Assessment of a Patient with Lung Disease

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1. The History in Lung Disease

Although the symptoms of respiratory disease may not accurately reflect the degree of functional impairment, a carefully taken clinical history often allows a correct diagnosis to be made (Hampton et al. 1975). The definition of chronic bronchitis is, in fact, based on the clinical history of cough and sputum production. The findings of the history can be evaluated by special investigations.

Major Symptoms

Dyspnoea

Dyspnoea is a feeling that breathing is difficult, laboured or uncomfortable, and there are a number of factors which may lead to the sensation. In some patients it may be difficult to differentiate dyspnoea from pain associated with breathing and, indeed, the limitation of expansion from pleuritic pain may produce a feeling of dyspnoea. Although dyspnoea may be a feature of anxiety it is important to remember that acute dyspnoea is a very distressing situation in itself, quite capable of inducing anxiety in most patients. There are a number of causes of dyspnoea related to the work of breathing, the efficiency of the respiratory muscles and the drive to breathing.

Increased Work of Breathing

The work required for adequate ventilation is increased if there is obstruction to airflow from airway narrowing or if the lungs or the chest wall are abnormally stiff. Both situations increase the mechanical load on breathing and are often associated with dyspnoea.

Neuromuscular Problems

In such conditions as myasthenia gravis and muscular dystrophies the response to the neurological output from the respiratory centre may be inadequate, resulting in a feeling of inability to take in enough air. However, in neuromuscular disorders there may be a paradoxical absence of dyspnoea despite considerable functional impairment. The same situation is found in chronic airflow obstruction associated with CO_2 retention, the 'blue bloater'.

Increased Drive to Breathing

In acidosis and hypoxia there is an increased chemical drive to breathing which stimulates deep, rapid respiration. However, a sensation of dyspnoea may not be prominent if there is not an underlying pulmonary cause for the situation. Dyspnoea may also be found in severe anaemia where the oxygen carrying capacity of the blood is diminished. In chronic airflow obstruction with hypercapnia, the 'blue bloater', the hypercapnia fails to maintain its drive to breathing and dyspnoea is usually absent, despite the hypercapnia and airflow obstruction. In conditions such as pulmonary embolism, pneumonia and pulmonary oedema there seems to be an inappropriately high drive to breathing resulting in hypocapnia. This may be related to stimulation of juxtacapillary, 'J', receptors. Dyspnoea in pneumothorax is probably related to stimulation of a pulmonary deflation reflex. Often a combination of factors is involved in individual cases, for instance in asthma, increased drive, increased load, anxiety and poor muscle function of the diaphragm with overinflation may all be involved.

The pattern of dyspnoea should be assessed. Although orthopnoea is usually described in pulmonary congestion, it is not unusual in chronic airflow obstruction. Shortness of breath waking the patient in the early hours of the morning is often seen in asthma, especially during recovery from an acute attack. Other characteristics of asthma are the day-to-day variation in the severity of the dyspnoea and the relationship to time of year, exercise and environmental factors. Asthma precipitated by extrinsic factors often occurs immediately after exposure but may be delayed

for five or six hours, so that asthma induced by exposure at work may only develop after returning home. This makes the relationship more difficult to detect, so that it is important to assess any change in symptoms during weekends or holidays. In chronic airflow obstruction there usually is a slow progression of disability, whereas acute dyspnoea is most often caused by pneumothorax, pulmonary embolism or asthma. The degree of limitation imposed by dyspnoea should be assessed. One system of grading this is shown in Table 1, although it is often best to record the reported exercise tolerance. Although there is not a precise relationship to physiological assessment, the one second forced expiratory volume (FEV_1) is usually less than 0.6 l in patients in grade 5.

Chest Pain

The raw retrosternal pain of acute tracheitis is often associated with virus infections of the upper respiratory tract. Retrosternal pain may be produced by mediastinal lesions such as acute mediastinitis, mediastinal pleurisy or mediastinal tumours. In mediastinal pleurisy the pain is usually continuous rather than typically pleuritic in nature. Occasionally the hilar lymphadenopathy of sarcoidosis is associated with central chest pain.

The commonest chest pain related to the respiratory system is pleuritic pain. This is made worse by the movements of breathing and coughing; it often varies with position and may be related to exercise where respiratory excursions increase. Irritation of the

Table 1. Grading of dyspnoea.

Grade	Description
1	Normal
2	Short of breath on walking up mild hills or stairs
3	Short of breath on walking at a normal pace on level ground
4	Short of breath on walking at own pace on level ground for 100 metres
5	Short of breath on washing, dressing or walking a few paces

diaphragmatic pleura may present as pain in the tip of the shoulder because of the common innervation from the third, fourth and fifth cervical nerves.

Pleuritic pain may occur with rib fractures such as cough fractures which are especially common in patients on long-term corticosteroid therapy. Invasion of the ribs by malignant tumours, however, usually produces a constant, severe, aching pain. Pleuritic pain may be mimicked by epidemic myalgia or Bornholm disease, where breathing is often rapid and shallow to avoid the pain. This condition is distinguished by the tenderness of the overlying muscles and the occurrence in epidemics.

Wheezing

It is important to ask both the patient and any close relative about a history of wheezing, as patients with long-standing wheezing may not consider it relevant. Wheezes have the same significance whether they are heard at the mouth or on listening with the stethoscope. It may be possible to distinguish from the history the loud monophonic wheeze or stridor of tracheal or laryngeal narrowing from the generalized wheezing at many pitches which is a feature of more widespread airway narrowing.

Cough

A cough is the commonest presenting symptom of chest disease. Many smokers regard a morning cough as quite normal and it is not uncommon for patients to admit to sputum production but not to a cough. Coughing may be stimulated by the presence of sputum, foreign bodies, irritant gases, particulate matter or just apposition of bronchial walls. In widespread airflow obstruction it is common to find increased bronchial irritability so that such stimuli as cold air, dry air or increased depth of inspiration produce cough or increased dyspnoea. This is also a feature of the recovery phase from viral infections of the upper respiratory tract. Coughing may also be voluntary and may be a habit or a symptom of anxiety. In asthmatic children a nocturnal cough may be the only presenting symptom noticed by the parents.

Cough is common in association with upper respiratory tract

infections, but requires further investigation if it persists for longer than three weeks. Only relatively large airways are cleared by coughing. Below subsegmental levels airway clearance relies upon the mucociliary escalator carrying mucus or particulate matter up to the level at which coughing becomes effective.

Sputum Production

The volume of sputum, colour, consistency, odour and the time of production should be assessed. It is often difficult to quantify the volume of sputum from the history, but large volumes are typical of bronchiectasis and lung abscesses where the sputum is usually purulent and often foetid from anaerobic infection. In 20 per cent of cases of alveolar cell carcinoma large volumes of pink, frothy sputum are produced.

The sputum may be very viscid in asthma and there may be Curschmann's spirals, which are the casts of small bronchi or the firm brownish plugs which are typical of allergic bronchopulmonary aspergillosis. The expectoration of such casts or plugs may be associated with the relief of acute exacerbations of dyspnoea.

Haemoptysis

Haemoptysis is usually produced in small quantities and massive haemoptysis is an uncommon symptom, although it may be found in bronchiectasis, tuberculosis, mycetomas in old cavities and occasionally with bronchial neoplasms. Haemoptysis is most commonly due to inflamed mucosa in association with acute respiratory tract infections or exacerbations of chronic bronchitis. The initial radiograph is normal in more than half of the patients presenting with haemoptysis and very few extra diagnoses are made on subsequent radiographs taken a month or so later (Poole and Stradling 1964). It is important to investigate haemoptysis fully to rule out tuberculosis and bronchial neoplasms but most series show that neoplasms only make up about four per cent of haemoptyses (Table 2).

In cases of bronchial carcinoma the haemoptysis has usually lasted for longer than two weeks at the time of presentation and

usually consists of just blood streaking in the sputum. When the haemoptysis is associated with acute pulmonary symptoms the most common diagnoses are pneumonia and pulmonary infarction, but it must be remembered that pneumonia may be associated with an underlying adenoma or carcinoma.

Other Factors

Smoking History

Smoking is the most important predisposing factor in chronic airflow obstruction and in bronchial carcinoma. The risk of bronchial carcinoma is related to the number and type of cigarettes smoked and smokers of more than 20 cigarettes a day are at a 40 times greater risk than non-smokers (Doll and Hill 1964). The increased carcinoma risk declines slowly for 10 years after stopping smoking. Therefore, details should be recorded of the age of starting and stopping smoking, the amount of tobacco consumed and the average consumption over the smoking period.

Table 2. Causes of haemoptysis.

Cause	Percentage of cases
Upper respiratory tract infection	24
No cause found	21
Bronchitis	17
Bronchiectasis	13
Pneumonia	6
Active tuberculosis	4
Quiescent tuberculosis	6
Cardiovascular	7
Bronchial carcinoma	4

From a review of 324 consecutive cases presenting to a chest clinic (Johnston et al. 1960).

Family History

The most important conditions to ask about in the family history are tuberculosis and atopic conditions. In emphysema the finding of other family members may raise the suspicion of α_1 antitrypsin deficiency. Screening the family may detect pre-symptomatic homozygotes at risk of developing emphysema in whom the onset may be delayed if they can be persuaded not to smoke.

Social History

In asthmatics it is important to ask closely about predisposing factors such as exposure to animal hairs or precipitation of attacks by food or drugs. Many patients do not consider common analgesics or preparations taken for constipation important enough to be mentioned as medications and they should be specifically questioned about such drugs as salicylates and liquid paraffin. Salicylates may precipitate asthma especially in patients with nasal polyposis and intrinsic asthma, and inhaled mineral oils may produce lipoid pneumonia. There is increasing awareness of the association of drugs with a number of respiratory diseases.

Occupational exposure may be relevant to a variety of disorders including asthma, extrinsic allergic alveolitis, pneumoconiosis and neoplasms. In asbestos exposure there is often a long latent period between exposure and development of bronchial carcinoma or mesothelioma. The association may be related to forgotten exposure, such as wartime manufacture of gas masks or servicing of brake linings. It may be related to the husband's occupation if the wife washed her husband's working clothes contaminated with asbestos dust, or even to residence close to an asbestos factory.

2. Examination of the Respiratory System

There are two main parts to the examination of the respiratory system: examination of the chest itself and the assessment of signs outside the chest which relate to respiratory disease. Either part may be examined first. The patient should be reclining comfortably at 45°, stripped to the waist and placed in a good light. The first stage should be general inspection, followed by palpation, percussion and, finally, auscultation. The signs found in common conditions are summarized in Table 3.

Inspection

Any abnormalities in the shape of the chest should be noted. These may be congenital features, such as pectus excavatum, or acquired deformities, such as kyphoscoliosis. Long-standing collapse or fibrosis of the lung may lead to depression of the overlying chest wall; this is most obvious when the upper lobes are involved. Hyperinflation of the lung in asthma or emphysema produces an anteroposterior diameter greater than the lateral diameter. The pattern of breathing should next be observed. The normal respiratory rate is 12 to 16 per minute and not the 20 per minute often recorded in hospital patients.

Abnormal patterns of breathing may give clues to the diagnosis (Table 4), although nervous patients tend to hyperventilate during the examination. Dyspnoeic patients often use their accessory muscles on inspiration and their abdominal muscles on expiration. Any local diminution of chest wall movement is best observed facing the patient from the foot of the bed; reduction in

chest wall movement is found with fibrosis, collapse, consolidation, effusion and pneumothorax. Abnormal veins may be present on the chest wall in superior vena caval obstruction. Gynaecomastia may be a sign of bronchial carcinoma or, more commonly, drug therapy such as spironolactone.

Palpation

The position of the mediastinum is assessed from the trachea and the apex beat. The trachea is often best felt with the patient sitting up. With an overinflated chest it may not be possible to feel an apex beat. The impulse of an hypertrophied right ventricle may be felt at the left sternal edge, although with an overinflated chest it is often best felt producing a downward impulse in the epigastrium. In pulmonary hypertension a dilated pulmonary artery may be visible and palpable in the second left interspace. The expansion of the left and right sides of the chest should be compared in upper and lower zones. This is best done with the spread fingers on the chest wall and the thumbs free of the chest, meeting in the midline.

Consolidation allows the transmission of the higher frequency components of speech to the surface and may be felt as increased tactile vocal fremitus (TVF). This is usually easier to detect as increased conduction of voice sounds on auscultation. Pleural friction rubs may sometimes be transmitted to the palpating hand.

Percussion

The information from percussion comes from both the sound and the vibrations felt in the percussed finger. Percussion should start over the clavicles which may be percussed directly and should then proceed down the chest comparing the two sides. The lateral part of the chest must always be examined as well as anterior and posterior surfaces. With overexpansion of the chest the cardiac dullness may be diminished or absent and the top level of the hepatic dullness may be abnormally low. In diaphragmatic paralysis the hepatic dullness may be high and it is sometimes possible to demonstrate that the level does not move with respiration.

Table 3. Signs found in common lung conditions.

Clinical diagnosis	Observation	Movement	Palpation	Percussion
Consolidation		Decreased locally	Mediastinum central ↑ TVF	Dull
Collapse		Decreased locally	Mediastinum towards lesion ↓ TVF	Dull
Fibrosis	Overlying chest wall deformity	Decreased locally	Mediastinum towards lesion ↑ TVF	Dull
Pneumothorax		Decreased locally	Mediastinum may move away from lesion ↓ TVF	↑ Resonance
Pleural effusion		Decreased locally	Mediastinum may move away from lesion ↓ TVF	Stony dull
Chronic bronchitis and emphysema	Prolonged expiration Overexpansion	Decreased excursion		↑ Resonance ↓ Cardiac and hepatic dullness
Asthma	Prolonged expiration Overexpansion	Decreased excursion		↑ Resonance ↓ Cardiac and hepatic dullness
Fibrosing alveolitis	Rapid shallow breathing	Decreased excursion		May be decreased resonance

[1] In upper lobe collapse bronchial breathing may be transmitted from adjacent trachea.

Breath sounds	Voice sounds	Wheezes	Crackles
Bronchial	Bronchophony Whispering pectoriloquy		Pan or late inspiratory
Decreased[1]	Decreased		
Bronchial	Bronchophony Whispering pectoriloquy		Coarse late inspiratory, gravity dependent
Decreased	Decreased		Occasionally click in time with heart beat in shallow left pneumothorax
Decreased	Decreased Aegophony at upper border		May be associated pleural crackles
Prolonged expiration	Decreased	Polyphonic expiratory	Late expiratory and early inspiratory audible at the mouth
Prolonged expiration	Decreased	Polyphonic expiratory or random monophonic	Late expiratory and early inspiratory audible at the mouth
		Sequential inspiratory	Fine late inspiratory not conducted to the mouth

Table 4. Patterns of breathing movements.

Locally decreased movement	Fibrosis, collapse, consolidation, effusion, pneumothorax
Rapid shallow breathing	Restrictive ventilatory defect Pleuritic pain
Rapid deep breathing	Metabolic acidosis Brain damage Fever Anxiety
Cheyne–Stokes or periodic breathing	Pulmonary oedema Brain damage Raised intracranial pressure Renal failure
Irregular or 'ataxic' breathing	Brain stem damage Psychogenic
Prolonged inspiration	Laryngeal or tracheal stenosis
Prolonged expiration	Chronic airflow obstruction Asthma
Grunting expiration	Pneumonia
Orthopnoea	Pulmonary oedema Chronic airflow obstruction
Platypnoea (dyspnoea in upright position)	Basal vascular shunts (congenital or portal hypertension)
Inward abdominal movement on inspiration	Severe airflow obstruction Bilateral diaphragm paralysis
Inward thoracic movement on inspiration	Cervical cord transection

Auscultation

The interpretation of lung sounds has been given a sounder physiological basis in recent years by the work of Forgacs (1978). The terminology is simplified by describing added sounds as either wheezes (continuous sounds) or crackles (interrupted sounds). Lung sounds generally have a high frequency range and may be well heard through any efficient stethoscope. However, use of the bell avoids artifactual sounds produced by skin or hair rubbing on the diaphragm.

Breath Sounds

The breath sounds have a frequency range of 200 to 600 Hz (cycles per second) and are heard throughout inspiration and at the beginning of expiration. In bronchial breathing there is little alteration or filtration of the sounds from the large airways. The sounds are similar to those heard over the trachea with an even frequency distribution from 200 to 2000 Hz. They are audible throughout expiration as well as inspiration and are louder than normal breathing. Breath sounds are decreased over hyperinflated lung, fluid or pneumothorax. However, the size of an effusion or pneumothorax makes little difference to the decrease in sound, which comes largely from reflection of sound at the pleural surfaces where sound conducting properties on the two sides of the pleura change.

Table 5. Crackles.

Clinical diagnosis	Timing	Number	Pitch	Conduction to mouth	Gravity dependence
Pulmonary oedema	Late inspiration	Many	High	Usually no	Yes
Fibrosing alveolitis	Late inspiration	Many	High	No	Yes (until fibrosis severe)
Chronic airflow obstruction	End expiratory and early inspiratory	Few	Low	Yes	No
Pneumonia	Through or late inspiration	Variable	Variable	No	No
Pleurisy	Inspiration, expiration or both	Variable	Usually low	No	May change with position
Bronchiectasis	Inspiration	Variable	'Coarse'	Yes	No

Crackles

Crackles are the result of the sudden opening of closed airways allowing the pressure in the airway above and below the closure to become equal. Nath and Capel (1974) observed the repetitive nature of crackles and related the timing to the underlying pathology. In fibrosing alveolitis and pulmonary oedema the crackles are heard late in inspiration and are usually gravity-dependent, although when the fibrosis is severe the crackles may not change with posture. The pattern of crackles changes on listening at sites a short distance apart. This, together with lack of conduction to the mouth, suggests that these crackles originate in small airways. The late opening of small airways implied from physiological tests lends additional support to this view.

End expiratory and early inspiratory crackles are heard in airflow obstruction. They are probably the result of the passage of boluses of air through intermittently obstructed airways, and are heard at the mouth, with or without the aid of a stethoscope. Other sounds to listen for are pleural crackles (Table 5), clicking sounds in pneumothoraces and a splash with air and fluid in the pleural cavity.

Wheezes (Table 6)

Wheezes are produced in airways whose walls are nearly in contact and oscillate to produce a single note. The pitch is dependent on the nature of the obstruction and the velocity of the gas passing through it, but is not dependent upon the density of the gas or the length and diameter of the adjacent airway. A fixed stenosis caused by a tumour, foreign body or stricture produces a monophonic wheeze often present on inspiration as well as expiration. If the narrowing is in a single lobar bronchus the wheeze may be out of phase with airflow at the mouth. In some situations wheezing may be absent despite severe airway narrowing. In airflow obstruction with carbon dioxide retention ventilation may be reduced to such an extent that airflow is insufficient to cause wheezing. In severe asthma the obstruction may be so far out in the bronchial tree that airflow is too slow to produce wheezing.

Table 6. Wheezes[1]

Wheezing	Associated conditions	Timing	Number of pitches	Audible at mouth
Monophonic	Large airway obstruction	Inspiratory and/or expiratory	One	Yes (stridor)
Polyphonic	Chronic airflow obstruction Asthma	Expiratory	Many	Yes
Random monophonic	Asthma	Inspiratory and expiratory	Few	Yes
Sequential inspiratory	Fibrosing alveolitis	Inspiratory usually late preceding crackles	Few	No

[1] Note that in severe asthma and in hypercapnic respiratory failure there may be a paradoxical absence of wheezing despite severe airway narrowing.

Voice Sounds

Consolidated lung allows the transmission of the higher frequency components of speech to the surface so that the spoken words become recognizable. Whispering is made up of mainly high frequency sounds which are normally filtered out but become audible over consolidated or fibrosed lung. Bronchophony and whispering pectoriloquy have the same implications as bronchial breathing. They may also be heard over a collapsed upper lobe because of the proximity of the trachea, even though the upper lobe bronchus is obstructed. A small amount of pleural fluid filters out the low frequency components of speech and this gives the voice sounds at the upper level of a pleural effusion a peculiar 'bleating' quality known as aegophony.

Other Signs Associated with Respiratory Disease

There are a number of signs which should always be looked for in assessing the patient with respiratory disease. Central cyanosis is best looked for in the buccal mucosa on the inside of the lower lip. It implies the presence of 5 g of desaturated haemoglobin but it is an unreliable sign. In a young asthmatic cyanosis indicates a severe ventilatory defect, which requires emergency treatment. The face may reveal the cyanosed, plethoric complexion of the 'blue bloater' with chronic hypercapnia and often polycythaemia. In superior vena caval obstruction the face appears swollen and cyanosed with watery eyes and distended non-pulsatile neck veins.

The hands should be examined for the bounding pulses, warm extremities and flapping tremor of carbon dioxide retention. Clubbing of the fingers is assessed by looking for an increase of the angle between nail and cuticle above the normal 140°, increased longitudinal and lateral curvature of the nail and sponginess of the nailbed. Clubbing is sometimes associated with hypertrophic pulmonary osteoarthropathy which presents as stiffness, swelling and tenderness at the ends of the long bones, most commonly at the knees, ankles, wrists and elbows. The usual cause is a bronchial neoplasm, although it is found in 50 per cent of cases of pleural fibromas.

The neck should be examined for the jugular pulse, lymphadenopathy and, in the presence of stridor, a thyroid extending retrosternally should be excluded. In right ventricular failure secondary to pulmonary disease there may be a raised jugular venous pressure, peripheral oedema and an enlarged liver. On auscultation a third heart sound may be present at the left sternal edge and occasionally a tricuspid regurgitant murmur. The jugular venous pressure may be difficult to assess in patients with airflow obstruction because of the high positive pleural pressure during expiration.

In the eyes, iridocyclitis is a feature of tuberculosis and sarcoidosis and phlyctenular conjunctivitis may be found in tuberculosis. Examination of the fundi may reveal choroidal