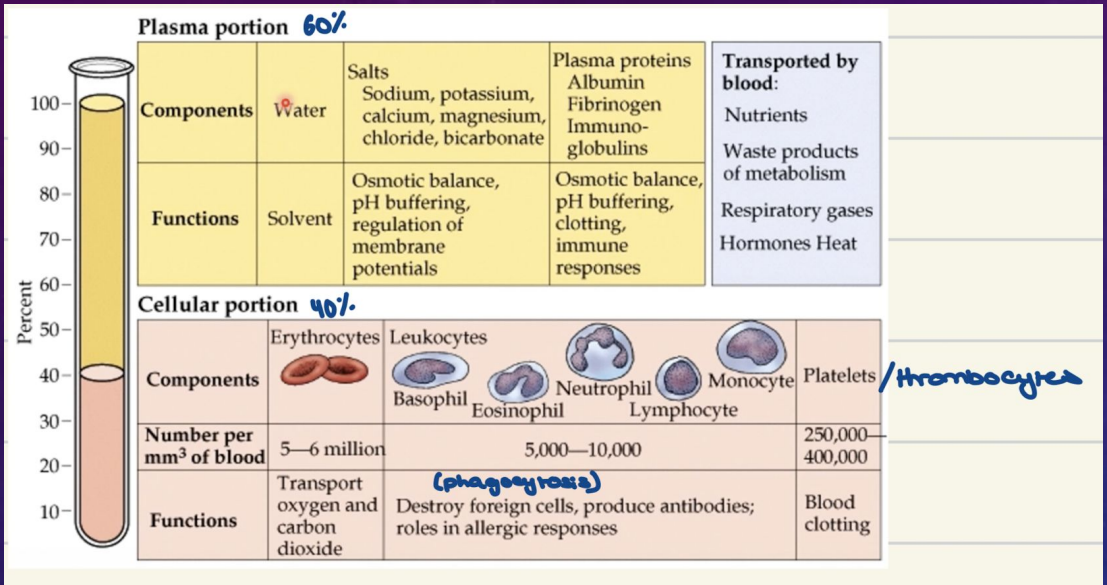
CARDIAC PHYSIOLOGY

BLOOD

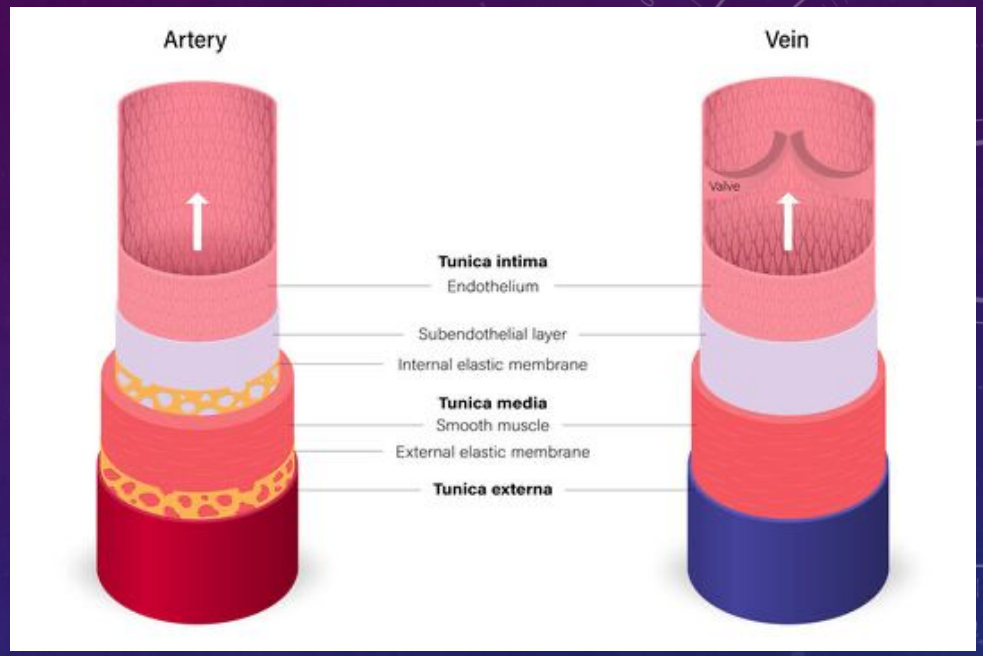


<< constituents of blood (2 parts):
1. Blood Plasma = mostly water with salts, proteins in it
2. Formed Elements = cells

Need good blood volume so enough blood gets to cells
i.e. enough o2 transported to cells

Ischaemia = lack of blood flow to cells
Infarcation = necrosis (cells die) due to ischaemia (lack of blood)

BLOOD VESSELS



- Both arteries and veins have 3 layers: tunica intima, tunica media and tunica externa
- Capillaries are single layered (just endothelium)

1. Tunica intima

- Simple squamous endothelial cells
- Sub endothelial cells
- **Arteries** have internal elastic membrane as part of tunica intima for elastic recoil to cope with high blood pressures

2. Tunica media

- Smooth muscle with autonomic contraction
- Contracts/relax in response to hormones e.g. **angiotensin 2 – potent vasoconstrictor**

3. Tunica externa

- Fibrous layer made of collagen fibres and fibroblasts

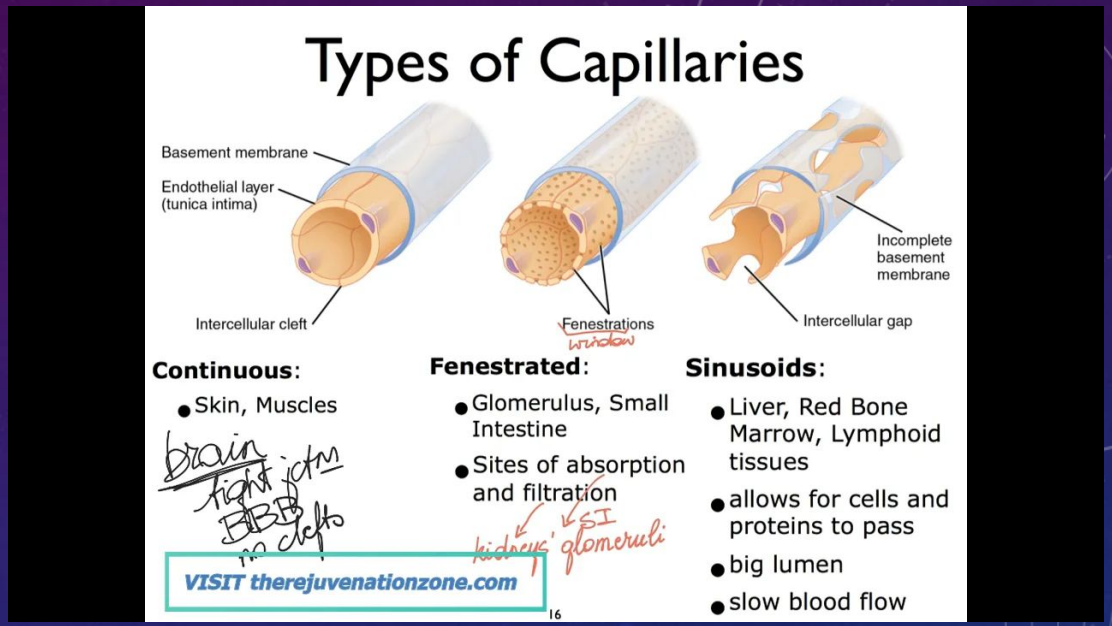
Vaso vasorum = when vessels have their own blood supply

Veins have valves to prevent backflow of blood

CAPILLARIES

- Composed of interlocking single layer of endothelial cells
- Materials eg nutrients, ions, water, o2 leave capillaries by 3 mechanisms:

1. Diffusion
2. Hydrostatic pressure
3. Pinocytosis



Types of capillaries

1. Continuous

- Widespread eg found in blood brain barrier, dermis
- Have pericytes which wrap around endothelium and control what enters/leaves capillaries

2. Discontinuous

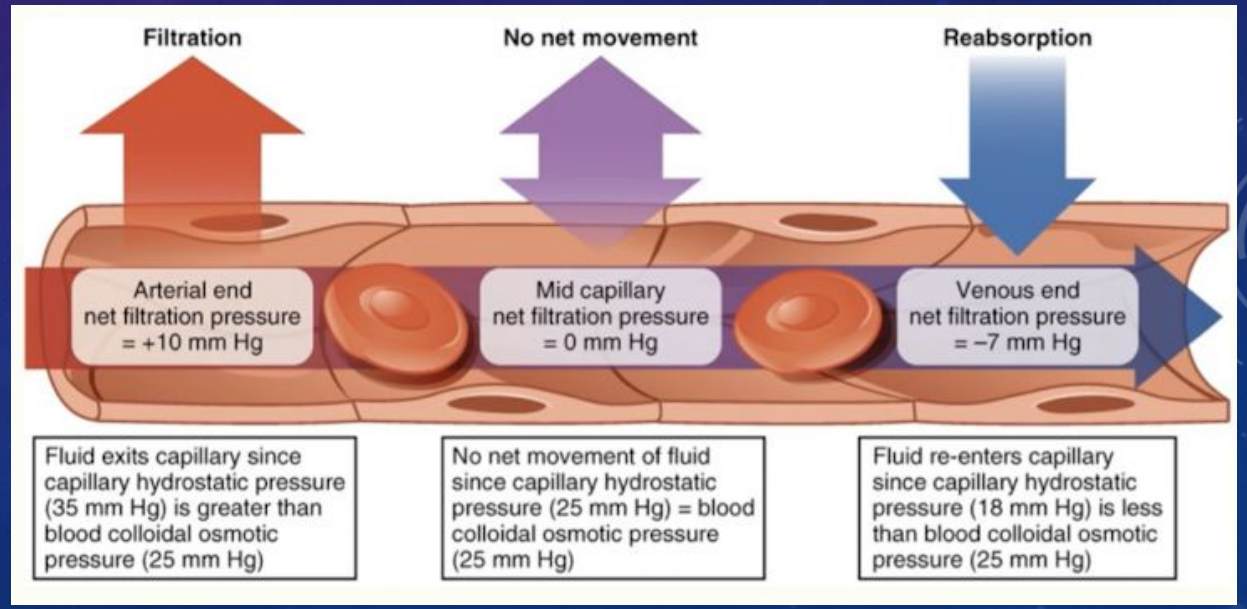
- Have really big holes to allow blood cell transfer
- Bone marrow, liver, spleen

3. Fenestrated

- 10x more permeable than continuous capillaries – good for fluid exchange
- Found in kidneys

NET FILTRATION PRESSURE

- Capillary hydrostatic pressure (CHP): blood in capillaries creates force against capillary walls. This force pushes blood **out** capillaries
- Blood colloidal osmotic pressure (BCOP): pressure created by proteins in blood. Helps draw water from tissues back **in** to capillaries to prevent excess fluid loss
- In arterioles: CHP > BCOP so fluid **OUT**
- In capillaries: pressures are balanced
- In venules: BCOP > CHP so fluid **IN**



CONTROLLING BLOOD VOL: RAS SYSTEM (LEARN)

- Kidney responsible for controlling blood volume via renin-angiotensin system

1. Macula densa cells in ascending loop of henle detect decreased flow of NaCl/decreased flow i.e. bp low or blood vol low

2. Renin Release: When blood pressure drops or blood volume decreases, juxtaglomerular cells in kidneys release an enzyme called renin into the bloodstream.

3. Angiotensinogen Conversion: Renin acts on a protein in the blood called angiotensinogen, converting it into angiotensin I.

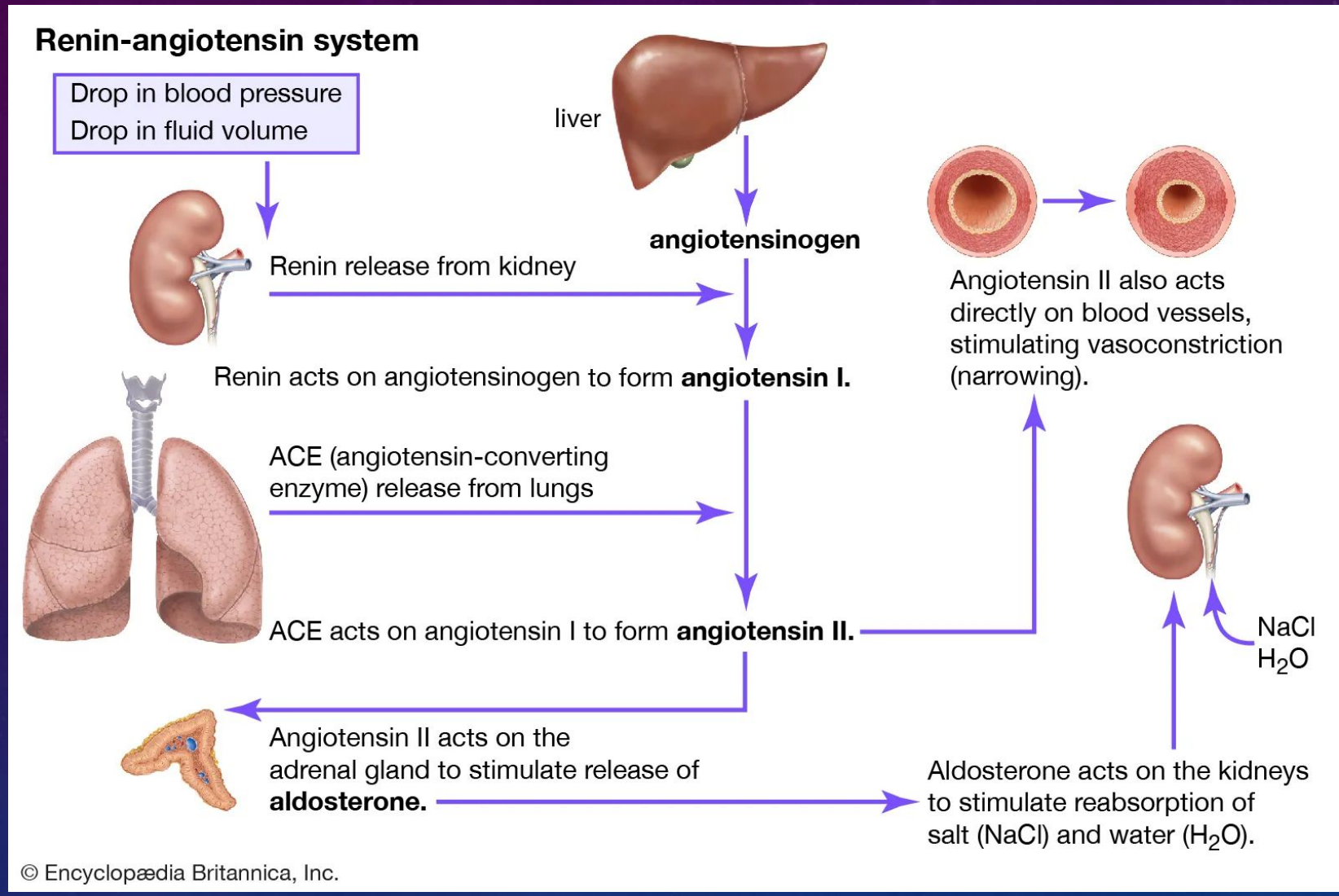
4. Angiotensin-Converting Enzyme (ACE) Activation: Angiotensin 1 converted to angiotensin 2 via ACE enzyme from lungs

5. Angiotensin II Effects:

1. **Vasoconstriction**: Angiotensin II causes blood vessels to narrow (vasoconstriction), increasing blood pressure.
2. **Aldosterone Release**: Angiotensin II stimulates the adrenal glands to release a hormone called aldosterone.

6. Aldosterone Effects: Aldosterone acts on the kidneys; increase reabsorption of sodium and water back into the bloodstream and increasing excretion of potassium. This leads to an increase in blood volume and blood pressure.

7. Negative Feedback Loop: As blood pressure and blood volume return to normal, the release of renin decreases, and the RAS is inhibited to prevent excessive vasoconstriction and fluid retention.


Renin-angiotensin system
Drop in blood pressure
Drop in fluid volume
liver
angiotensinogen
Renin release from kidney
Renin acts on angiotensinogen to form angiotensin I.
ACE (angiotensin-converting enzyme) release from lungs
ACE acts on angiotensin I to form angiotensin II.
Angiotensin II also acts directly on blood vessels, stimulating vasoconstriction (narrowing).
NaCl
H2O
Angiotensin II acts on the adrenal gland to stimulate release of aldosterone.
Aldosterone acts on the kidneys to stimulate reabsorption of salt (NaCl) and water (H2O).
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ANGIOTENSIN 2 EFFECTS ON CVS

1. Act on AT1 receptors (g-coupled receptors) > constrict vessels > increase blood pressure
2. Stimulates adrenal cortex to release aldosterone > increases sodium and fluid retention
3. Stimulate release of anti-diuretic hormone (ADH) > increase fluid retention
4. Cardiac and vascular hypertrophy (heart gets bigger)
5. Stimulates noradrenaline release > enhances sympathetic adrenergic function > everything increases eg blood pressure, vasoconstriction, heart rate etc

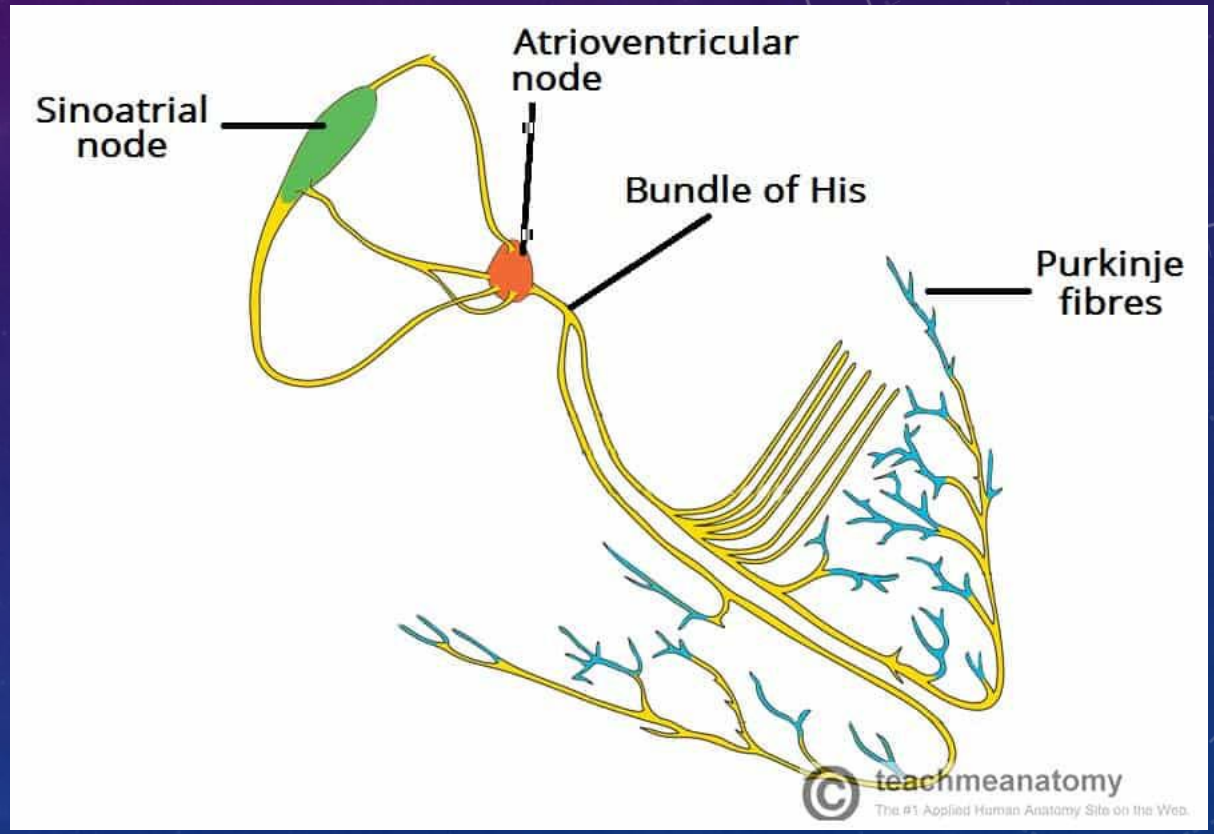
CARDIAC CONDUCTION SYSTEM

1. START in SinoAtrial Node. The cardiac impulse starts in the SA node which is in the right atrium (this causes the atria to contract)

2. We then move to the AtrioVentricular Node which is located at the start of the atrioventricular septum. The AV nodes is a delay to the impulse to stop the heart contracting all at once. It makes sure that the atria have fully pumped all of their blood into the ventricles before the AV valves close and the ventricles contract

3. From the AV node we go into the AtrioVentricular Bundle (Bundle of His) which sends the impulse down the AV septum. It divides into 2 main bundles for each ventricle

4. FINISH in the purkinje fibres which allow the rest of the ventricle muscle to contract



Credits: ruhaan yr3

DEFINITIONS TO HELP

Polarisation - diff. in charge between sides of a membrane

↳ polarised = intracellular more negative (voltage ↑ -ve, line downwards)

↳ depolarised = intracellular more positive (voltage ↑ +ve, line on graph goes up)

voltage = diff. in +ve charges from 1 side of a membrane to the other

↳ -80mV = fewer +ve charges inside

↳ resting membrane pot. of cardiomyocyte

current = flow of charged particles across a membrane

influx = flow into cell efflux = flow out of cell

My handwriting's neater than this promise

PACEMAKER ACTION POTENTIAL (AVN AND SAN)

impulse here takes place automatically

Phase 4 – funny currents

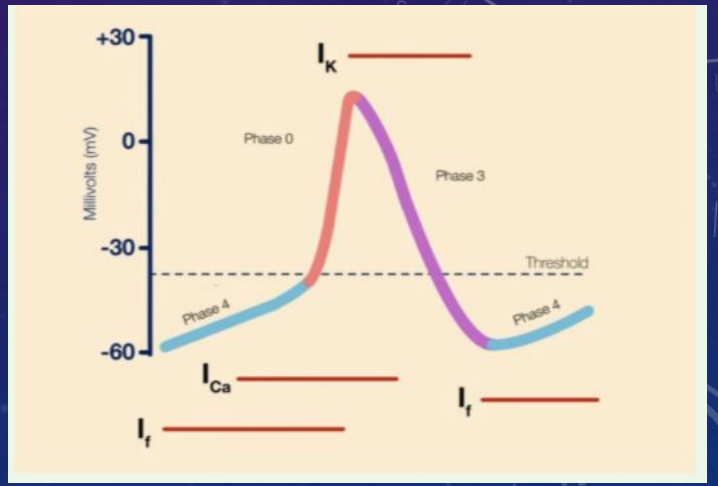
Ion channels that allow slow movement of Na+ into the cell are open which start to slowly depolarise the cell until it reaches potential threshold. These are the currents that cause SPONTANEOUS DEPOLARISATION of the cell

Phase 0 – rapid depolarisation

When the potential threshold is reached, L type calcium channels open and cause calcium to enter the cell and depolarise it (this is basc the contraction of the cell). The funny currents and Calcium channels slowly stop to initiate phase 3

Phase 3 - repolarisation

Outward K+ channels open and start to repolarise the cell (because the positive stuff is now going out). The cell becomes hyperpolarised its that negative.



NON-PACEMAKER ACTION POTENTIALS (MYOCYTES)

Phase 0 (Rapid depolarisation and therefore contraction)

The action potential from surrounding cells increases the membrane potential to its threshold potential, enough to start depolarisation. inward voltage gated sodium channels open to let Na+ into the cell

Phase 1 (Initial repolarisation)

The inward voltage gated sodium channels close and outward voltage gated potassium channels open which repolarise the cell

Phase 2 (plateau phase)

There is calcium influx through voltage gated calcium channels that open which slow down the repolarisation of the cell by the potassium ones in phase 1, that's why it looks flat on the graph.

Phase 3 (rapid repolarisation)

Calcium channels close and even more potassium channels open to rapidly make the cell negative inside until it reaches its baseline potential in phase 4

Phase 4 (Resting potential)

This is the flat line at the bottom. Its maintained by an ATPase sodium potassium pump which keeps the membrane more permeable to potassium

PHASE 0

RAPID UPSTROKE & DEPOLARIZATION

Voltage-gated Na+ channels open

PHASE 1

INITIAL REPOLARIZATION

Inactivation of voltage-gated Na+ channels.
Voltage-gated K+ channel begin to open.

PHASE 2

PLATEAU

Ca2+ influx through voltage-gated Ca2+ channels balances K+ efflux.

Ca2+ influx triggers Ca2+ release from sarcoplasmic reticulum and myocyte contraction.

PHASE 3

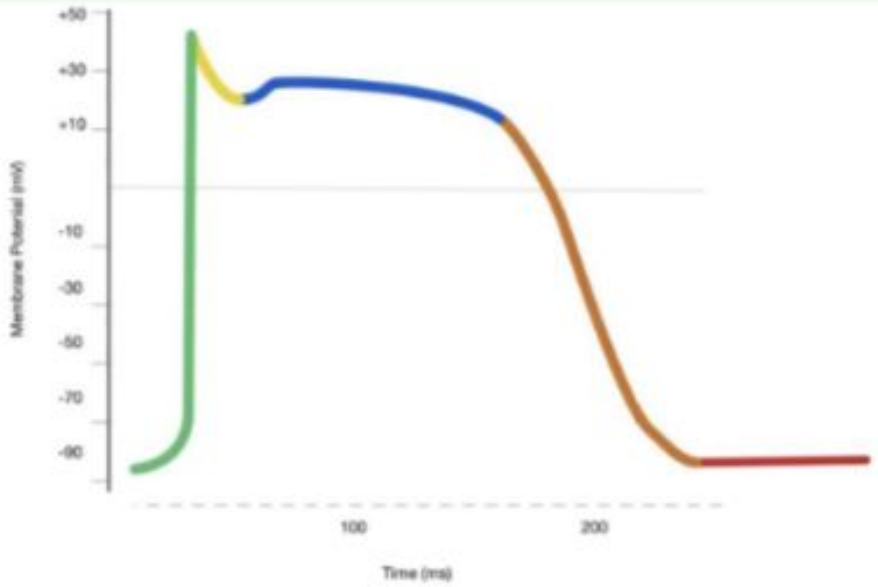
RAPID REPOLARIZATION

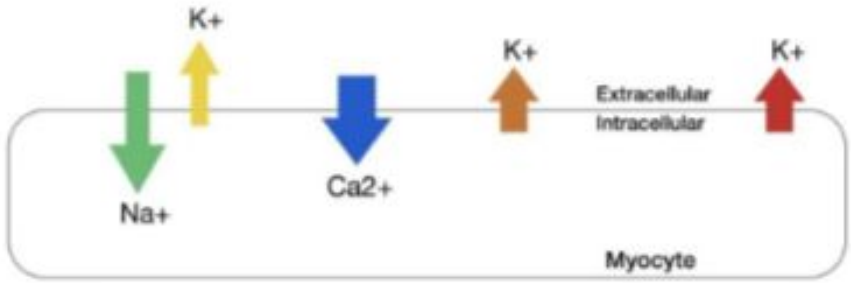
Massive K+ efflux due to open of voltage-gated slow delayed-rectifier K+ channels and closure of voltage-gated Ca2+ channels.

PHASE 4

RESTING POTENTIAL

High K+ permeability through K+ channels.

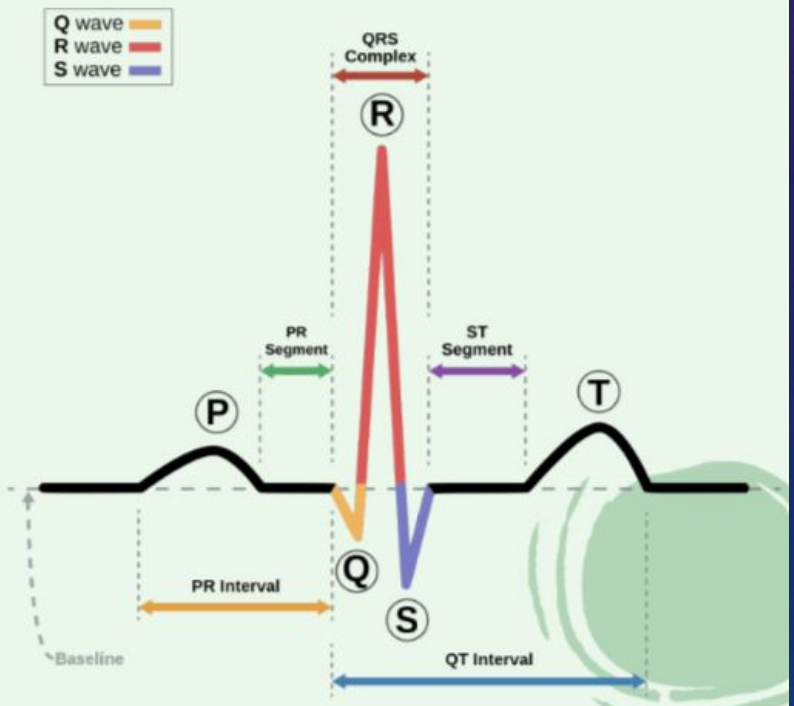




ECG

ECG

- P wave = depolarisation of the atria triggered by the SA node
- PR segment = time between the end of atrial depolarisation and the start of ventricular depolarisation. This is the time taken for the impulse to travel thru the AVN and bundle of His
- QRS complex = depolarisation of the ventricles which is the main pump of blood out of the heart.
- ST segment = end of QRS to the start of the T wave. This represents the beginning of ventricular repolarisation
- T wave = ventricular repolarisation



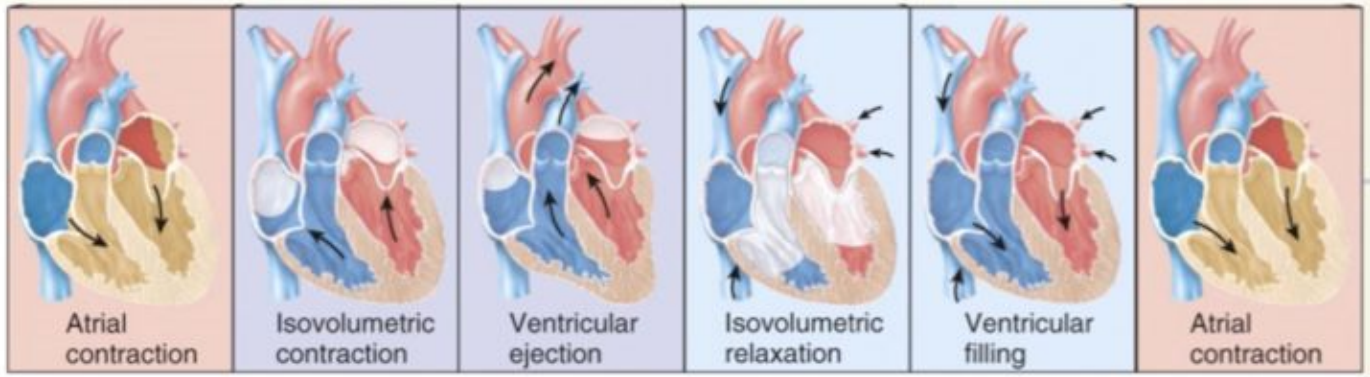
10 electrodes
- 12 leads

CARDIAC CYCLE

- **Atrial Contraction (Atrial Systole)**:
  - The cardiac cycle begins with atrial contraction, which occurs as the atria receive blood from the body and lungs. Atrial contraction helps push the remaining blood into the ventricles.
- **Ventricular Filling (Early Diastole)**:
  - After atrial contraction, the ventricles begin to fill with blood as the atria relax (atrial diastole). Blood flows passively from the atria into the ventricles through the open atrioventricular (AV) valves.
- **Isovolumetric Contraction (Ventricular Systole)**:
  - As the ventricles fill with blood, they contract (ventricular systole), increasing pressure inside the chambers. AV valves are closed as ventricular pressure > aortic pressure. The SL valves are still closed though, so pressure increases and volume stays constant. No blood is ejected
- **Ventricular Ejection (Ventricular Systole)**:
  - Once ventricular pressure exceeds the pressure in the aorta and pulmonary artery, the semilunar valves open, and blood is ejected from the ventricles into the pulmonary artery and aorta.
- **Isovolumetric Relaxation (Early Diastole)**:
  - After ventricular ejection, the ventricles begin to relax (ventricular diastole), and ventricular pressure decreases. All valves are closed, pressure decreases and volume is constant
- **Ventricular Filling (Late Diastole)**:
  - As the ventricles continue to relax, ventricular pressure drops further, falling below atrial pressure.
  - The AV valves open, and blood flows passively from the atria into the ventricles, completing the cardiac cycle and preparing for the next heartbeat

Phases of Cardiac Cycle

1. Atrial systole
2. Isovolumetric ventricular contraction
3. Rapid ventricular ejection
4. Isovolumetric ventricular relaxation
5. Rapid ventricular filling



(e) Phases of the cardiac cycle

CARDIAC OUTPUT, STROKE VOLUME, HEART RATE

- **CO = SV x HR**
- SV is determined by 3 factors:

1. Preload – vol of blood available to pump (in heart)
2. Contractility – force with which heart can contract
3. Afterload – arterial pressure against which ventricle needs contract

- Left Ventricle (LV) does not empty completely during systole.

- Stroke Volume (SV) = End Diastolic Volume (EDV) - End Systolic Volume (ESV)

Stroke Volume (SV)	End Diastolic Volume (EDV)	End Systolic Volume (ESV)
Amount of Blood transferred from LV to arterial system during Systole.	*Total volume of blood in ventricle at end of Diastole* ***Dependent on PRELOAD***	*Volume of blood remaining in ventricle at end of Systole.* ***Dependent on AFTERLOAD***
SV =Approx. 80-90ml	***EDV= Approx. 140ml***	***ESV= Approx. 50ml***

- In healty person SV should be > 60 ml.
- EF (ejection fraction) = SV \ EDV (normally about 55% - 75%.
- EF is an important measurement of cardiac efficiency.
- EF is used clinically to assess cardiac status in patients with heart failure.

MORE DEFINITIONS

- Systolic pressure = max. aortic pressure **after** ejection
- Diastolic pressure = lowest aortic pressure **before** ejection
- Difference between the two ^ = aortic pulse pressure
- Mean arterial pressure = diastolic + 1/3 pulse pressure

QUESTIONS

1. Which of the following hormones causes arterial vasoconstriction?

a. Noradrenaline
b. Acetylcholine
c. Anp
d. Endothelin
e. Insulin

2. Which pump maintains the resting membrane potential in myocytes?

a. ca2+ channels
b. Na+ channels
c. K+ channels
d. NA/K ATPase

3. What receptor does angiotensin 2 act on in kidney?

a. AT2 receptor
b. Macula densa cell receptors
c. AT1 receptor
d. ADH receptors

4. Which of the following is not an action of aldosterone?

a. increases blood osmolality
b. Increases sodium excretion by renal tubule
c. Increases potassium excretion by renal tubule
d. Inhibits noradrenaline release

- 1. a
- 2. d
- 3. c
- 4. b