

Essentials of Respiratory Disease

R. B. Cole

second edition

Essentials of Respiratory Disease

R. B. COLE, MA, MD (Cantab.), FRCP (Lond.)

*Consultant Physician,
North Staffordshire Hospital Centre,
formerly Senior Lecturer, Department of Medicine,
Queen's University of Belfast*

(内部交流)



Pitman Medical

First published 1971

Reprinted 1973

Second edition 1975

Pitman Medical Publishing Co Ltd
42 Camden Road, Tunbridge Wells,
Kent TN1 2QD

Associated Companies

UNITED KINGDOM

Pitman Publishing Ltd, London
Focal Press Ltd, London

USA

Pitman Publishing Corporation, New York
Fearon Publishers Inc, California

AUSTRALIA

Pitman Publishing Pty Ltd, Melbourne

CANADA

Pitman Publishing, Toronto
Copp Clark Publishing, Toronto

EAST AFRICA

Sir Isaac Pitman and Sons Ltd, Nairobi

SOUTH AFRICA

Pitman Publishing Co SA (Pty) Ltd, Johannesburg

© R B Cole 1975

ISBN 0 272 79366 3

Cat. No. 21 0442 81

Printed in Great Britain by
The Whitefriars Press Ltd, London and Tonbridge

Preface

Many of the commonest problems in clinical medicine are due to respiratory illnesses and I have always felt that a thorough understanding of the manifestations of pulmonary disease is an invaluable asset to the general physician, particularly in a field of medicine in which a knowledge of physiological principles plays so large a part in the logic of diagnosis and management. In this book I have tried to bring together the techniques of bedside diagnosis, the essential concepts of pulmonary physiology and the clinical characteristics of the commoner respiratory diseases, in the hope of providing a practical guide to clinical problems that may be useful to senior medical students and to doctors in their early postgraduate years.

In writing this book I have been constantly encouraged by Professor John Vallance-Owen, for whose interest and advice I am sincerely grateful. I also acknowledge with gratitude the valuable advice of those who were good enough to find time to read the manuscript, including my father, Dr Leslie Cole, and my friends and colleagues, Professor Peter Elmes, Dr Ross McHardy, Dr Ian Green, and Dr Leslie Capel.

My warmest thanks are due to Miss Anne Wilkie and to Mrs Joan Beattie who undertook the tedious task of typing the manuscript, and to Mr J. A. Robin and the staff of the Photographic Department, Belfast City Hospital, for their expert assistance with the diagrams. I am also grateful to Butterworth and Co Ltd for permission to reproduce certain material previously published in *Medical Progress* 1969/70.

R.B.C.
Belfast, 1970

Preface to the Second Edition

The preparation of a new edition of this book has given me a welcome opportunity to correct certain inaccuracies and ambiguities and to revise the text in the light of recent advances in the diagnosis and management of respiratory disorders. The reviewers of the first edition made many constructive comments which I have endeavoured to incorporate into this edition; few changes have seemed necessary in the first two sections of the book dealing with clinical methods and physiological principles but some rearrangements and additions have been made to the third section concerned with the commoner varieties of respiratory disease, including a chapter dealing with lung disorders induced by drugs. I have, however, tried to avoid excessive expansion of the text, for this remains a basic textbook designed for medical students and doctors in training rather than a reference book for specialists.

I would like to thank again all those whose help is acknowledged in the preface to the first edition, particularly Professor Peter Elmes who has once more given me invaluable advice. I am greatly indebted to Miss Diane Stirling for her able help in producing this revised book.

R.B.C
Belfast, 1975

Contents

	<i>Page</i>
<i>Preface</i>	v
<i>Preface to Second Edition</i>	vi
 PART I—CLINICAL METHODS	
1 The History	3
2 External Signs of Lung Disease	12
3 Examination of the Chest	19
4 Cardiovascular Aspects of Lung Disease	31
5 The Chest X-ray	40
 PART II—PULMONARY PHYSIOLOGY IN RELATION TO DISEASE	
6 Concepts of Pulmonary Physiology	51
The Alveolus	
Gas Exchange	
Breathing	
7 Testing Pulmonary Function	70
Tests of Gas Exchange	
Tests of Ventilatory Function	
Practical Assessment of Lung Function	
8 The Circulation and Lymphatic Drainage of the Lungs	90
9 Disordered Function in Lung Disease	97
 PART III—VARIETIES OF LUNG DISEASE	
10 Bronchial Asthma	115
11 Chronic Obstructive Lung Disease	128
Chronic Bronchitis and Emphysema	
Bronchiectasis	
Cystic Fibrosis	
12 Spontaneous Pneumothorax	148
13 Acute Infections of the Respiratory Tract	154
Acute Virus Infections	
Pneumonia	
Complications of Pneumonia	
Lung Abscess	

14	Pulmonary Tuberculosis	179
15	Fungal Infections of the Lungs	196
16	Sarcoidosis	202
17	Diffuse Fibrosing Alveolitis	210
18	Occupational Lung Disease	216
19	Malignant Disease of the Lungs	230
	Carcinoma of the Bronchus	
	Bronchial Adenoma	
	Mesothelioma of the Pleura	
	Malignant Invasion of the Lungs	
20	Pulmonary Manifestations of Connective Tissue Disorders	249
21	Pulmonary Thromboembolic Disease	258
22	Drug-induced Lung Disease	268
	<i>Bibliography</i>	277
	<i>Index</i>	285

PART I

Clinical Methods

1

The History

Throughout his interview with the patient the clinician's aim is to accumulate accurate clinical data which must be assessed mentally, tested by further questions, and stored in the mind in terms of a differential diagnosis. Besides listening to the patient it is important at this stage to observe every detail of his behaviour and appearance, noting all abnormalities and directing questions that may reveal their cause. In effect, during the interview a clinician must be a highly efficient and versatile computer, using his humanity and sympathy and his powers of observation as the vital means for obtaining the information needed to reach a correct diagnosis.

That the patient should be encouraged to give his own account of the symptoms is a well-tried maxim, and some patience may be needed before a general outline of the illness is revealed. Nevertheless, the main points that must be clearly understood at the end of this early stage of the interview provide a framework upon which the differential diagnosis must be built by direct questions. These main points are as follows:

1. The major symptoms and their duration.
2. The precise time and mode of onset of the first symptom.
3. The sequence and mode of onset of further symptoms.
4. The progress of the illness and of individual symptoms.

Once these points have been clearly established the clinician must turn to detailed probing of the patient by direct questions, designed not only to enlarge on the descriptions already obtained but also to acquire circumstantial evidence for one or other of the possible diagnoses that have been suggested by the patient's account. An example of this type of question might be, with regard to a primary complaint of haemoptysis, an enquiry into recent swelling or pain of a leg which might have seemed too slight or apparently too irrelevant to have been recounted by the patient but which, if present, would point to a possible diagnosis of pulmonary embolism.

It should be clear that these direct questions will cut across any

formal division of present, past or family history or systems of questioning that we all learn as junior students. Nevertheless, some such useful system should be deeply ingrained in the mind of the clinician so that once he has fully enlightened himself as to the patient's present illness he may survey the data he has already obtained and seek for unsuspected relevant information by formal questions about the patient's past history, his family, his occupation and social background that may lead to a modification or enlargement of the mental diagnosis he has already established.

By the time history-taking is completed the doctor should have a clear idea of the abnormal clinical signs he is likely to find on detailed examination of the patient, which will enable him to confirm or reject the various diagnoses that have already been suggested during the interview. However, he must bring to bear the same critical accuracy in clinical examination as in history-taking, remembering that new data may be revealed which will alter his earlier conclusions or raise new possibilities.

Symptoms of Respiratory Disease

These principles of the science of history-taking are applicable in all fields of medicine, but among diseases of the respiratory system certain symptoms are so common that more detailed consideration of their incidence and significance is necessary here. They are shortness of breath, cough, sputum, haemoptysis, and chest pain.

SHORTNESS OF BREATH

The sensation of shortness of breath, or dyspnoea, is a subjective one although the complaint may be supported by objective signs such as laboured or rapid breathing and evidence of hypoxia or hypercapnia. The sense organs and nervous pathways that convey the sensation are elusive, but it has been suggested that dyspnoea consists of an unpleasant awareness of inappropriate effort in performing the normal act of breathing (see page 68). This excessive effort may be demanded by neurochemical stimuli such as hypercapnia, or may be due to abnormal stiffness of the lungs as occurs in diffuse pulmonary fibrosis, or to weakness of the respiratory muscles as in myasthenia gravis.

Dyspnoea may present either as a complaint of persistent or progressive intolerance of exercise or as an acute attack, often paroxysmal. A complaint of reduced *exercise tolerance* should be defined precisely by questioning the patient's ability to walk on the flat or up an incline, to climb stairs and to perform activities such as carrying burdens, doing housework and gardening. These details should be recorded so that comparisons may subsequently be made,

particularly with regard to the effectiveness of treatment. The length of the history and the progress of the symptoms may give some guide to the nature of the underlying pathology, but exertional dyspnoea is a symptom of many conditions, not only diseases of the cardiac and respiratory systems but also of hyperthyroidism, obesity, anaemia, and anxiety. It is therefore an indication for enquiry into further symptoms which may reveal its cause, such as cough, wheeze, chest pain, palpitation and dependent oedema.

The mode of onset of dyspnoea may be more informative in diagnosis. Severe dyspnoea of sudden onset in a previously normal person suggests either *spontaneous pneumothorax*, which is often associated with pain or tightness in the chest, or *massive pulmonary embolism*. Recurrent paroxysms of dyspnoea suggest *bronchial asthma*, especially when precipitated by emotion, exposure to a known antigen, by exercise or by breathing cold air. Evidence of other forms of hypersensitivity and of a family history should also be sought. Bronchial asthma may present in an otherwise normal person or against a background of permanent exertional dyspnoea caused by recurrent attacks and by bronchial infection. The distinction between the dyspnoeic attacks of the middle-aged asthmatic and those of the chronic bronchitic is often difficult since both are liable to have chronic exertional dyspnoea with recurrent exacerbations due to bronchial infection.

Acute attacks of dyspnoea occurring when the patient is recumbent at night indicate impaired cardiac output, due usually to left ventricular failure or to severe mitral stenosis. The history of *paroxysmal nocturnal dyspnoea* is characteristic: the patient wakes from sleep with an intense feeling of suffocation, getting relief after a few minutes by sitting on the side of the bed or by standing at the window. Often he admits that this occurs when he slips down from his pillows during sleep, and enquiry into the number of pillows used by the patient may reveal his intolerance of the horizontal position, a condition known as *orthopnoea*. It is due to a reduction in the influence of gravity upon the peripheral circulation, causing increased pulmonary venous engorgement at a time when the pulmonary circulation is already overfilled because of left ventricular failure or mitral stenosis. Orthopnoea also occurs in patients with severe airways obstruction, but in this case it is due to the impaired ventilation that results from limitation of diaphragmatic movement when they are lying down.

Wheezing is due to bronchial narrowing, either by secretions or bronchospasm, and tends to be most marked during expiration, when slight bronchoconstriction occurs physiologically. It is therefore a characteristic symptom in asthma, bronchitis, and pulmonary oedema.

and is often associated with coughing as the patient attempts to clear obstructing mucus. Wheeze in a localised area, unaffected by coughing, may indicate partial obstruction of a bronchus due to a neoplasm or an inhaled foreign body.

A complaint of dyspnoea is an indication for further interrogation of the patient with regard to other symptoms of respiratory or cardiac disease, his past history of respiratory infection, a family history of asthma, his occupational background, and his smoking habits. In examination, the clinician must be alert for evidence of cardiac arrhythmias, cardiac failure, and mitral stenosis, for signs of pneumothorax or pleural effusion, and of infection or neoplasm in the airways and lung parenchyma, for evidence of diffuse pulmonary fibrosis, for skeletal abnormalities of the thorax, and for neuromuscular disorders.

COUGH, SPUTUM, AND HAEMOPTYSIS

Cough is due to irritation of the mucous membrane anywhere in the respiratory tract between the pharynx and the small bronchi. To the clinician, cough is most commonly an indication of infection of the airways or lung parenchyma, when inflammation and excessive mucus production act as irritant stimuli. Other important causes of cough are inhaled foreign bodies, most often occurring in children, pulmonary oedema, and irritation of the mucous membrane by bronchial tumours arising either within or outside the bronchi, or by enlarged lymph nodes encroaching upon the airway from the mediastinum.

An unproductive cough denotes airway irritation without telling much about the underlying cause; only rarely does the character of the cough itself provide information about the underlying pathology, as does, for example, the long, inspiratory 'whoop' of whooping cough or the 'bovine' cough which characterises adductor paralysis of a vocal cord due to interruption of the fibres of the recurrent laryngeal nerve by tumour or by aortic aneurysm. In general, the diagnosis of the cause of cough depends not on an analysis of the cough itself, on its painfulness, its paroxysmal nature, its precipitating factors, but upon associated symptoms and signs. Common examples are the history of nasal discharge and sore throat, which usually precede the cough of acute pharyngitis or laryngitis; and the association of haemoptysis with cough in a smoker, which arouses the suspicion of bronchial carcinoma.

The most informative coincidental feature of cough is the character of the material coughed up. Occasionally, the cough is actually dry, either because there is no excessive mucus production or because the

mucus is too viscid or the patient too weak to expectorate it. This type of dry cough is characteristic of the early stages of acute bronchitis and pneumococcal pneumonia, and during an acute attack of bronchial asthma. More commonly the patient protests that his cough is dry when in fact he swallows his sputum; in such a case a sputum specimen must be obtained by personal encouragement, if necessary with the help of postural drainage.

The patient's description of the *sputum* must be confirmed by inspection. Large quantities of sputum in excess of 50 ml in 24 hours suggests three possible diagnoses: if it is purulent, either chronic bronchitis or bronchiectasis is likely and the possibility of underlying cystic fibrosis should be borne in mind; in chronic bronchitis the sputum may be quite copious even when it is not purulent, but excessive quantities of thin watery sputum should raise the suspicion of alveolar cell carcinoma. Although it is commonly supposed that bronchiectasis is characterised by a large sputum volume it must be remembered that localised bronchiectasis, particularly in the well-drained upper lobes, may occur without much sputum.

The sputum may be black (if it contains smoke or carbon particles), and green, yellow, or khaki if it contains pus. An excessive number of eosinophils in the sputum may give it a lemon-yellow appearance. The observation of pus is an indication for bacteriological examination, to determine the infecting organism and its sensitivities. Specimens sent for bacteriological examination should be freshly taken and contain obvious pus. Muroid sputum is white, grey or 'clear', and is characteristic of quiescent chronic bronchitis, diffuse fibrosing alveolitis and bronchial asthma. During acute attacks of asthma the sputum is more than usually viscid and difficult to expectorate, becoming 'looser' and sometimes frothy when the attack begins to subside. Bronchial casts are often found in such sputum following an acute asthmatic attack.

The coughing up of blood-stained sputum, or *haemoptysis*, is an indication for thorough investigation to exclude serious underlying disease. If the sputum is 'rusty' in appearance due to admixture of haemoglobin breakdown products, pneumococcal pneumonia is strongly suggested. Pink frothy sputum occurs in pulmonary oedema because of the exudation of red cells with oedema fluid into the airways. Otherwise, the appearance of the sputum varies only as to the relative proportions of blood and mucus, from 'frank blood' to blood-staining or streaking. The quantity of blood produced and the frequency of the haemoptysis do not give a reliable guide to the underlying cause, but the relationship of the haemoptysis to other symptoms and signs may be of prime importance, as for example the coincidence of haemoptysis and pleuritic pain ten days after an

abdominal operation, indicating the diagnosis of deep venous thrombosis and pulmonary embolism.

The commoner causes of haemoptysis which should always be considered, and excluded by thorough investigation, are:

1. Bronchial tumour, either carcinoma or carcinoid.
2. Pulmonary infarction.
3. Pulmonary tuberculosis.
4. Acute pneumonia.
5. Bronchiectasis.
6. Lung abscess.
7. Mitral stenosis.
8. Pulmonary oedema.
9. Chronic bronchitis.
10. Trauma, particularly lung contusion.
11. Abnormalities of blood coagulation.

CHEST PAIN

Chest pain should be analysed in a fashion similar to that of pain elsewhere in the body, by enquiry into its: 1, mode of onset; 2, character; 3, site; 4, area of radiation; 5, duration; 6, intensity; 7, precipitating or aggravating factors; and 8, means of relief. The most characteristic pain of lung disease is that due to inflammation of the pleura. Typical pleurisy may come on gradually or abruptly, is sharp or knife-like in character, and usually occurs at the site of inflammation, although diaphragmatic pleurisy is often referred to the point of the shoulder, and pleurisy in the lower thorax may be referred to the back or abdomen. The pain is continuous and severe, but is characteristically sharply aggravated by movement, coughing or deep breathing, so that the patient adopts a pattern of rapid, shallow respiration and tends to avoid unnecessary movement. The commonest causes of pleuritic pain are pulmonary infection or infarction, but many other conditions may present with, or be complicated by pleurisy, including primary and secondary malignant tumours, tuberculosis, spontaneous pneumothorax, and rheumatoid arthritis. The development of pleural effusion is usually accompanied by some relief of pain, due probably to limitation of lung movement by the fluid and to separation of the inflamed pleural surfaces.

Retrosternal pain accompanies acute tracheitis and bronchitis, usually aggravated by coughing, and relieved when the patient begins to bring up sputum. A rather vague, indefinite aching pain often occurs in bronchial carcinoma situated in the hilar region, poorly localised but sometimes referred to the neck, back or abdomen. This pain is most troublesome at night and is relieved by activity or occupation.

In assessing the cause of chest pain the clinician must give due consideration to alternative possibilities such as myocardial ischaemia, aneurysm of the aorta, oesophageal obstruction or reflux, sub-diaphragmatic disease, Bornholm disease and affections of the intercostal muscles, herpes zoster, and diseases of the spinal cord, vertebral column and ribs.

Significance of Previous Illness and Family History

Enquiry into the past history may reveal that the present illness is either a recrudescence or a complication of a previous one. Similarly, the health of other members of the patient's family may have a direct bearing on his present illness. Since patients do not usually give a complete answer to questions such as, 'Have you ever had any serious illness in the past?', or 'Do you come from a healthy family?', specific questions must be put which are designed to throw light on the background of the present complaint. A past history of the following diseases may be relevant:

1. *Asthma*. This disorder is well-recognised by the patient who can often give a good account of spasmodic attacks going back to childhood. The opportunity should be taken to enquire about other allergies such as hay fever and skin disorders, and also about the incidence of asthma in other members of the patient's family.

2. *Bronchitis*. Recurrent illnesses with cough and purulent sputum are the customary precursors of chronic bronchitis, respiratory failure and cor pulmonale. Often such illnesses have been present since childhood when a severe attack of measles or whooping cough with secondary pulmonary infection may have initiated pulmonary damage.

3. *Pneumonia and Pleurisy*. These illnesses are often symptomatic of underlying lung disease, e.g. chronic bronchitis and emphysema. A history of recurrent attacks can sometimes be obtained in pulmonary thromboembolic disease, due to repeated episodes of pulmonary infarction. In the middle-aged a short history of recurrent pneumonia that fails to clear up satisfactorily is one of the commonest presenting features of bronchial carcinoma.

4. *Tuberculosis*. Previous tuberculosis infection may have caused lung damage which is responsible for bronchiectasis and pulmonary fibrosis. Recrudescence of quiescent pulmonary tuberculosis should also be considered.

5. *Cystic Fibrosis*. Enquiry into the childhood and family history of patients who present with symptoms and signs of bronchiectasis may rarely reveal a chronic respiratory history throughout early life, and similar illness or early death among one or more of the patient's siblings.

6. *Surgical operations*, especially those on the thorax or abdomen, radiotherapy, motor accidents, and any illness that imposes a period of prolonged bed rest may be responsible for pulmonary damage from fibrosis, infection, or pulmonary embolism.

Occupational and Environmental History

Occupational exposure to dust or antigenic material by inhalation is a common cause of lung disease, especially pulmonary fibrosis. The clinician should be careful to enquire not only into the patient's present occupation but into all other occupations in which he has been engaged throughout his working life. The actual nature of the work should be enquired into, with specific reference to the type of dust and the duration of exposure. In reaching a clinical diagnosis the following occupational hazards should be borne in mind:

1. As a cause of pulmonary fibrosis:
 - (a) The mining, quarrying or handling of coal, or of any rock containing silica.
 - (b) Sand-blasting.
 - (c) Foundry-work, particularly the freeing of sand from castings.
 - (d) The manufacture of china or earthenware.
 - (e) Stone-masonry.
 - (f) Grinding, using a sandstone grinder.
 - (g) Boiler-scaling.
 - (h) The handling of asbestos.
 - (i) The manufacture or use of beryllium.
 - (j) Farmers or horticulturists who are liable to handle mouldy hay.
 - (k) Mushroom growing.
 - (l) Handling sugar-cane pulp (bagassosis).
 - (m) Bird-fanciers, including chicken farmers and pigeon breeders.
2. As a cause of chronic obstructive airways disease:
 - (a) Spinning or manipulation of cotton or flax (byssinosis).
 - (b) Inhalation of cadmium fumes, e.g. among scrap-metal cutters.
3. As a cause of pulmonary malignant disease (carcinoma or mesothelioma):
 - (a) Exposure to asbestos dust (e.g. workers in the building or ship-building industries).
 - (b) Workers in the chromate industry.
 - (c) Miners exposed to radioactive dust.