Heart Chambers

Left Atria: oxygen- **rich** blood from pulmonary veins -> left AV valve
Right Atria: oxygen **poor** blood from vena cavae -> right AV valves
Left Ventricle: oxygen **rich** blood, from right at--> semilunar valves
Right Ventricle: oxygen **poor** blood from rigth atrium-> pulmonary artery

Nodes

SA Node	AV node	Bundle of His

SA node
initiates potential and **leads**
audtorhythmicity: 70 action potentials per minute

AV node
base of right atrium
audtorhythmicity: 50 A.P. / minute
follows SA node under normal conditions

Bundle of His
origin: AV node
projects to left and right ventricles

Purkinje Fibers
terminal fibers
extend from Bundle of His
follows faster SA node under normal conditions

Cardiac Cycle

Systole

contraction

Diastole

relaxation

End- diastolic volume

volume of blood at end of diastole, max amount that chamber will hold

Isovolumetric ventricular contraction

Cardiac Cycle (cont)

during contraction, valves closed, no blood leaves, increase in chamber pressure

Stroke volume

amount of blood pumped out of chamber

Isovolumetric ventricular relaxation

chamber pressure decreases

ACh: Parasympathetic release

SA Node

Ach: inc. permeability to K+ by delaying inactivation of K+ channels -> more hyperp-olarization

AV Node

inc permeability to K+, reduces excitability of node, delays response of input from SA node

Atrial contractile cells

shortens of action potentials: reducing Ca2+ permeability during plateau

Components

Heart	Blood Vessels	Blood

Valves : one way

Right AV Valve	Left AV valve	Aortic/ Pulmonary valve
tricuspid	bicuspic	semilunar

Pathways

Interatrial

cardiac cells pacemaker activity from right to left atrium

Internodal

cardiac cell pacemaker activity from SA to AV node

AV nodal delay

Slow through AV node due to thickness

Action potential in Contractile cells

Resting potenial

-90mV until excited

Rising phase

rapid, Na+ entry due to opening of fast Na+ channels, close K+ channels

Repolarization

at peak, open transient K+ efflux, inactivate Na+ channels

Plateu

Slow Ca2+ entry, open of L-type Ca2+ channels, reduce

Falling phase

K+ efflux, open voltage-gated K+ channels

Resting Potential

maintained by opening leaky K+ channels

Heart Sounds

Murmer:

turbulent flow of blood through malfunctioning valves

Stenotic valve:

whistling, stiff narrow valve

Insufficient valve:

swishing, structurally damaged valve

Rheumatic fever:

valvular stenosis, autoimmune disease

Cardiac muscle type

Straited:

striped lines

Involuntary

moves by itself

Sarcomere

Z line

sarcomere boundary

A band

thick filament with thin filament overlapp

H zone

where thin filaments don't reach

M line

middle, vertically down A band

I band

remaining thin filament that are not in A band

Muscle

Myofibril:

thin(actin) thick (myosin), sarcomeres connected together

Sarcomere:

smallest unit of muscle cell,

Myosin:

cytoskeletal protein

Thin filament

actin, tropomyosin, troponin

Types of Muscle

Endocardium - thin/ interior of each chamber

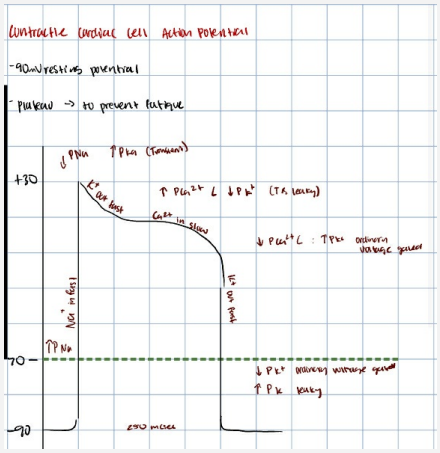
Myocardium - middle layer, thickest, interc-alated disks for connection

Epicardium - thin external membrane with pericardial fluid

Pacemaker Activity

Open funny channels:
NA+ inward and closes K+ outward
Reaches threshold of -40:
Open L-type Ca2+ and K+ channels

Action potential in Contractile cells



Electrical Activity

Autorhythmicity- capability to produce own rhythmic electrical activity due to
Pacemaker activity
Only 1% of Cardiac cells

Excitation Contraction Coupling

Ca2+ entry into cystol

T-tubule

contain: dyhyropyridine receptors-> open Ca2+ into cytosol

#crossbridges proportional to cytosolic Ca2+ concentration

Electrocardiogram

P wave

depolarization of atria

QRS complex

depolarization of ventricles

T- wave

Repolarization of ventricles

Cardiac Output

Cardiac Output:

heart rate x stroke volume

Heart rate:

beats per minute, regulated by both parasympathetic and sympathetic nervous system

Excitation Contraction Coupling

ACh

- released by motor neuron axon binds to motor end plate

- results in action potential that propagates to surface membrane and down t-tubule

- triggers Ca2+ release from SR

-Ca2+ released from lateral sacs

- bind to troponin, tropomyosin moves aside to open actin binding site

- myosin crossbridge attach to actin-> bend -> pull actin towards center of sarcomere

- Ca2+ taken up by SR

- tropomyosin goes back to blocking position.