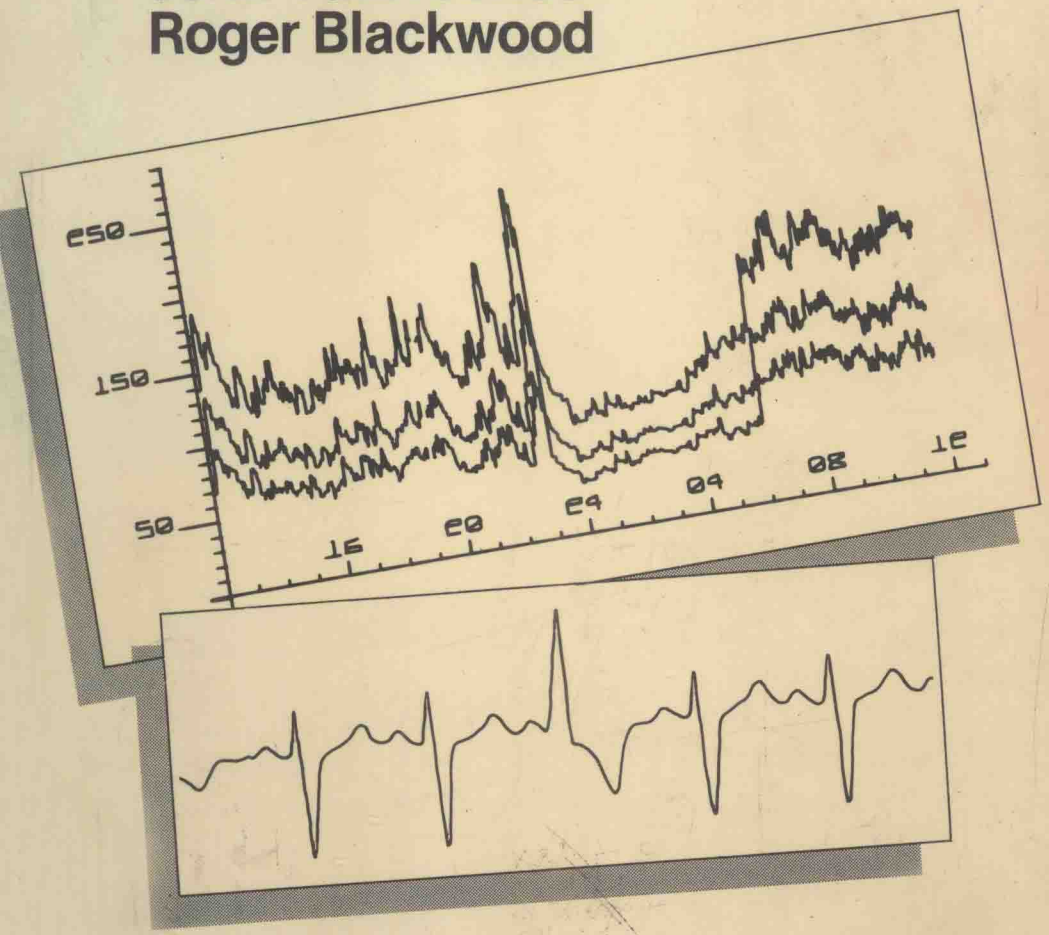
An outline of

# CARDIOLOGY

John Vann Jones
Roger Blackwood



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# An Outline of Cardiology

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1983 Bristol London Boston

*Published by:*
John Wright & Sons Ltd,
823–825 Bath Road,
Bristol BS4 5NU, England.

John Wright PSG Inc.,
545 Great Road, Littleton,
Massachusetts 01460, USA.

*British Library Cataloguing in Publication Data*

Jones, John Vann
Outline of cardiology
1. Cardiology
I. Title II. Blackwood, Roger
616. 1–2 RC681

ISBN 0 7236 0680 3

Library of Congress
Catalog Card Number:
83–50627

Typeset and printed in Great Britain by
John Wright & Sons (Printing) Ltd.
The Stonebridge Press, Bristol BS4 5NU

# Preface

The purpose of this book is to provide a short concise account of modern cardiology with emphasis on common conditions and problems. It is intended for junior hospital doctors finding themselves in their first cardiology job or studying for higher medical qualifications. It should also be of interest to medical students, to nurses or other paramedical people involved with cardiology patients, and to physicians in more general units or in other specialties who want to know a little more about diseases of the heart and circulation. We have included rather detailed descriptions of certain cardiac procedures, e.g. pacemaker insertion, because this is the sort of thing that can be difficult to glean from elsewhere.

We started writing this book when we were colleagues at the John Radcliffe Hospital, Oxford, and rather surprisingly it survived our translation to other places. We received a large amount of help, of course, not least from our secretaries Karen Fryman, Alison Nicholls and Hilary Griffin and from Ken Browne who drew all our illustrations where these are not real records that have been reproduced directly. David Bennett from Wythenshawe Hospital, Manchester, another former colleague, provided many of the traces produced in Chapter 9 and, indeed, they were 'poached' from his own book 'Cardiac Arrhythmias'. Dr Frank Ross provided most of the echocardiograms in the book. Professor Peter Sleight financed much of the initial expense and we are very grateful for his support. Finally, we would like to thank our wives, Anne and Libby, for their forbearance and encouragement.

J. V. J.
R. B.

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# 1

# The heart and circulation

## • Anatomy and Physiology

### Embryological Development of the Heart

The embryology of the heart is complex but basically starts with a single tube that has peristaltic action. At one end of the tube are gathered the veins (vitelline, cardinal and umbilical) and at the other end the arteries in the form of two dorsal aortae and six branchial arches. Remnants of these arches and aortae eventually go to form many parts of the vessels found in the adult or mature circulation, e.g. first—maxillary, second—stapedial; third—common carotid and proximal internal carotid; fourth—right subclavian and part of aorta; fifth—atrophies; sixth—pulmonary artery and ductus arteriosus (*Fig.* 1.1). The right dorsal aorta also becomes part of the right subclavian artery and the left dorsal aorta part of the aorta proper. It is soon obvious that there are four components to the heart tube, the sinus venosus, atria, ventricular inlet and ventricular outlet portions. Over the first 2 months the tube folds back on itself and rotates to the right. Septa appear and the great arteries form and migrate to their appropriate positions. The veins become incorporated partly into the atria and partly into the venae cavae. The basic human heart is formed in this way within the first 8 weeks of intrauterine life and it is hardly surprising that fairly frequently the development goes wrong.

In utero blood returns from the placenta to the right atrium where it either passes to the right ventricle and then to the pulmonary artery and then largely out across a patent ductus arteriosus, or it passes through the foramen ovale directly to the left atrium and hence to the systemic circulation. The pulmonary vascular resistance is high in utero encouraging blood to bypass the lungs and to go through the patent ductus (*Fig.* 1.2). At birth this resistance drops, the ductus and foramen ovale close and the adult pattern of circulation takes over (*Fig.* 1.3).

### Cardiac Anatomy

There are basically four cardiac chambers (*Fig.* 1.4). The superior and inferior venae cavae drain into the right atrium, a low pressure system with mean

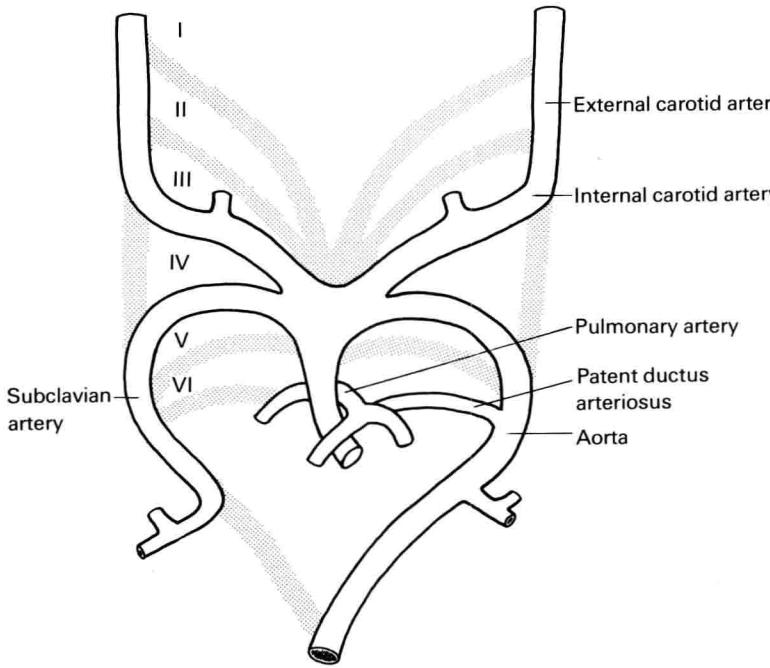


*Fig.* 1.1. The branchial arches and the formation of the great vessels.

pressures ranging from negative values to about +6 mmHg. Higher pressures than this cause venous distension and engorgement of the liver. Blood from the right atrium crosses the tricuspid valve into the right ventricle where the pressure is normally less than 30 mmHg systolic and 0–6 mmHg diastolic. This ventricle pumps blood across the pulmonary valve into the pulmonary artery where the systolic pressure also should not exceed 30 mmHg. Pressures in the peripheral pulmonary arteries are lower than in the main pulmonary artery by a few mmHg.

After oxygenation in the lungs the blood returns to the left atrium via four pulmonary veins. The pressure here is a maximum mean of 12 mmHg and is therefore considerably higher than in the right atrium. This higher pressure early in life holds the foramen ovale functionally shut until it becomes permanently closed considerably later. This potential opening is of great help to the cardiologist because it usually means that in young infants a cardiac catheter can be inserted through the foramen ovale from the venous side of the heart and into the left atrium.

Left atrial blood crosses the mitral valve into the left ventricle. Here pressures vary a lot with age but in adults the systolic pressure is at least 100 mmHg while at the end of diastole it should be less than 12 mmHg. An elevated end diastolic pressure is a useful indicator of left ventricular dysfunction. The left ventricle ejects its contents in systole over the aortic valve and into the aorta.

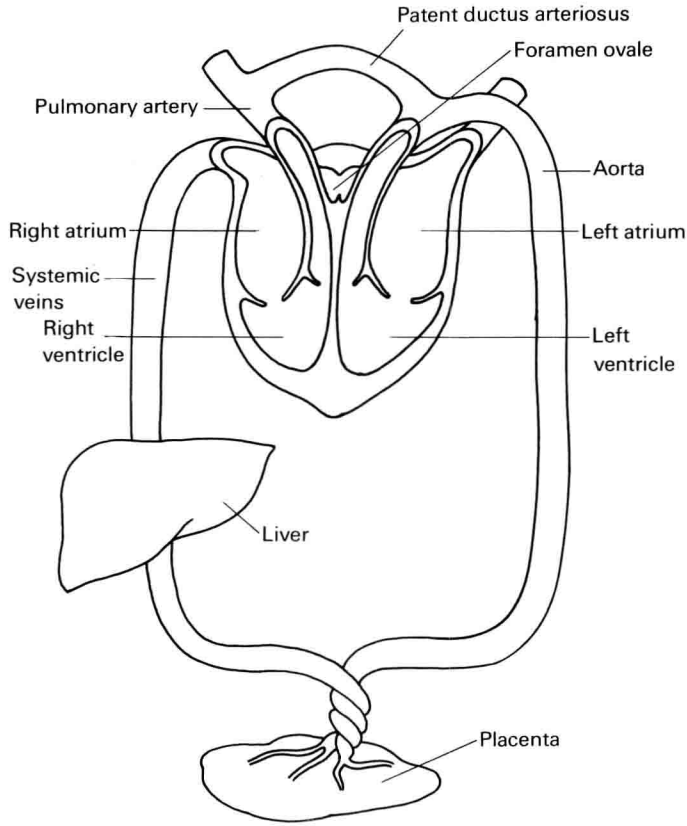


*Fig.* 1.2. The fetal circulation.

The heart's own blood supply comes from two major coronary arteries. The right coronary supplies the right atrium and right ventricle and a variable extent of the interventricular septum and inferior surface. It also gives off a branch to the sino-atrial node. The left coronary soon divides into two major branches: the left anterior descending artery which runs down the anterior surface of the heart supplying the anterior left ventricle and apex, and the circumflex artery which runs in the atrioventricular groove at the back of the heart supplying the structures there. There are two points to be made about this anatomical arrangement. The first part of the left coronary, the left main, is a vitally important vessel. Atheroma in this part is extremely dangerous as occlusion of the artery at this point would have disastrous consequences for the heart. In the atrioventricular groove the left circumflex lies beside the large coronary sinus and there is danger of damaging this vein at surgery. In addition, the circumflex, lying as it does at the back of the heart, is in any event relatively inaccessible to the surgeon when compared to the other vessels (*Fig.* 1.5).

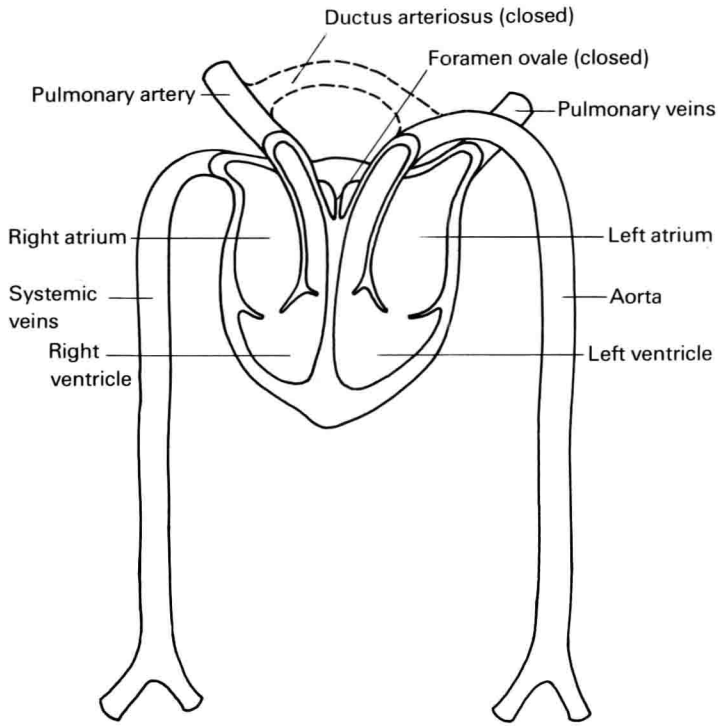


*Fig.* 1.3. The mature circulation.

The heart has four valves (*Fig.* 1.4). Those between the atria and the ventricles are the tricuspid and mitral valves. These are referred to as the atrioventricular (AV) valves and the first heart sound results from their closure at the start of systole. The second heart sound marks the end of systole and is due to the aortic and pulmonary valves closing. The aortic valve usually closes first as the high pressure aorta slams it closed. The pulmonary valve tends more to drift shut. Thus, the second heart sound is split with an aortic and pulmonary component audible. On inspiration this splitting is marked as more blood returns to the right side of the heart and delays pulmonary valve closure. Splitting of the second heart sound is particularly well heard in children and its absence or lack of variation should make you look more closely for heart disease.

**Cardiac Physiology**

The heart's basic function is to pump, but there are four such pumps which must work in proper sequence. In order to do this the pump has an internal wiring system that allows a proper sequence of filling, pumping and emptying. In addition, there are escape mechanisms to allow for failure of any part of the

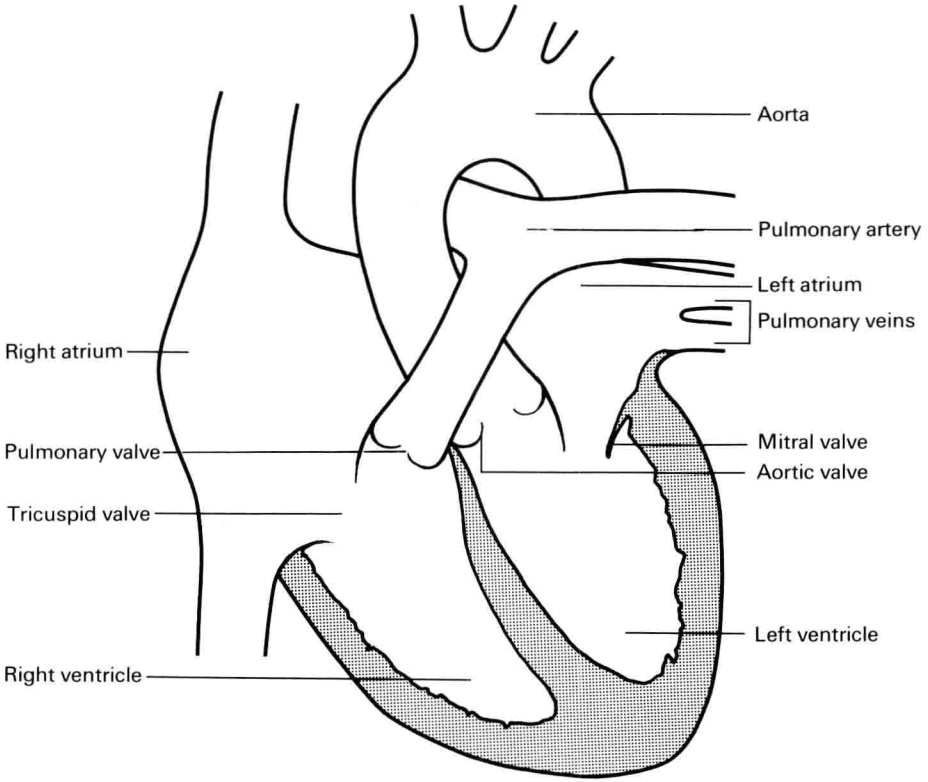


*Fig.* 1.4. The anatomy of the normal heart.

internal wiring system and there are external nerves which can alter the performance of the pump depending on the need of the body as a whole. As a final refinement there are receptors in the heart that can modify its performance and alter and respond to its work load.

**The Heart as a Series of Pumps**

The heart is best considered as two separate atrial pumps emptying into two ventricular pumps. Venous blood returns to the heart largely due to the negative intrathoracic pressure and to the skeletal muscle pumps compressing systemic veins. The atria fill passively but when they contract they tend to nip off the venae cavae so that blood, in the main, passes through the AV valves into the ventricles. The timing of atrial contraction is such that it occurs late in diastole and therefore really just primes the ventricle with a last top up. In other words, 80 per cent of venous return comes back to the atria, runs into the ventricles when the AV valves open and then finally the atria contract to pass another 20 per cent of the venous return into the appropriate ventricle. Therefore atrial systole contributes about 20

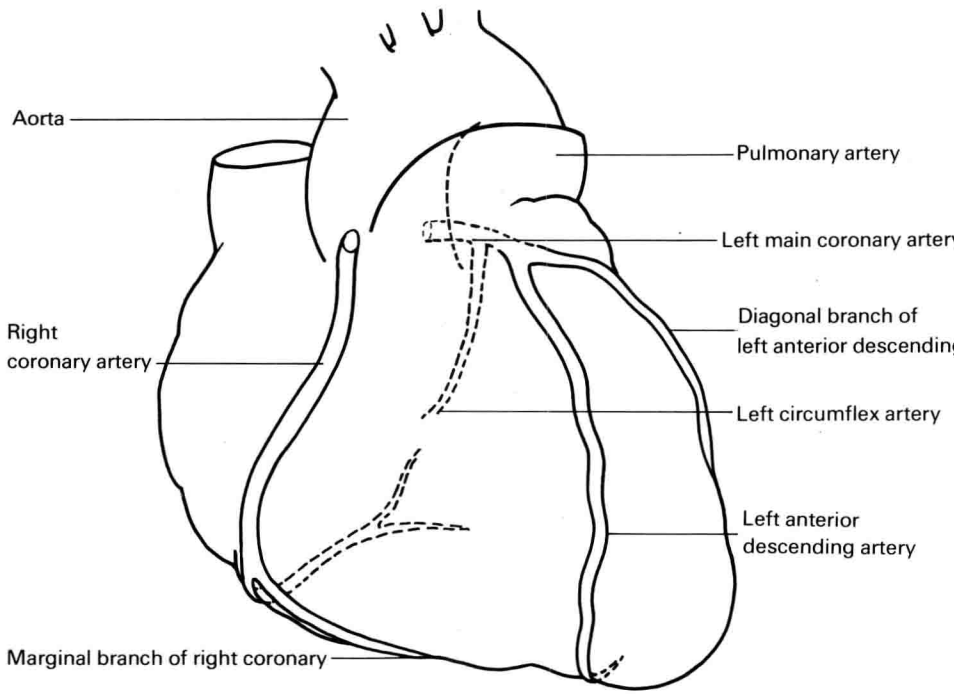


*Fig.* 1.5. The coronary arteries.

per cent of cardiac output. Where the ventricle is diseased or heart valves are damaged atrial systole can be much more important. For instance, it has been estimated that in patients surviving a myocardial infarction atrial systole may contribute to over 50 per cent of the cardiac output.

With atrial systole there is some regurgitation back into the systemic veins. This is seen as the 'a' wave in the jugular venous pulse; 'v' waves are also seen in time with ventricular systole but the exact mechanism of how they occur is under debate.

When the ventricles start to contract they close the AV valves as the intraventricular pressure rises. The ventricles effectively clamp round their contents (the period of isovolumic contraction) and the pressure rises rapidly until it exceeds the aortic or pulmonary artery pressure. The contents are then ejected (isometric contraction) and aortic and pulmonary pressures soon are greater than ventricular, the appropriate valves close and the ventricles relax. The cycle is then repeated (*Figs.* 1.6–1.7).

When blood is ejected into the aorta, the aorta distends and then recoils to force blood out into the peripheral arteries. As the aortic valve closes a notch, the dicrotic notch, appears on the pressure trace.

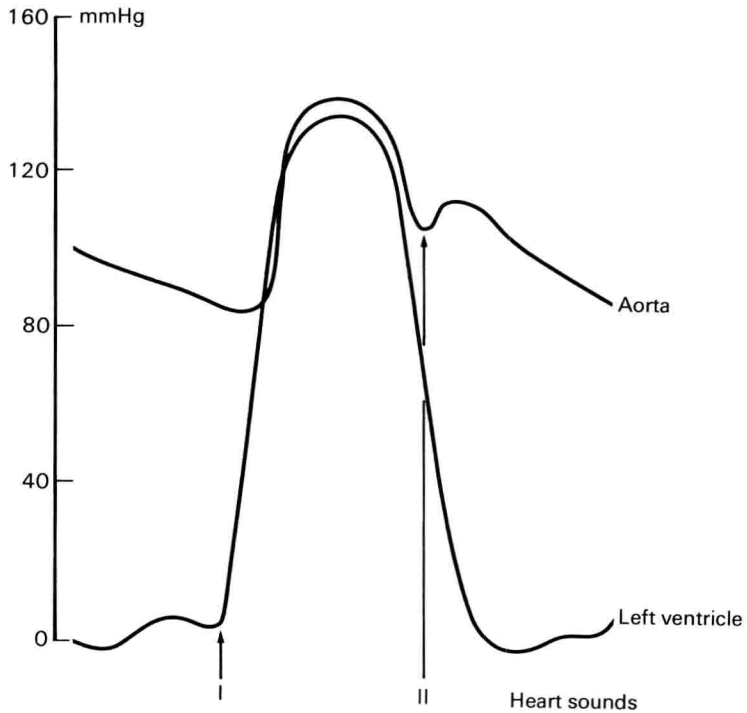


*Fig.* 1.6. Left heart pressures.

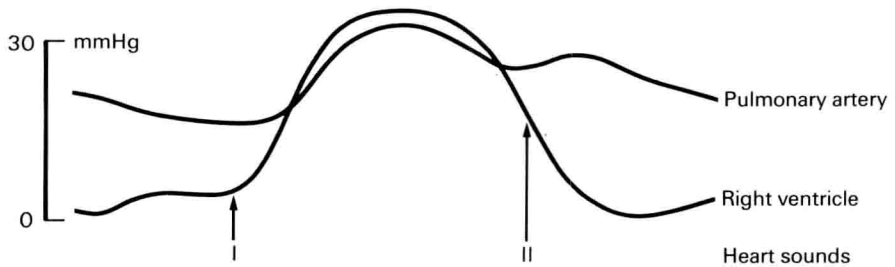


*Fig.* 1.7. Right heart pressures.

**The Frank–Starling Mechanism**

Two physiologists (Otto Frank and Ernest Starling) made many of the basic observations on intrinsic control mechanisms of the heart nearly a hundred years ago. It is now known that even the denervated heart exerts a

great deal of control over its own performance. There are three important physiological points:

1. If venous return increases, cardiac output increases.
2. If left atrial pressure increases, cardiac output increases.
3. When blood pressure rises, stroke volume and cardiac output decrease. This fall in cardiac output with increased peripheral resistance can be compensated for by an increased filling pressure.

It is not difficult to see, therefore, why left atrial pressure has to be higher than right atrial pressure because the systemic vascular resistance (and pressure) is higher than the pulmonary vascular resistance (and pressure). Thus both sides of the heart are operating this basic control mechanism and although pressures are different in this way cardiac output remains the same and in balance. As an example of this imagine the aorta being clamped. Stroke volume of the left ventricle falls because of the increased pressure. The lungs would rapidly fill with blood if the right ventricle kept up its output. The reduced venous return, however, reduces right atrial filling pressure and right ventricular output within a beat or two so that the two pumps are once more back in balance.

Starling's law of the heart states that as left ventricular end diastolic pressure increases (left atrial pressure) then cardiac output will increase in line with this provided the peripheral resistance is unchanged (*Fig.* 1.8). However, there is a limit to how far this can go and eventually the end diastolic pressure is so great and the filling so excessive the heart cannot cope. At this stage we talk of the descending limb of the Starling curve. Such a situation is seen in patients with clinical left ventricular failure.

When heart rate increases stroke volume is reduced and cardiac output stays constant, provided there is no change in peripheral resistance (*Fig.* 1.9). As

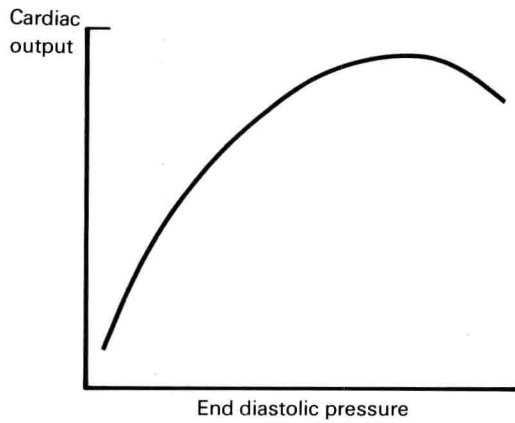


*Fig.* 1.8. The Frank–Starling curve in a normal heart.

the heart speeds up diastole is shortened and diastolic filling time reduced. Eventually, however, diastole is so short that cardiac filling is ineffective and cardiac output will fall. Such a situation is sometimes seen in patients with fast cardiac arrhythmias and explains why they feel faint or dizzy or may even lose consciousness.

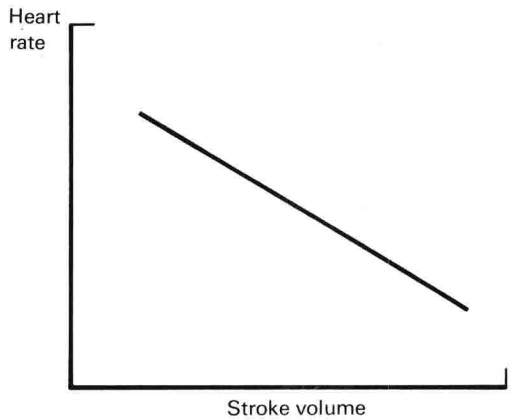


*Fig.* 1.9. Relationship between heart rate and stroke volume at constant blood pressure.

Although the heart has a great range of control over its own performance as an inherent property of the muscle itself, it is also influenced by extrinsic factors such as circulating catecholamines, parasympathetic and sympathetic nerves.

**Extrinsic Control Mechanisms**

The heart has an extrinsic innervation, mostly to the regions of the sino-atrial and atrioventricular nodes. This is both sympathetic and parasympathetic and there is a complex interplay between these two influences when the heart rate changes. The parasympathetic (vagal) discharge slows the heart (sometimes to the level of ventricular standstill or various degrees of heart block) while sympathetic discharge increases heart rate. In the normal resting situation the vagal or parasympathetic influence is dominant.

Circulating catecholamines increase myocardial contractility and cause a tachycardia. In particular the contractility of atrial muscle is improved so that there is increased ventricular filling and hence cardiac output is increased over and above that resulting from the increased heart rate.

In the denervated heart the cardiac muscle fibres have to be stretched to increase their contractility (up to a certain limit as shown by the Starling curve). Catecholamines improve myocardial contractility without increased stretch of muscle fibres so that there is an effective shift of the Starling curve to the left. Of

course sympathetic activity and catecholamines also affect the peripheral vasculature and the net result of sympathetic stimulation on cardiac performance depends upon the interplay of these various factors.

**Cardiac Receptors**

There are receptors throughout the myocardium that greatly influence, reflexly, its performance. The atrial receptors, whose impulses are then carried in myelinated nerve fibres, have been classified as type A and type B. Type A discharge in atrial systole and seem to respond to atrial wall tension while type B respond to venous distension of the atria. Activation of atrial receptors may cause tachycardia and an increase in urinary output possibly by inhibition of antidiuretic hormone (ADH) secretion. There are also atrial receptors with unmyelinated afferent fibres, discharging in response to atrial distension.

Ventricular receptors with both myelinated and unmyelinated afferent fibres have been described. The myelinated are fewer and located at the apex. They may serve to prevent gross over-distension of the heart. Unmyelinated receptors are more widespread in the left ventricle and seem to respond to the level of end diastolic pressure. Both these receptors, when activated, result in negative inotropism (reduction in contractility) and negative chronotropism (reduction in heart rate).

There are many other powerful reflex influences on the heart from receptors elsewhere, e.g. the peripheral arterial baroreceptors, the chemoreceptors and pulmonary or lung receptors. All of these can alter sympathetic or parasympathetic discharge to the heart.

**Electrical Conduction in the Myocardium** (*Fig.* 1.10)

The pacemaker of the heart lies in the sino-atrial (SA) node at the junction of the superior vena cava and right atrium. Electrical impulses then pass through the atrial muscle, but not by any established pathway, to the atrioventricular (AV) node. Here there is an inbuilt delay mechanism and then the impulse passes to the bundle of His and then out to the right and left bundle branches. There is no anatomical justification for dividing the left bundle into anterior and posterior divisions as has been done by cardiologists although from the functional point of view this is a useful division. The left bundle is extensive and blocks may occur in it at various parts and it is useful to have nomenclature to describe this.

The passage of electricity through the atria corresponds to the P wave of the ECG. Delay in the AV node gives the isoelectric period between P wave and QRS. The QRS itself results from depolarization of all the ventricular muscle fibres while the T wave is ventricular muscle being repolarized. Repolarization of atrial muscle is lost in the QRS complex while a U wave is seen after the T wave in some individuals. Its cause is not known.

**Electrical Events at Cellular Level**

When a microelectrode is inserted into a cardiac muscle cell the action potential recorded is altogether different from that seen with skeletal muscle.

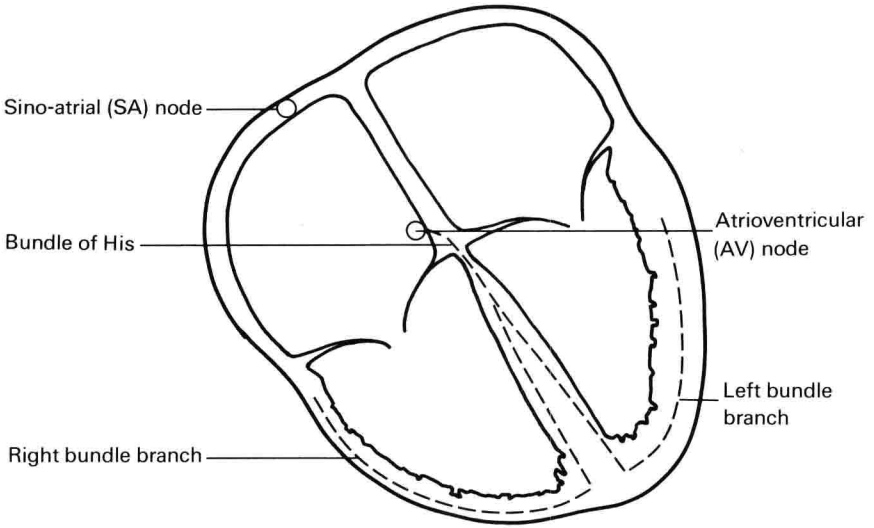


*Fig.* 1.10. Intracardiac conduction pathways.

*Fig.* 1.11 shows a typical cardiac muscle action potential although this differs in different parts of the heart. For instance it is usually of less duration in atrial and conducting tissue fibres. This example would be more typical of a ventricular muscle cell.

There are two important points to note about this action potential. First of all it has a plateau which makes the action potential of long duration (300 ms+).

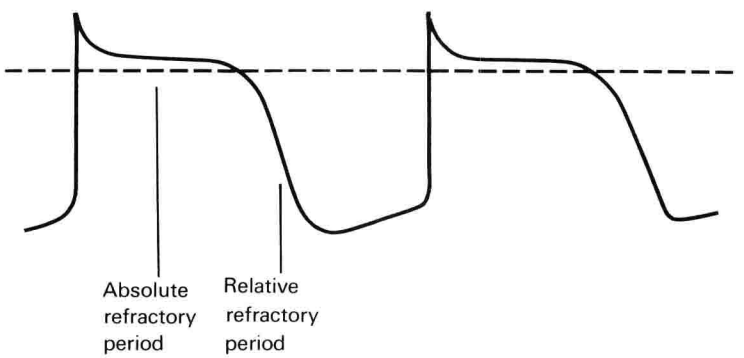


*Fig.* 1.11. Action potential in a cardiac muscle cell.

Thus there is a limit to how fast the cell can beat. Unlike with skeletal muscle tetany cannot be induced in cardiac muscle by repeated stimuli; each contraction is separate. Another stimulus can evoke an action potential at certain points in this pattern (relative refractory period). This is noteworthy because at this point the myocardial cell is unstable and arrhythmias may result. Many anti-dysrhythmic drugs have their mode of action on different parts of the action potential. The second point to note is that between action potentials there is a steady rate of electrical leakage (depolarization) so that at a certain point (*Fig.* 1.11) another action potential becomes inevitable. This leakage can be affected by autonomic nerves, catecholamines and drugs as well as by electrolyte balance. The rate of leakage varies but is fastest in tissues of the SA node so that it is the effective heart pacemaker. It does mean that other muscle fibres can take over as pacemakers if the SA node is damaged.

The exact ionic changes that accompany the action potential are not certain but initially there seems to be a rapid influx of sodium into the cell followed by a slow influx of calcium. Potassium is lost fairly rapidly to begin with and then almost half way through the action potential it moves back into the cell. During the slow decay period between action potentials potassium is lost from the cells and sodium enters. These events are of importance to the pharmacology of new drugs and already a large group (the calcium antagonists) has been developed as a result of this knowledge. Calcium does, of course, have profound effects on myocardial contractility with the result that different members of the calcium antagonist group have relatively greater or lesser effects on conduction or contractility in the heart.

**Peripheral Blood Vessels**

The heart has two sources of work. It has to take blood that is delivered to it and pass it on to either body or lungs. This is called the *preload* of the heart and is largely determined by venous return and the capacitance of the veins. If the veins are dilated venous return falls, preload is reduced and cardiac work is reduced. When the heart ejects blood the resistance to this is called the *afterload* and is a function of the arterial vessels, especially the small resistance vessels. Thus not only do arteries and veins deliver and collect blood from the various tissues and organs but they can themselves greatly influence cardiac performance. When afterload is reduced stroke volume and cardiac output can increase markedly. The situation with preload is more complex depending on whether the heart is healthy or not. Once again knowledge of this pathophysiology has led to therapeutic developments and vasodilators in the treatment of heart failure either by preload or afterload reduction or both are established drugs.

The blood vessels are under nervous control, much of it reflex in origin. There are alpha-adrenergic receptors which result in vasoconstriction and beta-adrenergic receptors which result in vasodilatation. Nervous influences are greater on the arterial side but the veins are assuming more importance as we understand more about their physiology.