

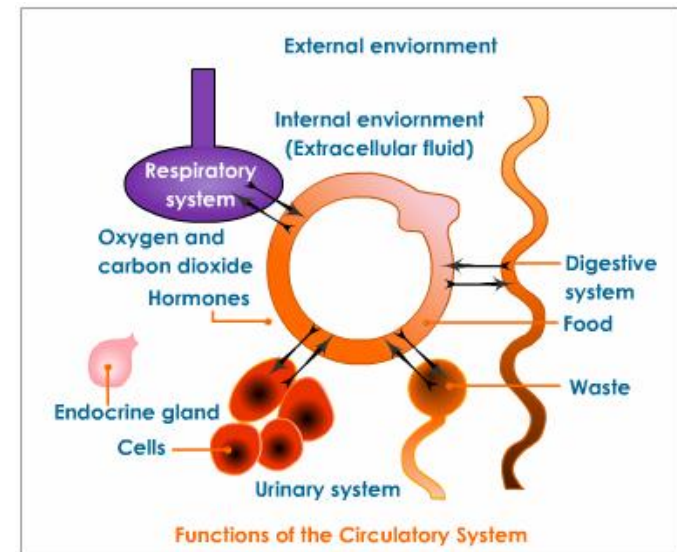
CARDIOVASCULAR PHYSIOLOGY



Heart pumps 7200 lt of blood a day!

- CVS is composed of heart, blood vessels and blood.
- The heart pumps blood through a closed system of vessels.
- Arteries take blood away from the heart and the veins carry blood back to the heart.

Primary function of CVS is transport of materials.



The heart is divided by a central wall, **septum**, into left and right halves.

Each half consists of atrium and ventricle.

Atrium receives blood returning to the heart;
ventricle pumps blood out into the blood vessels.

Right side receives blood from the tissue and sends it to the lungs for oxygenation.

Left side receives newly oxygenated blood from lungs and pumps it to tissues.

Blood in the right side is colored blue indicating deoxygenated blood which contains less oxygen.

Blood pumped out of the left ventricle enters a large artery known as **aorta**.

Aorta branches into a series of smaller arteries finally lead into network of capillaries.

From small veins into larger veins. The veins from upper part join to form superior vena cava.

The veins from lower part join to form inferior vena cava.

Coronary arteries nourish the heart muscle itself. From these arteries, to capillaries, then into coronary veins which empty directly at the right sinus.

Why blood flows?

- So that oxygen and nutrients can get to all parts of the body.
- Liquids and gases flow down pressure gradients from regions of higher pressure to regions of lower pressure.

Pressure created by the contraction of ventricles is called driving pressure

If blood vessels dilate, BP falls;
if contracts, BP increases.

As blood flows through the vessel, it loses energy. Resistance is the tendency to oppose blood flow.

A small change in diameter has a large effect.

Blood flow $\propto \Delta P/R$

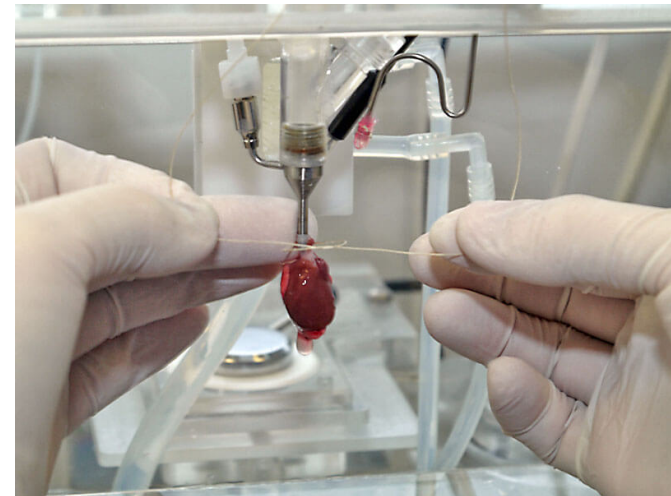
The heart valves ensure one-way flow

During ventricular contraction, AV valves remain closed to prevent blood flow backward into the atria.

Semilunar valves prevent blood that has entered the arteries from flowing back into the ventricles during ventricular relaxation.

Heart is composed of cardiac muscle or myocardium.

Most of cardiac cells are excitable, but about %1 of them can generate AP without any outside signal.



Signal comes from specialized myocardial cells called **pacemaker cell**.
They set the rate of heartbeat.

Cardiac Muscle

- Small, have a single nucleus
- Cardiac muscle cells branch
- Have cell junctions called **intercalated disks**; composed of desmosomes and gap junctions
- Larger t-tubules
- Smaller sarcoplasmic reticulum
- Many mitochondria
- Rhythmic contractions
- Does not fatigue as easily as skeletal
- Require high O₂

Only way to get more O₂ is via coronary arteries.

Reduced blood flow from narrowing of a coronary vessel by a clot or fatty deposit can damage or even kill myocardial cells.

Excitation-contraction (EC) coupling in cardiac muscle

Action potential enters from adjacent cell.

Voltage-gated Ca^{2+} channels open. Ca^{2+} enters cell.

Ca^{2+} induces Ca^{2+} release through ryanodine receptor-channels (RyR).

Local release causes Ca^{2+} spark.

Summed Ca^{2+} Sparks create a Ca^{2+} signal

Ca^{2+} ions bind to troponin to initiate contraction.

Relaxation occurs when Ca^{2+} unbinds from troponin

Ca^{2+} is pumped back into the sarcoplasmic reticulum for storage

Ca^{2+} is exchanged with Na^{+} .

Na^{+} gradient is maintained by the Na^{+} - K^{+} -ATPase.

Myocardial Contractile Cells

Action potential of a cardiac contractile cell

Resting membrane potential is -90mv .

Na^+ passes through double gated voltage channels

Plateau results from decreased K^+ and increased Ca^{++}

Plateau end when flux is reversed

Myocardial Contractile Cells

Cardiac muscle must relax. Prevention of tetanus is important. By the time a second AP can take place, myocardial cell has almost completely relaxed. No summation occurs.

Without an input, autorhythmic (Pacemaker) cells can generate AP, because of their unstable membrane potential which starts at -60 mV and slowly drifts upward through threshold. It never 'rests'!

Modulation of Heart Rate by the Nervous System

Sympathetic stimulation targets I_f channels to open rapidly.

Parasympathetic stimulation targets K^+ and Ca^{++} channels, it hyper-polarizes the cell and slows depolarization

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Electrical Conduction in Myocardial Cells

1% of myocardial cells are designed to spontaneously generate an action potential.

They can contract without outside signal = autorhythmic.

Pacemaker cells do not have sarcomeres

Electrical Conduction in Heart

SA node depolarizes.

Electrical activity goes rapidly to AV node via internodal pathways.

Depolarization spreads more slowly across atria. Conduction slows through AV node.

Depolarization moves rapidly through ventricular conducting system to the apex of the heart.

Depolarization wave spreads upward from the apex.

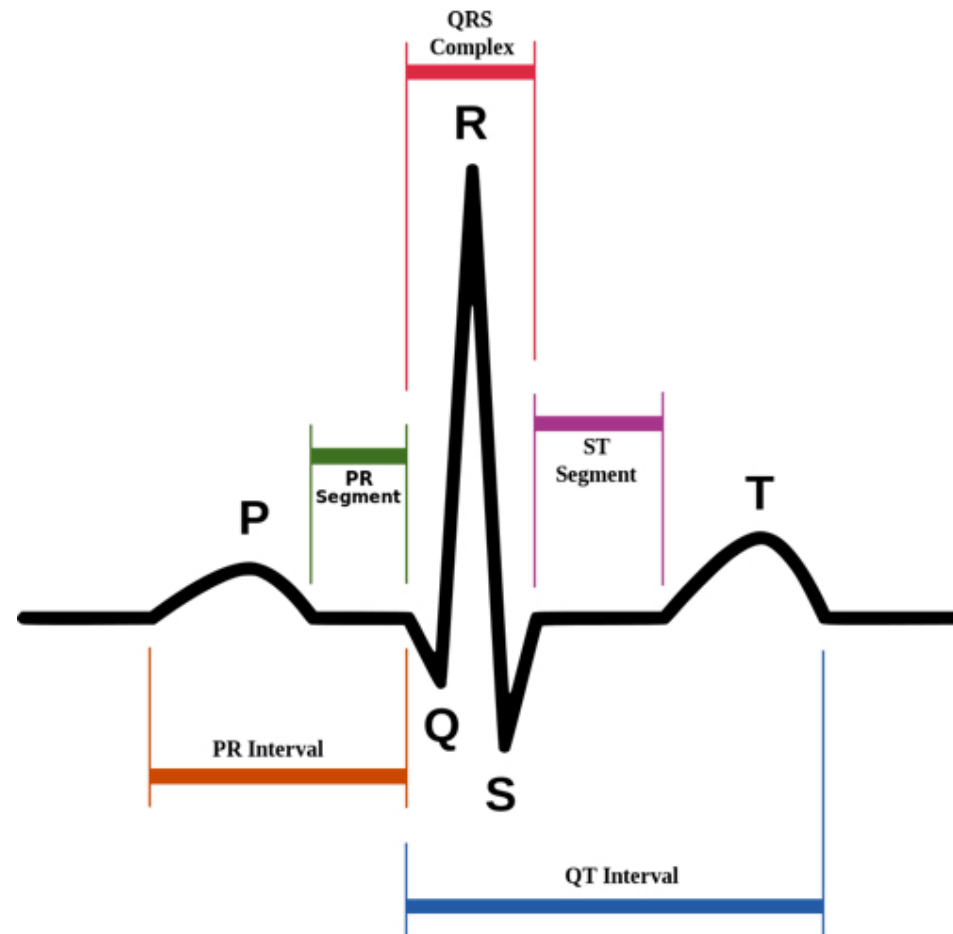
SA node set the pace of the heartbeat at 70 bpm, AV node (50 bpm) and Purkinje fibers (25-40 bpm) can act as pacemakers under some conditions.

The Electrocardiogram

Waves; the parts of the trace that go above or below the baseline.

Segments; sections of baseline between two waves.

Intervals; combinations of waves and segments.



Electrical Activity

Correlation between an ECG and electrical events in the heart

Cardiac cycle begins with both atria and ventricle at rest. ECG begins with atrial depolarization.

Electrical Activity

The P wave reflects the activity of the atria. The atria contract from top to bottom so the P-wave ends after full atrial depolarization

Electrical Activity

The P-Q segment reflects the flow of current along the interventricular septum via the AV node and AV bundle. This is the time when the ventricles are relaxed and filling with blood

Electrical Activity

The QRS complex occurs while the ventricles contraction (depolarize) from the apex & upwards. At the end of the contraction all blood volume to be expelled as been pushed out.

S-T segment happens during ventricular repolarization (relax)

Electrical Activity

The T-wave indicates ventricular repolarization- meaning that the muscle is coming back to a resting state. At this point the chambers are ready to receive blood

ECG provides information on

- Heart rate and rhythm
- Conduction velocity
- Conditions of tissues

Mechanical events of the cardiac cycle

1. The heart at rest: atrial and ventricular diastole.
2. Completion of ventricular filling: atrial systole.
3. Early ventricular contraction and the first heart sound.
4. The heart pumps: ventricular ejection
5. Ventricular relaxation and the second heart sound.

Cardiac Cycle

Left ventricular pressure-volume changes during one cardiac cycle

The ventricle has completed a contraction, has relaxed; contains the minimum amount of blood; its pressure is at its minimum value.

When ventricular contraction begins, the mitral AV valve closes. With both valves closed, blood in the ventricle has nowhere to go. Ventricles continue to contract, causing the pressure to increase rapidly during isovolumic contraction.

Once ventricular pressure exceeds the pressure in the aorta, aortic valve opens. Pressure continues to increase as the ventricle contracts further but ventricular volume decreases as blood is pushed out into the aorta.

At the end of each ventricular contraction, the ventricle begins to relax. As it does so, ventricular pressure decreases. Once pressure in the ventricle falls below aortic pressure, semilunar valve closes, ventricle again becomes a sealed chamber.

Stroke volume is the amount of blood pumped by one ventricle during contraction.

$$SV = EDV - ESV$$

$$135 \text{ mL} - 65 \text{ mL} = 70 \text{ mL} , \text{ average } SV$$

Cardiac output is the volume of blood pumped by one ventricle in a given period of time.

$$\begin{aligned} &72 \text{ bpm} \times 70 \text{ mL/beat} \\ &= 5040 \text{ mL/min , average CO} \end{aligned}$$

Inotropic agent; any chemical that affects contractility
If it increases contractility; positive inotropic (NE, E)
If it decreases contractility; negative inotropic

Ejection Fraction:

Percentage of EDV ejected with one contraction

SV/EDV