CHAPTER 14

The heart as a pump

Two muscular pumps in series move the whole blood volume round the body about once a minute at rest. This chapter follows one heartbeat from the first electrical signal to the last drop of blood ejected, and then asks what sets the size of each beat.

The human heart is a pair of pumps built into a single organ. The right side receives venous blood from the body and drives it through the lungs; the left side receives the oxygenated blood and drives it round the systemic circulation. Because the two are connected in series, they must, over any period longer than a few beats, move exactly the same volume of blood. Most of what follows concerns the left ventricle, but the principles apply to both sides of the heart: the right ventricle does the same work against a pressure about one-fifth as high.

14.1 The cardiac cycle

The sequence of events from the beginning of one heartbeat to the beginning of the next is the cardiac cycle. At a resting heart rate of 72 beats per minute each cycle lasts 0.83 s, of which ventricular systole (contraction) takes about 0.3 s and diastole (relaxation) the remaining 0.5 s. When the heart rate rises, diastole shortens far more than systole: at 180 beats per minute the whole cycle lasts 0.33 s and less than half of it is left for filling.

The flow of blood through the heart is governed entirely by pressure differences and by four one-way valves. The mitral and tricuspid valves separate atria from ventricles; the aortic and pulmonary valves guard the exits of the ventricles. A valve opens when the pressure behind it exceeds the pressure ahead of it and closes when the gradient reverses. Nothing else moves them. It is convenient to divide the cycle into five phases.

Atrial systole. Contraction of the atria pushes a final volume of blood into ventricles that are already mostly full. At rest this atrial kick adds only 10–20 % of ventricular filling; it matters more at high heart rates and in a stiff ventricle, and its loss in atrial fibrillation is felt mainly on exertion.

Isovolumetric contraction. As the ventricle begins to contract, its pressure rises above atrial pressure and the mitral valve closes, producing the first heart sound. For about 0.05 s all four valves are shut. The ventricle contracts around a fixed volume of blood and its pressure climbs steeply.

Ejection. When left ventricular pressure exceeds aortic pressure, about 80 mmHg, the aortic valve opens and blood leaves the ventricle, rapidly at

first and then more slowly. Ventricular and aortic pressures rise together to a peak of about 120 mmHg.

Isovolumetric relaxation. As the muscle relaxes, ventricular pressure falls below aortic pressure; the aortic valve closes and gives the second heart sound, and a brief notch, the incisura, appears on the aortic pressure trace. Again the ventricle is a closed chamber, now relaxing at constant volume.

Ventricular filling. Once ventricular pressure drops below atrial pressure, the mitral valve opens and blood that has collected in the atrium during systole rushes in. Most filling occurs in this first third of diastole; the middle third adds little, and atrial systole completes the process.

14.2 The pressure–volume loop

Plotting left ventricular pressure against volume turns the cycle into a single closed curve, the pressure–volume loop (**Fig. 14.1**). The ventricle ends diastole holding about 120 mL, the end-diastolic volume, and ends systole with about 50 mL, the end-systolic volume. The difference, some 70 mL, is the stroke volume. The fraction of the end-diastolic volume that is ejected, here 70/120 or 58 %, is the ejection fraction; values between 55 and 70 % are normal, and the ejection fraction is the single number most often used to describe the pumping performance of a ventricle.

The area enclosed by the loop is the external work done by the ventricle in one beat, the stroke work. Each side of the loop corresponds to one phase of the cycle, and each corner to the opening or closing of a valve, so a single loop records the whole beat.

14.3 Cardiac output

The volume of blood pumped by each ventricle per minute is the cardiac output. It is the product of stroke volume and heart rate: 70 mL × 72 beats per minute gives about 5 L/min in a resting adult, which means that the entire blood volume passes through each side of the heart roughly once a minute. Because output scales with body size, it is often divided by body surface area; the resulting cardiac index is about 3 L/min per square metre. Dur-

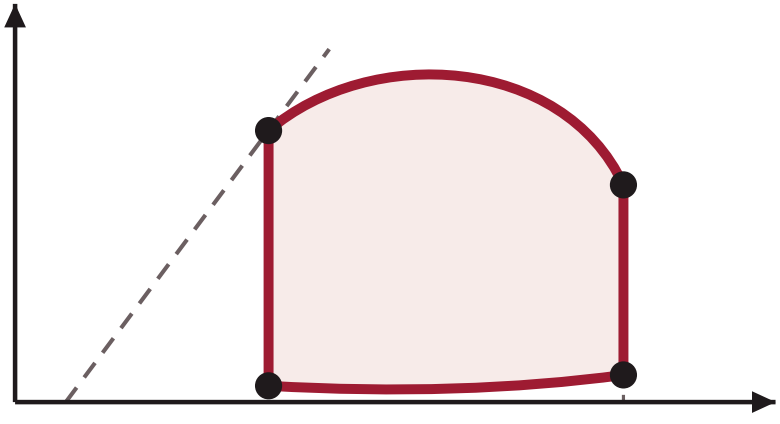

Figure 14.1. Pressure–volume loop of the left ventricle at rest: volume from 0 to 150 mL across, pressure from 0 to 140 mmHg up. The dots, anticlockwise from bottom right: mitral valve closes (120 mL), aortic valve opens (80 mmHg), aortic valve closes, mitral valve opens (50 mL). Dashed: the end-systolic pressure–volume relation.

ing maximal exercise cardiac output rises four- to fivefold in an untrained young adult and to 30–35 L/min in an endurance athlete, whose larger heart achieves this mainly through a greater stroke volume.

Every change in cardiac output is a change in heart rate or in stroke volume, or in both. The rest of this chapter considers each in turn.

14.4 The sinoatrial node and the conduction system

Cardiac muscle does not need a nerve to contract. The beat starts in the sinoatrial node, a small strip of specialised muscle in the wall of the right atrium near the entry of the superior vena cava. Its cells have no stable resting potential: after each action potential the membrane depolarises slowly, largely through the so-called funny current, until it reaches threshold and fires again. Isolated from all nervous influence, the node fires about 100 times a minute. The resting rate of 60–80 beats per minute is lower because the vagus nerve continuously releases acetylcholine onto the node and slows this pacemaker depolarisation.

From the node the impulse spreads through both atria and converges on the atrioventricular node, which conducts at only about 0.05 m/s. The resulting delay of roughly 0.1 s lets the atria finish emptying before the ventricles contract. Beyond the node the impulse runs down the bundle of His and its branches into the Purkinje fibres, which conduct at up to 4 m/s and activate the whole ventricular mass within about 0.08 s.

The sinoatrial node sets the pace only because it is the fastest. If it fails, the atrioventricular node takes over at 40–60 beats per minute, and the Purkinje fibres, if conduction through the node is blocked, at 15–40.

14.5 Regulating stroke volume

Three factors determine how much blood the ventricle ejects with each beat: the volume it holds before it contracts, the pressure it must overcome, and the strength of its contraction.

Preload and the Frank–Starling mechanism. The more a ventricle is filled during diastole, the more forcefully it contracts and the more blood it ejects. This intrinsic property of heart muscle is the Frank–Starling mechanism, after the German physiologist Otto Frank and the British physiologist Ernest Starling, who described it in 1895 and 1914. Stretching a cardiac muscle fibre brings its sarcomeres towards an optimal length of about 2.2 µm, at which the overlap of actin and myosin filaments, and the sensitivity of the filaments to calcium, are greatest. The degree of stretch before contraction is the preload, usually taken as the end-diastolic volume. The mechanism keeps the two ventricles in step: if the right ventricle pumps a little more for a few beats, more blood reaches the left ventricle, which stretches and ejects the extra volume.

Afterload. The afterload is the load the ventricle works against once it starts to eject, which for the left ventricle is essentially the aortic pressure. A sudden rise in afterload means the aortic valve opens later and closes earlier, so the ventricle ejects less and is left with a larger end-systolic volume.

Over the next few beats the Frank–Starling mechanism restores the stroke volume at the cost of a larger heart.

Contractility. A ventricle can also eject more blood from the same end-diastolic volume if its fibres contract more strongly. This property, contractility or inotropy, is raised above all by the sympathetic nervous system. Noradrenaline released from sympathetic nerves acts on beta-1 adrenergic receptors of the muscle cells, increases calcium entry during each action potential, and makes each contraction stronger and shorter. The same transmitter acting on the sinoatrial node increases the heart rate, so sympathetic stimulation raises cardiac output through both of its factors at once.

These three mechanisms rarely act alone. During exercise, venous return increases the preload, sympathetic activity raises contractility and heart rate, and dilatation of the muscle arterioles keeps the afterload from rising in proportion to the flow.

HEART FAILURE

In heart failure the heart cannot pump enough blood for the needs of the body at normal filling pressures. When the ejection fraction falls to 40 % or less, the condition is called heart failure with reduced ejection fraction; many patients, however, have a normal ejection fraction and a stiff ventricle that fills poorly. Older texts use the term cardiac insufficiency.

14.6 Listening to the heart

The closure of the valves can be heard through the chest wall, and auscultation with the stethoscope, which the French physician René Laennec introduced in 1816, remains the first examination of the heart. The first heart sound, low-pitched and relatively long, marks the closure of the mitral and tricuspid valves at the start of systole; it is best heard over the apex. The second, shorter and sharper, marks the closure of the aortic and pulmonary valves at the end of ejection. During inspiration the fall in intrathoracic pressure increases venous return to the right ventricle, which then ejects for slightly longer, so the pulmonary valve closes a few hundredths of a second after the aortic valve: the second sound is heard split.

A third heart sound in early diastole comes from the rapid inflow of blood into the ventricle. It is normal in children and young athletes, but after the age of about forty it usually means a dilated, failing ventricle. A fourth sound, just before the first, is produced by atrial systole against a stiff ventricle.

Between the sounds the heart is normally silent, because blood flows smoothly through open valves. A murmur is the noise of turbulent flow: through a narrowed valve that must still open (stenosis) or through a valve that fails to close (regurgitation). Its timing in the cycle tells the examiner which valve is at fault; an aortic stenosis, for example, produces a murmur during ejection, between the first and the second sounds.

CHAPTER 15

Vessels and blood pressure

The heart supplies the pressure; the vessels decide where the blood goes. This chapter explains how a few micrometres of change in the radius of small arteries redistribute the cardiac output, and how the brainstem keeps arterial pressure steady from one heartbeat to the next.

Blood leaves the left ventricle in pulses and reaches the capillaries as a steady stream. Between the two lies a branching system of tubes whose walls differ in thickness, in elastic tissue and in smooth muscle, and whose properties explain most of what follows: why arterial pressure has a systolic and a diastolic value, why pressure falls so steeply in the smallest arteries, and why most of the blood in the body is found in the veins.

15.1 The vascular tree

The aorta and its large branches are arteries rich in elastic fibres. They expand as each stroke volume enters and recoil during diastole, so that flow continues between beats. Smaller arteries carry more smooth muscle and distribute blood to the organs. The arterioles, 10–100 µm across, have the thickest walls for their size and are the main site of resistance to flow: as **Tab. 15.1** shows, mean pressure falls from about 85 to 35 mmHg across them. By contracting or relaxing their smooth muscle, the arterioles set both the total resistance of the circulation and the share of the cardiac output that each organ receives.

The capillaries are tubes of a single layer of endothelial cells, 5–8 µm in diameter, just wide enough for a red cell to squeeze through. There are some

Segment	Blood volume (%)	Mean pressure (mmHg)
Arteries	13	100 to 85
Arterioles	7	85 to 35
Capillaries		35 to 15
Venules and veins	64	15 to 0
Heart	7	–
Pulmonary circulation	9	15 to 8

Table 15.1. Blood volume and mean pressure along the circulation of a resting adult.

ten billion of them, with a total exchange surface of 500–700 m^2, and blood moves through them at about 0.3 mm/s, a thousandth of its mean velocity in the aorta. Blood returns through venules and veins, whose walls are thin and easily distended. At rest the systemic veins hold about 64 % of the blood volume, which is why they are called capacitance vessels.

15.2 Flow, pressure and resistance

Flow through any vessel obeys a relation analogous to Ohm's law: flow equals the pressure difference between the two ends divided by the resistance, $Q = (P_1 - P_2)/R$. Applied to the whole systemic circulation, with a mean arterial pressure of 93 mmHg, a right atrial pressure close to zero and a cardiac output of 5 L/min, it gives a total peripheral resistance of about 19 mmHg·min/L.

What determines the resistance of a vessel? For steady, streamlined flow of a fluid through a rigid tube, the answer is Poiseuille's law, derived in the 1840s by the French physician Jean Poiseuille from experiments on water flowing through glass capillaries. The resistance is proportional to the length of the tube and to the viscosity of the fluid, and inversely proportional to the fourth power of the radius, r^4. Halving the radius of an arteriole multiplies its resistance by sixteen; a rise in radius of only 19 % doubles the flow through it at the same pressure. This extreme sensitivity is what gives the arterioles their control over the circulation. The viscosity of blood, about three to four times that of water, depends mainly on the haematocrit, and it rises steeply when the haematocrit exceeds 60 %.

Poiseuille's law assumes laminar flow, in which the blood moves in concentric layers, fastest at the centre. When velocity is high, the vessel wide or the wall irregular, the layers break into eddies. The tendency to such turbulent flow is expressed by the Reynolds number, the product of the density of blood, its velocity and the diameter of the vessel divided by the viscosity. Above about 2000 flow becomes turbulent, and turbulence is audible: a murmur over a narrowed valve, a bruit over a stenosed artery, and the Korotkoff sounds heard when blood pressure is measured with a cuff.

Vessels arranged in series add their resistances; vessels arranged in parallel add their conductances, so the total resistance of a parallel network is lower than that of any one of its branches. The organs of the body are supplied in parallel, which allows each to adjust its own blood flow without much effect on the others.

15.3 Arterial pressure

In a healthy young adult arterial pressure rises to about 120 mmHg with each ejection, the systolic pressure, and falls to about 80 mmHg before the next, the diastolic pressure. The difference, 40 mmHg, is the pulse pressure. Because diastole lasts about twice as long as systole at rest, the average pressure over the cycle lies closer to the diastolic value. It is estimated as the diastolic pressure plus one-third of the pulse pressure:

$$\mathrm{MAP} = \mathrm{DBP} + (\mathrm{SBP} - \mathrm{DBP})/3 = 80 + 40/3 = 93\ mmHg$$

This mean arterial pressure is the pressure that drives blood through the systemic circulation. At high heart rates diastole shortens and the true mean moves towards the arithmetic mean of systolic and diastolic pressure.

The pulse pressure depends mainly on two quantities: the stroke volume and the arterial compliance, the change in arterial volume per unit change in pressure. The elastic arteries store about half of each stroke volume during systole and return it during diastole, an arrangement known as the Windkessel effect after the air chamber of eighteenth-century fire engines, which turned the strokes of the pump into a steady jet. With age the aorta stiffens, compliance falls, and the same stroke volume produces a higher systolic and a lower diastolic pressure.

HYPERTENSION

Sustained hypertension is diagnosed when the arterial pressure measured at rest on repeated occasions is 140/90 mmHg or higher; American guidelines since 2017 use 130/80 mmHg. In older people a high systolic pressure with a normal or low diastolic pressure, isolated systolic hypertension, reflects the loss of aortic compliance.

15.4 Capillary exchange

Across the capillary wall, fluid movement is determined by the balance between hydrostatic pressure, which pushes fluid out, and the colloid osmotic pressure of the plasma proteins, which holds it in. These Starling forces favour filtration at the arterial end of the capillary, where the hydrostatic pressure is about 35 mmHg against an oncotic pressure of 25 mmHg, and reabsorption at the venous end, where the hydrostatic pressure has fallen to about 15 mmHg. Of the 20 L or so filtered each day in the systemic capillaries, some 17 L return directly to the blood; the rest is carried back by the lymph vessels. When filtration outstrips this return, fluid gathers in the tissues as oedema.

15.5 The baroreceptor reflex

Arterial pressure is held within a few millimetres of mercury of its set point from moment to moment by the baroreceptor reflex. Baroreceptors are stretch-sensitive nerve endings in the wall of the carotid sinus, at the bifurcation of each common carotid artery, and in the aortic arch. Their firing rises with each systolic distension. Signals from the carotid sinus travel in the glossopharyngeal nerve and those from the aortic arch in the vagus nerve; both end in the nucleus tractus solitarius of the medulla.

A rise in pressure increases baroreceptor firing, which inhibits the sympathetic outflow to the heart and vessels and increases vagal activity. Heart rate and contractility fall, the arterioles dilate, and pressure returns towards normal. A fall in pressure has the opposite effects. The carotid receptors respond between about 60 and 180 mmHg and are most sensitive around the normal mean arterial pressure. Within one or two days, however, they reset to any pressure that is sustained, so the reflex corrects rapid changes but cannot, by itself, set the long-term level of arterial pressure, which depends on the handling of salt and water by the kidneys.

The reflex is tested every time a person stands up. Some 500 mL of blood pools in the veins of the legs, venous return and stroke volume fall, and arterial pressure starts to drop; within a few seconds the reflex raises the heart rate and constricts the arterioles. When it fails, as it may in the elderly or in

diseases of the autonomic nerves, standing causes a fall in systolic pressure of 20 mmHg or more, orthostatic hypotension, with dizziness or fainting.

15.6 Venous return

Whatever the heart pumps out must first come back to it. The flow of blood from the veins into the right atrium, the venous return, is driven by a small pressure difference: about 7 mmHg between the venules and the right atrium, where the central venous pressure is close to zero. Because the veins are so compliant, a small change in their tone moves a large volume of blood. Sympathetic constriction of the veins can shift several hundred millilitres from the peripheral veins towards the heart within seconds, raising the preload and, through the Frank–Starling mechanism, the stroke volume.

Two pumps outside the heart help. In the legs, the deep veins run between the muscles and contain one-way valves every few centimetres. Each contraction of the calf squeezes the veins and drives blood upwards, and the valves stop it from falling back; this skeletal muscle pump can lower the venous pressure at the ankle of a walking person from about 90 mmHg to 25 mmHg or less. When the valves fail, the veins of the legs dilate and become tortuous, as varicose veins. In the chest, each inspiration lowers the intrathoracic pressure and raises the abdominal pressure, drawing blood from the abdominal veins into the thorax: the respiratory pump.

Over any period longer than a few beats, venous return and cardiac output are equal. This is why the heart, although it generates the pressure, is not the only organ that sets the output: in a healthy person at rest, the cardiac output follows the venous return, and the venous return follows the metabolic needs of the tissues, each of which controls its own blood flow through its arterioles.

Index

Numbers in bold mark the principal discussion of a term. A range such as 301–3 means that the discussion runs across those pages. See sends you to the entry that holds the page numbers; see also to a related entry.

Principles of Human Physiology, Part IV, chapters 14 and 15, a sample set with Postext. Set in Literata and Libre Franklin (SIL OFL 1.1). Text and drawing: original, CC BY 4.0. The book and its authors are fictional.