POSTGRADUATE MEDICINE

Fifth edition

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PREFACE TO FIFTH EDITION

Medical textbooks fall into two main groups: those intended for medical students and those which are large, comprehensive and intended mainly for reference purposes. It seemed to me that there was need for a book intended to be read from cover to cover which served the needs of those engaged in general medicine and which attempted to bridge the gap between the theoretical knowledge of the final year medical student and the practice of sound, safe and orthodox medicine.

About the large standard medical text-book, Professor Tony Mitchell says trenchantly

> . . . because it is so massive and because I walk to work I could not take it home. It is like an early bible, chained to its lectern so that you need to come to it rather than taking it with you . . . . Dinosaurs died out because they were too big, could not adapt to change and because their brains were too far from their genitalia. In my view the same fate will befall the single BIG book.

The first book to recognize that MRCP candidates could benefit from a book written specifically for them was Maurice Pappworth's *Primer of Medicine*, the second was *Postgraduate Medicine*. Since then the whole style of the examination (and examiners) has changed and many books, clinical courses and mock examinations have been produced (and many have subsequently fallen by the wayside!). *Postgraduate Medicine* has retained its popularity. From the correspondence I receive from current candidates and from many who passed the Membership examination up to two decades ago the book's philosophy, content and style are found useful. Earlier editions have been 'pirated' in at least four different Far Eastern cities which is flattering for authors (but does seem to worry publishers!) and the book has been translated into Spanish and Italian.

For the fifth edition I have revised the whole book continuing to try and cater for those who require a reasonably concise – albeit personal overview of general medicine. The scale of the book has also made it useful for candidates for postgraduate diplomas other than the MRCP – it is popular with candidates for MRCGP, FRCS, MRCOG, FRCR and MRCPath examinations. Basic medical knowledge is assumed, but where I know that biochemical or physiological principles are often misunderstood I have sometimes gone back to first principles.

It might be questioned these days whether there is still a place for a single-author review of general medicine. However, it is known that consultant physicians with a special interest spend at least 80% of their time practising general medicine; candidates for the MRCP diploma are still required to cover the whole span of general medicine – it does not therefore seem unreasonable that their examiners should be expected to do the same.

I take heart from a quotation from Sir Geoffrey Vickers' *The Art of Judgement*:

> Even the dogs may eat the crumbs which fall from the rich man's table; and in these days, when the rich in knowledge eat such specialized food at such separate tables, only the dogs have a chance of a balanced diet.

The eponyms are a personal quirk; they either intrigue or sometimes antagonize but provided candidates don't take them too seriously it must be of some passing interest to any reader that, for example, Kussmaul had to have a sword swallower on whom to attempt the first gastroscopy or that Marey, to prove his law, had to raise the blood pressure of a horse by squeezing its aorta by means of his hand in the horse's rectum or Koch at the age of 50 ran off with a chorus girl or that Henle had to have the permission of the Church to propound the view that the body is made of individual cells.

The bane of a book is its index; once again I am grateful to Mr Frank Wallis of Stratford upon Avon who has prepared a most thorough index. Peter Altman and Helena Watson of Chapman and Hall have been as helpful, encouraging and cheerful as one could have wished. It is a particular pleasure for me to be associated with the original publishers of my favourite author – Anthony Trollope.

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1990

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1 CARDIOLOGY

1.1 INTRODUCTION

The availability of sophisticated investigations has not replaced the need for careful clinical assessment. There is good evidence that in three-quarters of patients careful history-taking leads to the correct diagnosis; in two-thirds of patients the physical examination only confirms the working diagnosis. In only 1 in 12 patients do subsequent investigations alter a diagnosis made on clinical grounds (Hampton *et al.*, 1975).

1.1.1 Interpreting Laboratory Results (Fraser and Fogarty, 1989)

When a single specimen is assayed several times the results differ each time and the variation is known as 'analytical imprecision'. In addition, each substance measured may have cyclical variations in the body (e.g. diurnal or circadian) and also there are inherent biological fluctuations around the normal value – these are termed 'within-subject variations'. The 'critical difference' between two sequential estimations is the difference which has to occur before statistical significance can be attached to the result. The 'significant critical difference' ($P < 0.05$) for various investigations are often suprisingly large, e.g.

Sodium	3%
Potassium	14%
Urea	30%
Creatinine	14%
Calcium	5%
Glucose	15%
Hb Alc	21%
Haemoglobin	8%
Platelets	25%

1.2 DYSPNOEA

The grading of dyspnoea is helpful in recording the progress of symptoms and in briefly communicating the degree of disability. The

accepted convention is:

Grade 1. Dyspnoea on severe exertion such as running up two flights of stairs.

Grade 2*A*. Dyspnoea on moderate exertion such as walking normally up two flights of stairs.

Grade 2*B*. Dyspnoea on mild exertion such as walking slowly up one flight of stairs.

Grade 3. Dyspnoea on minimal exertion such as walking from room to room.

Grade 4. Dyspnoea at rest.

It is of value to record the extent of interference with the patient's way of life, for example, dyspnoea which prevents a housewife from doing the family shopping or a husband from driving the family car is usually severe. It is helpful to record the exact disability which breathlessness imposes on a patient. For example can a housewife make one, two or three beds 'on the trot' without having to rest in between each, can she sweep out one room without resting, can she carry the week's shopping home or can she run downstairs to answer the door? The number of flights of stairs that can be climbed at a normal pace is a useful guide; or if the patient has only tried one flight – does he think he could climb a second flight if there was one? A simple individualized hospital corridor walking test can be used which shows good correlation with patient's actual disability and can be useful in assessing progress or decline (Butland *et al.*, 1982).

Dyspnoea is a subjective, uncomfortable awareness of breathing. It is mainly due to excessive use and fatigue of the respiratory muscles. The pulmonary venous congestion which occurs with left-sided heart failure results in congestion of the interstitial lung tissue and airways diminishing the elastic properties of the lungs and increasing the ventilatory effort needed to transfer air through the airways. Normal expiration is probably triggered by a number of reflexes of which the best known is the Hering[1] – Breuer[2] reflex, in which stretch receptors in the alveolar walls are stimulated during inspiration, and impulses pass via the vagus to the respiratory centre which initiates relaxation of the inspiratory muscles. Expiration is a passive process due to the inherent elastic properties of the lungs. The normal rate and depth of breathing at rest are probably mainly due to the inherent rhythmicity of the respiratory centre secondarily modified by afferent reflexes from the lungs and, during exercise, by alterations of the gas tensions in the blood. Congestion of the alveolar walls will accelerate the afferent reflexes (increasing the rate of breathing). Later, breathing becomes further accelerated because of hypoxia of the respiratory centre, due to alveolar-wall oedema interfering with the diffusion of

oxygen from the alveoli into the blood. Early cardiac failure is accompanied by dyspnoea before there is any alteration in pH, oxygen or carbon dioxide content of arterial blood perfusing the respiratory centre. The rapid relief of cardiac dyspnoea by morphia is probably due to a decrease in awareness of breathing as well as depression of the respiratory centre and inhibition of the vagus, slowing the rate of breathing and thereby reducing fatigue of the respiratory muscles.

Paroxysmal nocturnal dyspnoea when lying flat at night, and orthopnoea (*Gk.* orthos: straight) are due to reduced mechanical advantage of the diaphragm, redistribution of oedema fluid from dependent parts, and reduced sensitivity of the respiratory centre during sleep, leading to failure of early compensatory mechanisms and increase in cardiac output and venous return in the recumbent position.

Occasionally, patients who are dyspnoeic due to a pulmonary embolus prefer to lie flat; the reason for this is that they feel faint if they sit upright because they have a low cardiac output.

Breathlessness when the patient lies in a particular position is seen in the uncommon occurrence of a pedunculated tracheal or bronchial polyp.

1.3 CARDIAC PAIN

Ischaemic cardiac pain arises from pain receptors in the myocardium, and is transmitted via the sympathetic nerves to the upper thoracic sympathetic ganglia and thence to the upper five thoracic spinal nerves. This explains the radiation of cardiac pain in the distribution of T 1–5 and the relief of pain by division of these sympathetic ganglia. These nerves supply the upper oesophagus, accounting for the frequent similarity of oesophageal pain and angina; they also supply some of the muscles and ligaments surrounding the shoulder joints, accounting for the reflex spasm and disuse of the left shoulder which may occur following cardiac infarction. Disuse of the joint may be accompanied by a periarthritis and calcification. The left joint is much more frequently involved than the right but the converse is true when periarthritis is due to excessive use, because most people are right-handed. The first thoracic nerve supplies sensation to the inner side of the upper arm and occasionally to the lower arm and little finger accounting for the radiation of cardiac pain down the arm. Distinction between oesophageal and cardiac pain may sometimes be made by infusing dilute hydrochloric acid or dilute sodium bicarbonate through an oesophageal tube, and noting whether this induces the patient's pain. Following a large meal, T-wave changes may occur in the ECG in the absence of ischaemic heart disease, and are due to

slight alteration in the position of the heart. Occasionally, pain due to reflux oesophagitis, motor inco-ordination of the oesophagus and hiatus hernia may induce ischaemic ECG changes, and may be relieved by trinitrin.

The pain of pericarditis is occasionally confused with angina. The lower part of the parietal pericardium alone is pain-sensitive and is supplied by the phrenic nerve (C 4–5). Gross distension of the pericardium with fluid gives rise to a dull ache in the front of the chest which may be referred to the back of the neck and shoulder in the distribution of C 4–5. Usually the occurrence of a pericardial effusion in acute pericarditis leads to a lessening of pain because of separation of the inflamed visceral and parietal pericardium. The characteristic pain of pericarditis is usually sudden in onset and frequently pleuritic in nature, due to involvement of contiguous diaphragmatic parietal pleura. Acute pericarditis is accompanied by superficial inflammation and necrosis of the myocardium, which is responsible for the accompanying ST elevation in the ECG and for any similarity between the pain of pericarditis and myocardial ischaemia. However, no pathological Q waves occur in the ECG in pericarditis.

Dissection of the aorta may be similar to myocardial infarction, as shock is a common accompaniment in both. The pain of dissection is unlike that of myocardial infarction in that it is usually 'tearing' and it is maximal at its onset and gradually wanes, whereas the pain of myocardial infarction is frequently preceded by premonitory pain. The pain of dissection usually radiates to the back and abdomen; following aortic dissection, occlusion of peripheral arterial pulses is less common than is generally believed. Dissection involving the aortic valve leads to severe aortic incompetence. The dissection may continue over several days and if the abdominal aorta is involved adominal pain is usually severe and continuous; bleeding into the peritoneum gives rise to paralytic ileus and signs of peritonitis. The clue may be the presence of a pathological arterial bruit. Occasionally, bleeding is into the gut. An unexplained and puzzling feature of dissection of the thoracic aorta is a gap of several days between the onset of the tearing pain of dissection and enlargement of the aortic knuckle on the chest X-ray.

Other causes of chest pain which may simulate the pain of myocardial ischaemia include pain of musculoskeletal origin, Tietze[3] syndrome (tenderness and swelling of upper costochondral junctions), pleuritic pain, and bronchial carcinoma, especially if it causes rib erosion.

1.4 THE PULSE

It is traditional for doctors to feel the radial pulse; however, the character and rhythm of the pulse are best assessed in the more proximal pulses – the carotids and brachials are the most convenient. Occasionally the radial pulses are most suitable for detecting pulsus paradoxus and the femorals for detecting a collapsing pulse.

The normal peripheral arterial pulse is made up of three components. The first, known as the percussion wave, is due to a forward moving column of blood expanding the peripheral arteries as a result of the relatively high resistances it meets in the arterioles as compared with the main arteries. The second is the tidal wave and is probably caused by two separate mechanisms – one is reflection of a pressure wave from the high-resistance arterioles back up the column of blood, and the other is transmission of a wave along the wall of the arteries, beginning at the aorta with the ejection of blood from the left ventricle. The third wave – the dicrotic wave – is due to transmission down the column of blood of a wave resulting from bulging downwards, into the ventricle, of the cusps of the closed aortic valve (Fig. 1.1).

In aortic stenosis the ejection of blood from the ventricle is prolonged and there is delay in arrival of the full percussion wave at the periphery. The tidal wave is less affected. If the stenosis is severe, the percussion wave is delayed beyond the tidal wave which is felt as a notch on the upstroke of the pulse tracing – the anacrotic notch (*Gk.* ana – up; krotos – stroke). The tighter the stenosis, the more delayed will be the percussion wave and the lower on the upstroke will be the anacrotic notch.

The collapsing pulse of aortic incompetence or arteriovenous shunting is due to disappearance of the dicrotic wave, since leaking

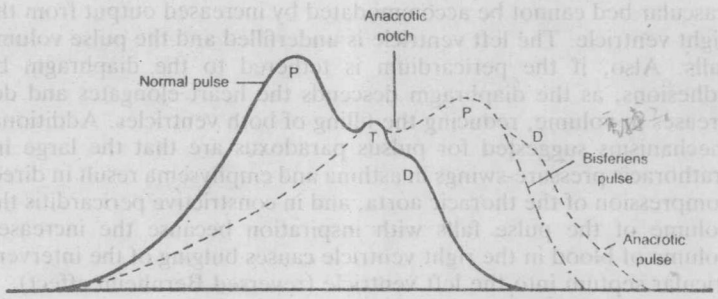

FIG. 1.1.—Components of the normal and abnormal peripheral pulse. P, Percussion wave; T, Tidal wave; D, Dicrotic wave.

valves will not abruptly stop retrograde flow and will not reflect a pressure wave towards the periphery. The bisferiens pulse (*L.* to beat twice) of combined stenosis and incompetence has two easily palpable impulses, the first due to the tidal wave, as in the anacrotic pulse, and the second to an apparently forcible percussion wave due to disappearance of the dicrotic wave. The dicrotic pulse of widespread peripheral arteriolar dilatation, usually due to fever, is a combination of a rapid upstroke due to early run-off into the arterioles, and an easily palpable dicrotic notch on the downstroke (*Gk.* dictrotos – twofold beating). The characteristic pulses of hypertension and atherosclerosis of the major vessels are similar in that there is a rapid and strong upstroke due in the one case to a high arteriolar resistance, and in the other to loss of elasticity of the aorta.

Other abnormal pulses are:

Pulsus alternans, in which regular alternate beats are diminished in volume. It is specially easy to appreciate this by palpating the radial pulse while deflating a sphygmomanometer cuff around the upper arm – as the pressure falls there will be a sudden doubling of the pulse rate.

Pulsus bigeminus (or coupling), in which every alternate beat is an extrasystole and therefore of diminished volume, but following this extrasystole there is a long compensatory pause; unlike pulsus alternans every beat is not equidistant (*L.* geminus – a twin).

Pulsus paradoxus occurs in constriction of the heart, whether by thickened pericardium, pericardial effusion or, rarely, in severe asthma when there is a gross trapping of air within the chest. During inspiration there is a fall in pulse volume. It is important to note that this is not paradoxical but is an exaggeration of the normal. During inspiration the pulmonary vascular bed increases in size due to traction on the vessels by the expanding lung. If the heart is constricted, the volume of the right ventricle is fixed during inspiration and expiration, therefore, the increased volume of the pulmonary vascular bed cannot be accommodated by increased output from the right ventricle. The left ventricle is underfilled and the pulse volume falls. Also, if the pericardium is tethered to the diaphragm by adhesions, as the diaphragm descends the heart elongates and decreases in volume, reducing the filling of both ventricles. Additional mechanisms suggested for pulsus paradoxus are that the large intrathoracic pressure-swings in asthma and emphysema result in direct compression of the thoracic aorta, and in constrictive pericarditis the volume of the pulse falls with inspiration because the increased volume of blood in the right ventricle causes bulging of the interventricular septum into the left ventricle (reversed Bernheim effect).

1.5 PERIPHERAL ARTERIAL INSUFFICIENCY

Relative ischaemia of a limb is suggested on inspection by:

Atrophic shiny skin with loss of hair
Brittle deformed nails
Persistent skin infections
Skin which looks permanently red, or cyanosed, due to chronic anoxia, causing the superficial vessels to become permanently dilated.

The skin of the ischaemic limb is cooler than the normal side; before deciding for certain about differences in temperature between the two sides make sure that the limbs are in the same position and that they have been exposed for the same length of time. The peripheral pulses should be carefully palpated at rest, and if there is a suspicion of arterial insufficiency and the pulses appear equal in volume at rest, it is most important to exercise the limbs and palpate the pulses again. Occasionally peripheral pulses appear equal at rest, but after exercise the pulse on one side may disappear due to blood being diverted by the exertion to the leg with the most patent arteries.

1.5.1 Simple Confirmatory Tests of Arterial Insufficiency

1. With the limbs horizontal press the skin of both limbs in corresponding positions. After removing the pressure blanching will be seen; the blanched area should normally begin to flush in 5 s. If the circulation is completely obstructed and the skin permanently cyanosed, blanching will not occur. This situation exists in early gangrene.
2. Elevation of both limbs to 45° normally does not result in much change of colour of the limbs. If part of the limb becomes pale, the arterial supply is impaired. With the limbs in this position the skin compression test can be carried out as above; flushing of the blanched area should occur within 10 s.
3. If elevation of the limbs to 45° results in pallor, the patient should be asked to hang the legs over the couch so that they are below the level of the body. In this position the pink colour should return to the skin within 10 s. At 10 s the veins on the dorsum of the feet should also have filled when the legs are in a dependent position.