

### In this article...

- Functioning of the heart and its components – chambers, valves, arteries and veins
- The conduction system, heart rhythm and cardiac cycle (diastole and systole)
- Cardiac examination – inspection, palpation and auscultation

# Cardiac system 1: anatomy and physiology

## Key points

The heart is a muscle that contracts and relaxes, pumping blood through the body

The heart cavity is divided into two atria and two ventricles separated by cardiac valves

Blood supply goes into the heart via the coronary arteries and is drained via the coronary veins

The heart has its own conduction system, the sinoatrial node being its natural pacemaker

During the cardiac cycle, the chambers of the heart contract and relax, and blood flows from high-pressure to low-pressure areas

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**Abstract** The heart is a complex organ that pumps blood through the body with an intricate system of muscle layers, chambers, valves and nodes. It has its own circulation system and receives electric impulses that make it contract and relax, which triggers a sequence of events forming the cardiac cycle. A solid and methodical understanding of how the heart works is key to understanding what can go wrong with it. This first article in a two-part series covers anatomy and physiology, and the second part discusses pathophysiology.

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The heart is the key organ of the cardiovascular system – the body's transport system for blood. A muscle that contracts rhythmically and autonomously, it works in conjunction with an extensive network of blood vessels running throughout the body. Basically, the heart is a pump ensuring the continuous circulation of blood in the body. This article describes the heart's anatomy and physiology.

### What is the heart?

The heart weighs around 350g and is roughly the size of an adult's clenched fist. It is enclosed in the mediastinal cavity of the thorax between the lungs, and extends downwards on the left between the second and fifth intercostal space (Fig 1). If one draws an imaginary line from the middle of the left clavicle down to below the nipple, this is where the most forceful part of the heart, the apex beat, can be felt.

The heart has a middle muscular layer, the myocardium, made up of cardiac muscle cells, and an inner lining called the

endocardium. The inside of the heart (heart cavity) is divided into four chambers – two atria and two ventricles – separated by cardiac valves that regulate the passage of blood.

The heart is enclosed in a sac, the pericardium, which protects it and prevents it from over-expanding, anchoring it inside the thorax. The pericardium is attached to the diaphragm and inner surface of the sternum, and is made up of:

- The fibrous pericardium, composed of a loosely fitting but dense layer of connective tissue;
- The serous pericardium or epicardium, composed of the parietal and visceral layers;
- A film of serous fluid between the fibrous and serous pericardium that allows them to glide smoothly against each other.

### Atria and ventricles

The atria receive blood returning to the heart, while the ventricles receive blood from the atria – via the atrioventricular valves – and pump it into the lungs and

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the rest of the body (Fig 2a). The left atrium (LA) and left ventricle (LV) are separated from the right atria (RA) and right ventricle (RV) by a band of tissue called the septum.

The RA receives deoxygenated blood from the head and neck and from the rest of the body via the superior and inferior vena cava, respectively. The RV then pumps blood into the lungs (through the pulmonary trunk, which divides into the right and left pulmonary arteries), where it is oxygenated. The oxygenated blood is returned to the LA via the pulmonary veins and passes into the LV through the cardiac valves. From the LV, it is delivered to the whole body through the aorta.

The RV does not need a huge amount of force to pump blood into the lungs, compared with the LV, which has to pump blood into the rest of the body. The LV has a thicker wall and its cavity is circular, while the RV cavity is crescent-shaped with a thinner wall (Marieb and Hoehn, 2015).

### Cardiac valves

When working correctly, the cardiac valves (Fig 2b) ensure a one-way system of blood flow. They have projections (cusps) held in place by strong tendons (chordae tendinae) attached to the inner walls of the heart by small papillary muscles.

The RA and RV are separated by the tricuspid valve, which has three leaflets. The tricuspid valve allows deoxygenated blood to move from the RA into the RV. From the RV, blood passes through the pulmonary valve (situated between the RV and the pulmonary artery), allowing deoxygenated blood to enter the lungs.

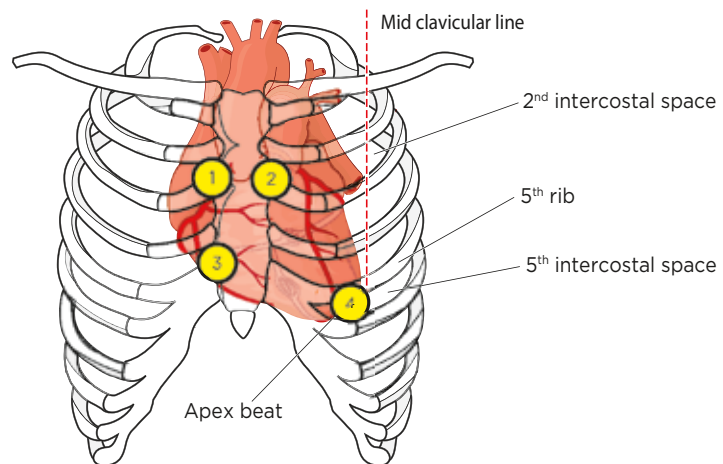
On the left side of the heart, oxygenated blood from the lungs enters the LA from the pulmonary vein. The LA is separated from the LV by the mitral valve (also called bicuspid valve, as it has two leaflets (Fig 2b)) and blood flows through this valve into the LV. It then passes through the aortic valve into the aorta, which transports oxygenated blood throughout the body.

### Coronary circulation

The heart itself requires a richly oxygenated blood supply to support its activity. This is delivered via the right and left coronary arteries, which lie on the epicardium and penetrate the myocardium with deeper branches to supply this highly active layer of muscle.

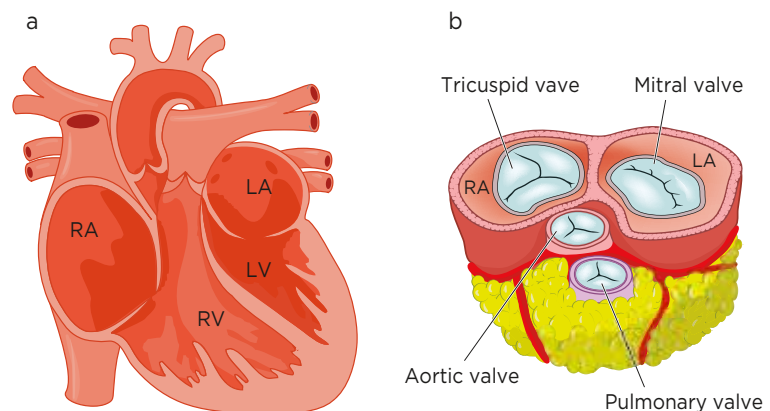
The right and left coronary arteries arise from vascular openings at the base of the aorta, called the coronary ostia. The left coronary artery runs towards the left side of the heart, dividing into the left anterior

Fig 1. Location of the heart and landmarks



Particular valves can be best heard in certain positions: 1 = aortic valve; 2 = pulmonary valve; 3 = tricuspid valve; 4 = mitral valve

Fig 2a and 2b. Heart chambers and cardiac valves



LA = Left atrium; LV = Left ventricle; RA = Right atrium; RV = Right ventricle

descending artery and the left circumflex artery. The right coronary artery runs down the right side of the heart dividing into the marginal artery (lateral part of the right-hand side of the heart) and posterior descending artery (supplying the posterior part of the heart) (Fig 3).

The coronary arteries provide an intermittent supply of blood to the heart, predominantly when the heart is relaxed (during diastole), as the entrance to the

coronary arteries is open at that point of the cardiac cycle. Table 1 shows which regions of the heart are supplied by which coronary arteries.

The venous drainage system of the heart uses the coronary veins, which follow a course similar to that of the coronary arteries. The coronary sinus is a collection of coronary veins (small, middle, great and oblique veins, left marginal vein and left posterior ventricular vein) that drain into the RA

Table 1. Coronary arteries and regional blood supply to the heart

Coronary artery	Anatomical region of the heart supplied by it
Right coronary artery	Inferior aspect
Left anterior descending artery	Anteroseptal and anteroapical aspects
Left circumflex artery	Anterolateral aspect
Right coronary artery	Posterior aspect

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at the posterior aspect of the heart. Two thirds of the cardiac venous blood is returned to the heart via the coronary sinus, while one third is returned directly into the heart (with the anterior cardiac veins opening directly into the RA and the smallest coronary veins into all four chambers).

### The conduction system and heart rhythm

The cardiac muscle has the ability to undergo depolarisation (change in the excitation of a cell), which leads to a contraction of the muscle cells.

In the heart, the electrical changes needed to generate a cardiac impulse are regulated by its own conduction system, which starts with a sequence of excitation in a specialised area of cardiac cells, the sinoatrial node (SAN), situated in the right atrium. This is the heart's natural pacemaker. When working properly, it sets the heart rhythm (sinus rhythm) and initiates impulses that act on the myocardium, stimulating cardiac contraction. The cardiac impulse passes from the SAN into the atria, which starts to contract, and the impulse is transmitted to another mass of specialised cells, the atrioventricular node (AVN).

The AVN is situated in the inter-atrial septum, a band of tissue between the RA and LA that provides a pathway of conduction between the atria and the ventricles. There is a slight delay (of 0.1 seconds) of the impulse at the AVN because the fibres of the AVN are smaller, which gives the atria time to contract and empty into the ventricles before ventricular contraction occurs.

The impulse then travels down into a large bundle of specialised tissue, the Bundle of His, which conducts it down the ventricles. The Bundle of His subsequently splits into the right and left bundles in the inter-ventricular septum. Purkinje fibres then continue down to the inferior aspect of the heart, before looping upwards and travelling in the lateral aspects of the RV and LV (Fig 4).

### Cardiac cycle

The chambers of the heart contract and relax in a coordinated fashion. The contraction phase is referred to as 'systole' and the relaxation phase, when the heart fills up again, as 'diastole'. The RA and LA synchronise during atrial systole and diastole, while the RV and LV synchronise during ventricular systole and diastole. One complete cycle of these events is referred to as the cardiac cycle.

During the cardiac cycle, the pressure in the cardiac chambers increases or falls, affecting valve opening or closure, thereby

Fig 3. **Coronary arteries**

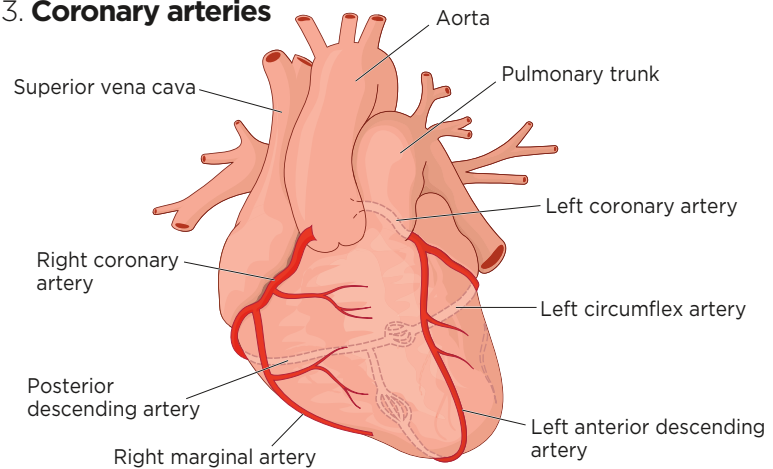
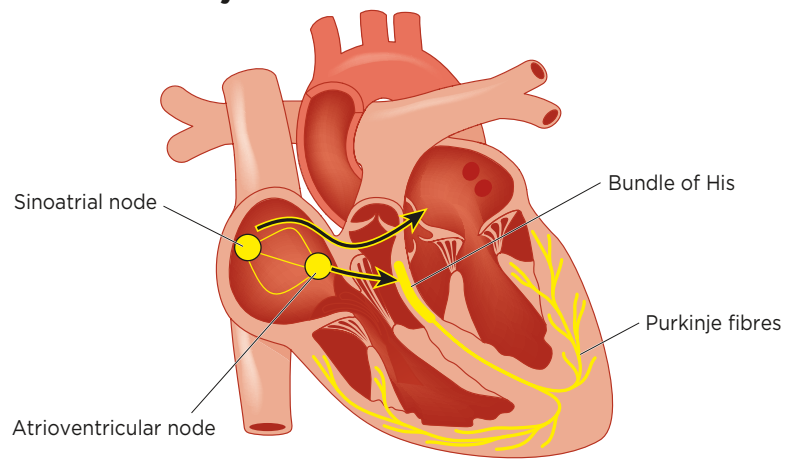


Fig 4. **Conduction system of the heart**



regulating blood flow between the chambers. Pressures in the left side of the heart are around five times higher than in the right side, but the same volume of blood is pumped per cardiac beat.

The cardiac cycle can be broken down into a sequence of events based on the principle that any blood flow through the chambers depends on pressure changes, as blood will always flow from a high-pressure to a low-pressure area (Marieb and Hoehn, 2015). The process is shown in Fig 5 and described below.

### Atrial systole and ventricular filling

At this part of the cardiac cycle, the pressure in the heart is low and the blood from the circulation passively fills the atria on both sides. This culminates in the opening of the atrioventricular valves and blood moving into the ventricles. Around 70% of ventricular filling occurs during this phase. After depolarisation of the atria (P wave on an electrocardiogram [ECG]), the atria contract compressing blood in

the atrial chambers and push residual blood out into the ventricles.

This signifies the last part of the ventricular resting phase (diastole) and the blood within the ventricles is referred to as the end diastolic volume (EDV). The atria then relax and then the electrical impulse is transmitted to the ventricles, which undergo depolarisation (QRS wave on an ECG).

### Ventricular systole

At this point, the atria are relaxed and the ventricles begin to contract. This contraction of the ventricles leads to an increase in ventricular pressures within the cavity. As the pressure rises, it exceeds the pressure within the arteries, forcing the opening of the aortic and pulmonary valves as blood is ejected from ventricles and into these large vessels.

### Isovolumetric relaxation

At this point, the ventricles relax and any blood remaining in the chamber is called end systolic volume (ESV). The ventricular pressure precipitously drops and as this

occurs, blood within the aorta and pulmonary trunk momentarily backflow and the aortic and pulmonary valves close. This backflow causes a brief rise in the pressure in the aorta giving a characteristic change in the pressure of the cardiac cycle called the dicrotic notch. While the ventricles have been in systole, the atria are in diastole and fill again ready for the next cardiac cycle.

### Cardiac output and stroke volume

Cardiac output (CO) is the amount of blood pumped out by the heart in one minute. CO can be calculated using a simple equation: the stroke volume (SV) – the volume of blood pumped by the ventricles with each heart beat – multiplied by the heart rate.

First, one needs to calculate the SV – the difference between the EDV (the volume of blood left in the ventricles during diastole) and the ESV (the volume of blood remaining in the ventricles after it has contracted).

If the EDV is 120ml and the ESV is 50ml, the SV will be:

- 120ml (EDV) - 50ml (ESV) = 70 ml/beat (SV)

Once the SV has been determined, the CO can be calculated. If the SV is 70mls and the heart rate is 70bpm, the CO will be:

- 70ml (SV) x 70bpm (heart rate)  
= 4,900ml/min (CO)

The CO can vary; for example, it will increase in response to metabolic demands such as exercise or pregnancy. In pathological states such as heart failure, the CO may not be sufficient to support simple activities of daily living or to increase in response to demands such as mild-to-moderate exercise (Jarvis and Saman, 2017).

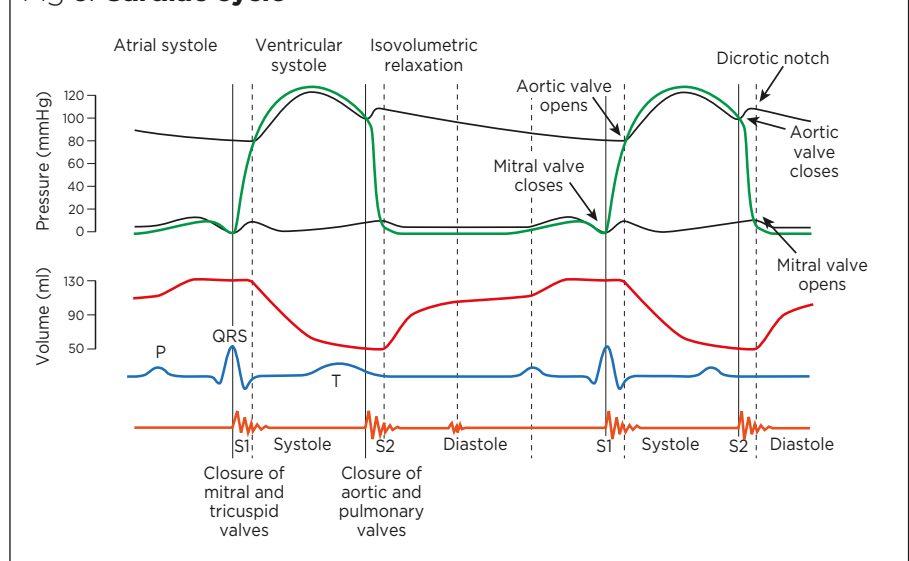
### Frank-Starling law

A physiological principle underpinning cardiac function is the Frank-Starling law, which proposes that the critical factor affecting SV is the preload – that is, the blood that goes from the returning circulation into the heart during its filling.

The amount of preload determines the volume of blood that can leave the heart (the CO) and influences the stretch and tension on the individual muscle cells which make up the cardiac fibres. The SV increases in response to preload. As a result of the filling, increased pressure in the ventricles increases the stretching of the cardiac muscle fibres. This stretch culminates in increased contractility of the heart and increased CO. Up to a certain physiological limit, the preload and contractility of the heart are positively correlated. This explains how exercise can improve cardiac performance.

Many hormones and chemicals can affect the contractility of the heart. Factors

Fig 5. Cardiac cycle



that enhance contractility – such as adrenaline and thyroxine – are said to have a positive inotropic effect. Conversely, factors that decrease contractility – such as calcium channel blockers – are said to have a negative inotropic effect on the heart (Marieb and Hoehn, 2015).

### Clinical examination

Clinical examination of the heart requires several steps (Douglas et al, 2013) in an orderly sequence of inspection, palpation and auscultation, starting from the patient's hands.

There should be careful assessment of the pulse (whether it is strong/weak/slow rising), its rate per minute, its character and rhythm (regular or irregular). The venous pressure in the neck veins (jugular pressure) should be assessed to help understand fluid status; it can reveal signs of heart failure or valve disease.

Palpating the anterior chest wall (precordium) allows clinicians to assess the force of the heart: a failing valve can be felt as a thrill, while hypertrophy of the heart muscle may lead to a heave. The apex beat should be felt to ensure it is where it should be – on the mid-clavicular line at the fifth intercostal space (Fig 1); this is part of the routine clinical examination of the heart (Douglas et al, 2013).

During the cardiac cycle, there are two sounds associated with each heart beat and these are audible with a stethoscope (Fig 5). Both signal the closure of the cardiac valves: the first heart sound (S1) represents the closure of the mitral and tricuspid valves, and the second heart sound (S2) is due to the closure of the aortic and pulmonary valves (Fig 5). These two heart sounds

create what can be described as a 'lub-dup' sound heard between pauses.

In certain physiological states, auscultation may reveal extra heart sounds that may require further investigation. Listening to each of the valves of the heart can reveal useful information; for example, incompetence or narrowing of the valves will cause a 'whooshing' sound, referred to as a murmur.

Any failure of the pump function of the heart (for example, in heart failure) can lead fluid to back up into the lungs, in which case auscultation may reveal crackles. The legs should also be assessed for any signs of fluid accumulation (peripheral oedema) (Jarvis and Saman, 2017).

### Conclusion

Cardiac anatomy and physiology is complex. A useful resource on this topic is available online ([www.cvphysiology.com](http://www.cvphysiology.com)). The second article in this series, covering cardiac pathophysiology, will show how using a methodical system can facilitate a more comprehensive understanding of what goes wrong in cardiac diseases. **NT**

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### Cardiac system series

- Part 1: Anatomy and physiology
- Part 2: Pathophysiology

### Date

- Jan
- Feb