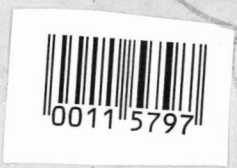
# cardiology for students

MAX ZOOB

# Cardiology for Students

**Max Zoob** M.D., F.R.C.P.,
Consultant Cardiologist, Regional Cardiac Unit, Brook General Hospital, London.

Foreword by J.F. Goodwin, M.D., F.R.C.P., F.A.C.C.
Professor of Clinical Cardiology, Consultant Physician, Royal Postgraduate Medical School, London.




CHURCHILL LIVINGSTONE
Edinburgh London and New York 1979

**CHURCHILL LIVINGSTONE**
Medical Division of Longman Group Limited

Distributed in the United States of America by Longman Inc., 19 West 44th Street, New York, N.Y. 10036 and by associated companies, branches and representatives throughout the world.



ISBN 0 443 01530 9

**Library of Congress Cataloging in Publication Data**

Zoob, Max.
Cardiology for students.

Includes index.
1. Heart-Diseases. 2. Cardiovascular system-Diseases.
I. Title. [DNLM: 1. Heart diseases – Diagnosis.
WG141 Z87c]
RC681.Z66 616.1'2 76–54864

Printed in Singapore by
New Art Printing Co Pte Ltd

# Foreword

The subject of cardiovascular disease has now become so vast and so sub-specialised that it is difficult to encompass it effectively within a textbook of medicine and difficult to know how much detail to include.

Dr Zoob's short textbook whets the appetite of the students and cardiovascular nurse and the same time provides a source for reference that is easily assimilable and effectively illustrated by simple drawings.

The style is straightforward and readable, the phraseology sometimes suggesting a nostalgia for the incisive style of the late Paul Wood, to whom Dr Zoob pays tribute in his preface. Dr Zoob has followed in general the format of Wood's classic textbook, beginning with structure and function and proceeding to symptoms, signs, investigations and then specific cardiovascular disorders.

The aim has been to concentrate on broad principles so that fine description of details of diagnosis and treatment are not to be expected.

In an era when the increasing complexity and sophistication of instrumentation threaten to overwhelm the clinician and sometimes obscure important basic clinical aspects the second chapter, which clearly describes symptoms and signs of cardiovascular disease, is particularly welcome and will be of great value to the student.

Dr Zoob is to be commended also for his handling of references. He has avoided the twin pitfalls of too extensive referencing on the one hand and total omission of all references on the other. Each chapter is followed by a short list for recommended reading.

The final section on actions and unwanted effects of drugs is a useful addition and could be expanded in future editions.

I have much pleasure in wishing Dr Zoob every success in this venture.

1978 J. F. Goodwin

# Acknowledgements

It is a pleasure to acknowledge my indebtedness to the teachers who first interested me in cardiology and especially in the value and techniques of bedside observation. They include Sir George Pickering, the late Dr Paul Wood, Sir John McMichael, and Professor John Goodwin.

I am greatly indebted to my friend and colleague Dr E. Gaertner who has read the manuscript meticulously and helped me with much stimulating and constructive criticism and advice.

It is a pleasure to acknowledge the excellent work of Mr. Frank Price who made the line drawings, and my secretaries Mrs P. Buxey and Mrs E. C. Mills.

I am grateful to Hoechst Pharmaceuticals for permission to adapt illustrations from their booklet on Echocardiography.

London, 1978 Max Zoob

# Introduction

The last 30 years have seen remarkable changes both in the total incidence of heart disease and in the frequency of its various forms. The most spectacular alteration has been a nearly two-fold increase in coronary artery disease which has made cardiovascular disorders the commonest cause of death in the Western World. Another remarkable change, the significance of which is probably not yet fully apparent, has been the appearance of the primary cardiomyopathies as partially defined though at present fairly uncommon entities. At the same time the frequency of rheumatic and syphilitic heart disease has diminished strikingly. Consideration of incidence and frequency are particularly important in diagnosis, which is after all a question of probabilities. Common conditions are more likely to be encountered than rare ones and though the former may be reasonably diagnosed in the presence of some unusual or atypical features, the latter may not. Rarities should be thought of frequently but diagnosed rarely and then only on particularly secure grounds.

Incidence and frequency must of course be reflected in any textbook so that the amount of detail is related to the commonness of the disorder described. But this cannot be the sole criterion of proportions for several reasons. Thus rheumatic heart disease—though now much less common than ischeamic or hypertensive heart disease—merits a full presentation since this ensures a training in auscultation and haemodynamic concepts applicable to the whole of cardiology. The same is true to a lesser extent of congenital heart disease which is comparatively rare. Again, a knowledge of the cardiomyopathies should draw attention to the considerable numbers of patients with florid heart disease whose aetiology is totally unknown. This should lead to a preoccupation with aetiological diagnosis and help to eradicate the prevalent habit of thinking of 'congestive heart failure' as a complete diagnosis. It should also discourage the habit of making a facile diagnosis of a common condition in a patient lacking some of its usual signs or presenting unusual ones—rarities should be considered frequently though diagnosed rarely.

These were some of the considerations which determined the proportions of this book. Short sections usually imply uncommon disorders and when a larger section has been devoted to them their relative frequency has been noted.

# Contents

# 1. The Structure and Properties of Heart Muscle and Heart Failure

Throughout biology, structure can be seen to be intimately related to function and study of the one enhances understanding of the other.

In recent years electron microscopy of heart muscle cells (fibres) has revealed the structural basis for some of the fundamental properties of heart muscle. Each muscle cell is composed of longitudinal fibrils which are divided by dark transverse bands (Z lines) into the individual basic units of contraction, the sarcomeres, whose structure is shown in Fig. 1.1. Thin (50 Å diam.) actin filaments arise from the Z lines,

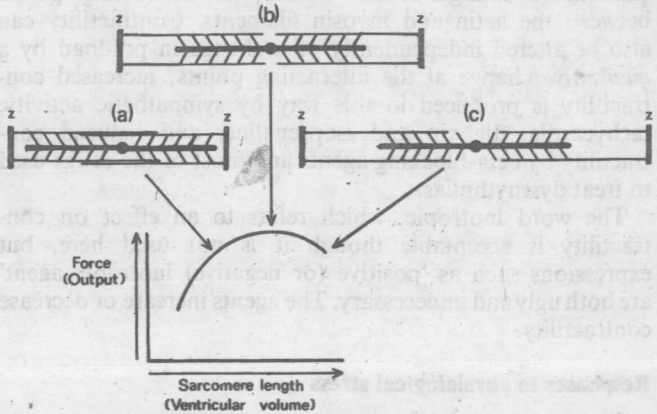



Fig. 1.1 The relationship between structure and function of the sarcomere (see text). (a) The sarcomere is insufficiently stretched so that the overlapping parts of the actin fibrils cannot be engaged by the bridges of the myosin filaments. The force and output developed is submaximal and the muscle is operating on the ascending limb of Starling's curve shown below. (b) At the peak of the curve stretching is optimal and all the bridges are engaged. (c) The sarcomere is overstretched and some of the bridges are disengaged. Developed force is sub-optimal corresponding to the falling limb of the curve.

interdigitate with the thicker (100 Å diam.) centrally placed myosin filaments and are connected to them by bridges. During contraction the Z lines approximate and although the cells shorten the actin and myosin filaments do not. They must therefore slide over one another, impelled it is thought by forces generated at the points of contact of the bridges. The maximum force of contraction occurs when all the bridges are operative (Fig. 1.1(b)). If the muscle is stretched too little, the ends of the actin filaments overlap and cannot be engaged, while if it is overstretched some bridges are disengaged (Fig. 1.1(a) & (c)). This is the ultra-structural basis for the physiological observation embodied in Starling's Law, that the force of contraction increases with fibre length until the latter is optimal, and then diminishes (Braunwald 1971). The term myocardial contractility refers to the force and velocity of contraction of the myocardium; its alteration by a change in preliminary stretching (pre-load), just described, depends on a *quantitative* change in the number of interacting points between the actin and myosin filaments. Contractility can also be altered independently of a change in pre-load by a *qualitative* change at the interacting points; increased contractility is produced in this way by sympathetic activity, tachycardia, digoxin and isoprenaline, and reduced contractility by beta-blocking agents and most of the drugs used to treat dysrhythmias.

The word inotropic, which refers to an effect on contractility is acceptable though it is not used here, but expressions such as 'positive (or negative) inotropic agent' are both ugly and unnecessary. The agents increase or decrease contractility.

**Responses to physiological stress**

There are two basic determinants of cardiac output—the heart rate and the stroke volume. The heart rate is governed by vagal activity which tends to slow it and by sympathetic activity mediated by the catecholamines adrenaline and noradrenaline. These tend to increase the rate and also increase the force and velocity of contractions. Probably sympathetic and para-sympathetic effects work in harmony to produce changes in cardiac output; thus increased vagal activity and diminution of catechol amine production may

reduce the heart rate and cardiac output during sleep. An increased cardiac output which occurs normally after food, on emotion, stooping, or during exercise is probably mediated mainly by the sympathetic nervous system through an increased production of catecholamines. The contrasting actions of adrenaline, noradrenaline, isoprenaline and their antagonists are shown in Table 1.1. It will be seen that

Table 1.1 The effects of catecholamines and drugs blocking $\alpha$ and $\beta$ sympathetic receptors with the resultant effects on the circulation.

| *Compound* | Action on Sympathetic Receptors | | | *Result* |
|---|---|---|---|---|
| | *Heart* | *Peripheral Vessels* | | |
| | ($\beta$) | constriction ($\alpha$) | dilatation ($\beta$) | |
| Noradrenaline | + | ++ | − | B.P. ↑<br>Periph: Flow ↓<br>Cardiac Output Unchanged<br>Systolic Pressure ↑<br>Diastolic Pressure ↓ |
| Adrenaline | ++ | + | ++ | Muscle Flow ↑<br>Skin Flow ↓<br>Cardiac Output ↑<br>Systolic Pressure ↑<br>Diastolic Pressure ↓ |
| Isoprenaline | +++ | − | +++ | Peripheral Flow ↑<br>Cardiac Output ↓ |
| Propranolol | Blocks | | | Heart Slowed ↑<br>Cardiac Output ↓ |
| Phentolamine | − | Blocks | − | Vasodilatation<br>B.P. ↓<br>Cardiac Output ↓ |

noradrenaline causes generalized vasoconstriction affecting the arteries of muscle, heart and skin, with a total increase in vascular resistance and pressure and reflex slowing of the heart. By contrast, adrenaline produces effects ideally suited to the increased cardiac output required during exercise and other physiological activities. It increases the heart rate, dilates the arteries of the heart and muscles where extra blood flow is required and it constricts the skin vessels where increased flow is not needed. As a result the total vascular resistance falls, the systolic blood pressure rises and the diastolic pressure

falls. The cardiac output increases so that the venous return increases, increasing the filling pressure of the right heart thus increasing the force of contraction of the right ventricle, according to Starling's law. The left atrium and ventricle follow suit in due course with a resultant further increase in cardiac output. An increased contractility results from the increased sympathetic activity. It has been observed that dynamic exercise involving movement of the body such as walking and running produces relatively slight increases in cardiac work and blood pressure while static work such as maintaining weight against gravity or tasks involving straining or gripping causes a great increase in blood pressure and a correspondingly large increase in cardiac work. This is highly relevant to the type of exercise and work which should be allowed to patients suffering or convalescent from heart disease. They can usually be permitted to engage in dynamic forms of work and exercise but it may be necessary to advise against or restrict static exercises or work involving heavy static strain.

These normal mechanisms of increasing cardiac output can be altered by drugs, especially those which block the beta adrenergic receptors. Propranolol is the best established of these; it reduces the heart rate and decreases the force of contraction. Isoprenaline is a powerful beta receptor stimulant increasing the heart rate and force of contraction and dilating skeletal blood vessels and bronchi. Phenoxy-benzamine (phentolamine) blocks the alpha-receptors leading to vasolilatation in the skin and it also stimulates production of catechol amines so that there is increased cardiac output with fall of blood pressure and dilatation of the bronchi. The action of these drugs is summarised in Table 1.1.

**Response to pathological stresses**

There are two main categories of pathological stress:

1. Increased volume work
2. Increased pressure work

Increased volume work is demanded of the heart if any of its valves leak. Then, if a normal quantity of blood is to be delivered to the body, the heart must pump an additional amount corresponding to that which leaks back through the

faulty valve. Increased volume work is also demanded of the heart if there is an abnormal congenital communication between the left and right sides of the heart allowing a shunt or short-circuit in the circulation. Then too, if a normal volume is to be delivered to the body an additional quantity must be pumped by the heart corresponding to the quantity taking the shortened route. This is explained in detail in the chapter on haemodynamics.

Whatever the cause the heart responds to the increased volume load by increased force of contraction, according to Starling's law. The dilatation becomes permanent and is followed by hypertrophy. The filling pressure rises proportionately. The rapid filling of a dilated ventricle which is the essential feature of the condition gives characteristic clinical findings. The ventricle can be felt to have a large amplitude pulsation (a hyperkinetic ventricle). The rapid filling produces an audible 3rd sound in the first third of diastole (p. 35).

The increased diastolic pressure is communicated to the atrium and to the venous and capillary territories up-stream with consequences described under left and right heart failure in subsequent pages. Although dilatation of the ventricle produces increased force of contraction it is not without disadvantages. Laplace showed that the greater the cavity size the greater is the wall tension needed to produce a given intraventricular pressure. The reduced efficiency of the dilated ventricle is shown by the increased oxygen requirement needed for a given amount of heart work and a reduced rate of rise of intraventricular pressure.

The heart performs increased pressure work when it is forced to eject against increased resistance as in systemic or pulmonary hypertension or when there is obstruction at the aortic or pulmonary valves. The heart responds by hypertrophy—its individual fibres thickening and lengthening. Although the ratio between capillaries and fibres appears to remain one to one, some difficulty of diffusion of oxygen from the capillary to the centre of the cell may occur as the cell becomes very thick with some consequent impairment of function. Hypertrophied muscle is uncompliant and tends to resist stretching. A powerful atrial contraction is needed to ensure adequate filling. This may produce an audible pre-

systolic sound which together with a powerful but small amplitude thrust are the characteristic clinical findings of a ventricle performing excessive pressure work (a hyperdynamic ventricle). The pressure, at first raised only during atrial contraction in pre-systole, later becomes elevated throughout diastole and this elevated pressure is transmitted from the ventricle to atrium and venous and capillary territories with consequences described below.

*Heart failure*

It is remarkable but true that there is no satisfactory definition of heart failure. It has been stated to mean an inability of the heart to pump sufficient blood to meet the normal requirements of the body. The latter however cannot be defined, and surely if the normal requirements are not met the patient must die. Failure cannot be defined simply in terms of cardiac output or cardiac index (output per square metre of body surface area). These measurements may fall within the wide normal range although the patient shows the clinical features generally accepted as those of heart failure. A definition which is clinically applicable was that proposed by Sharpey Shafer who emphasised that the failing heart is unable to adjust its output to an altered inflow. This can be demonstrated at the bedside by asking the patient to perform the Valsalva manoeuvre in which he expires forcibly against a closed glottis with the nostrils held closed. This manoeuvre abruptly reduces the venous return to the heart which normally responds with a diminution of cardiac output, reduction of the stroke volume and consequent fall in pulse pressure. The reduction of stroke volume can be felt at the brachial or radial pulse and the reduction in pulse pressure can be measured with a sphygmomanometer. On release of the forced expiratory effort there is a sudden increase of stroke volume, pulse pressure and heart rate. If there is heart failure these parameters are unchanged by the manoeuvre which simply increases the systolic and diastolic arterial pressure owing to the rise in intra-thoracic pressure.

In addition however to this useful and attractive definition the more widely understood and accepted sense of the term 'congestive heart failure' and 'left' and 'right heart failure'

must be described. The terms imply the presence of raised pulmonary or systemic venous pressure together with the presence of pulmonary or systemic oedema consequent on water and salt retention. Formerly it was thought that the mechanism was quite simple. The pressure was presumed to rise behind the 'failing ventricle' so that the venous and capillary pressures rose exceeding the osmotic pressures and therefore caused transudation in the extra-cellular compartment and hence oedema. Subsequently however it was found that in many cases water and salt retention preceded the rise of venous pressure which followed secondarily. Sometimes the two events occurred almost simultaneously. The mechanism of the salt and water retention has still not been fully elucidated. The reduction in the glomerular filtration fraction seems to be only a minor factor and increased tubular reabsorption of sodium through the action of aldosterone can be detected only in a proportion of severe cases. However these mechanisms may be present transiently in mild cases but disappear before they are detected. The distribution of the oedema probably depends on the site of elevation of the venous pressures. If there has been a pressure or volume load on the left ventricle as in hypertension or mitral reflux, the left atrial and pulmonary venous pressures are raised and pulmonary oedema usually occurs when the pulmonary venous pressure exceeds the normal osmotic pressure of 25 to 30 mmHg. Persistently raised pulmonary capillary pressure may cause a slow transudation of fluid into the alveolar walls which become fibrosed so that higher pressures may be tolerated before pulmonary oedema occurs. When it does however, the result is the same, serous fluid seeps into the alveolar walls and thence into the alveoli themselves to be coughed up by the patient as pink frothy sputum. The clinical picture is described below.

If the pressure or volume load has been on the right ventricle as in pulmonary hypertension or atrial septal defect the systemic venous pressure is raised as can be appreciated by inspection of the jugular veins, and the liver is enlarged. Oedema develops in the dependant parts where the hydrostatic pressure exceeds the colloid osmotic pressure; ascites and pleural effusions may develop by a similar mechanism.

Pulmonary oedema is commonly referred to as 'left ventricular failure' and oedema with raised venous pressure, liver enlargement and perhaps pleural effusions, is often referred to as 'right ventricular failure'. These terms however are unfortunate. It has previously been noted that the cardiac output is not always below normal when these features are present; indeed in many cases the *work* of the heart is actually increased. For example, in arterial hypertension the blood pressure often rises during an attack of pulmonary oedema and if the output is normal the heart is performing greatly increased work. Oedema results from the very high pulmonary venous pressure. Similarly on the right side severe pulmonary hypertension and tricuspid reflux greatly increase the work of the heart which is actually contracting extremely forcefully at a time when oedema develops and right heart *failure* is said to be present. It would therefore seem much better simply to speak of pulmonary or systenic oedema and to state their cause.

**The direct impairment of myocardial function**

Myocardial function may be directly impaired in two ways, the myocardium may be affected by disease or its effective working may be hampered by disease of the pericardium or rarely the endocardium.

a. *Myocardial disease.* The most common is ischaemic disease resulting from coronary artery atheroma. Infarction or diffuse fibrosis reduces the contractile power of the left ventricle. Other disorders reducing the contractility of the muscles include myxoedema, amyloid disease, haemochromatosis and other metabolic cardiomyopathies and hypertrophic cardiomyopathies of unknown cause. Cardiomyopathies are also associated with hereditary neurological myopathies. Cardiac and vascular responses to the impairment of myocardial function produced by these disorders is similar to those produced by increased pathological loads already described. There is increased diastolic volume with increased filling pressure in order to maintain cardiac output. This is accompanied by an audible third heart sound or gallop rhythm. The heart is clinically and radiologically enlarged and hypertrophy may follow. Water and salt retention ensues and there may

be pulmonary or dependent oedema with the clinical features described below.

b. *Pericardial constriction or effusion.* This may severely impair filling of the heart. The pulmonary venous pressure and the systemic venous pressure rise to a great height but the heart, encased by dense fibrous tissue or surrounded by incompressible fluid within the pericardial sac, cannot enlarge to accommodate entering blood and its output is therefore limited and fixed. Sodium and water retention follow with consequent oedema and often ascites and pleural effusions. The high venous pressure results in gross liver enlargement. The clinical picture closely resembles that due to 'right heart failure' but in pericardial constriction the heart is small.

*Pulmonary oedema*

*Definition.* Transudation of fluid from capillaries into the alveolar walls and alveolar spaces of the lungs.

*Mechanism and causes.* Transudation may occur when the hydrostatic pressure exceeds the colloid osmotic pressure of about 25 mmHg. The normal mean left atrial pressure is below 12 mmHg. It is elevated if the left atrium has difficulty in emptying. This is the case if the left ventricle is uncompliant due to hypertrophy. The causes of hypertrophy include hypertension and aortic valve disease. The left ventricular end-diastolic pressure may be raised through myocardial disease, including myocardial infarction and cardiomyopathy and this would of course be communicated to the left atrium, pulmonary veins and capillaries. The left atrial pressure would also be elevated by mitral reflux and mitral stenosis and rarely too by a ball thrombus or myxoma obstructing the mitral valve. The exact level of pressure at which pulmonary oedema occurs is variable. If the left atrial pressure and pulmonary capillary pressure are persistently elevated to the colloid osmotic pressure of 25 mmHg, a slow transudation of fluid into the alveolar wall may occur, leading to thickening and fibrosis which tends to prevent further exudation until higher pressures are reached. By contrast if there is a sudden increase of pressure above the critical value of 25 mmHg oedema occurs abruptly. Any sudden elevation of pressure such as occurs on exercise, emotion, sexual intercourse or