MEDICINE FOR ANAESTHETISTS

EDITED BY M D VICKERS

FOREWORD BY WILLIAM W MUSHIN

Blackwell Scientific Publications

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BLACKWELL SCIENTIFIC PUBLICATIONS

OXFORD LONDON EDINBURGH MELBOURNE

Osney Mead, Oxford, OX2 0EL
8 John Street, London, WC1N 2ES
9 Forrest Road, Edinburgh, EH1 2QH
P.O. Box 9, North Balwyn, Victoria, Australia.

First published 1977
Reprinted 1978, 1979

British Library Cataloguing in Publication Data.

Medicine for Anaesthetists.
1. Pathology
I. Vickers, Michael Douglas Allen
616'.002'4617 RB111

ISBN 0-632-00156-9

Distributed in U.S.A. by
Blackwell Mosby Book Distributors
11830 Westline Industrial Drive
St Louis, Missouri 63141,
in Canada by
Blackwell Mosby Book Distributors
86 Northline Road, Toronto
Ontario, M4B 3E5,
and in Australia by
Blackwell Scientific Book Distributors
214 Berkeley Street, Carlton
Victoria 3053

Printed and bound in Great Britain by
Billing & Sons Limited
Guildford, London and Worcester

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Foreword

Of the many factors which affect the safety of anaesthesia, the careful and accurate assessment of the health of the patient by the anaesthetist must rank high in importance. By this means not only can the best type of anaesthetic be selected but any hazards to which the patient will be exposed by virtue of his pre-existing general medical disease can be avoided or minimized. Prophylactic measures can be taken preoperatively, extra vigilance for warning signs can be maintained during and after operation, and any special drugs or instruments kept ready in case of need. There is therefore a high priority for every anaesthetist to be sufficiently well versed in general medicine to play his proper part in bringing the patient safely through the anaesthetic and surgical operation. This need was recognized by the Faculty of Anaesthetists at its inception of the FFARCS diploma, by the inclusion in it of a test in clinical medicine in relation to anaesthesia, by clinical, oral, and written examination.

However, instruction in this field, to say nothing of source material in the literature, is not easy to come by. Practising internists have, by and large, little opportunity for acquiring knowledge or experience of the problems posed by their patients when subjected to anaesthesia, or of the risks they face. As a result, in the past, such advice as they gave was often absurd, perhaps ludicrous, and sometimes downright dangerous. Textbooks on internal medicine are no better. They either omit all mention of anaesthesia, or refer to it so sparsely that they give little help to the clinical anaesthetist.

Nearly all the authors of this book are anaesthetists and they have undertaken their task with courage, knowledge, and enthusiasm. They show courage in entering with confidence a field of medicine to which others might claim territorial rights, but which in reality belongs to all physicians, not least to anaesthetists. Their knowledge has been acquired in the most fruitful of places in medicine—the bedside. The present day anaesthetist has long ago extended his activities beyond the operating theatre and the description of him as the clinical physiologist of the surgical team is apt. The authors are enthusiastic fortunately, for a new venture such as this book (it is the first of its kind in this country) needs that quality of vigour which only enthusiasts, sure of their objectives, can give it.

This book instructs both learner and teacher. It will be an invaluable source of reference for research and practise alike. It will raise the standard of knowledge of clinical medicine among anaesthetists. As a result, the standard of anaesthesia will still further improve, leading to safer anaesthesia, more successful surgery, and improved health in the community.

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Preface

Unless an anaesthetist is also a physician, his expertise is ultimately technical. The last 20 years have indeed seen great technical advances in anaesthesia and this has been founded on the application of physiological and pharmacological knowledge. It would now be fair to claim that an individual in normal health is exposed to no measurable risk from modern anaesthesia.

The extension of this degree of safety to the unfit is unlikely to be achieved by any mere technical advances. This will require an understanding of the pathophysiology of all those conditions which render a patient less than optimally fit; in short, a knowledge of medicine in its widest sense.

To be able to cope safely with all such patients the truly professional practitioner of the art and science of anaesthesia has to have the breadth of approach of a general physician. Medicine, however, has seen the growth of specialisms and has led to the evolution of the cardiologist, neurologist, gastroenterologist, rheumatologist, nephrologist and many other '-ologists'. The anaesthetist cannot hope to match such specialists in their own field, nor should he try to do so. He must, however, keep sufficiently up to date with all of them to know the importance of their knowledge to the practice of anaesthesia.

This is the justification for a textbook of medicine for anaesthetists. But what form should it take? A standard test, even if simplified or of restricted scope is not what anaesthetists need. They rarely need, for example, to make initial diagnoses and their approach is much more problem-orientated than that of the traditional physician. They are less concerned with what the problem *is*, as with what it *means* in terms of function. Can the patient be improved in the time available? How does one decide when the situation is optimal? What else *might* go wrong with the patient? These are the sort of questions to which anaesthetists need to know the answers.

The structure of the book has resulted from an attempt to obtain this perspective. Some subjects have been covered by individuals who are acknowledged authorities in both the anaesthetic and medical aspects of their subject. Most have been tackled by a team of two in which an anaesthetist has chosen a physician or other expert colleague to assist him. I have invited three physician colleagues to contribute chapters on their respective fields and in these chapters the anaesthetic viewpoint is my own.

Multiple authorship inevitably brings the problem of sub-dividing integrated subject matter into more or less logical parts and introduces problems of deciding where to cover topics which can be put into more than one place: cross-referencing may save space but it does so at the expense of readability; on occasions it has seemed better to allow some duplication in order to allow a more complete presentation of a

subject. This has added a little additional length to what I fear is already a rather overlong work. I hope the reader will find that it improves his understanding, assessment and management of the medical status of all those patients who are presented to him with chronic or acute illnesses. It is these patients to whom we need to give our most expert attention and who offer the greatest margin for improvement in current anaesthetic practice.

M. D. Vickers

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Chapter 1
Heart Disease

J. N. LUNN AND J. R. MUIR

Studies of the results of surgery in patients with cardiac disease have revealed that the overall hospital mortality is significantly greater in patients with heart disease than in other patients. The mortality increases with increasing severity of functional impairment. However, the surgical and anaesthetic techniques can be almost completely exonerated since these deaths do not usually occur within the first 24 hours after surgery. The mortality is, furthermore, not clearly related to the duration of the surgery; indeed, there is some evidence of a negative correlation between the incidence of postoperative death and duration of surgery for specific operations. The majority of patients who die do so as a result of recognizable complications of heart disease such as pulmonary embolus, intractable cardiac failure or myocardial ischaemia.

It is this increased hospital mortality following surgery in patients with coincidental heart disease which makes the study of cardiology so important for anaesthetists. Recognition that heart disease is present in an individual patient is an important step in the identification of risk and in the anticipation of the probable effect of anaesthesia upon cardiac function.

THE ASSESSMENT OF THE PATIENT

The assessment of patients who may have heart disease involves the careful integration of information derived from the history, the physical examination, the electrocardiogram, the chest X-ray and other special investigations. All these parts of the assessment are of value, but the history and the physical examination are the most important, since they determine the type and scope of special investigations. In the particular context of the work of the anaesthetist the history and physical examination are essential.

THE HISTORY

The importance of a clear history will be readily understood if it is appreciated that the diagnosis of severe coronary arterial disease may be dependent solely on a history of angina pectoris, when the physical examination and electrocardiogram may be entirely normal.

PAIN

Cardiac ischaemic pain results from an inadequate supply of oxygen to the myocardium, and it may take the form of angina pectoris, acute coronary insufficiency or acute myocardial infarction.

Angina pectoris is characteristically described as a crushing 'band-like' pain or discomfort across the chest which may radiate to one or both arms, into the neck or the jaw. The patient may confuse it with indigestion. It is usually closely associated with effort, being precipitated by exertion and relieved by rest within a few minutes, but may, in some patients, be related to excitement or emotional disturbance. It may, on occasions, occur at rest or in bed at night in patients with severe coronary arterial disease when it is precipitated by the increase in cardiac output in the supine position. It is of great importance to distinguish this form of nocturnal angina from oesophageal pain due to acid regurgitation, and this distinction is made difficult since both types of pain may be relieved when the patient sits up. *Nocturnal angina* almost always occurs in patients who also have severe angina of effort although the story of effort angina may be difficult to elicit due to voluntary restriction of exertion by the patient.

The pain of acute coronary insufficiency is more severe and prolonged than angina pectoris and may occur at rest, but it is not associated with laboratory evidence of myocardial necrosis. While the serial electrocardiograms may show transient changes in the S–T segments or the T waves, they do not show the characteristic changes of acute myocardial infarction.

In acute myocardial infarction the pain is similar to angina pectoris, but it usually comes on at rest, is more severe in nature and lasts for a considerable length of time.

The restriction of oxygen supply to the myocardium, which is the basis of ischaemic heart pain, is usually the result of arteriosclerotic narrowing or obstruction in the coronary arterial tree. The balance between oxygen supply and oxygen demand in the myocardium may, however, on occasions be upset by other mechanisms acting either on their own or in association with coronary arterial disease. Systemic hypertension and aortic stenosis, which both present the left ventricular muscle with an increased pressure load (after-load), may cause cardiac ischaemic pain.

Angina may also be precipitated in patients with relatively mild coronary arterial disease by any of the *hyperkinetic circulatory states*. In thyrotoxicosis the cardiac output is increased but the efficiency of energy utilization by the myocardium is reduced. In severe anaemia there is reduction in oxygen delivery and an increased cardiac output. Angina is surprisingly rare in cyanotic heart disease, although it may occur when there is a considerable pressure overload of a ventricle. *Syphilitic aortitis* affecting the coronary ostia and *polyarteritis nodosa* involving the coronary arteries may also cause angina.

The importance of establishing clearly the nature of chest pain cannot be over-stressed. The damage to a patient that may follow the incorrect labelling of chest pain as cardiac ischaemic pain is incalculable, for, once the spectre of coronary arterial disease has been raised in a patient's mind, it is extremely difficult to restore the patient's confidence in his heart.

The differential diagnosis is discussed in detail later in the chapter.

DYSPNOEA

Dyspnoea is an important and usually progressive symptom and is graded according to the New York Heart Association Scale (Table 1.1). In heart disease dyspnoea is

usually related to a rise in the pulmonary venous pressure, but it may also occur in patients with primary pulmonary hypertension or severe pulmonary stenosis when the pulmonary venous pressure is normal.

Table 1.1. New York Heart Association functional classification

Class 1	No limitation of activity.
Class 2	Some limitation on heavy exertion, but patient can climb 1 flight of stairs or walk three blocks without shortness of breath.
Class 3	Some limitation on ordinary activity with difficulty walking 3 blocks or climbing 1 flight of stairs.
Class 4	Shortness of breath at rest.

When a subject lies flat there is a temporary increase in the stroke volume of the right ventricle as compared to that of the left and the left atrial pressure increases. When left atrial pressure is already raised, secondary to left ventricular disease or mitral stenosis, further pulmonary congestion will occur in the supine position and the patient complains of increasing dyspnoea. This symptom, *orthopnoea*, is usually confined to patients who have at least grade III or IV dyspnoea on exertion. They may also suffer from attacks of *acute paroxysmal nocturnal dyspnoea* caused by the same mechanism. In these attacks the patient is awoken from sleep by sudden severe breathlessness which appears to be life-threatening: florid pulmonary oedema may then develop. These symptoms are usually relieved by sitting up.

Dyspnoea, due to acute pulmonary oedema or to pulmonary emboli, may occasionally develop suddenly in a patient whose effort tolerance was previously normal.

HAEMOPTYSIS

This is an important symptom and may be due to pulmonary infarction, rupture of a small intrapulmonary bronchial vein, or be associated with paroxysmal nocturnal dyspnoea or pulmonary oedema. Recurrent winter bronchitis is a common presenting feature of mitral stenosis, and blood streaking of the sputum may occur in these attacks.

SYNCOPE

This is associated in particular with complete heart block when the syncopal episodes are referred to as Stokes–Adams attacks. It is also an important symptom of aortic stenosis, atrial myxoma and pulmonary embolus.

PALPITATIONS

Palpitations do not necessarily indicate cardiac pathology and may be due to paroxysmal tachycardia (including paroxysmal atrial fibrillation) or recurrent extrasystoles. Attacks of *paroxysmal tachycardia* usually start and end suddenly, lasting from a few seconds to many hours. The patient may be conscious of a rapid regular palpitation and may feel faint if the heart rate is very high. Paroxysmal tachycardia may occur in the absence of heart disease but when it is associated with dyspnoea or ischaemic pain the possibility of occult heart disease exists. *Paroxysmal atrial fibrillation* can usually be easily distinguished from paroxysmal tachycardia since in the former the patient is

well aware of the irregularity of the pulse. Paroxysmal atrial fibrillation usually indicates significant underlying disease, the three commonest types being rheumatic heart disease, ischaemic heart disease and thyrotoxicosis. *Recurrent extrasystoles* occur both in normal subjects and in those with heart disease. The sufferer is conscious of a sudden heavy beat, which is the accentuated post-extrasystolic beat since the filling and emptying of the ventricle following the premature extrasystole is greater than normal. On occasions extrasystoles may occur in runs. When this happens the patient may complain of episodes of completely irregular palpitation, which are then historically indistinguishable from atrial fibrillation.

OEDEMA

Patients in cardiac failure may complain of swelling of the ankles which may spread to involve the whole lower half of the body. The appearance of oedema is a relatively late symptom of cardiac failure and does not usually appear until right heart failure with elevation of the jugular venous pressure has developed, but this is not the principal cause of the oedema. There is an abnormal retention of sodium by the kidneys in cardiac failure which results in a secondary retention of water with a consequent increase in the extracellular and plasma volumes. This leads to the appearance of peripheral oedema.

The mechanisms underlying the sodium retention by the kidneys in heart failure are poorly understood. Cardiac failure results in a redistribution of cardiac output between different organs. There is a particularly marked reduction in the renal blood flow due to renal arteriolar constriction and in extreme cases it may fall to 25 per cent of control levels. There is no doubt that there is diminished glomerular filtration and increased tubular reabsorption of sodium in cardiac failure but this is not as marked as the reduction in cardiac output, and therefore it appears unlikely that it is an important factor in sodium retention.

Hyperaldosteronism occurs in some patients with severe failure and in these cases the increased retention of sodium and excretion of potassium and hydrogen, due to the action of aldosterone on the distal tubule, certainly increase the severity of the oedema. In the majority of patients with cardiac failure, however, there is no evidence of hyperaldosteronism and the mechanism underlying the increased retention of sodium is unknown.

The retention of water is almost always secondary to sodium retention, but on occasions there is an active retention of water, which results in a fall in the serum sodium concentration. This may be due to an inappropriate secretion of antidiuretic hormone (ADH).

The distribution of peripheral oedema in cardiac failure is largely determined by gravity. In the mobile patient it occurs in the feet and ankles, whilst in the bedridden patient a pad of oedema may accumulate over the sacrum. Other causes of peripheral oedema which need to be distinguished are venous incompetence, deep venous thrombosis, inferior vena-caval obstruction, lymphatic obstruction, low serum albumin, pre-eclamptic toxaemia, and acute glomerulonephritis.

Ascites, the accumulation of fluid within the peritoneal cavity, may occur in advanced failure. When it does, the fluid is a transudate with a low protein content (< 30 g/litre), as opposed to an exudate such as might be associated with carcinomatosis of the peritoneum. Ascites is particularly common when the venous pressure is very high (tricuspid incompetence and constrictive pericarditis). The very high

jugular venous pressures can result in the development of cardiac cirrhosis and portal hypertension, and this may play a part in the development of ascites which is disproportionate to the degree of peripheral oedema.

PHYSICAL EXAMINATION

The physical examination of the cardiovascular system is a logical exercise in which auscultation plays only a part, albeit an important part. If careful attention is paid to the general examination of the patient and in particular to the character and form of the arterial and venous pulsations, and to the nature of the cardiac impulse, considerable progress can be made towards a correct diagnosis even before the heart is auscultated.

INSPECTION

The general appearance and build of patients with heart disease is usually unremarkable. However, a tall thin individual whose arm span exceeds his height, with a high-arched palate, and dislocation of the lens, probably has Marfan's syndrome, a condition associated with cystic medial necrosis of the aorta. Similarly, certain types of congenital heart disease are often associated with other congenital abnormalities. One such association is the frequent occurrence of abnormalities of the endocardial cushion with Down's syndrome.

Cardiac disability may be secondary to another disease process. For example, anaemia secondary to bleeding from a carcinoma may cause angina or cardiac failure, and rapid atrial fibrillation in a patient with a thyroid mass should suggest thyrotoxicosis, rather than primary heart disease.

Clubbing of the fingers may vary in degree from simple obliteration of the angle between the nail bed and the finger, to 'drumsticking' of the terminal phalanges. It occurs in cyanotic congenital heart disease, chronic suppuration in the chest, carcinoma of the bronchus, subacute bacterial endocarditis and occasionally in hepatic cirrhosis. It may also rarely be congenital.

Cyanosis is a blue or blue-grey discoloration of the skin. It is due to the presence of a significant amount of desaturated haemoglobin in the capillary bed immediately below the skin. *Peripheral* cyanosis is due to an increased extraction of oxygen in the tissues due to a reduced flow of blood through the capillary beds in the skin. This occurs when the cardiac output is substantially reduced or, in normal people, as a result of peripheral vasoconstriction due to cold. Cyanosis of this type is not seen in the mucous membranes that are warm. In *central* cyanosis the skin is warm and the discoloration is readily apparent in the tongue. It is due to mixing of desaturated blood with oxygenated blood in the heart, great vessels or lungs. Ventilation-perfusion abnormalities in the lungs cause central cyanosis which can be distinguished from that due to cardiac abnormalities by allowing the patient to breathe a high concentration of oxygen which reduces the cyanosis.

Abnormal haemoglobins, such as methaemoglobin or sulphaemoglobin, can cause a blue colour in the skin, and are usually due to drugs, but on very rare occasions, methaemoglobinaemia may be congenital.

ARTERIAL PULSE

Rate and rhythm may be judged from palpation of the radial artery, but the volume and character of the pulse wave can only be assessed accurately from the carotid artery, because the pulse wave alters as it is propagated into the peripheral arteries.

The *character and volume* of the pulse can best be considered together. Pulses of small volume may be of two types; small volume with a normal upstroke wave, which occurs in any condition in which the stroke volume is small, such as shock or severe mitral stenosis; and small volume with a slow upstroke and a sustained character (Fig. 1.1), which is characteristic of a fixed obstruction to the outflow of the left ventricle.

Fig. 1.1. Diagram of aortic pressure trace of a plateau pulse. (A normal trace is shown in broken lines for comparison.) The normal dicrotic notch is seen on both traces.

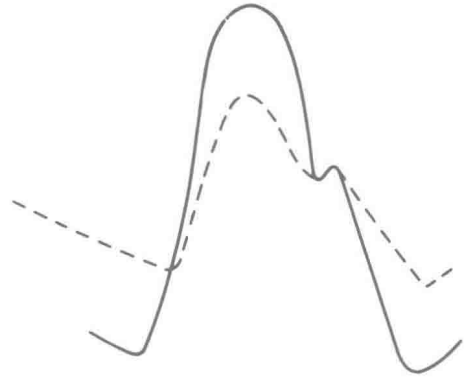

Fig. 1.2. Diagram of a collapsing pulse.

The obstruction impedes the ejection of blood from the ventricle into the aorta throughout the isotonic phase of systole and causes a plateau pulse; on an arterial pressure tracing it may be possible to identify a notch, the anacrotic notch, on the upstroke of the pulse wave (Fig. 1.1). A plateau pulse in adults almost always indicates the presence of aortic valve stenosis, but in children it may occasionally be due to supravalvar or subvalvar aortic stenosis. In hypertrophic cardiomyopathy, there may be asymmetric ventricular hypertrophy particularly involving the ventricular septum. Early in systole the flow of blood into the aorta is unimpeded but as systole continues the outflow becomes obstructed by the hypertrophied muscle of the ventricular septum and the anterior cusp of the mitral valve which moves abnormally. This results in a characteristic small jerky pulse.

A large-volume collapsing pulse occurs in any condition in which the total resistance in the systemic arterial system is low. This may be due to exercise, fever, pregnancy or to pathological causes. This pulse (Fig. 1.2) is characteristic of aortic incompetence, patent ductus arteriosus, aorto-pulmonary window, large arterio-venous fistulae, severe anaemia and thyrotoxicosis. In mixed aortic stenosis and incompetence a bifid pulse, 'pulse bisferiens', may on occasions be felt (Fig. 1.3). In mitral incompetence the carotid pulse may be of large volume but is not collapsing in character. (Irregularities of the arterial pulse are considered in the section on dysrhythmias.)

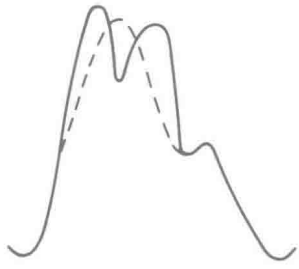

Fig. 1.3. Diagram of pulsus bisferiens.

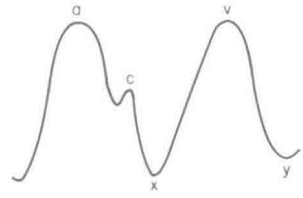

Fig. 1.4. Diagram of a normal jugular venous pulse.

JUGULAR VENOUS PULSE

The level of the jugular venous pressure and the character of its wave form reflects changes on the right side of the heart. In the normal subject the jugular venous wave (Fig. 1.4) can be just seen in the supraclavicular fossa with the subject reclining at 45°.

The *a* wave is caused by atrial contraction, and is followed by the *x* descent. This drop in the wave is due to the pulling down of the atrio-ventricular ring in early systole, and it is interrupted by the *c* wave which is thought to be caused by the bulging of the closed tricuspid valve into the atrium. The *v* wave represents the passive filling of the atrium against the closed tricuspid valve, and is followed by the *y* descent, when the valve opens at the beginning of diastole.

The jugular venous pressure wave is altered by rhythm disturbances. For example, since the *a* wave depends on atrial contraction, it is absent in atrial fibrillation. In atrial flutter rapid 'flutter' waves can be seen, and in complete heart block 'cannon' waves may occur. Cannon waves are caused by the atrium contracting against a closed tricuspid valve, when atrial and ventricular contraction are completely dissociated. When right ventricular compliance is lowered, the pressure which the atrium has to generate to expel blood into the ventricle rises. This causes an increase in the height of the *a* wave. A prominent *a* wave is characteristic of right ventricular hypertrophy with sinus rhythm. This may be caused by primary pulmonary hypertension, pulmonary emboli, pulmonary hypertensive mitral valve disease, pulmonary stenosis or the Eisenmenger syndrome. If the tricuspid valve is incompetent the *x* descent is replaced by a high *c-v* wave which is often loosely referred to as a *v* wave. This wave is due to the transmission of the ventricular pressure pulse to the right atrium across the incompetent valve.

THE CARDIAC IMPULSE

The cardiac impulse (apex beat) is normally situated in the fifth intercostal space within the mid-clavicular line. Its position may be altered either as the result of enlargement of the heart or by displacement by extracardiac factors. The cause of any displacement is more important than the precise location of the impulse. When the heart is enlarged it is necessary to identify which chamber or chambers are involved, and to determine whether a pressure or a volume overload is responsible. If the right ventricle is working against a pressure overload, as in pulmonary stenosis or pulmonary hypertension, the palpating hand to the left of the sternum may actually be lifted by