



Control of the Human Heart

This Factsheet explains, in detail, the way in which the human heart is controlled. Common misunderstandings shown by A - C grade students are summarised.

One complete heartbeat is equal to one complete **cardiac cycle**. A cardiac cycle consists of

- **diastole** - the whole heart relaxes and fills with blood,
- **atrial systole** - the atria contract to force their blood contents into the ventricles.
- **ventricular systole**, when the ventricles contract to force their blood contents into the pulmonary and aortic arches.
- The heart then returns to diastole, relaxing and filling with blood from the veins.
- Valves in the veins and heart prevent backflow of blood.

The control of the heart falls into two areas:

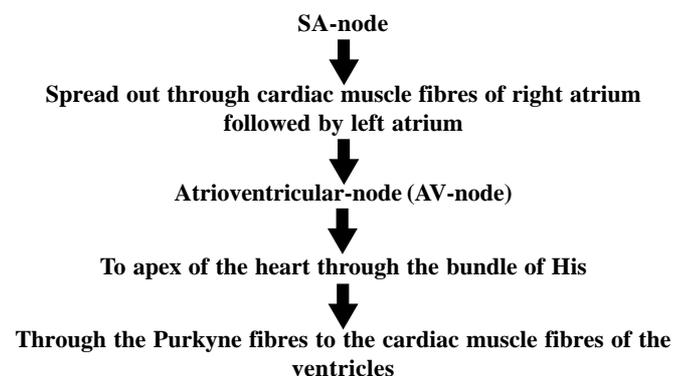
1. **Coordination of the cardiac cycle.** The heart itself does this, through its own internal conducting system.
2. **Control of the frequency and force of the heartbeat.** The autonomic nervous system does this, - both sympathetic and parasympathetic divisions are involved, in a balanced way.

The coordination of the cardiac cycle by the heart itself

- Cardiac muscle fibres are **myogenic**. This means they contract and relax rhythmically of their own accord, without intervention from the nervous system. They even beat rhythmically if kept outside the body in warm, isotonic, oxygenated saline.
- Cardiac muscles in the different areas of the heart beat at different inherent rates. Isolated atrial muscle will beat on average at 60 times per minute but isolated ventricular muscle only averages 20 beats per minute. Cardiac muscle of the sinus, a small area in the wall of the right atrium, has the quickest inherent rate, averaging 72 beats per minute.
- The internal conducting system of the heart enables the inherent rate of the sinus to be imposed on the atria and ventricles, so the sinus is called the **pacemaker** or **sino-atrial node**. It enforces an average rhythm of 72 beats per minute, **on the whole heart**.

The sino-atrial node (SA-node) generates electrical impulses, similar to nerve impulses, which pass through the conducting system and cardiac muscle fibres of the heart. An impulse consists of a wave of **depolarisation**, which causes cardiac muscle to contract, followed by a wave of **repolarisation**, which causes cardiac muscle to relax.

The pathway of impulses from the SA-node through the heart is:



In older textbooks, the Purkyne fibres are called Purkinje fibres. **Purkyne** is the spelling you should use according to current international guidelines on the use of biological terms.

In the heart wall round the junction of atria and ventricles, is a ring of tough fibrous tissue. It acts as a skeleton for attachment of the cardiac muscles of atria and ventricles. Contractions of the atrial and ventricular muscles must be towards the fibrous ring, so ensuring that the blood flow is from atria to ventricles and from the apex of the ventricles into the arches.

The fibrous tissue has high electrical resistance, preventing the passage of impulses from atria to ventricles except at the AV-node in the top of the ventricular septum.

Fig 1. Vertical section through the heart showing its conducting system

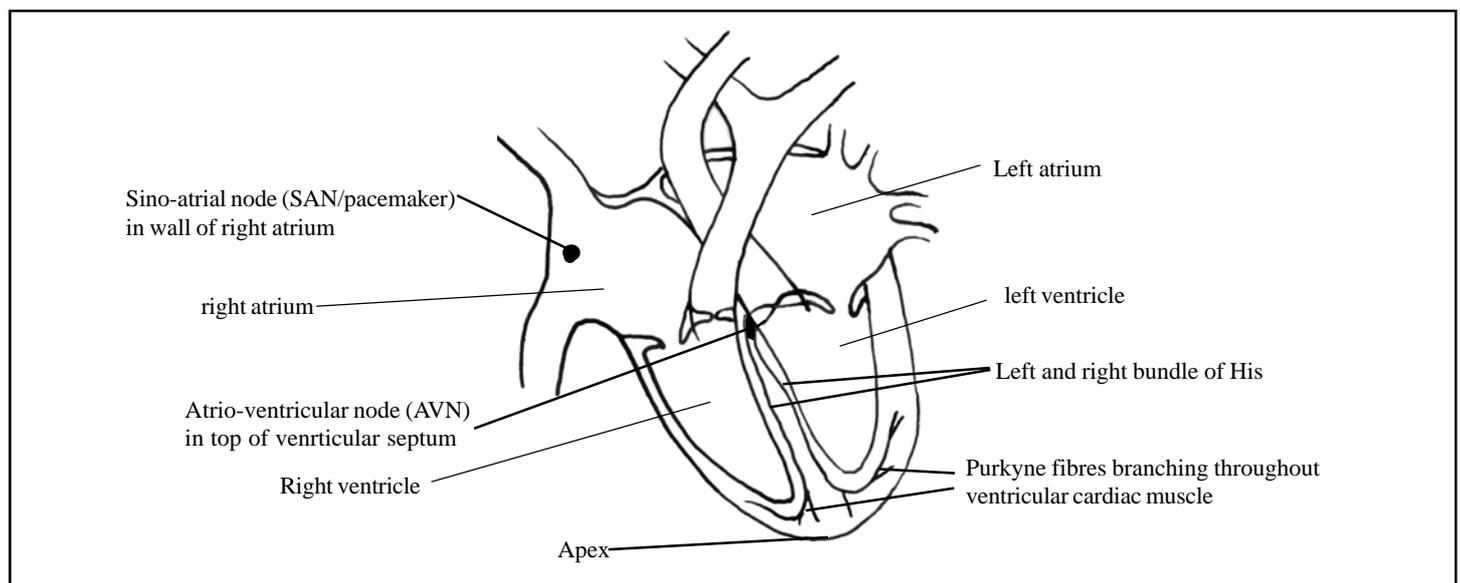
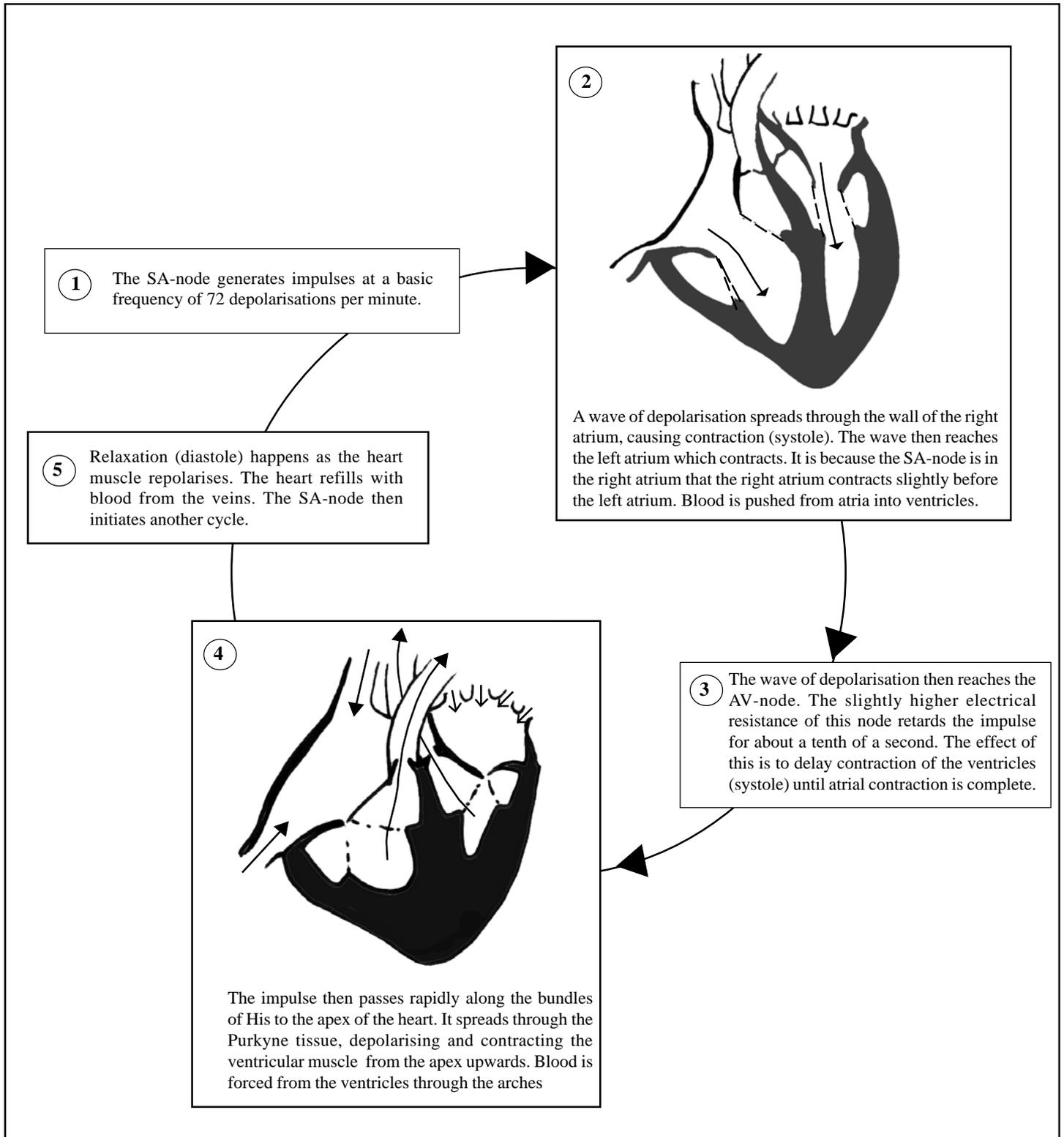


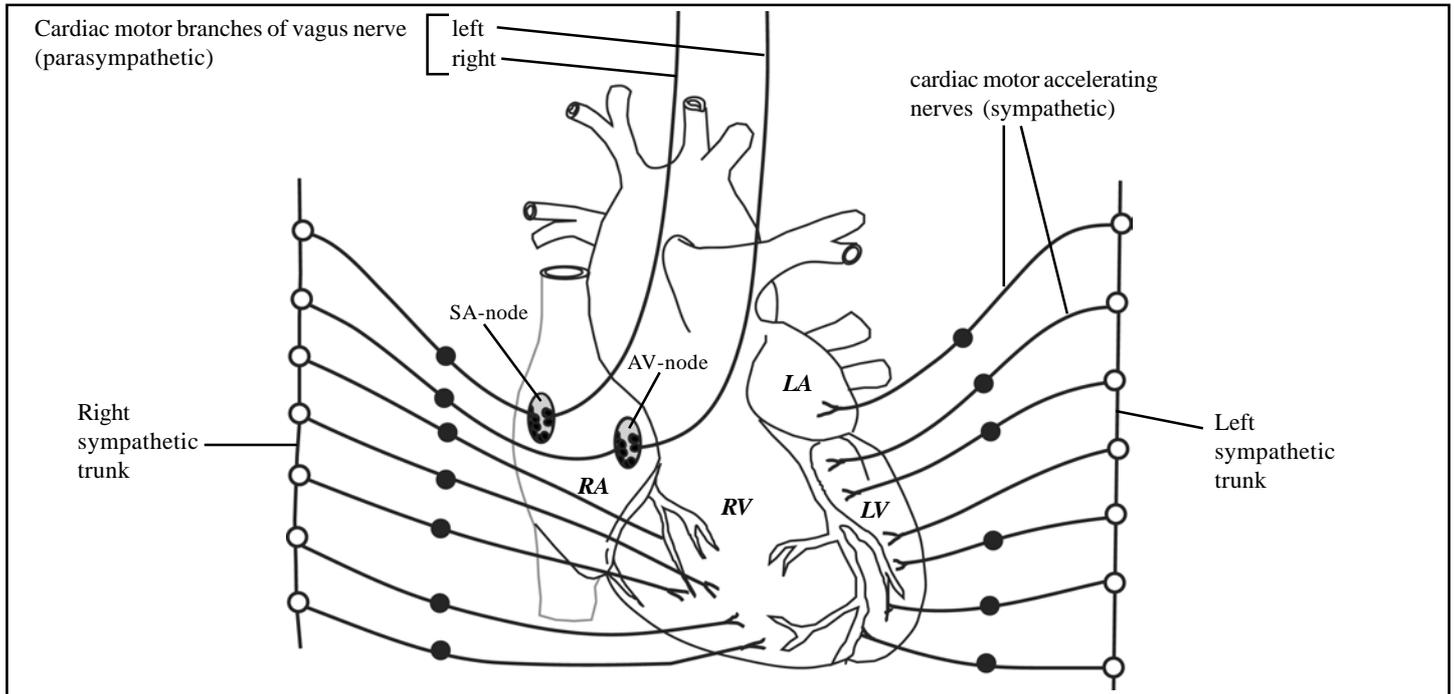
Fig 2. Sequence of events in control of the cardiac cycle



Exam Hint: Candidates often forget the skeleton of the heart and that it causes impulses to be directed via the AVN. They often forget the delay in impulse passage caused by the AVN – this is crucial to explaining why ventricular systole follows atrial systole.

Regulation of the heart by the autonomic nervous system

Although cardiac cycles (heart beats) are internally regulated by the heart, the frequency and force of beats are controlled by the autonomic nervous system.

Fig 3. Ventral view of heart showing the autonomic nerve supply

Note that:-

- Sympathetic nerves to the heart go directly to the SA-node, AV-node and ventricular cardiac muscle. Raised sympathetic stimulation increases the impulse frequency of the SAN and reduces the delay time at the AV-node. This accelerates the heart – increasing the **frequency** and **force** of the heart beat. Decreased stimulation has the reverse effects.
- Parasympathetic nerves to the heart (branches of the vagus nerve) go directly to the SA-node and AV-node. Raised parasympathetic stimulation decreases the impulse frequency of the SAN and increases the delay time at the AVN. This reduces the frequency and force of the heart beat. A decrease in parasympathetic stimulation increases the impulse frequency of the SAN and decreases the delay time at the AVN, thus complementing the effects of sympathetic stimulation.

Exam Hint: Candidates often forget to include the roles of the autonomic nervous system, or they confuse the functions of sympathetic and parasympathetic systems. Candidates rarely indicate that the heart rate is a result of balance between sympathetic and parasympathetic stimulation.

Try not to think of the heart as an isolated organ – it is an integral part of the body and its cardiac output must adjust to meet the needs of the body.

Cardiac output is the volume of blood pumped from the heart, measured in $\text{dm}^3 \text{min}^{-1}$.

If body activity increases, cardiac output must increase, to carry more food and oxygen to the active tissues. If body activity reduces, cardiac output can be reduced.

$$\text{Cardiac output} = \frac{\text{stroke volume of the heart}}{\text{the heart}} \times \frac{\text{number of beats per minute}}{\text{minute}}$$

The **stroke volume** of the heart is the volume of blood pumped out of the heart per beat. In a resting adult heart it is about 70 cm^3 per beat. A resting heart averages 72 beats per minute. The average resting cardiac output is therefore $70 \times 72 = 5040 \text{ cm}^3 \text{min}^{-1}$. ($5.04 \text{ dm}^3 \text{min}^{-1}$).

In **strenuous exercise**, the stroke volume may reach 115 cm^3 per beat with 120 beats per minute.

The cardiac output would be $115 \times 120 = 13.8 \text{ dm}^3 \text{min}^{-1}$.

The stroke volume increases as the force of cardiac muscle contraction increases. This is due to raised sympathetic stimulation coupled with reduced parasympathetic stimulation.

The stroke volume decreases as the force of cardiac muscle contraction decreases. This is due to raised parasympathetic stimulation coupled with reduced sympathetic stimulation.

Two **cardiac control centres** in the medulla of the brain operate to regulate the cardiac output in response to information they receive from receptors.

- **The cardioacceleratory centre (CAC)** increases cardiac output by stimulating the sympathetic nerves and inhibiting the parasympathetic nerves to the heart.
- **The cardioinhibitory centre (CIC)** reduces cardiac output by stimulating the parasympathetic nerves and inhibiting the sympathetic nerves to the heart.

Exam Hint: A common students' error is to omit reference to the CAC and CIC, but to refer to the **vasomotor centre** in the medulla as the controller of cardiac output. The **vasomotor centre regulates the diameter of blood vessels**, especially arterioles in the skin and abdominal viscera. The vasomotor centre does not directly control the cardiac output of the heart.

The synaptic transmitter substance between sympathetic motor neurones and the heart is **nor-adrenaline**. This is why the hormones **nor-adrenaline and adrenaline** act on the heart, mimicking sympathetic stimulation, to increase cardiac output. This happens mainly in times of stress or emergency. The synaptic transmitter of all parasympathetic synapses is **acetylcholine**. Acetylcholine causes a reduction of cardiac output.

The CAC and CIC respond according to impulses they receive from **baroreceptors**. Baroreceptors are pressure receptors and sense blood pressure at particular points in the circulatory system.

Stimulation or inhibition of cardiac control centres occurs by three reflex pathways:-

- **The carotid sinus reflex** is concerned with maintaining a suitable blood supply to the brain. The carotid sinus is a swelling at the base of the carotid artery to the brain. The carotid sinus wall contains baroreceptors. If blood pressure rises, the carotid sinus wall stretches stimulating the baroreceptors to send impulses to the CIC. This reduces cardiac output so blood pressure falls. When the sinus wall becomes less stretched, the baroreceptors are no longer stimulated, allowing the CAC to dominate. This increases cardiac output so blood pressure rises.
- The **aortic reflex** operates in the same way as the carotid reflex but is concerned with the general systemic blood pressure. It relies on baroreceptors in the aortic arch wall.
- The **Bainbridge reflex** regulates venous pressure. If this pressure rises, baroreceptors in the walls of the venae cavae and right atrium send impulses to the CAC, increasing cardiac output. This makes the heart draw blood from the venae cavae faster so the venous pressure falls.

Practice questions

- (a) Beta-blockers are drugs which inhibit the sympathetic nerves to the heart. Explain why they are used to treat sufferers from high blood pressure. **3**
 - (b) Calculate the stroke volume of the heart when the heart rate is 120 beats per minute and the cardiac output is $15.6 \text{ dm}^3 \text{ min}^{-1}$. Show your working. **2**
 - (c) Explain what happens to the following when cardiac output increases?
 - (i) The venous return to the heart. **2**
 - (ii) The arterial blood pressure. **2**
 - (iii) The venous blood pressure. **2**

Total 11 marks
- (a) Outline how the conducting system of the heart co-ordinates the cardiac cycle. **8**
 - (b) Wolff (Wolfe)-Parkinson-White Syndrome is a rare heart defect with an incidence of 1.5 per 1000 people. Sufferers have an extra conducting pathway between the atria and the ventricles, due to a gap in the fibrous skeletal ring separating atria from the ventricles. The condition causes palpitations due to premature atrial contractions or to premature ventricular contractions which initiate from the top of the ventricles (not the apex). The condition is rarely fatal but may cause light-headedness and blackouts. Suggest explanations for the premature atrial and ventricular contractions. **3**

Total 11 marks
- (a)
 - (i) What is a baroreceptor? **1**
 - (ii) Where are baroreceptors that help to regulate cardiac output situated? **3**
 - (b)
 - (i) What are the cardiac control centres in the medulla? **2**
 - (ii) How do the cardiac control centres operate? **4**

Total 10 marks

Answers

- (a) sympathetic stimulation of the heart increases cardiac output; increased cardiac output contributes to increased blood pressure; beta-blockers reduce sympathetic stimulation, so reducing cardiac output so reducing blood pressure; **3**
 - (b) $15.6 = \text{stroke volume} \times 120$;
stroke volume = $15.6 \div 120 = 0.13 \text{ dm}^3 \text{ per beat} / 130 \text{ cm}^3 \text{ per beat}$ **2**
 - (c)
 - (i) venous return would increase;
because venous return must equal cardiac output (to maintain efficient blood flow); **2**
 - (ii) arterial blood pressure would increase;
because the arteries are receiving a greater volume of blood from the heart at each beat; **2**
 - (iii) venous blood pressure would fall;
because the heart is drawing more blood from the veins at each beat; **2**
(remember: capillaries reduce the arterial blood pressure to the venous blood pressure).
- (a) SA-node generates impulses imposing a rate on the complete heart; wave of depolarization causes contraction, repolarisation causes relaxation;
wave of depolarization spreads out over right atrium which contracts;
followed by left atrium which contracts;
wave can only pass to ventricles via AV-node due to high electrical resistance of fibrous tissue (between atria and ventricles);
AV-node imposes a delay on impulse passage delaying ventricular contraction;
wave passes rapidly through the bundles of His to the apex of the ventricles;
spreads out through Purkyne tissue to ventricular cardiac muscle, causing contraction; **8**
 - (b) the normal wave of depolarization through the atrial walls will reach both the AV-node and the extra conducting pathway;
the AV-node causes a delay but the abnormal pathway will immediately pass the wave of depolarization to the top of a ventricle, initiating contraction;
as normal waves of depolarization pass up the ventricles from the apex some pass through the abnormal pathway to an atrium, initiating contraction; **3**
- (a)
 - (i) a receptor which monitors (blood) pressure **1**
 - (ii) in the (wall of the) carotid sinus;
in the (wall of the) aorta;
in the (walls of the) venae cavae; **3**
 - (b)
 - (i) cardioacceleratory centre/CAC; **2**
cardioinhibitory centre/CIC;
 - (ii) CAC: increases cardiac output by increasing sympathetic stimulation to the heart;
and inhibiting parasympathetic stimulation to the heart;
CIC: decreases cardiac output by inhibiting sympathetic stimulation to the heart;
and increasing parasympathetic stimulation to the heart; **4**

Acknowledgements:

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