Intro

Myocardium	middle of heart wall, contains cardiac muscle
How are CM cells connected?	intercalated discs, forms desmosomes and gap-junctions
Functional Syncytium	group of CM cells that contract in coordination with each other (gap junctions)
Autorhythmicity	creates its own electrical activity (no NS input)
Pacemaker Cells	creates pacemaker activity, grouped together in nodes
Cardiac Contractile Cells	99% of cardiac muscle cells, actually performs contraction but is not autorhythmic
Other Characteristics	involuntary (autonomic neuro fibers), striated, lots of mito + myoglobin, longer AP than smooth/skeletal muscle

Pacemaker Flow

SA Node	70 APs/min, main node
Where is SA node?	right atrium near superior vena cava
AV Node	50 APs/min, follows SA node
Where is AV node?	base of right atrium
Bundle of His	tract of pacemaker cells that start at AV node -> ends at left and right ventricles
Purkinje Fibers	30 APs/min, follows SA node
Where are Purkinje Fibers?	from end of Bundle of His through ventricular myocardium
Interatrial Pathway	pacemaker pathway from right to left atrium

Pacemaker Flow (cont)

Internodal Pathway	pacemaker pathway from SA node to AV node
AV Nodal Delay	activity delay of 100ms going through AV node
Why is AV Nodal Delay important?	allows for ventricles to contract after atrial contraction

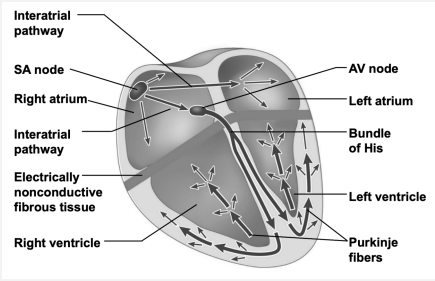
Pacemaker Activity

Nodes	controls rate and coordination of contractions
How many nodes?	2 nodes, SA and AV
Pacemaker potentials	depolarization of membrane potential until threshold (triggers AP)
First half of pacemaker potential	funny channels open -> Na+ in, K+ channels close (K+ remains inside)
Second half of pacemaker potential	funny channels close, T-type Ca2+ channels open -> takes potential to threshold
Threshold	T-type Ca2+ channels close, L-type Ca2+ channels open -> potential reaches peak
Falling Phase	K+ channels open (K+ out), L-type Ca2+ channels close -> fall back to original potential
Major ions for pacemaker activity	K+, Na+, Ca2+
Timing	both Ca2+ channels are crucial for keeping rhythm (T-type channels: gradual depolarization, L-type: fast depolarization)

Pacemaker Activity (cont)

Pacemaker potential value	-60mV

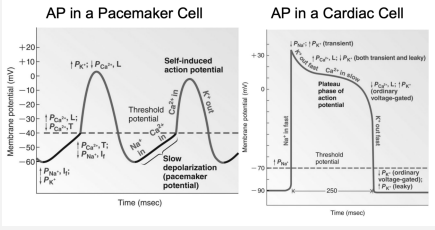
Excitation Pathway



Contractile Cardiac Muscle Cells

Resting potential value	-90mV
Rapid rise	opening fast Na+ channels, Na+ in
Brief repolarization	limited K+ efflux, coupled with inactivation of Na+ channel
Plateau Phase	Ca2+ entry (opens L-type channels), coupled with reduced K+ efflux (K+ channels close)
Rapid falling	opening ordinary voltage-gated K+ channels (K+ out)
Resting potential	back to resting potential by closing ordinary K+ channels and opening leaky K+ channels
AP and Contractile response	contraction happens during plateau phase

Contractile AP vs. Pacemaker AP



Excitation-Contraction Coupling	
Dyhydr-opyridine receptors	acts like voltage-gated Ca2+ channels. When AP reaches T-tubules, these receptors activate and allows Ca2+ flow
Sarcop-lasmic Reticulum	entry of Ca2+ causes calcium release from Sarcoplasmic Reticulum
Contra-ction	number of activated cross--bridges is proportional to Ca2+ conc. in cytosol
Calcium-I-nduced Calcium-Release	opening of L-type Ca2+ channels -> activation of dyhydropyridine receptors -> amplified release of Ca2+ from sarcoplasmic reticulum
Refractory Period and Contra-ction	refractory period and length/st-rength of contraction is directly proportional (longer refractory period = contractile length/st-rength increases)

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