Cardiovascular Physiology

Heart Physiology

Introduction

The cardiovascular system consists of the heart and two vascular systems, the systemic and pulmonary circulations. The heart pumps blood through two vascular systems - the low pressure pulmonary circulation in which gas exchange occurs, and then the systemic circulation, which delivers blood to individual organs, matching supply to metabolic demand. Blood pressure and flow is largely controlled by the autonomic nervous system and is also influenced by surgery and anaesthetic drugs.

The heart

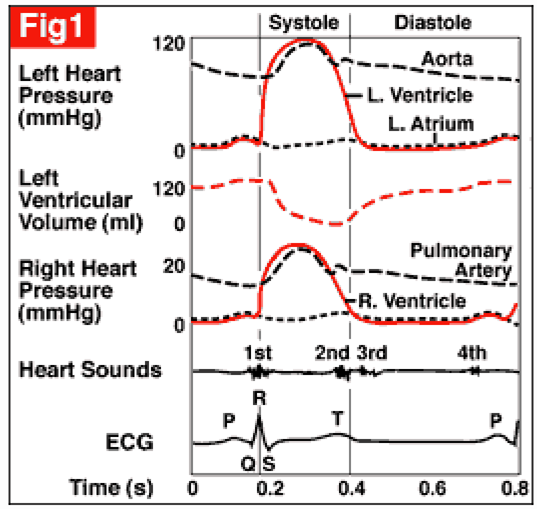
The heart comprises four chambers, and is divided into a right and left side, each with an atrium and a ventricle. The atria act as reservoirs for venous blood, with a small pumping action to assist ventricular filling. In contrast, the ventricles are the major pumping chambers for delivering blood to the pulmonary (right ventricle) and systemic (left ventricle) circulations. The left ventricle is conical in shape and has to generate greater pressures than the right ventricle, and so has a much thicker and more muscular wall. Four valves ensure that blood flows only one way, from atria to ventricle (tricuspid and mitral valves), and then to the arterial circulations (pulmonary and aortic valves). The myocardium consists of muscle cells which can contract spontaneously, also pacemaker and conducting cells, which have a specialised function.

Electrophysiology of the heart

Myocardial contraction results from a change in voltage across the cell membrane (depolarisation), which leads to an action potential. Although contraction may happen spontaneously, it is normally in response to an electrical impulse. This impulse starts in the sinoatrial (SA) node, a collection of pacemaker cells located at the junction of the right atrium and superior vena cava. These specialised cells depolarise spontaneously, and cause a wave of contraction to pass across the atria. Following atrial contraction, the impulse is delayed at the atrioventricular (AV) node, located in the septal wall of the right atrium. From here His-Purkinje fibres allow rapid conduction of the electrical impulse via right and left branches, causing almost simultaneous depolarisation of both ventricles, approximately 0.2 seconds after the initial impulse has arisen in the sinoatrial node. Depolarisation of the myocardial cell membrane causes a large increase in the concentration of calcium within the cell, which in turn causes contraction by a temporary binding between two proteins, actin and myosin. The cardiac action potential is much longer than that of skeletal muscle, and during this time the myocardial cell is unresponsive to further excitation.

The cardiac cycle

The relationship between electrical and mechanical events in the cardiac cycle is summarised in Figure 1.



There is a similar cycle on both sides of the heart, but the pressures in the right ventricle and pulmonary arteries are less than those in the left ventricle and aorta.

Systole refers to contraction, while diastole refers to relaxation. Both contraction and relaxation can be isometric, when changes in intraventricular pressure occur without a change in length of the muscle fibres.

The cycle starts with depolarisation at the sinoatrial node leading to atrial contraction. Until this time blood flow into the ventricles has been passive, but the atrial contraction increases filling by 20-30%.

Ventricular systole causes closure of the atrioventricular valves (1st heart sound), and contraction is isometric until intraventricular pressures are sufficient to open the pulmonary and aortic valves, when the ejection phase begins.

The volume of blood ejected is known as the stroke volume. At the end of this phase ventricular relaxation occurs, and the pulmonary and aortic valves close (2nd heart sound). After isometric relaxation ventricular pressures fall to less than atrial pressures. This leads to opening of the atrioventricular valves and the start of ventricular diastolic filling. The whole cycle then repeats following another impulse from the sinoatrial node.

The coronary circulation

Myocardial blood supply is from the right and left coronary arteries, which run over the surface of the heart giving branches to the endocardium (the inner layer of the myocardium). Venous drainage is mostly via the coronary sinus into the right atrium, but a small proportion of blood flows directly into the ventricles through the Thebesian veins, delivering unoxygenated blood to the systemic circulation. Oxygen extraction by the tissues is dependent on consumption and delivery. Myocardial oxygen consumption is higher than in skeletal muscle (65% of arterial oxygen is extracted as compared to 25%). Therefore any increased myocardial metabolic demand must be matched by increased coronary blood flow. This is a local response, mediated by changes in coronary arterial tone, with only a small input from the autonomic nervous system.

Cardiac Output

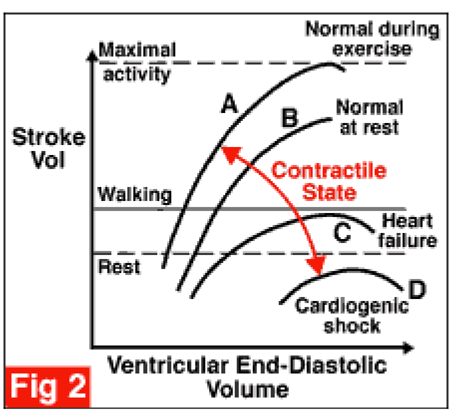
Cardiac output (CO) is the product of heart rate (HR) and stroke volume (SV):

CO = HR x SV

For a 70kg man normal values are HR=70/min and SV=70ml, giving a cardiac output of about 5litre/min. The cardiac index is the cardiac output per square metre of body surface area - normal values range from 2.5-4.0 litre/min/m^2.
Heart rate is determined by the rate of spontaneous depolarisation at the sinoatrial node (see above), but can be modified by the autonomic nervous system. The vagus nerve acts on muscarinic receptors to slow the heart, whereas the cardiac sympathetic fibres stimulate beta-adrenergic receptors and increase heart rate.

Stroke volume is determined by three main factors: preload, afterload and contractility. These will be considered in turn:

Preload is the ventricular volume at the end of diastole. An increased preload leads to an increased stroke volume. Preload is mainly dependent on the return of venous blood from the body. Venous return is influenced by changes in position, intra-thoracic pressure, blood volume and the balance of constriction and dilatation (tone) in the venous system. The relationship between ventricular end-diastolic volume and stroke volume is known as **Starling's law of the heart**, which states that the energy of contraction of the muscle is related/proportional to the initial length of the muscle fibre. This is graphically illustrated in Figure 2 by a series of "Starling curves".



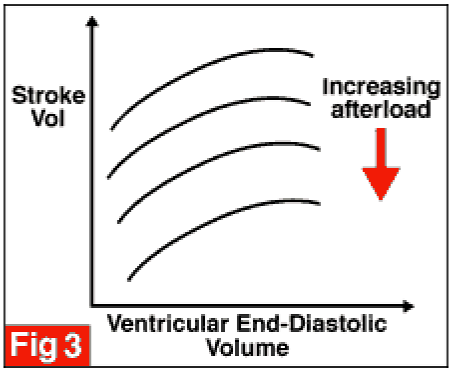
Curves A and B illustrate the rise in cardiac output with increases in ventricular end-diastolic volume (pre-load) in the normal heart. Note that with an increase in contractility there is a greater cardiac output for the same ventricular end- diastolic volume.

In the diseased heart (C and D), cardiac output is leass and falls if ventricular end-diastolic volume rises to high levels, as in heart failure or overload.

As volume at the end of diastole (**end-diastolic volume**) increases and stretches the muscle fibre, so the energy of contraction and stroke volume increase, until a point of over-stretching when stroke volume may actually decrease, as in the failing heart. Cardiac output will also increase or decrease in parallel with stroke volume **if there is no change in heart rate**.

The curves show how the heart performs at different states of **contractility**, ranging from the normal heart to one in cardiogenic shock. This is a condition where the heart is so damaged by disease that cardiac output is unable to maintain tissue perfusion. Also shown are increasing levels of physical activity which require a corresponding increase in cardiac output.

Afterload is the resistance to ventricular ejection. This is caused by the resistance to flow in the systemic circulation and is the **systemic vascular resistance**. The resistance is determined by the diameter of the arterioles and pre-capillary sphincters; the narrower or more constricted, the higher the resistance. The level of systemic vascular resistance is controlled by the sympathetic system which, in turn, controls the tone of the muscle in the wall of the arteriole, and hence the diameter. The resistance is measured in units of dyne.sec/cm^5. A series of Starling curves with differing afterloads is shown in Figure 3, demonstrating a fall in stroke volume as afterload increases.



The relationship between stroke volume and afterload. A series of curves illustrates the effects of increasing afterload on systemic vascular resistance. As afterload increases, the patient moves to a lower curve, with a lower stroke volume for the same ventricular end-diastolic volume (preload).

The relationship between systemic vascular resistance and the control of arterial pressure is discussed below.

Contractility describes the ability of the myocardium to contract in the absence of any changes in preload or afterload. In other words, it is the "power" of the cardiac muscle. The most important influence on contractility is the sympathetic nervous system. Beta-adrenergic receptors are stimulated by noradrenaline released from nerve endings, and contractility increases. A similar effect is seen with circulating adrenaline and drugs such as ephedrine, digoxin and calcium. Contractility is reduced by acidosis, myocardial ischaemia, and the use of beta-blocking and anti-arrhythmic agents.

Cardiac output will change to match changing metabolic demands of the body. The outputs of both ventricles must be identical, and also equal the venous return of blood from the body. The balancing of cardiac output and venous return is illustrated during the response to exercise. Blood vessels dilate in exercising muscle groups because of increased metabolism, and blood flow increases. This increases venous return and right ventricular preload. Consequently more blood is delivered to the left ventricle and cardiac output increases. There will also be increased contractility and heart rate from the sympathetic activity associated with exercise, further increasing cardiac output to meet tissue requirements.