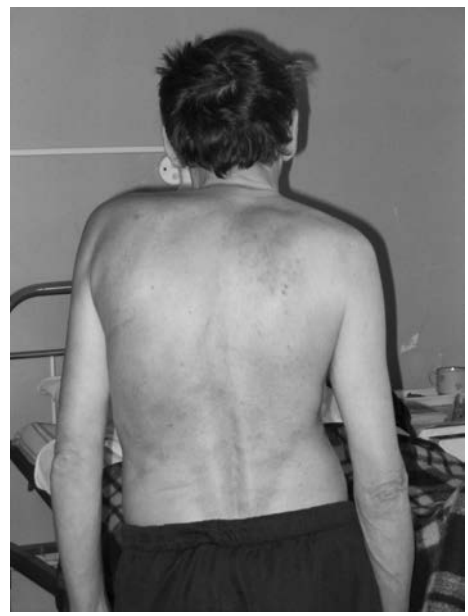

Fig. 3. Kyphoscoliotic chest.

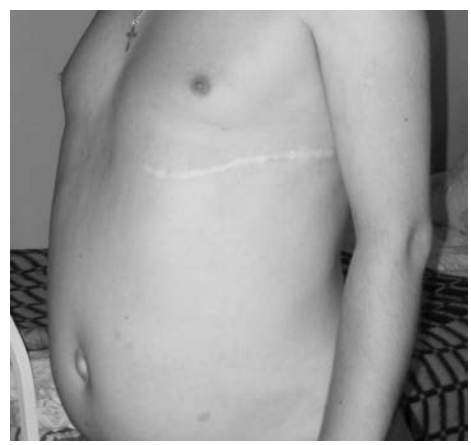

Fig. 4. Hyperinflated chest.

The chest at rest looks as it would at the end of full inspiration, and any further inspiration from this position is achieved by an upward movement of the ribcage effected by the accessory respiratory and abdominal muscles (Fig. 5).

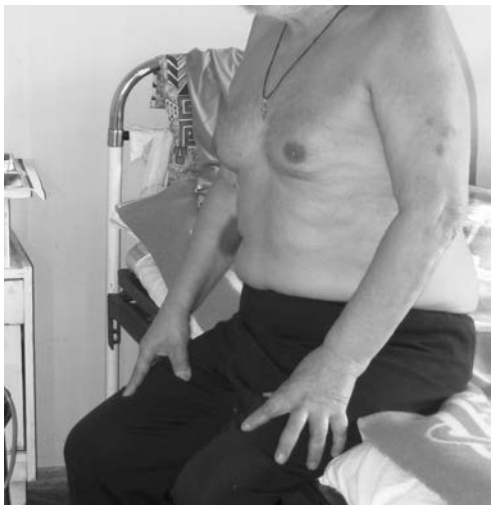

Fig. 5. Posture of the patient in severe dyspnea («forced position»).

These changes of hyperinflation are often associated with emphysema. In severe cases the patient may sit at the edge of the bed with raised shoulders in an effort to increase the intrathoracic capacity for the lungs to expand further during inspiration. A more acceptable and polite term for barrel-shaped chest is **hyperinflated chest**.

A note should be made of any scars of previous operations which may have a link with the present problem.

Thoracoplasty scars are seen in older patients who had this operation done for tuberculosis before effective chemotherapy became available. This procedure constitutes removal of some ribs and thereby allows the underlying part of the lung collapse and «rest». The trachea may be deviated to the site of the thoracoplasty and there may be bronchial breathing audible over the collapsed area.

Look at the chest wall for any skin lesions or swellings such as **gynaecomastia** (Fig. 6). Sometimes pulmonary symptoms may be related to the intrathoracic metastases of breast cancer (Fig. 7).

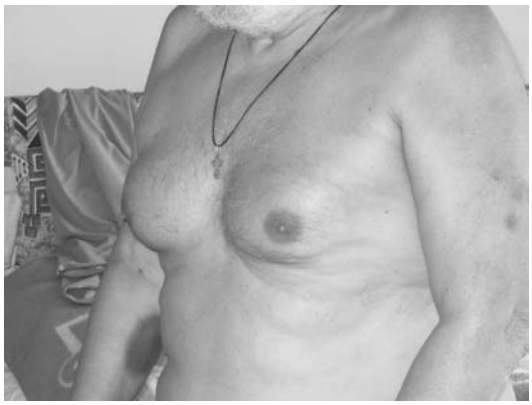

Fig. 6. Gynaecomastia.

Chest wall movements should be observed carefully for their direction (e.g. mainly outwards or upwards) and for the symmetry of the two sides. Normally, thoracic expansion is a symmetrical process. If respiratory differences are detected, even when slight, they indicate definite disease on the side of the lagging respiratory excursion. Abnormalities in thoracic expansion may be unilateral or bilateral, or may be limited to certain definite areas of the thorax. Bilateral general increase in expansion may be observed physiologically after active physical effort, particularly in persons not accustomed to such effort. Pathologically, it occurs during paroxysms of bronchial asthma which may lead to emphysema. Bilateral general decrease is usually seen in the small chests of elderly patients with atrophic emphysema. A similar decrease may occur in disease of the chest wall, such as intercostal muscle paralysis, and disease of the lungs or pleura, such as bilateral pneumonia, tuberculosis or pleurisy with effusion. The pain of diaphragmatic pleurisy and intercostal neuralgia may also cause a general diminution in respiratory excursion. Unilateral diminution or delayed expansion is tuberculosis of the lungs, but it may also result from pneumonia, pleuritis with its attendant pain, pleural effusion or pneumothorax. In chronic cases, such inequalities are generally due to local adhesions.

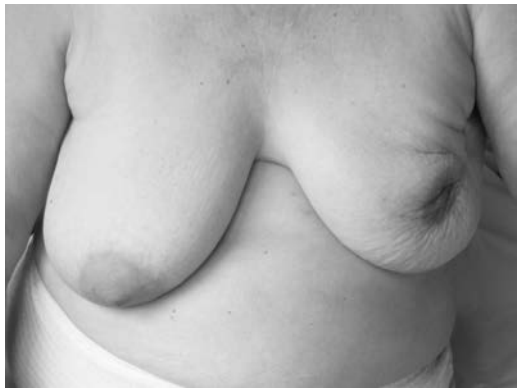

Fig. 7. Cancer of the left breast.

The lower rib spaces should be seen to bulge during inspiration, that occurs in chronic obstructive airways disease in which the diaphragms are flat and low and the high negative pressure causes intercostal recession.

Breathing

During the process of inspection you should try to listen carefully to the patient's breathing, since the information so gained will be vital in the final synthesis of the diagnosis. The bell of the stethoscope, placed in front of the patient's mouth, can be useful in listening to both phases of the respiration and any accompanying noises.

While you listen to the breathing you need to answer six questions:

1. What is the rate?
2. Is there an unusual rhythm or pattern?
3. What is mutual relationship of inspiration and expiration?
4. Is breathing audible?
5. What is the nature of the accompanying noise(s)?
6. Is the patient using accessory respiratory muscles?

The breathing rate should be counted when the patient is not conscious of it; it can be done during the earlier part of the inspection. The normal rate is between 14 and 18 breaths a minute. In opiate or barbiturate poisoning this may fall to below eight breaths a minute whereas in acute bronchopneumonia the rate may exceed 40 a minute. The relationship between inspiration and expiration should be determined. Normally, the inspiration is active and longer whereas expiration is shorter and accomplished by the passive recoil of the lungs. In small airways obstruction the expiration becomes active and prolonged, due to a greater pressure gradient from small to major airways.

The deep inspiration and shorter expiration which follows immediately gives the respiration its normal **rhythm**. **Shallow breathing** with short inspiration and expiration occurs either when breathing is restricted (e.g. obesity, pulmonary fibrosis) or is painful as in chest wall disease and pleurisy, or in anxiety states. **Kussmaul breathing** with deep inspiration and expiration typically occurs in metabolic acidosis (e.g. diabetic ketoacidosis, renal failure, methyl alcohol poisoning, etc.). **Cheyne-Stokes breathing** comprises periods of apnea alternating with a gradual resumption of respiration with increasing depth which then declines to another period of cessation of breathing. This pattern of breathing is also termed periodic or cyclical breathing and occurs in advanced cardiac and respiratory failure, narcotic drug poisoning and in cerebrovascular disease. **Pursed lip breathing** is a sign of severe small airways obstruction, as can be found in asthma and emphysema, but it also occurs occasionally in left heart failure.

It is an attempt by the patient to create an effective pressure gradient, by narrowing the outlet, to drive the air during expiration through the diseased airways.

You should ask yourself whether the breathing is audible to the unaided ears and, if so, what is the accompanying noise. Loud, featureless respiration may simply be the result of an increased rate as happens during exercise in normal subjects. **Stridor** is a harsh high-pitched musical or vibratory noise mostly during the inspiration, caused by obstruction in the pharynx, larynx or trachea. **Wheeze** is a high-pitched, musical noise caused by the high velocity of air passing through narrowed small tubes. It is usually heard during the entire or the end of expiration in asthma, emphysema and chronic bronchitis. **Inspiratory click** is a single click or series of clicking noises caused by the movement of the bolus of air moving secretions in the major tubes in acute tracheitis, tracheobronchitis and bronchiectasia. Similar noises may be heard during expiration (**expiratory clicks**) in these conditions.

Finally, you should note whether the breathing is labored and the patient is using accessory respiratory muscles. There may be indrawing of the supraclavicular fossae and flaring of the *alae nasi* during inspiration, and the patient may purse his lips during expiration.

Examination of the front of the chest

It is convenient to the patient particularly if he is unwell, if you carry out all the procedures of palpation, percussion and auscultation on the front of the chest, and then repeat these over the back. To have to ask your patient to sit up and lie down for each procedure is tedious for him and will disturb your concentration.

Palpation

In carrying out a complete clinical assessment of a patient, palpation of the chest may be conveniently undertaken for both the cardiovascular and respiratory systems. Besides, the location of the apex beat may have been altered by a respiratory disorder such as collapse of the lung or a pleural effusion.

As outlined in Table 6 there are **six palpation procedures** which are relevant to the examination of the respiratory system. These can be conveniently carried out on the front of the chest with the backrest adjusted at 45 degrees to the bed.

Table 6. Palpation procedures

1. General palpation
2. Expansion
3. Axillae
4. Trachea
5. Vocal fremitus

First, you should have a general feel for any **tenderness** or **swelling** as over costochondritis, abscess, a rib fracture, or a malignant deposit in a rib.

Second, you should study the expansion of the ribcage during inspiration. It may be done by placing the fingers over the rib spaces and approximating the thumbs in a straight line **at the end of expiration**. As the patient breathes in the thumbs will separate giving you a good idea of whether the two sides are moving equally and by how much the lungs expand at the end of the inspiration. It is a good practice to measure the expansion after maximal expiration with a tape measure and compare the result with the distance between the two thumbtips at the end of a full inspiration.

These two values will tend to approximate as you gain more experience.

Third, you should palpate in the axillae on either side to look for any glandular swellings.

Fourth, you should feel for the trachea in the suprasternal notch with your middle finger while keep-

ing the ring and index fingers on the two high points of the manubrium as your reference.

As you feel the tracheal rings with your exploring fingertip you can decide whether the trachea is centrally placed or deviated to either side. You should take this opportunity using your fingertip to feel if several rings move downwards as patient breathes in.

This **tracheal tag** is a sign of chronic airways obstruction where the hyperinflated ribcage moves upwards during inspiration, lifting your fingertip against stationary tracheal rings. In these patients the **cricomanubrium distance** is reduced. In normal subjects this distance is usually about three finger breadths.

Although the glands in the neck are best palpated by approaching the patient from behind, this opportunity should be used to feel for any swellings.

Fifth, vibrations generated by the voice, or vocal fremitus, can be felt on the surface of the chest (Fig. 8) if the intervening air passages in the lungs contain air and are not clogged up with any secretions or masses. You can test the vocal fremitus by placing palms or more sensitive ulnar border of your hand on the chest while the patient repeats «ninety nine» in a deep clear voice (Fig. 9).

The corresponding areas on the chest must be tested simultaneously by both palms in symmetrical areas. Vocal fremitus is increased through a consolidated lung (lobar pneumonia) and decreased when the corresponding bronchi are obstructed, or if there is a pleural effusion. It is useful in distinguishing consolidation from pleural effusion, both of which produce a dull note on percussion.

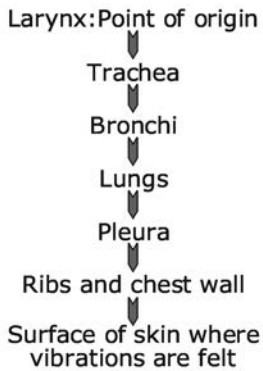

Fig. 8. Transmission of vocal fremitus.

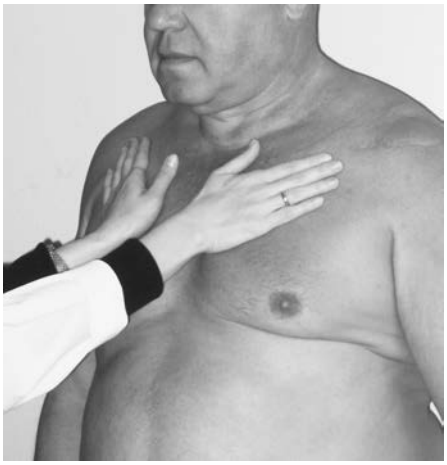

Fig. 9. Assessment of the vocal fremitus.

Comparative percussion

When a blow is delivered with the middle finger of one hand (the right hand in a right-handed person) on the firmly placed middle finger of the other hand, the underlying air is set in vibration producing a resonant sound. The stroke should be delivered from the wrist and finger joints to give you control over the force of the blow and over the precision of the site where it lands (Fig. 10).

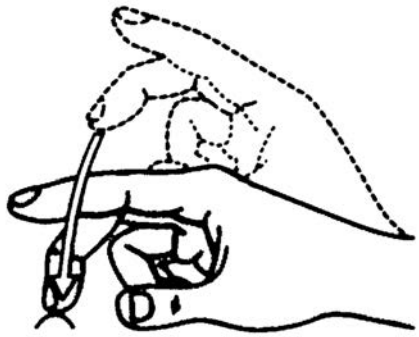

Fig. 10. Technique of percussion

The art of percussing properly is acquired by constant practice. In the early stages you can percuss on your friends, to improve the mechanics of your actions making them fluid, controlled and uniform.

To percuss the front of the chest, you should start by percussing over the clavicle on one side, then on the other side, and then percuss on each ribspace and compare the note elicited over the corresponding note on the other side.

It takes some experience to recognize the quality of the percussion note, which may range from very resonant (tympanic) to dull (Table 7).

As you percuss over the heart on the left side and below the 5th space on the right side, the sound will change to a higher-pitched dull note. This **cardiac dullness** may be reduced or obliterated altogether and the **hepatic dullness** may be lowered when the lungs are hyperinflated as in emphysema. On the left side as you percuss over the area overlying the stomach, the note

Table 7. Percussion notes and interpretation

• Resonant	Normal chest
• Tympanic	Hollow viscus (lung abscess, pneumothorax)
• Hyperresonant	Emphysema
• Hyporesonant	Pulmonary fibrosis, pulmonary collapse
• Dull	Pleural thickening, pulmonary infarction / collapse / consolidation / fibrosis
• Stony dull (flat)	Pleural effusion

will become tympanic due to the presence of air in the hollow viscus. After completing percussion of the front, you should percuss over both axillae.

As you percuss downwards, on the left side you will encounter the splenic dullness and on the right side the hepatic dullness. The areas over both these organs may be resonant if air has leaked through a perforated peptic ulcer.

Resonance is increased in emphysema and may become tympanic when the pleural cavity contains air (pneumothorax). Resonance is decreased if the underlying pleura is thickened or contains fluid (pleural effusion) and if the lung is consolidated by the products of infection, or if there is granulomatous or neoplastic infiltration or fibrosis. Careful and light percussion can reveal even small areas of consolidation or collapse. The note obtained over the pleural cavity containing fluid is stony dull like that elicited by percussing on a stone or brick wall! Apart from excessive dullness of the note, the pleximeter and percussing fingers feel some resistance from the underlying chest wall each time the blow is delivered.

Topographic percussion

In order to determine the exact size of the various organs or to differentiate the borders of two organs that lie adjacent to one another, they must be of different densities. Thus, by percussion it is easy to determine where the lung ends and the heart begins because of the different densities of these organs. However, it is extremely difficult to differentiate between heart and liver dullness, or between the dullness of pleural effusion and liver dullness since the densities so closely approximate one another.

Superficial and deep percussion refer to the force of the percussion blow. Heavy percussion gives a deeper penetration so that greater masses of tissue are set into vibration and a louder sound is produced. Such percussion, therefore, has an exceedingly restricted field of usefulness. A heavy blow creates so much lateral vibration that the sound produced by the structures immediately under the pleximeter finger is obscured.

Light percussion is essential for topographic diagnosis. Heavier percussion may be used when the superficial tissues are thick, in order to set up audible pulmonary vibrations, but at the same time accurate delimitation between organs becomes more difficult. Heavy percussion is absolutely useless for purposes of outlining organs or for detection of small areas of pulmonary infiltration. Deep percussion tends to bring out the note of the lung as a whole and, therefore, drowns out any slight degrees of dullness.

It is, therefore, wisest for students to learn from the beginning to use a light percussion stroke, and to do this, the pleximeter finger must be lightly but snugly applied, the stroke must be gentle and its duration short, so as to set into vibration a small acoustic sphere. The pleximeter finger must be placed firmly against the chest wall in the interspace paralleling the ribs. If it is held too lightly, a small pocket of air forms between the finger and the chest wall and is sufficient to impair what might otherwise be a resonant note; if it is held too tightly against the chest wall, the finger becomes tired, the patient becomes annoyed and there is interference with the normal resiliency of the chest wall. The fingers adjoining the pleximeter finger should not rest on the chest wall since they tend to dampen the vibrations.

The normal limits of pulmonary resonance correspond accurately to the anatomic boundaries of the lung. With light percussion the inferior limits of the lung are found at the level of the sixth rib in the midclavicular line, the eighth rib in the midaxillary line and the tenth rib in the scapular line.

The lower limit of pulmonary resonance should in all instances be examined by percussion during both forced inspiration and expiration (Fig. 11); normally the difference in space between these two extremes measures 3 to 4 cm. This space represents the complemental pleural space, and by this means the degree of respiratory mobility is attained. This respiratory mobility is diminished or absent in diseases of the lung such as emphysema, pleural diaphragmatic adhesions and conditions that interfere with movement of the diaphragm.

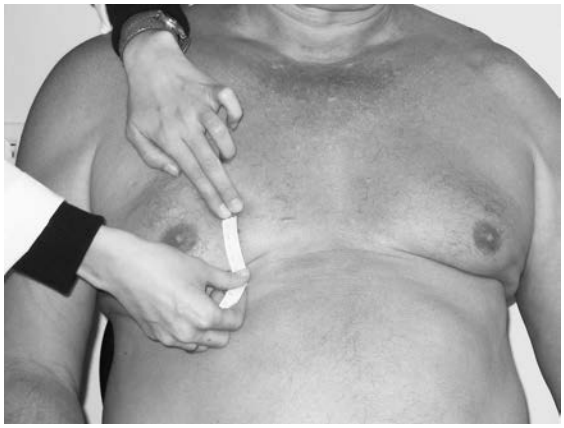

Fig. 11. Measuring the respiratory mobility of the right lung at midclavicular line.

Krönig's area is a zone or isthmus of vesicular resonance extending across the trapezius muscle on each side of the neck corresponding to the apex of the lung (Fig. 12). Normally, this area measures about 5 cm., and contraction of this zone denotes disease of the lung apex.

On the left side near the lower costal margin, a tympanitic area, called Traube's semilunar space, is encountered. This area is bounded above by the lower border of the left lung, below by the spleen, internally by the left lobe of the liver and externally by the costal margins. It contains the fundus of the stomach, and the tympanitic note obtained by percussion is occasioned by the air content of the stomach. When the

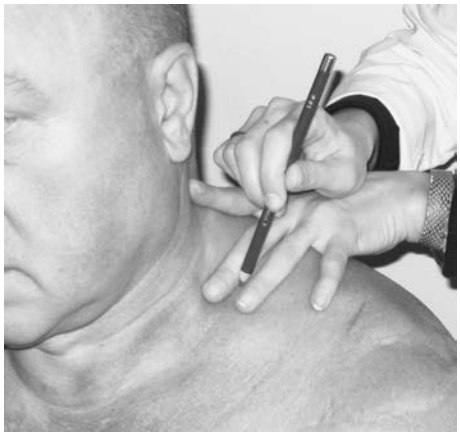

Fig. 12. Percussion of the outer and inner borders of the Krönig's area.

stomach is filled with food, the tympanitic note is decreased or disappears, as it also does in cases of pericardial effusion and left pleural effusions.

Auscultation

As explained earlier a good quality stethoscope should be used. Since most respiratory noises are low-pitched, the bell chest-piece should be used for listening over the chest. The patient should remain still and breath through the mouth and the chest-piece should be placed first on the apex and then on each rib space on either side.

Breath sounds

Breath sounds are generated in the trachea and major bronchi and are conducted through the lungs to the stethoscope placed on the chest wall.

Inspiration is audible longer than expiration which follows without a pause and the sounds have a rustling quality.

This is the normally heard vesicular respiration and the only sure way of recognizing it is to listen frequently over normal chests (try your friends).

Harsh vesicular breath sounds are somewhat louder with a prolonged expiration sometimes heard when there is partial obstruction of a bronchus as in bronchitis and asthma. It can be confused with bronchial breathing particularly by a beginner using the diaphragm. The important distinguishing feature between **harsh vesicular** and **bronchial breathing** is that in the former there is no pause between inspiration and expiration whereas there is a definite interval between the two in bronchial breathing. Bronchial breath sounds have a blowing quality which can be appreciated if you whistle air in and out of your mouth, but keep the tip of your tongue against the inside of your **upper** incisors.

Bronchial breathing is heard whenever the breath sounds generated in a major bronchus are conducted to the surface either through a cavity or an airless lung (consolidation, collapse, fibrosis). Bronchial breathing heard over a cavity is usually low-pitched and is therefore called **cavernous**. High-pitched bronchial breathing is typically heard over consolidation and this is sometimes called **tubular**.

Bronchovesicular breathing, or the breath sound combining both vesicular and bronchial elements, is heard anteriorly in the right upper zone (conducted tracheal sounds), or behind where a main bronchus may be near the surface. The expiration is prolonged and has a bronchial character. It is normally heard in a thin person and should not be confused with bronchial breathing.

Breath sounds are diminished or absent over thickened pleura, pleural effusion, pneumothorax or whenever there is fibrosis, collapse or infection in the underlying lung. Some people mistakenly state that air entry is diminished into such an area where they do not hear any breath sounds. It should be appreciated that breath sounds are not generated in the alveoli but in the major air passages and conducted through the intervening lungs and pleura to the stethoscope.

Vocal resonance

Vocal resonance is the auscultatory equivalent of vocal fremitus; sounds generated in the larynx are conducted to the surface of the chest where they can be heard through the bell of the stethoscope. When a patient repeats a resonant word such as «ninety-nine» the sounds are conveyed through somewhat attenuated unless interrupted by consolidation in which case the sound is louder and clearer. The sound may be absent or diminished in intensity if conducted through pleural fluid or thickening, collapsed lung or pneumothorax. The term **bronchophony**, or **egophony**, is applied when the sounds are heard clearly as if directly spoken in the ear. In such cases whispered words (usually a soft sounding word such as «sixty-six») are also clearly heard and the phenomenon is called **whispering pectoriloquy**. Both these signs are associated with bronchial breathing and are characteristically heard over consolidation or cavity communicating with bronchus.

Adventitious sounds

The lack of agreement about the classification of adventitious sounds seems to stem from confusion about how they are caused. There seems to be an impression that breath sounds are caused by the entry of air into the alveoli, and crepitations or crackles are caused by the movement of fluid (hence the old term «moist rales») in and out of these small air spaces. As stated above, this belief underlies the usual comment «no air entry» when no breath sounds are heard over pleural thickening or collapse of part of a lung.

There are **four** reasons why breath sounds or crackles cannot be explained by the movement of air and fluid into the bronchioles and alveoli. **First**, it is difficult to imagine how the movement of air in microlitre quantities through the air passages with a diameter of no more than a micrometer can generate the rustling

sound one hears through the stethoscope. **Second**, the mid-inspiratory crackles usually heard over the lung bases in left ventricular failure have a fixed time-relationship with inspiration which would not hold constant if they were caused by the movement of fluid. **Third**, crackles are also heard in pulmonary fibrosis when there is no reason to expect any interstitial fluid. **Fourth**, inspiratory crackles can be made to disappear by change of posture and not by coughing.

These objections can be explained if we accept that breath sounds are generated by movement of air through the major air passages, and that crackles are caused by the opening up of the alveoli that are collapsed under weight of the fluid-laden lungs, by infected secretions or by fibrotic bands. This explains why these sounds always appear at the middle or towards the end of inspiration when the inspiratory effort reaches these collapsed alveoli at the lung bases and crackle-opens them. In view of these considerations the various adventitious sounds can be produced under the following pathophysiological circumstances.

Late inspiratory crackles. The opening up of multiple collapsed alveoli produces discontinuous, non-musical crackling, clicking or bubbling sounds during the middle or late phase of inspiration, sometimes spilling over the early part of expiration. These sounds can be imitated by rubbing together a few hairs between the thumb and finger in front of your ear.

Coughing may accentuate or even bring about inspiratory crackles. The probable explanation is that coughing expels air out of the alveoli at the lung bases and these alveoli vacuum – close and then the rush of air during late inspiration produces explosive clicking sounds. Conversely, the intensity of these sounds can be reduced or they can even be made to disappear if the pressure compressing the alveoli generating these sounds can be reduced. You can do this by placing the bell of your stethoscope at the highest point on the back where you can hear the crackles, and then, with the chest-piece still on the same place, ask the patient to bend forwards as far as possible, thus relieving the pressure over the alveoli where the crackling sounds will become fewer in number and with reduced intensity.

Inspiratory crackles are characteristically heard over the lung bases in left heart failure and fibrosing alveolitis, but they may be restricted to one area in lobar pneumonia and localized fibrosis, and over the apex in tuberculosis.

Inspiratory and expiratory crackles. As stated above, inspiratory crackles do not occur at random but appear in the same sequence from breath to breath, suggesting that pressure and volume changes determine their occurrence. However, in the late stages of pulmonary oedema and in inflammatory conditions of bronchi, the larger airways may be flooded with oedema fluid or bronchial secretions, and then the crackles will be heard during both phases of respiration. In these cases the crackles appear at random and are modified by coughing.

Early inspiratory crackles. In severe airways obstruction, the larger airways, already narrow, tend to close prematurely in expiration and open early in subsequent inspiration producing crackling sounds. They are confined to the early phase of inspiration, and repeat from breath to breath in the same sequence. These sounds originate in larger airways in asthma, emphysema and chronic bronchitis, and are not modified by change of posture or coughing.

Wheezes. Wheezes are high-pitched sounds which can be heard without the stethoscope especially at the end of expiration. Polyphonic wheezes consist of a cluster of continuous musical noises and are caused by high velocity of air flow through narrowed small bronchi in asthma, emphysema and chronic bronchitis. Healthy subjects can generate polyphonic wheezes towards the end of a forced expiration as the bronchi are compressed and the velocity of air is increased. In diffuse airways obstruction due to asthma and chronic bronchitis, wheezes occur at submaximal respiration and may occur even at tidal breathing.

In severe airways obstruction, there may be a paradoxical absence of wheezes as the air flow through the maximally narrowed bronchi is very slow. The absence of wheezes under such circumstances is an ominous sign.

Monophonic wheezes. Random monophonic wheezes arise as continuous bleating, musical noises from individual airways, which are narrowed due to spasm, oedema, secretions or by extramural compression by a tumour. The commonest cause of monophonic wheezes is bronchitis when they are heard together with crackles. Coughing may alter their pitch or clear them. **Unilateral or localized monophonic wheezes** are strong evidence of bronchial obstruction by a neoplasm or a foreign body.

Pleural crackles (rub). These are coarse, non-musical sounds and are heard at some point during both phases of respiration. Pleural crackles are localized to a small area of the chest. They can be imitated by scratching the scalp while the corresponding ear is blocked. The sound is caused by the two inflamed surfaces of the pleura rubbing against each other during respiration and disappears when sufficient fluid accumulates to separate the two layers of the pleura. A pleural rub may be confused with a monophonic wheeze or lung crackles, but it is usually confined to one area, has a fixed relation to **both** phases of respiration, and is not changed by coughing. Sometimes a pleural rub is palpable.

A pleural rub may be confused with a pericardial friction rub, particularly when the inflamed pleura is close to the apex of the heart. The resulting **pleuropericardial rub** usually assumes the rhythm of the heartbeat.

Examination of the back of the chest

You should now explain to your patient that you would like to examine the back of his chest and that he should sit up, leaning slightly forwards with forearms crossed in front (Fig. 13).

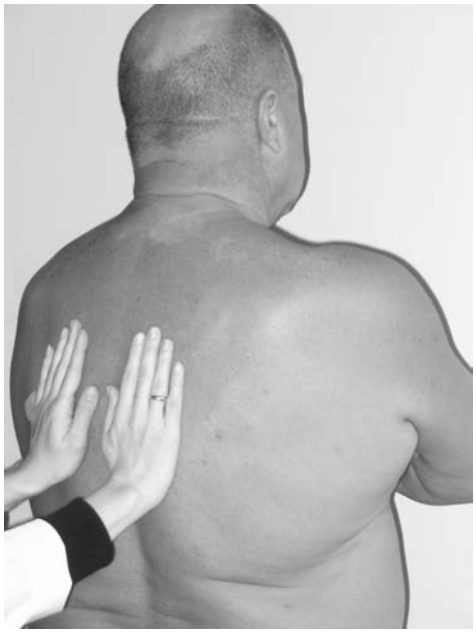

Fig. 13. Assessment of the vocal fremitus at the interscapular area.

If the patient is acutely ill you may have to accomplish this part of the examination after turning the patient over to lie on his side.

This approach does not afford a comprehensive examination of the back but, if it is convenient to the patient, you may get a satisfactory comparison of the two sides by rolling the patient first on one side then on the other.

The examination of the back should be carried out sequentially in the same way as for the front but two points require special mention. First, as you approach the patient from the back to measure the expansion of the ribcage, you may unwittingly ignore the apices. Sometimes it is easily done either in the stress of an emergency setting or in the final MB or Membership Examination. All procedures should start at the apices including the test for expansion. The upward movement of the thumbs meeting in the midline will indicate whether both the apices are moving equally or not. Similarly, percussion and auscultation should be started at the apices.

The second important point relates to percussion over the back which requires special care. Unlike the front of the chest where light percussion can be very informative, the back is covered by thick muscles and fat and therefore heavy percussion is needed, especially in obese subjects, to get a resonant note from normal lungs. However, as you percuss downwards you will encounter the various solid structures such as the liver overlapping the lungs, and relatively lighter percussion may be needed to define the lower border of the lungs. Sometimes dull lung bases are diagnosed erroneously in normal subjects with high diaphragms. In difficult cases it may be necessary to percuss the bases several times with varying degree of percussing force.

Additional bedside tests

Sputum

Examination of the respiratory system is not complete unless you have examined the products of expectoration, either by inspecting the spittoon, or by looking at a handkerchief or tissues which the patient may have used. Naked-eye examination of the sputum can give important clinical information:

i) Consistently large volumes (at least $^1/_2$ cup/day) suggest bronchiectasia.
ii) Rupture of an abscess, empyema or cyst into a bronchus may produce a sudden increase in volume.
iii) Infected sputum is usually yellow or green because of the large number of polymorphs it contains.
iv) Blood in the sputum produces a pink tinge in the typical frothy sputum of left ventricular failure, deep red flecks in bronchial carcinoma and pulmonary embolism, and a rusty colour in pneumococcal pneumonia.
v) Black sputum may be found in workers in a dusty environment or after smoke inhalation.
vi) Thick viscid sputum, sometimes taking the form of bronchial casts, is often seen in asthma and in allergic bronchopulmonary aspergillosis.
vii) Rupture of a hepatic amoebic abscess into the lung gives an «anchovy sauce» appearance to sputum.
viii) Rupture of a hepatic hydatid cyst into a right lower lobe bronchus produces bile-stained sputum.

Microscopic examination of sputum may show asbestos fibres, Charcot-Leyden crystals derived from eosinophils, fungal spores or clumps of pathogenic bacteria.

Sputum culture is of value in identifying bacteria and fungi and in testing for drug sensitivity. Culture normally takes 24–48 hours for pyogenic bacteria (up to 8 weeks for mycobacteria). Therapy should often be started without waiting for the result of culture.

For sputum cytology, as much sputum as possible should be sent fresh to the laboratory. The results are excellent with central tumours (80–90% positive), but much less successful with peripheral tumours.

Peak expiratory flow measurement

The maximum flow rate during a maximum expiration (after full inspiration) can be measured with a portable Wright's peak expiratory flow rate.

Normal values for men range from 400 to 650 l/min, and for women from 350 to 500 l/min. Airways obstruction causes reduction in the peak expiratory flow rate. It is useful in assessing severity and for monitoring progress of airways obstruction.

Skin tests

Skin-prick testing may be useful in establishing patient's immediate (type I) sensitivity to common allergens, thus confirming the patient's atopic state, and providing useful information about the possible role of allergens in disease.

Skin-prick testing may be useful in urticaria, asthma, rhinitis, allergic conjunctivitis and other allergic conditions, though false positive results are common in atopic eczema.

In skin-prick testing, a tiny quantity of allergen is introduced into the superficial layers of the skin. First, the volar surface of the forearm is cleaned, prick sites are marked, and drops of allergen extract in appropriate concentration are placed on the skin. The test should always include a negative control of 0.5% phenol saline, the suspending solution for the allergens, and histamine 1% as a positive control. A lance or a standard needle is introduced through each drop at 45° to the skin surface to a depth of about 1 mm; the skin is lifted slightly, and the lance withdrawn. The procedure is painless, and puncture sites should not bleed. The skin is blotted dry, and resultant reaction is assessed at 15–20 minutes. The maximum reaction is usually seen after 15–20 minutes. The saline control should be negative (unless the patient has dermographism). The histamine control should be positive. The presence of a positive skin response indicates the presence of specific IgE antibody in the blood, and there is a reasonable correlation between the size of the weal and the significance of different inhaled allergens in a single patient. Positive results are best recorded by measuring the diameter of the weal in millimetres, using a transparent gauge or a ruler. The interpretation of the response depends on the clinical history.

A true positive skin-prick reaction indicates that specific IgE is fixed to mast cells in the skin and has led to vasoactive response caused by release of histamine. When the allergen concentration is high, or the patient's sensitivity is extreme, a late skin reaction may also follow 4–6 hours (or even as late as 24–48 hours) after the test, with erythema, swelling and induration. Many asthmatics have multiple positive skin test reactions to common allergens, showing the ease with which they make IgE antibodies to common allergens in the environment.

When performed in patients with asthma, with appropriate positive (histamine) and negative (diluent) controls, skin test results correlate well with the results of bronchial challenge testing (which is not performed routinely), and thus give useful information on the allergens involved; however, the results must always be correlated with clinical history. For inhaled allergens, up to 15% of positive results are false positives, but fewer than 5% of negative results are false negatives. However, skin testing is relatively unreliable for ingested allergens including food, partly because of the nature of the available allergen preparations and partly because reactions to ingested substances are not always mediated by IgE.

Only a small number of allergens are needed for routine skin-prick testing in patients with asthma or rhinitis. A typical skin test battery can include four antigens, together with positive and negative controls (Table 8).

Table 8. Allergen extracts commonly used in skin testing

Routine short screen for atopy
- Phenol saline (negative control)
- Histamine (positive control)
- House dust mite*
- Grass pollen**
- Aspergillus fumigatus
- Cat

Additional allergens which may be used
- Tree / weed / other plant pollens
- Alternaria
- Cladosporium
- Dog
- Any other relevant animals
- Any relevant foods
- Any other relevant inhaled allergens

*) — Mixed «house dust» extracts may be used in the routine screen; alternatively allergens from *Dermatophagoides pteronyssinus* and/or *Dermatophagoides farinae* may be used.
**) — In some regions it is appropriate to include an alternative pollen antigen in the routine screen (e.g. birch in Scandinavia, ragweed in North America). The mix of grass pollens used may also vary between regions.

Additional antigens can be added when there is a clear possibility of the involvement of other antigens. The role of some allergens cannot be successfully investigated by skin-prick testing, and occupational allergens are also usually better identified by other means.

Strongly positive tuberculin tests are of value in diagnosing tuberculosis in individual patients, and tuberculin testing also has a role in screening contacts and pre-BCG (Bacillus Calmette-Guérin) assessment.

Blood tests

Venous blood samples taken for automated blood counts may provide major clues or confirmation of a suspected respiratory disease. A high haemoglobin concentration may be a reflection of polycythaemia, either primary or secondary and a low haemoglobin may cause breathlessness. The total white cell count may be elevated in a range of acute bacterial infections and its subsequent fall is a reflection of successful therapy. Normal or low white cell counts are found in mycoplasma or viral infections. Eosinophilia suggests an allergic component or parasitic infection.

A range of serological tests that depend on agglutination, precipitation and complement fixation provide evidence of the presence in the patient's serum of specific antibodies against viral, bacterial, fungal, protozoal and helminthic infections. Samples of blood should be tested on admission and repeated after 10–14 days to detect a rising titer.

Radioallergoabsorbent tests (RASTs) on venous blood are an alternative to skin-prick tests as a method of identifying specific IgE antibodies.

Blood gases

The presence of respiratory failure may be suspected by the signs of central cyanosis. It is important to define the type and extent of failure of oxygenation and this is best done by measurement of arterial blood gas tensions (PaO_2 and $PaCO_2$), oxygen saturation (SaO_2) and pH. Arterial blood sampling can be carried out from the femoral, brachial or radial arteries, but the most common site is the radial artery in the patient's nondominant arm. Firm pressure should be applied after withdrawal of the needle to prevent local haematoma formation.

The response to drugs and therapeutic response to oxygen can then be monitored easily. Haemoglobin saturation reflects oxygen carriage by the blood and thus the adequacy of tissue oxygenation (if perfusion is satisfactory) and the requirement for oxygen therapy. This can be measured noninvasively by pulse oximetry. The widespread introduction of pulse oximeters has been a great benefit in many areas of medicine, as oxygen saturation may be monitored noninvasively via a probe on a finger or earlobe. The estimation of oxygen saturation is not accurate at very low levels but, in the usual range for all but the most severe respiratory failure, oximeters are accurate if cardiac output and local circulation are adequate.

The arterial partial pressure of carbon dioxide ($PaCO_2$) is a good indication of ventilation, low values indicating hyperventilation and vice versa; it is often more important than the PaO_2 in assessing the need for assisted ventilation.

Pulmonary function tests

Simple pulmonary function tests may easily be done at home or at the bedside using a peak flow meter or gauge. This gives reasonably reliable and repeatable results and can be used to monitor therapy in asthma and chronic obstructive airways disease. While using mini peak flow meter the patient takes a deep breath, and then makes a maximal expiratory effort through the instrument. The procedure is repeated three times and the highest peak expiratory flow (PEF) is recorded. This can be compared with a normogram that shows the patient's sex, age and weight, and plotted on a chart to show the progress or response to treatment.

By use of a spirometer (Fig. 14) and other equipment, a number of volume and flow rates can be estimated (Table 9). A spirometer provides a simple means of assessing air flow obstruction. The patient takes a maximal inspiration and then exhales as fast as possible for as long as possible. The volume expired against time is measured, and the forced expiratory volume in one second (FEV_1) and the forced vital capacity (FVC) can be simply calculated from the graph produced.

i) The peak expiratory flow (PEF) is the fastest flow rate recorded during expiration.

Fig. 14. The procedure of pulmonary function testing.

Table 9. Common tests of respiratory function

Test	Abbreviation
• Peak expiratory flow	PEF
• Forced expiratory volume in one second	FEV_1
• Forced vital capacity	FVC
• Relaxed vital capacity	RVC
• Total lung capacity	TLC
• Residual volume	RV
• Functional residual capacity	FRC
• Maximum expiratory flow at lower lung volumes	MEF_{50} etc.
• Airways resistance	R_{AW}
• Specific airway conductance	$_SG_{AW}$
• Transfer factor	TL_{CO}
• Transfer factor per unit lung volume (diffusion coefficient)	K_{CO}

ii) The forced expiratory volume in 1 second (FEV_1) is the volume of gas expired in the first second of expiration.

iii) The forced vital capacity (FVC) is the total volume of gas expired.

iv) Flow-volume loops are particularly helpful in the assessment of airway obstruction.

v) After a full expiration some gas remains in the lung, the residual volume (RV); in order to measure the volume of gas in the lungs at full inspiration (total lung capacity, TLC) and obtain the residual volume by subtracting the vital capacity; TLC is usually measured by helium dilution; a subject re-breathes a known volume and concentration of helium, which is diluted in the lung so that the TLC can be calculated by measuring how much the helium has been diluted.

vi) Airways resistance can be measured by body plethysmography. Body plethysmography allows the simultaneous measurement of thoracic gas volume, airways resistance and specific conductance. Such techniques may provide valuable additional information, especially in relation to the assessment of the effects of drug therapy.

vii) There are methodological difficulties in measuring oxygen transfer from lung to blood, so carbon monoxide transfer factor (TL_{CO}) is commonly measured; the transfer of carbon monoxide depends on how much haemoglobin can be «seen» by the inspired gas; therefore transfer is low when the pulmonary capillary bed is damaged or obscured by inequalities of ventilation or perfusion; it is also low in anaemia and can be increased with polycythemia or if haemorrhage into the lung has occurred.

The results of simple spirometry provide much useful information and even simple lung function tests are of great value in following the course of disease and response to treatment.

Bronchial challenge testing with allergen extracts, histamine or methacholine is valuable in the assessment of airway hyperresponsiveness in asthma, but is not in routine clinical use in most centres.

Imaging

A posteroanterior (PA) chest X-ray will often provide valuable diagnostic information about the nature and location of respiratory disease.

A lateral chest X-ray is helpful in identifying the position of abnormalities seen on the posteroanterior film and may occasionally show significant abnormalities not seen on the standard film.

Tomography allows a radiograph of a specific slice of the chest; it is particularly useful for demonstrating lung cavities, the lumen of the trachea and major bronchi, and the position and nature of abnormal shadows noted on the plain radiograph.

Computerised axial tomography (CT) of the lung is most commonly used for the assessment of the extent of lung cancer, but is being increasingly used in diffuse lung disease and has replaced bronchography as the first-line investigation of suspected bronchiectasia.

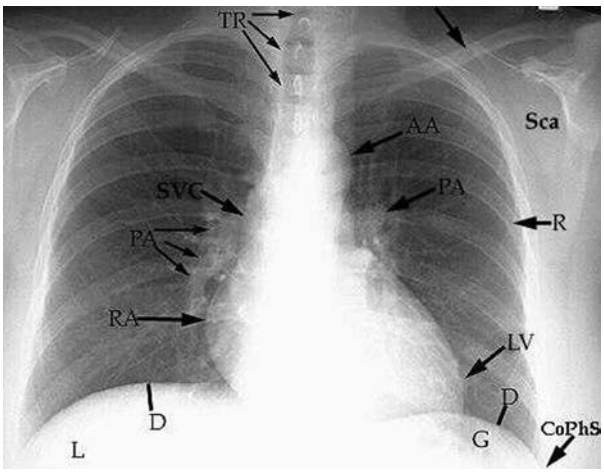

Fig. 15. The normal X-ray of the chest (PA — pulmonary artery; TR — trachea; CL — clavicle; AA — aortic arch; SVC — superior vena cava; RA — right atrium; CoPhS — costophrenic sulcus; LV — left ventricle; D — diaphragm; G — gastric air bubble; L — liver; Sca — scapula; R — rib).

MRI shows cardiovascular structures in the chest. Its role in pulmonary disease is still developing.

Pulmonary and aortic angiography may be used to define the anatomy of the arterial tree.

Radionuclide scans of lung (V/Q scan) are particularly useful in suspected pulmonary embolism. In this technique, xenon gas is inhaled and gamma-camera ima-ge is produced of the alveolar distribution of the radio-activity. Then an intravenous injection of microaggregates of human albumin (each 100 μ) labelled with 99m-Tc is injected. These microspheres embolize harm-lessly in the lung vessels and the distribution of the radioactivity is a reflection of the pulmonary blood supply.

Bronchoscopy

The flexible bronchoscope has made bronchoscopy easier, safer and less traumatic than before. In general, the flexible bronchoscope is simpler, quicker, safer and less traumatic to use than the rigid bronchoscope, but rigid bronchoscope allows larger biopsy samples to be obtained. Fibreoptic bronchoscopy is a simple technique that can be performed on the conscious patient. The bronchoscope is usually passed through the nose. Excellent views can be easily obtained of all major and segmental bronchi and samples of mucus collected via the aspiration channel. Videobronchoscopes are now replacing fibreoptic bronchoscopes in many centres.

In addition, brush biopsies, suction catheters and biopsy forceps can be passed through this channel and bronchoalveolar lavage can be performed. Samples for culture, cytology and histology can be easily obtained.

Bronchoscopy is the investigation of choice for mass lesions on the central and mid-zones, especially if carcinoma is thought to be obstructing bronchi.

In those who are very frail, however, sputum cytology may be the investigation of choice.

Lung biopsy is possible by advancing the biopsy catheter through the lung parenchyma under fluoroscopic control.

The use of bronchoscopy to obtain good specimens for microbiology and lung histology has reduced the requirement for open lung biopsy in patients with pulmonary infiltrates. Bronchoscopy can be useful therapeutically in the removal of secretions or foreign bodies causing airway obstruction, and some central tumours are amenable to laser resection via the bronchoscope.

Mediastinoscopy and thoracoscopy are often techniques that are of value in the investigation of selected patients with mediastinal and pleural disease.

Pleural aspiration and biopsy

Aspiration of pleural fluid is of major value for both diagnostic and therapeutic reasons (Fig. 17). Naked-eye inspection may suggest the presence of pus, the

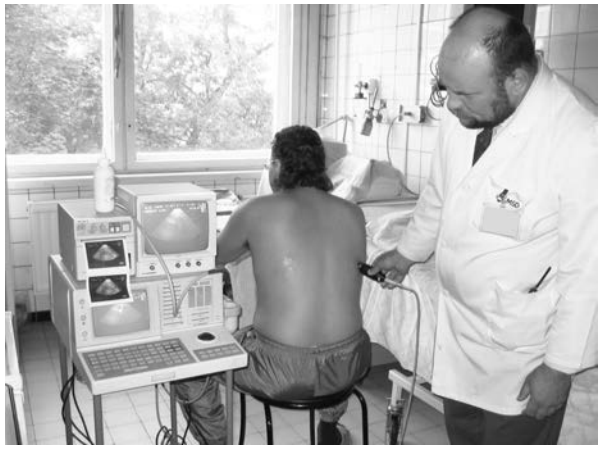

Fig. 17. Location of the pleural fluid by ultrasound method.

effusion may be blood-stained suggesting carcinoma or pulmonary embolism, or milky white (chylous) as a result of obstruction of the thoracic duct, usually by tumour.

i) Pleural transudates are associated with generalized oedema and are pale in colour with a specific gravity of less than 1015, total protein less than 2.5 g/100 ml.
ii) Pleural exudates represent inflammation and are usually darker in colour with a specific gravity above 1018 and a total protein greater than 3 g/100 ml.
iii) Total white cells count and cell type are of value; polymorphs indicate bacterial infection and lymphocytes suggest tuberculosis; culture of the fluid may show the organism responsible and cytology — the diagnosis of tumour.

At the time of pleural aspiration, it is often convenient to carry out a pleural biopsy with a side-cutting needle. This is «blind», but relatively non-traumatic. Pleuroscopy and mediastinoscopy can be used to provide direct vision for biopsy. If the diagnosis is already made, then an opportunity to instil antibiotic, cytotoxic or sclerosants may be taken. At pleural biopsy the needle is inserted into the pleural cavity and may be used to aspirate a pleural effusion. Then it can be turned round so that the notch present in the shaft engages with the parietal pleural surface. The inner cutting trocar is then advanced to cut a small biopsy that may include the intercostal muscle as well as pleura and any tumour present. This is an effective way of diagnosing pleural involvement by malignant disease.

Pleural aspiration and biopsy are not always harmless procedures. They may result in damage to the lung or abdominal organs, pneumothorax or haemothorax. In the longer term, biopsy of mesothelioma may result in spread of the tumour.

Lymph-node aspiration and biopsy

Aspiration of a palpable node, usually in the neck, may provide a rapid cytological diagnosis, and biopsy of node is a further possibility in suspected malignant disease. When no nodes are felt, removal of scalene node may provide diagnostic information.

Upper respiratory tract disorders

The common cold is discussed earlier, and rhinitis, sinusitis, tonsillitis, pharyngitis and laryngitis may also occur as a part of a number of childhood exanthemata such as measles, and in infectious mononucleosis and streptococcal infection.

Other medical disorders of the upper airway include other forms of rhinitis (Table 10).

Table 10. Some common causes of rhinitis

Allergic	Non-allergic		
Perennial	Infectious	Drugs	Irritant
House dust mite	Acute	Aspirin	Moulds
Pets Occupational causes	Chronic (consider immunodeficiency/mucociliar problems	β-blockers	
Seasonal	Nasal hyperreactivity	Systemic diseases	Hormonal
Pollens			Hypothyroidism
			Pregnancy Irritant

Allergic rhinitis may be seasonal, most commonly caused by allergy to grass or other pollens (hay fever), or perennial, most commonly caused by allergy to the faeces of the house-dust mite. Allergic rhinitis produces a greyish appearance in the nasal mucous membrane, especially when chronic. Skin-prick testing is often helpful in diagnosis.

Non-allergic or vasomotor rhinitis produces similar symptoms, but the cause is usually obscure.

Allergic rhinitis is often associated with other atopic disorders, including conjunctivitis, asthma, urticaria and eczema. It usually responds to topical corticosteroid or sodium chromoglycate therapy. Unlike asthma, however, most of the symptoms of allergic rhinitis often respond to oral antihistamines.

Nasal polyps are benign, oedematous, inflammatory swellings, which originate in the mucosa of the ethmoid sinuses or middle turbinates and protrude into the nasal cavity, causing obstructive symptoms. They are particularly common in a group of patients who also have asthma and are sensitive to aspirin and dietary salicylates, and are also associated with other respiratory tract disorders (Table 11).

They may be viewed via a nasal speculum or endoscopically, and extent may be assessed by CT scan. Nasal polyposis is a relatively frequent accompaniment of asthma and is particularly common in adult asthmatics with sensitivity to aspirin. Occasionally they may be large enough to distort the external appearance of the nose. They may respond to topical steroid therapy, but systemic steroids or surgical removal may sometimes be needed to control obstructive symptoms.