Diagnosis and Management of Respiratory Diseases

GRAHAM K. CROMPTON
MB FRCPE

Diagnosis and Management of Respiratory Diseases

GRAHAM K. CROMPTON
MB FRCPE

Consultant Physician, Respiratory Unit
Northern General Hospital, Edinburgh
Senior Lecturer in Medicine and Respiratory Diseases
University of Edinburgh

BLACKWELL SCIENTIFIC PUBLICATIONS
OXFORD LONDON EDINBURGH
BOSTON MELBOURNE

Foreword

For many years Dr Graham Crompton has been educating me in respiratory disease, to my great advantage. I am delighted that he is now applying his talents to a wider audience. Utilising to the full his extensive clinical experience and his skills as a teacher, he has succeeded in crystallising out the essentials of respiratory disease for the benefit of undergraduate students and junior staff in training. I particularly like, and so will they, the summary sections in which he lists the most important points to be remembered about a particular disease. Though he gives science its due, he does not, unlike some such books written by medical scientists, give it more than its due. This is primarily a book for budding clinicians and I am sure that it will help to bring them to full flower. I wish it every success.

Sir John Crofton

Preface

This book is the first of a series that will cover the major fields of medicine and the sub-specialities.

It is a comprehensive account of the diagnostic features and management of respiratory disorders presented clearly to facilitate easy reference. Each topic is set out clearly under separate headings, and the chapters are summarised by special points of emphasis.

The book is a valuable aid for the clinician faced with a problem and it will be useful also to candidates preparing for a higher degree, general practitioners and medical students who require a concise review of up-to-date knowledge presented in authoritative style without being dogmatic.

Contents

Chapter 1

Clinical Findings

Introduction

There are six common symptoms of respiratory disease. Cough and breathlessness are features of almost all respiratory disorders. However, by taking an accurate history of each individual symptom and its relationship to others it is often possible for the correct diagnosis to be suggested by the history alone. A likely diagnosis or short list of possible diagnoses should be formulated before physical examination and the presence, or absence, of abnormal physical signs can then be integrated with the history to produce a rational clinical diagnosis.

There are five common groups of bronchopulmonary disorders which are responsible for the vast majority of symptoms and abnormal physical signs. These are:

- Simple infections
- Tuberculosis
- Tumours
- Respiratory disorders secondary to cardiovascular disease:
  - Pulmonary oedema
  - Pulmonary thrombo-embolic disease
- Obstructive airways diseases.

If these common disease categories are considered in the assessment of all symptoms, abnormal physical signs and radiographic abnormalities, diagnosis is usually relatively straightforward in all but a few rare diseases.

Symptoms

The six common symptoms are:

Cough
Sputum
Haemoptysis
Breathlessness
Wheeze
Pleuritic chest pain.

Cough

Cough is the most frequent symptom of all and is a manifestation of almost all airways diseases and many pulmonary pathologies. Coughing involves a number of respiratory manoeuvres:

An initial inspiratory manoeuvre.

Occlusion of the glottis by closure of the vocal cords.

Contraction of the respiratory muscles against a closed glottis to produce a positive pressure within the airways.

Rapid opening of the vocal cords to allow explosive exit of air (and secretions) from the trachea.

Hence the clinical characteristics of 'normal' cough can be altered by:

1. Inability to breathe in efficiently, e.g. weakness or paralysis of the respiratory muscles: feeble cough. Obstruction to the larynx or main airways: inspiratory croup or stridor.
2. Inability to close and open the glottis, e.g. left vocal cord paralysis: bovine cough. Laryngeal oedema: croup.
3. Inability to use respiratory muscles efficiently, e.g. polyneuritis, myasthenia gravis: feeble cough or inability to cough.
4. Inefficient explosive expiration – as well as respiratory muscle and vocal cord abnormalities the usually forceful release of air from the larynx can be impaired by airways obstruction – e.g. chronic bronchitis: ineffective paroxysms of coughing associated with expiratory wheeze.

Although most diseases cause cough which has no specific characteristics, valuable diagnostic information can be obtained from the history such as the time cough is most troublesome, its duration and association with other symptoms. For instance,

cough troublesome first thing in the morning after a good night's sleep suggests chronic bronchitis, whereas cough associated with wheeze in the middle of the night is more typical of asthma. Persistent distressing cough in an adult smoker must always raise the suspicion of bronchial carcinoma.

The association of sputum production with cough is obvious. A persistent unproductive or 'dry' cough in an adult could indicate bronchial carcinoma, and a productive or 'loose' cough of long duration and brought on by changes in posture is characteristic of bronchiectasis. In the early stages of infections of the respiratory tract cough is often unproductive.

Sputum

Sputum production is always indicative of disease. Some patients, especially children and women, cough up sputum but swallow it. Careful assessment of the appearance of sputum is extremely important and useful information can be obtained from estimating sputum volume. Viscosity varies considerably.

SPUTUM APPEARANCE

Sputum may be serous, mucoid, purulent or mucopurulent.

Serous sputum indicates excessive non-infected production of bronchopulmonary secretion. Serous sputum is frothy or watery and is often described by patients in these terms – e.g. acute pulmonary oedema. A rare but frequently quoted cause of copious watery sputum is alveolar cell carcinoma.

Mucoid sputum, often described by patients as clear, grey or white, indicates excessive secretion of bronchial mucus and is characteristic of chronic bronchitis. Because of the chronicity of symptoms production of mucoid sputum is often accepted as normal and may not be readily admitted.

Purulent and mucopurulent sputum. Sputum containing leucocytes is a turbid yellow or green colour and in the vast majority of cases this indicates bacterial infection. Very rarely an abundance

of eosinophils can produce 'purulent' sputum. Patients often use the terms 'yellow', 'green' or 'dirty' to describe purulent sputum, but care must be taken in the interpretation of 'dirty sputum' as this can merely be a description of inhaled soot by urban dwellers.

The estimation of the amount of pus in the sputum is a good clinical guide of the severity of bacterial infection and a reliable means of assessing response to treatment.

Offensive smelling purulent sputum often said to have a foul taste usually means deep seated bronchopulmonary infection, e.g. lung abscess and bronchiectasis. Occasionally jet black sputum (melanoptysis) can be produced by patients with coalworker's pneumoconiosis.

Other appearances. Blood-staining of sputum must always be regarded as indicative of serious pathology (see haemoptysis). Rusty or orange coloured sputum occurs in pneumococcal infections.

SPUTUM VOLUME

Sputum volume varies tremendously in different pathologies and also in patients with the same disease. Large volumes of purulent sputum suggest bronchiectasis or lung abscess.

SPUTUM VISCOSITY

In general mucoid sputum is more viscid than purulent. Patients with chronic bronchitis often have more difficulty in coughing up mucoid than purulent sputum. The sputum in asthma is characteristically viscid and plugging of bronchi is a major complication of severe episodes. Some patients with asthma produce tubular casts of bronchi (see bronchial casts – asthma).

Haemoptysis

Coughing up blood must always be assumed to be of serious significance and always warrants appropriate investigation. It is rarely difficult to distinguish haemoptysis from haematemesis but sometimes bleeding from the nose and pharynx may simulate

haemoptysis. The amount of blood coughed up can range from large volumes (frank haemoptysis) through blood staining to streaking or flecking of sputum. The most frequent causes of haemoptysis are pulmonary infarction, bronchial carcinoma, tuberculosis and bronchiectasis. These diseases must always be considered in all patients who cough up blood. Useful diagnostic clues are:

1. Frank haemoptysis usually indicates pulmonary infarction, tuberculosis or bronchiectasis.
2. Haemoptysis preceded by, or associated with, purulent sputum often indicates bronchiectasis, suppurative pneumonia or lung abscess.
3. Blood streaking of sputum must always be regarded as an ominous symptom since the most common cause is bronchial carcinoma.
4. Recurrent haemoptysis over a number of years is more likely to be due to a relatively benign disease, e.g. bronchiectasis.

Breathlessness (dyspnoea)

Breathlessness can be said to be present when a patient is aware that breathing involves conscious effort. There are many respiratory causes of breathlessness but the majority can be loosely divided into two main groups:

Breathlessness associated with airways obstruction

Airways obstruction increases the work of breathing and is associated with wheeze or stridor.

Breathlessness associated with impairment of inflation of the lungs in the absence of airways obstruction (restrictive disease)

Chest wall causes: Neuromuscular – polyneuritis, poliomyelitis and myasthenia gravis. Skeletal – severe chest deformity and ankylosing spondylitis.

Pleural causes: Pleural effusion, pleural fibrosis and pneumothorax.

Pulmonary causes: Any condition which decreases pulmonary compliance (increases the 'stiffness' of the lungs) – fibrosis in its many forms, tumour, especially lymphatic carcinomatosis, pneumonia and oedema.

Hypoxaemia if severe and especially if of sudden onset can cause breathlessness by stimulation of receptors in the carotid bodies and aorta. Chemoreceptor stimulation can augment breathlessness in diseases such as acute pulmonary oedema and is possibly the major cause of dyspnoea in conditions such as massive pulmonary embolism.

Breathlessness is not only a manifestation of almost all bronchopulmonary disease and many of the cardiovascular system, but it is the only respiratory symptom that is a common manifestation of hysteria (hysterical hyperventilation).

IMPORTANT HISTORICAL POINTS

1. Duration
2. Speed of onset (see Table 1.1)
3. Association with other symptoms particularly wheeze and chest pain
4. Relationship to exertion
5. Spontaneous breathlessness at night – 'paroxysmal nocturnal dyspnoea' – is a very common symptom of cardiac disease but nocturnal breathlessness is also characteristic of bronchial asthma.

GRADING OF BREATHLESSNESS

Breathlessness is always made worse by exertion and it is useful clinically to assess the severity of this symptom by relating disability to everyday activity – e.g. breathlessness when walking up hills, on the level or at rest.

Wheeze (and stridor)

Wheeze is frequently associated with breathlessness and is a manifestation of obstructive airways diseases such as asthma and chronic obstructive bronchitis. It is clinically most marked during expiration but only a minority of patients appear to be aware of

TABLE 1.1. Rapidity of onset of breathlessness.

Acute onset minutes/hours	Subacute onset days/weeks	Chronic onset months/years
Pneumothorax	Pleural effusion	Chronic bronchitis
Foreign body inhalation	Bronchial asthma	Emphysema
Pulmonary embolism	Exacerbation of chronic bronchitis	Bronchial carcinoma
Bronchial asthma	Pneumonia	Fibrosing alveolitis
Pulmonary oedema	Pulmonary oedema	Lymphatic carcinomatosis
Pneumonia	Bronchial carcinoma	Sarcoidosis
Acute bronchitis	Tuberculosis	Pneumoconiosis
Allergic alveolitis	Lymphatic carcinomatosis	Tuberculosis
Hysterical hyperventilation		Thrombo-embolic pulmonary hypertension

this. The relationship of wheeze to exertion, changes of temperature and inhalation of bronchial irritants and possible allergens must be determined. It is also important to enquire about nocturnal exacerbations.

Stridor due to partial obstruction of major airways may be mistaken for wheeze, but stridor is always worse on inspiration. In cases where early stridor is suspected it can be accentuated by asking the patient to cough and then to breathe deeply through a widely open mouth.

Pleuritic pain

Pleuritic pain has many causes, but the most common are:

Infection (simple and tuberculous)
Pulmonary infarction
Malignant disease.

It is usually severe and often described as knife-like, and is characteristically made worse by breathing and coughing. Diaphragmatic pleural pain is often referred to the shoulder. The features of pleural pain are so classical that it is rarely mistaken for other types of chest pain except for fractured ribs.

Central chest pain of non-pleuritic type may simulate cardiac pain and is occasionally produced by tumours of the mediastinum, acute mediastinitis and mediastinal emphysema. Central pain, often described as 'burning', is a common symptom of acute inflammation of the trachea (acute tracheitis).

Examination

General observations

Breathless patients like to sit upright and those with obstructive airways disease fix the shoulder girdle to give the accessory muscles more purchase to aid expiration. Pleuritic pain may induce the patient to compress the chest with the hands in an attempt to reduce chest wall movement and always induces shallow rapid breathing. Patients dyspnoeic because of obstructive airways disease have prolongation of expiration with clearly audible expiratory wheeze. In contrast, the restrictive diseases cause rapid breathing without expiratory difficulty. The presence

or absence of cough is readily apparent. Less ill patients should be observed after exertion (moving in bed, undressing or walking) to assess the degree of breathlessness. Purse-lip breathing always means severe airways disease associated with air trapping (emphysema).

Cyanosis, if immediately apparent, reflects severe hypoxaemia. Colour of the tongue is the best clinical guide of hypoxaemia of respiratory origin (central cyanosis).

Assessment of higher cerebral functions is of extreme importance in severely ill patients. Hypoxia of acute onset, unless very profound, induces a state of severe respiratory distress associated with extreme anxiety. Profound hypoxia can be associated with confusion but disordered cerebral function usually means acute carbon dioxide retention (respiratory acidosis) as well as hypoxia. Drowsiness, confusion and coma occur in severe hypercapnia and sweating, twitching and coarse tremor are also characteristic features.

The hands

Careful examination of the hands is vital:

1. *Finger clubbing.* The respiratory causes of finger clubbing include bronchial carcinoma, intrathoracic suppuration (bronchiectasis, lung abscess and empyema) and fibrosing alveolitis.
2. *Flapping tremor.* Frequently present in uncompensated respiratory acidosis which is much more common than the other cause, liver failure.
3. *Skin temperature of the hands.* Carbon dioxide retention is associated with warm extremities.

Warm, blue flapping hands are found in acute respiratory acidosis.

The neck

The neck must be carefully examined systematically. Engorgement of neck veins can be due to increased intrathoracic pressure during expiration, right ventricular failure or superior vena caval obstruction. Palpation will determine the position of the trachea and enlargement of supraclavicular nodes. Subcutaneous emphysema is usually easily detectable in the neck by palpation.

The eyes

Conjunctival oedema is present in many patients with ventilatory failure and cor pulmonale. Horner's syndrome may indicate an apical bronchial carcinoma. Examination of the fundi will reveal engorgement of retinal veins and occasionally papilloedema in patients with ventilatory failure, and a thorough search for choroidal tubercles must be made in patients thought to have miliary tuberculosis.

The upper respiratory tract

Examination of the nose, mouth, pharynx and larynx, in selected cases, is of course essential. Nasal mucosal swelling and polyp formation is common in patients with allergic disorders.

The chest

There is a tendency for the inexperienced to denigrate the value of clinical examination of the chest and place too much emphasis on radiological findings. Precise diagnosis, however, depends upon the proper interpretation of abnormal clinical findings together with information provided by the chest X-rays. The routine examination of the chest by inspection, palpation, percussion and auscultation is often performed well by inexperienced clinicians, but lack of appreciation of the significance of abnormal findings may lead to misinterpretation. The relevance of each abnormality must be considered as it is detected (e.g. diminished expansion on inspection) so that abnormal signs subsequently elicited (e.g. dull percussion note) should be anticipated in order to allow appreciation of the significance of auscultatory findings (e.g. absent air entry – diagnosis – pleural effusion or collapse). Such an examination technique is much more rewarding than trying to assess the relevance of a host of abnormalities at the end of physical examination.

INSPECTION

The rate and depth of breathing as well as the presence of expiratory wheeze, inspiratory stridor and obvious pain associated with breathing must be noted.

CHEST SHAPE ABNORMALITIES

The most important abnormalities of chest configuration are:

Increase in antero-posterior diameter (barrel chest)

This usually reflects long-standing chronic obstructive airways disease but can be due to air trapping in severe asthma. Genuine increase in AP diameter is always associated with decreased chest expansion and accompanied by use of the accessary muscles (sternomastoids, scaleni and trapezii) which lift the thorax on inspiration but do not necessarily increase expansion. Thus chest movement and expansion are precise terms and are not interchangeable.
Indrawing of intercostal spaces is evidence of marked airways obstruction and is often seen in patients with barrel chest deformity.

Pectus carinatum ('pigeon' chest)

Chronic inflation of the lungs in children, usually due to asthma, causes the characteristic forward bowing of the sternum and indrawing of the lower lateral ribs due to muscular pull of the insertions of the diaphragm (Harrison's sulci).

Thoracic kyphoscoliosis

This is not due to respiratory disease but often causes respiratory problems in adult life because of the ventilation–perfusion imbalance it can produce. Scoliosis produces more respiratory embarrassment than kyphosis.

Pectus excavatum ('funnel' chest)

This congenital deformity of depression of the lower sternum is often cosmetically embarrassing but rarely causes respiratory disease.

Deformities due to accidental trauma and thoracic operations

Most deformities due to uncorrected chest trauma and thoracoplasty operations (rarely performed today) can lead to ventilation–perfusion imbalance.

Lesions of chest wall not associated with deformity. Scars, bruising, eruptions, nodules, swelling, dilated veins, etc. can all be evidence of intrathoracic pathology.

Decreased expansion in absence of chest deformity. Asymmetrical expansion may be apparent on inspection in some cases of gross pleural and pulmonary pathology.

PALPATION

Routine palpation of the chest wall in the search for nodules, localised tenderness, breast lesions and enlarged axillary lymph nodes should precede assessment of chest expansion.

CHEST EXPANSION

Common abnormalities of chest expansion are given in Table 1.2. In absence of chest deformity, the important abnormalities are:

Bilateral diminution of expansion

This is a feature of:

1. All patients with pleuritic pain – pain limits inspiratory effort – chest shape frequently normal – common.
2. Hyperinflation of lungs causing fixation of ribs in the inspiratory position – chest shape abnormal (barrel chest) – common.
3. Bilateral pulmonary diseases causing gross restriction of expansion – e.g. fibrosing alveolitis, lymphatic carcinomatosis – uncommon.
4. Bilateral pleural abnormalities causing restrictive defect – e.g. bilateral pleural effusions and fibrosis – uncommon.
5. Musculo-skeletal diseases – causing restriction of chest

TABLE 1.2. Some causes of decreased chest expansion.

Bilateral	Unilateral
Lung hyperinflation e.g. emphysema asthma	Lung hyperinflation obstructive emphysema (rare)
Restrictive lung diseases e.g. fibrosing alveolitis allergic alveolitis lymphatic carcinomatosis sarcoidosis	Pulmonary disorders atelectasis consolidation localised fibrosis large peripheral tumour
Pleural diseases e.g. pleuritic pain bilateral effusions (rare) bilateral fibrosis (rare)	Pleural diseases pneumothorax pleural effusion empyema pleural fibrosis pleural tumour
Musculo-skeletal disorders e.g. myasthenia gravis polyneuropathies ankylosing spondylitis kyphosis	Musculo-skeletal disorders scoliosis

expansion – e.g. myasthenia gravis, polyneuropathies or ankylosing spondylitis – uncommon.

Unilateral diminution of chest expansion

This is an important and common abnormal finding. The side of decreased chest expansion can always be regarded as the abnormal side. The commonest causes are:

1. Pleural – effusion, fibrosis, tumour and pneumothorax.
2. Pulmonary – collapse, consolidation, localised fibrosis and rarely large peripheral tumour.

PERCUSSION

The percussion note may be normal, dull or hyper-resonant. It is easier to be confident about the presence of dullness than hyper-resonance.