The walls of the ventricles are much thicker than the walls of the atria, because the ventricles need to develop much more force when they contract. Their contraction has to push the blood out of the heart and around the body. For the right ventricle, the force required is relatively small, because the blood goes only to the lungs, which are very close to the heart. The left ventricle, however, has to develop sufficient force to push blood around all the rest of the body. Therefore, the thickness of the muscular wall of the left ventricle is much greater than that of the right. Figure **9.8** shows the pressure changes in the left side of the heart and the aorta during two consecutive cardiac cycles. You can see that the pressure developed in the left ventricle is much greater than that in the left atrium.

SAQ 9.1

From Figure **9.8**, identify the time at which each stage shown in Figure **9.6** (page **166**) is occurring.

Control of the heart beat

Cardiac muscle differs from the muscle in all other areas of the body in that it is **myogenic**. This means that it **naturally** contracts and relaxes; it does not need to receive impulses from a nerve to make it contract. If cardiac muscle cells are cultured in a warm, oxygenated solution containing nutrients, they contract and relax rhythmically, all by themselves.

However, the individual heart muscle cells cannot be allowed to contract at their own natural rhythms. If they did, parts of the heart would contract out of sequence with other parts. The cardiac cycle would become disordered and the heart would stop working as a pump. The heart has its own built-in controlling and coordinating system which prevents this happening.

The cardiac cycle is initiated in a specialised patch of muscle in the wall of the right atrium, called the **sino-atrial node**. It is often called the **SAN** for short, or **pacemaker**. The muscle cells of the SAN set the rhythm for all the other cardiac muscle cells. Their natural rhythm of contraction is slightly faster than that of the rest of the heart muscle. Each time the muscles of the SAN contract, they set up a wave of electrical activity which spreads out rapidly over the walls of the atrial walls. The cardiac muscle in the atrial walls responds to this excitation wave by contracting, at the same rhythm as the SAN. Thus, all the muscle in both atria contracts almost simultaneously.

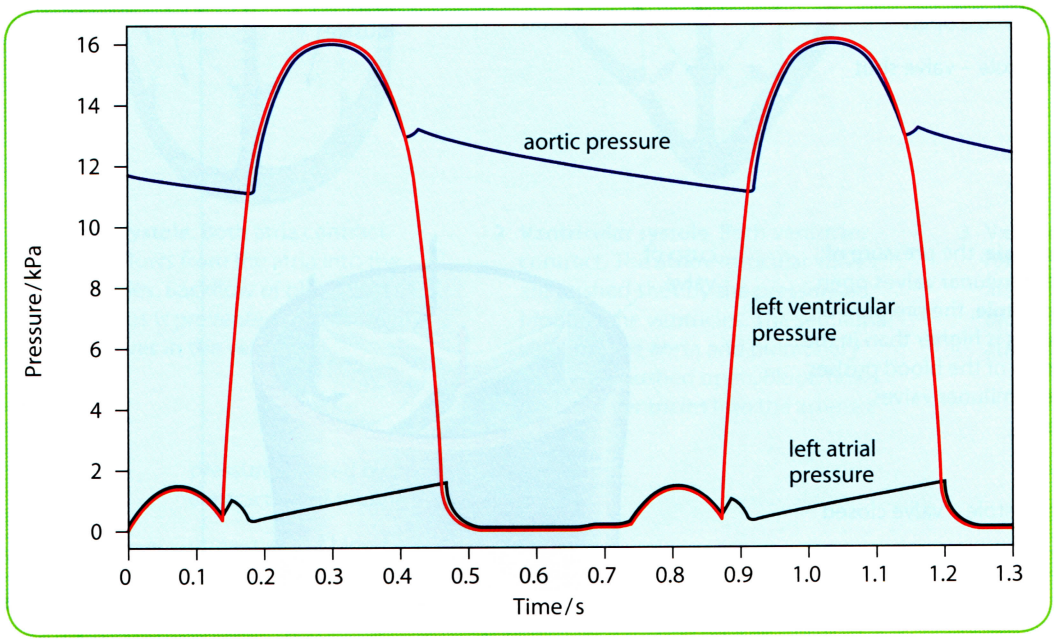

Figure 9.8 Pressure changes in the left side of the heart during the cardiac cycle.

As we have seen, the muscle of the ventricles do not contract until **after** the muscles of the atria. (You can imagine what would happen if they contracted at once.) This delay is caused by a feature of the heart that briefly delays the excitation wave in its passage from the atria to the ventricles. There is a band of fibres between the atria and ventricles which does not conduct the excitation wave. Thus, as the wave spreads out from the SAN over the atrial walls, it cannot pass into the ventricle walls. The only route through is a patch of conducting fibres situated in the septum, the

the **atrioventricular node**, or AVN (Figure 9.9). The AVN picks up the excitation wave as it spreads across the atria and, after a delay of about 0.1 seconds, passes it on to a bunch of conducting fibres called the **Purkyne tissue**, which runs down the septum between the ventricles. This transmits the excitation wave very rapidly down to the base of the septum, from where it spreads outwards and upwards through the ventricle walls. As it does so, it causes the cardiac muscle in these walls to contract, from the bottom up, so squeezing blood upwards and into the arteries.

In a healthy heart, therefore, the atria contract and then the ventricles contract from the bottom upwards. Sometimes, this coordination of contraction goes wrong. The excitation wave becomes chaotic, passing through the ventricular muscle in all directions, feeding back on itself and re-stimulating areas it has just left. Small sections of the cardiac muscle contract while other sections are relaxing. The result is **fibrillation**, in which the heart wall simply flutters rather than contracting as a whole and then relaxing as a whole. Fibrillation is almost always fatal, unless treated instantly. Fibrillation may be started by an electric shock or by damage to large areas of muscle in the walls of the heart.

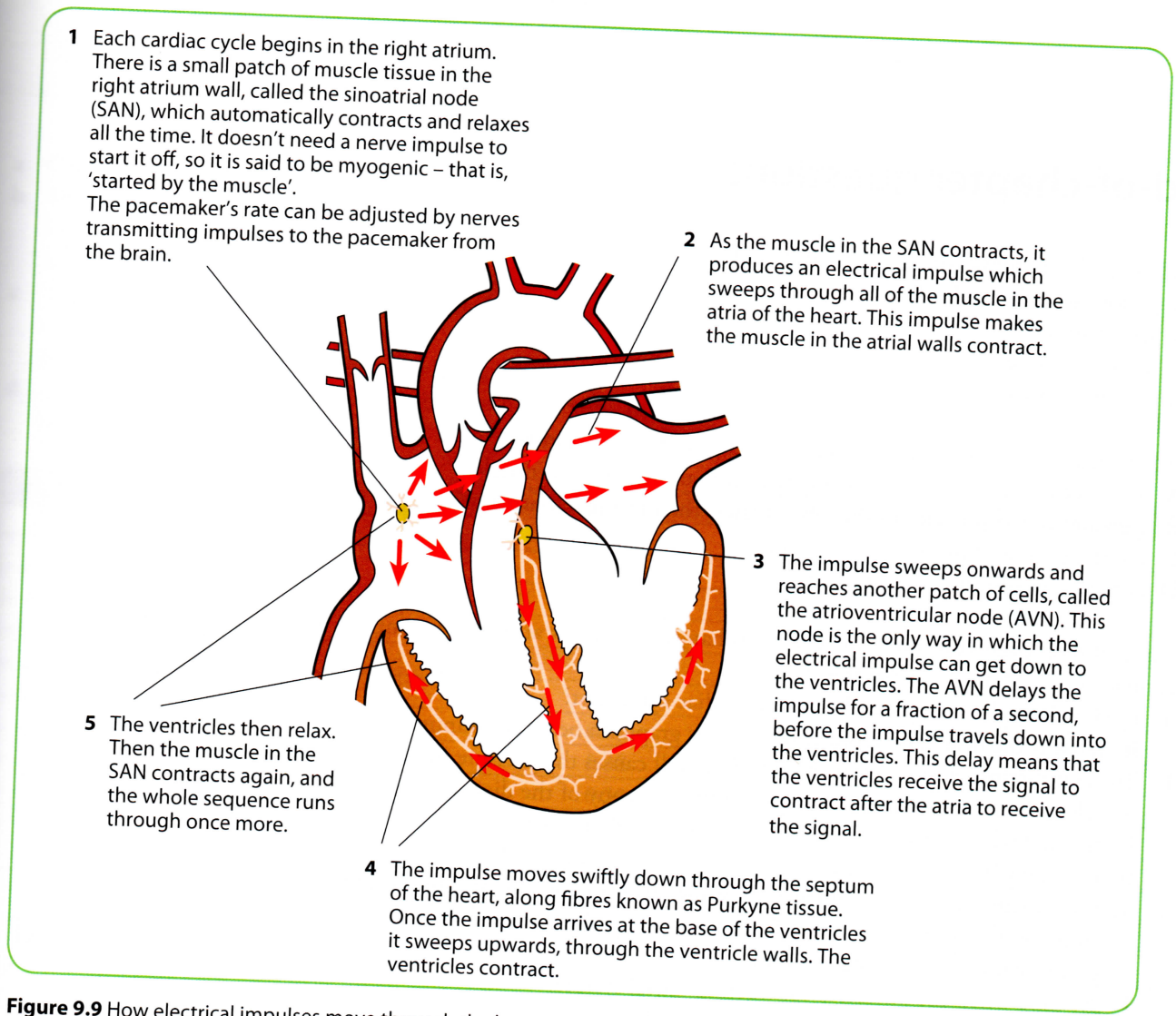

Figure 9.9 How electrical impulses move through the heart.

Electrocardiograms (ECGs)

It is relatively easy to detect and record the waves of excitation flowing through heart muscle. Electrodes can be placed on the skin over opposite sides of the heart, and the electrical potentials generated recorded with time. The result, which is essentially a graph of voltage against time, is an **electrocardiogram** (ECG) (Figure 9.10).

The part labelled P represents the wave of excitation sweeping over the atrial walls. The parts labelled Q, R and S represent the wave of excitation in the ventricle walls. The T section indicates the recovery of the ventricle walls.

Figure 9.10 A normal ECG.

End-of-chapter questions

1 Where is the mammalian heart beat initiated?
- **A** atrioventricular node
- **B** left atrium
- **C** Purkyne tissue
- **D** sinoatrial node

2 What causes the bicuspid valve to close during ventricular systole?
- **A** a greater blood pressure in the left atrium than in the left ventricle
- **B** a greater blood pressure in the left ventricle than in the left atrium
- **C** contraction of muscles in the septum
- **D** contraction of muscles in the valve

3 Figure 9.8 on page 168 shows the pressure changes in the left atrium, left ventricle and aorta throughout two cardiac cycles. Make a copy of this diagram.
- **a** **i** How long does one heart beat (one cardiac cycle) last?
  ii What is the heart rate represented on this graph, in beats per minute?
- **b** The contraction of muscles in the ventricle wall causes the pressure inside the ventricle to rise. When the muscles relax, the pressure drops again. On your copy of the diagram, mark the following periods:
  i the time when the ventricle is contracting (ventricular systole)
  ii the time when the ventricle is relaxing (ventricular diastole).
- **c** The contraction of muscles in the wall of the atrium raises the pressure inside it. This pressure is also raised when blood flows into the atrium from the veins, while the atrial walls are relaxed. On your copy of the diagram, mark the following periods:
  i the time when the atrium is contracting (atrial systole)
  ii the time when the atrium is relaxing (atrial diastole).