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Control of heart rate

Hormonal and nervous mechanisms involved in controlling human heart rate

Factors affecting heart rate

The heart pumps blood all around the body in the double-circulatory system. The blood supplies tissues with oxygen, glucose, fatty acids, amino acids and other useful products, and also removes waste products such as urea and carbon dioxide to prevent accumulation of such products, which would lead to inhibition of cell metabolism.

These requirements of the cells vary according to level of activity, and there are a number of factors which can affect this, so it is important that the heart can adapt to meet the requirements of the body at any one given time. Factors affecting heart rate include breathing rate, signals from the brain and hormones (e.g. adrenaline).

Regulation of heart rate

Heart muscle is **myogenic**, so initiates its own contractions, and involuntary. There is an area of tissue, called the **sinoatrial node (SAN)**, which can initiate an action potential to travel along the atrial walls as a wave of excitation, causing them to contract. This is where one heartbeat originates. The signal then spreads through the **atrioventricular node (AVN)** and down the **Purkyne tissue** to the ventricular apex, and finally through the ventricles, causing them to contract.

The heart has an underlying frequency of 60-80bpm (beats per minute). Whilst heart rate cannot be controlled wholly, the brain can send signals to tweak this frequency. These signals are sent to the SAN through nerves from the brain to the heart. It is the **medulla oblongata** in the brain which sends these signals to the SAN. There are two separate controls from the medulla oblongata:

- 1 the **accelerator nerve** causes the rate of signals from the SAN to increase, resulting in an increased heart rate
- 2 the **vagus nerve** reduces the rate of contractions, so resulting in a reduced heart rate

The medulla oblongata receives various signals itself, so that it can communicate with the SAN how to respond appropriately to external changes. The types of signal it can receive are:

- movement of limbs is detected by the stretch receptors (**propioceptors**), which will send signals to the *cardiovascular centre* in the medulla oblongata to inform it that more oxygen may soon be needed, so heart rate is increased to provide that extra oxygen
- **chemoreceptors** in the carotid arteries, aorta and brain detect the changes in pH produced when excess carbon dioxide (due to, for example, exercise) reacts with water in the blood plasma, lowering the pH, and so the chemoreceptors send signals to the cardiovascular centre to increase heart rate
- when exercising stops, the concentration of carbon dioxide in the blood falls, which reduces the activity of the accelerator nerve pathway, so the heart rate decreases, preventing further carbon dioxide from being removed

If the SAN stops working, the heart cannot contract, and so death is inevitable. This is why an artificial **pacemaker** would be fitted to a patient. The SAN is the natural pacemaker, but an artificial pacemaker is capable of sending signals to the heart to keep it contracting and to keep blood flowing around the body. They are around 4cm long and will be inserted under the skin and fat on the chest, sometimes inserted in the chest cavity.

Similarly, if the AVN stops working, the signal from the SAN cannot be passed onto the ventricular walls, and so those contractions stop. So some pacemakers are fitted to act upon the AVN, in order to resume normal contractions, as in these cases, the SAN will be functioning normally.