

The Heart

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The heart is situated between the two lungs and behind the sternum in the thorax.

It is surrounded by a sac, the **pericardium**, composed of an outer layer of white fibrous tissue and two inner membranes; the space between the membranes is filled with pericardial fluid.

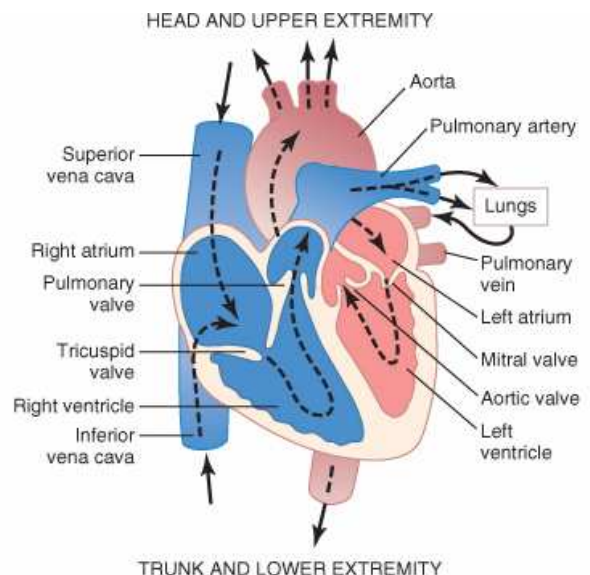
There are four chambers in the heart, the two upper thin walled **atria (auricles)** and the two lower thick walled **ventricles**.

The right side of the heart is completely separated from the left. The right side deals with deoxygenated blood – the left side with oxygenated blood.

As the atria contract, they force blood into the ventricles.

As the ventricles contract, the **auriculoventricular valves** (bicuspid or mitral on the left, tricuspid on the right) snap shut while the **semilunar valves** open, allowing blood to be pumped into the pulmonary artery and the dorsal aorta.

The auriculoventricular valves are attached by fibrous cords, the chordae tendineae which in turn attach to papillary muscles. When the ventricles contract, the papillary muscles contract, tightening the cords and preventing the valves being turned inside out.



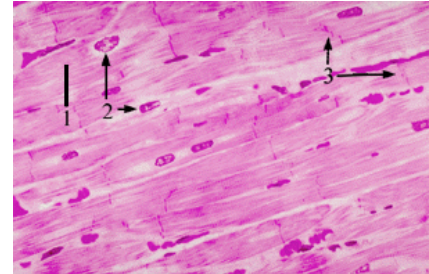
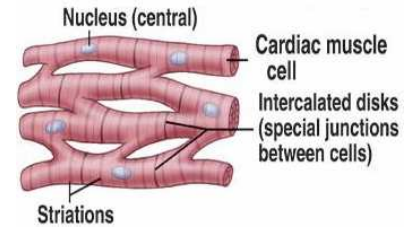
Cardiac Muscle

As the name implies, cardiac muscle is the muscle that makes up the wall of the heart. It consists of involuntary, striated, branched fibres attached to the walls of the heart.

Cardiac muscle is unique in that it shows some features of skeletal muscle and some features of smooth muscle. Cardiac muscle is similar to skeletal muscle in that it is striated and multinucleate, and similar to smooth muscle in that the nuclei are centrally located and many cells are required to span the length of the muscle.

It differs from both skeletal muscle and smooth muscle in that its cells branch and are joined to one another via intercalated discs.

Cardiac muscle also differs from the other two muscle types in that contraction can occur even without an initial nervous input. The cells that produce the stimulation for contraction without nervous input are called the pacemaker cells.



Cardiac Muscle

1. Cardiac Muscle Cell
2. Nuclei
3. Intercalated Discs

The Cardiac Cycle

The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called the cardiac cycle. Each cycle is initiated by spontaneous generation of an action potential in the S-A node. It involves repeated contraction and relaxation of heart muscle – contraction is termed systole and relaxation diastole.

Atrial diastole: The atria are relaxed and blood normally flows from the great veins into the atria. As blood continues to fill the atria, the pressure in the atria rises above that of the ventricles and the auriculoventricular valves open. Almost 80 % of the blood flows directly through the atria into the ventricles even before the atria contract.

Atrial systole: The two atria contract simultaneously forcing the remaining blood into the ventricles.

Ventricular systole: Immediately after ventricular contraction begins, the ventricular pressure rises abruptly, causing the auriculoventricular valves to close. The closing of the auriculoventricular valves gives rise to the first heart sound described as “lub”.

The ventricle then builds up sufficient pressure over 0.02 to 0.03 second to push the semilunar (aortic and pulmonary) valves open. During this period, contraction is occurring in the ventricles, but there is no emptying.

This is called the period of **isovolumic or isometric contraction**, meaning that tension is increasing in the muscle but little or no shortening of the muscle fibres is occurring.

This is followed by a period of ejection as the ventricular pressures push the semilunar valves open. Blood pours out of the ventricles into the aorta and the pulmonary artery.

Ventricular diastole: At the end of systole, ventricular relaxation begins suddenly, allowing both the right and left intraventricular pressures to decrease rapidly. The elevated pressures in the distended large arteries that have just been filled with blood pushes blood back toward the ventricles, which snaps the aortic and pulmonary valves closed.

The closing of the valves gives rise to the second heart sound described as “dub”.

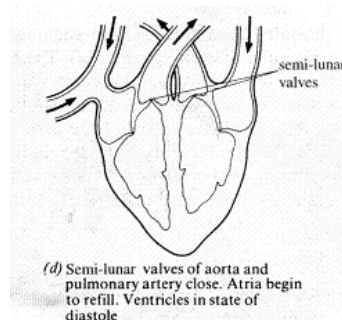
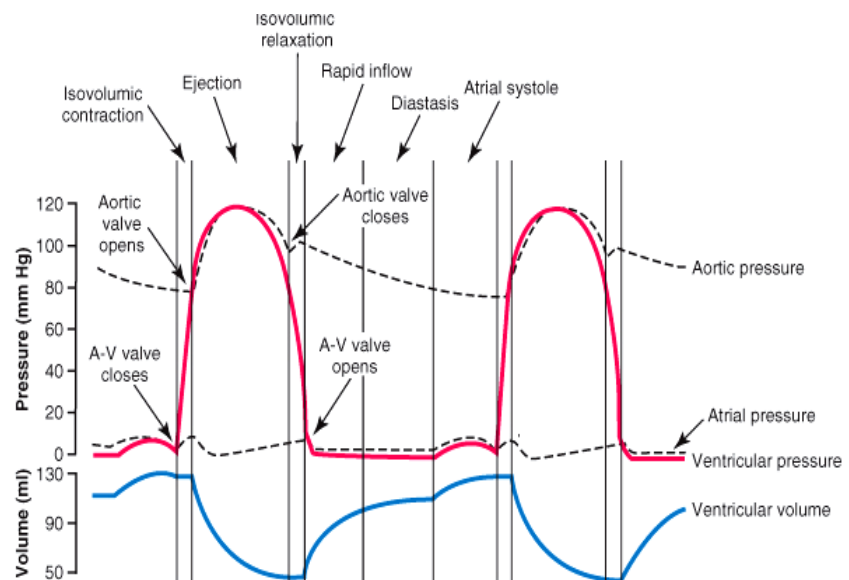
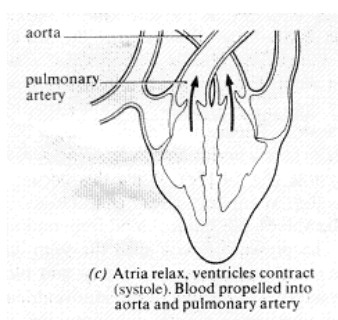
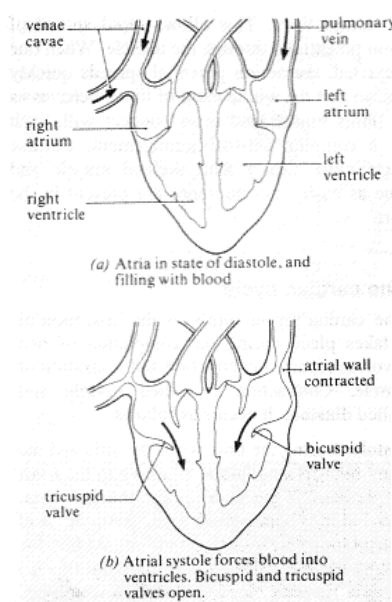
For another 0.03 - 0.06 second, the ventricular muscle continues to relax, even though the ventricular volume does not change, giving rise to the period of **isovolumic or isometric relaxation**. During this period, the intraventricular pressures decrease rapidly back to their low diastolic levels.

During diastole, normal filling of the ventricles increases the volume of each ventricle to about 110 to 120 ml. This volume is called the end-diastolic volume.

Then, as the ventricles empty during systole, the volume decreases by about 70 ml, which is called the **stroke volume** output.

The remaining volume in each ventricle, about 40 to 50 ml, is called the end-systolic volume.

One complete heartbeat is composed of one systole and one diastole and lasts about 0.8 seconds.



Events of the cardiac cycle

The pressure curves in the right ventricle and pulmonary artery are similar to those in the aorta, except that the pressures are only about one sixth as great.

When a person is at rest, the heart pumps around 5 l of blood each minute.

During severe exercise, the heart may be required to pump four to seven times this amount.

The basic means by which the volume pumped by the heart is regulated are

(1) intrinsic cardiac regulation of pumping in response to changes in volume of blood flowing into the heart

(2) control of heart rate and strength of heart pumping by the autonomic nervous system.

The Frank-Starling mechanism of the heart

Within physiologic limits, the greater the initial length of the cardiac muscle, the greater is the force of contraction.

As a result, a greater quantity of blood is pumped into the aorta.

Myogenic Stimulation of Heart rate

The heart of humans is myogenic (*myo*: muscle; *genic*: giving rise to) – innervation only modulates heart rate.

Some cardiac fibres have the capability of self-excitation, a process that can cause automatic rhythmical discharge and contraction. This is especially true of the fibres of the heart's specialized conducting system, including the fibres of the sinus node.

The specialized excitatory and conductive system of the heart that controls cardiac contractions include

the **sinoatrial (S-A node)** in which the normal rhythmical impulse is generated. The SA node is located in the posterolateral wall of the right atrium immediately below the opening of the superior vena cava.

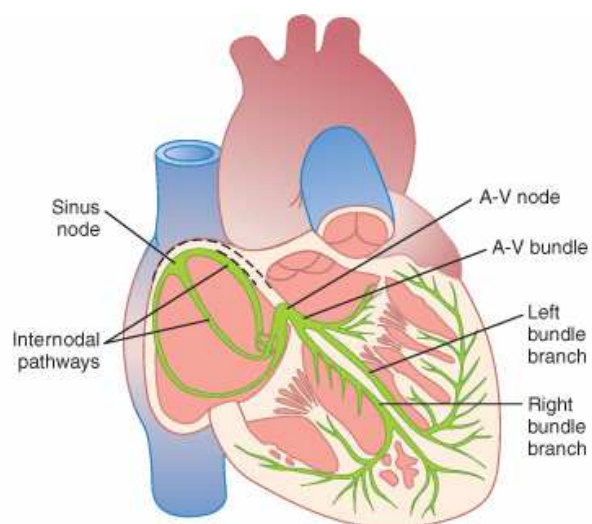
the **internodal pathways** that conduct the impulse from the S-A node to the A-V node

the **atrioventricular (A-V) node** located in the posterior wall of the right atrium immediately behind the tricuspid valve

the **A-V bundle (common bundle branch, bundle of His)** which conducts the impulse from the atria into the ventricles

the **left and right bundle branches of Purkinje fibres**, which conduct the cardiac impulse to all parts of the ventricles.

The conducting system is necessary as the fibrous tissues which make up the septa, particularly the auriculo-ventricular septum will not allow conduction by cell to cell coupling.



Mechanism of Sinus Nodal Rhythmicity

The "resting membrane potential" of the SA nodal fibre between discharges has a negativity of about -55 to -60 mV, in comparison with -85 to -90 mV for the ventricular muscle fibre.

The cause of this lesser negativity is that the cell membranes of the SA nodal fibres are naturally leaky to Na^+ and Ca^{2+} ions. Positive charges of the entering Na^+ and Ca^{2+} ions neutralize much of the intracellular negativity.

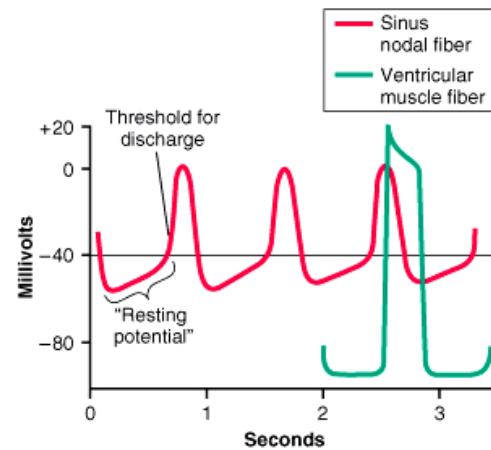
Cardiac muscle has three types of membrane ion channels that play important roles in causing the voltage changes of the action potential.

They are (1) fast sodium channels (2) slow sodium-calcium channels and (3) potassium channels.

The natural leakiness of the SA node membrane to Na^+ and Ca^{2+} allows Na^+ and Ca^{2+} to enter and gradually raise the "resting" potential to its threshold value of -40 mV.

When the potential reaches a threshold voltage of about -40 mV, the Na^+ - Ca^{2+} channels become "activated," causing the action potential.

The K^+ channel then opens and an efflux of K^+ occurs causing the potential to drop back to -ve.



Action potentials recorded from inside a sinus nodal fiber for three heartbeats and, by comparison, a single ventricular muscle fiber action potential.

The fast Na^+ channels open only if the potential drops below -60 mV (ideally -85 to -90 mV) which is the resting potential for ventricular muscle.

Opening of the fast Na^+ channels is responsible for the rapid upstroke spike of the action potential observed in ventricular muscle.

The "plateau" of the ventricular action potential is caused primarily by slower opening of the slow sodium-calcium channels. Finally, opening of K^+ channels returns the membrane potential to its resting level, a state known as hyperpolarization.

The hyperpolarization state carries the "resting" membrane potential down to about -55 to -60 mV at the termination of the action potential.

Then, more and more K^+ channels close progressively, the inward-leaking Na^+ and Ca^+ ions once again overbalance the outward flux of K^+ , and this causes the "resting" potential to drift upward once more, finally reaching the threshold level for discharge at a potential of about -40 mV.

The Rate of Rhythmicity

SA node: 70 – 80/ min
AV node: 40 – 60/ min
Ventricle: 20 – 40/min

Velocity of impulse conduction

Atrium: 0.3 m/sec
AV node: 0.02 m/sec
Purkinje system: 1.5 – 4.0 m/sec

The S-A node rather than the A-V node or the Purkinje fibres controls the heart's rhythmicity as the discharge rate of the sinus node is considerably faster than the natural self-excitatory discharge rate of either the A-V node or the Purkinje fibres.

Each time the S-A node discharges, its impulse is conducted into both the A-V node and the Purkinje fibres, also discharging their excitable membranes. But the S-A node discharges again before either the A-V node or the Purkinje fibres can reach their own thresholds for self-excitation.

Therefore, the new impulse from the sinus node discharges both the A-V node and the Purkinje fibres before self-excitation can occur in either of these.

The normal route of conduction is a one way route from the S-A node through internodal pathways to the A-V node then through A-V bundle, left and right bundle branches of Purkinje fibres and finally throughout the ventricles before it dies out. Meanwhile another impulse originates at the S-A node.

The impulse, after traveling through the internodal pathways, reaches the A-V node about 0.03 second after its origin in the sinus node.

This is followed by a total delay of about 0.13 second in the A-V node and A-V bundle system. Thus, there is a total delay of 0.16 second before the excitatory signal finally reaches the contracting muscle of the ventricles.

The velocity of impulse is slower at the A-V node to prevent auricles and ventricles contracting simultaneously.

A special characteristic of the A-V bundle is the inability, except in abnormal states, of action potentials to travel backward from the ventricles to the atria. This prevents re-entry of cardiac impulses by this route from the ventricles to the atria, allowing only forward conduction from the atria to the ventricles.

Also, except at the A-V bundle, the atrial muscle is separated from the ventricular muscle by a continuous fibrous barrier, the auriculo-ventricular septum which acts as an insulator to prevent passage of the cardiac impulse through any other route besides forward conduction through A-V bundle itself.

The Purkinje system transmits action potentials at a velocity of 1.5 to 4.0 m/sec allowing almost instantaneous transmission of the cardiac impulse throughout the entire remainder of the ventricular muscle.

Once the impulse reaches the ends of the Purkinje fibres, it is transmitted through the ventricular muscle mass by the ventricular muscle fibres themselves.

An important property of cardiac muscle is the presence of a **refractory period**.

If an impulse is applied during the contraction (from the onset of depolarization to approaching the threshold value) irrespective of the strength of the stimulus, no further contraction will occur. This is termed the absolute refractory period.

The normal refractory period of the ventricle is 0.25 to 0.30 second, which is about the duration of the action potential.

There is an additional relative refractory period of about 0.05 second between the periods from the threshold value to approaching the resting potential during which the muscle is more difficult than normal to excite but nevertheless can be excited by a very strong excitatory signal.

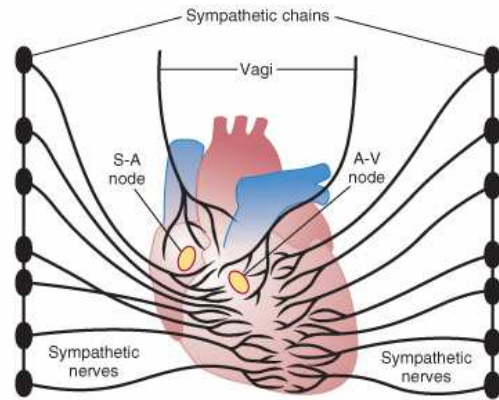
Control of Heart Rhythmicity

a) by Sympathetic and Parasympathetic Nerves

The heart is supplied with both sympathetic and parasympathetic nerves.

The parasympathetic nerves (the vagi) are distributed mainly to the S-A and A-V nodes, to a lesser extent to the muscle of the atria, and very little directly to the ventricular muscle.

The sympathetic nerves are distributed to all parts of the heart, including ventricular muscle.



Stimulation of the parasympathetic nerves to the heart causes acetylcholine to be released at the vagal endings. This decreases the rate of rhythm of the S-A node, and transmission of impulse to A-V node, slowing transmission of cardiac impulse into ventricles.

The acetylcholine increases the permeability of the fibre membranes to K^+ ions, allowing rapid leakage of K^+ out of the fibres and causing increased negativity inside the fibres.

Sympathetic stimulation causes the opposite effects: it increases the rate of sinus nodal discharge, the rate of conduction and the level of excitability in all parts of the heart. It also increases the force of contraction of atrial and ventricular cardiac musculature.

Stimulation of the sympathetic nerves releases norepinephrine at the sympathetic nerve endings. The precise mechanism by which this hormone acts is unclear, but it probably increases permeability of the fibre membrane to Na^+ and Ca^{2+} ions, causing a more positive resting potential and an increased rate of upward drift toward the threshold level for self-excitation.

b) by Hormones

Adrenaline and noradrenaline (epinephrine and norepinephrine) secreted by the medulla of the adrenal glands increase the heart rate, thus increasing cardiac output and blood pressure.

Thyroxine also leads to an increase in cardiac output.

Low pH and high temperature also increase the heart rate.

Tachycardia (*tachys*: swift; *cardia*: heart) is a condition describing increased heart rate. It may be caused by a variety of factors which increase sympathetic activity or an increase in thyroxine. Severe tachycardia may involve changes in the electrical activity of the heart.

Bradycardia (*bradys*: slow; *cardia*: heart) is a condition describing a decrease in heart rate below the normal rhythm. Long term training (eg. by athletes) increases stroke volume; to maintain a constant cardiac output, the resting heart rate is reduced.

Underactivity of the thyroid gland or changes in the electrical activity of S-A node can also cause bradycardia.