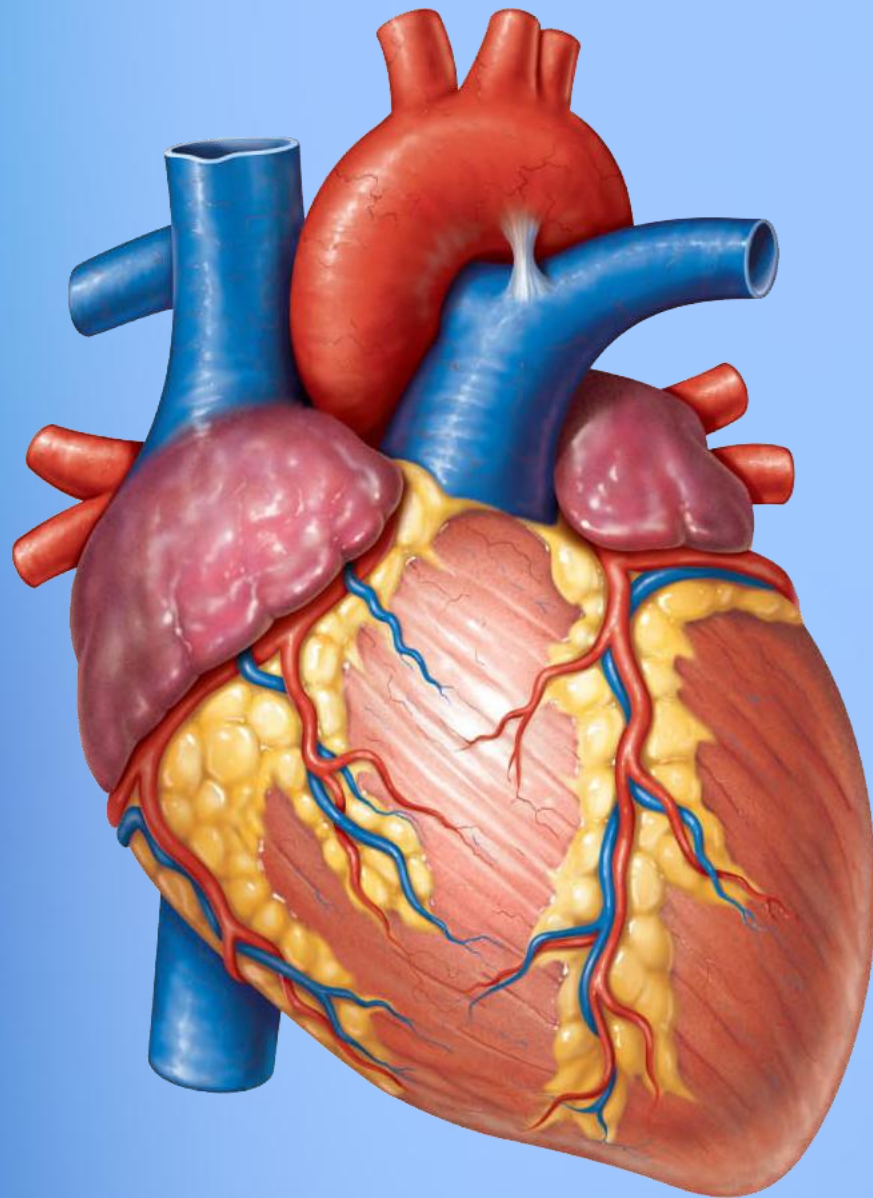


EXCITATION AND CONTRACTION OF HEART MUSCLES



Cardiovascular Block

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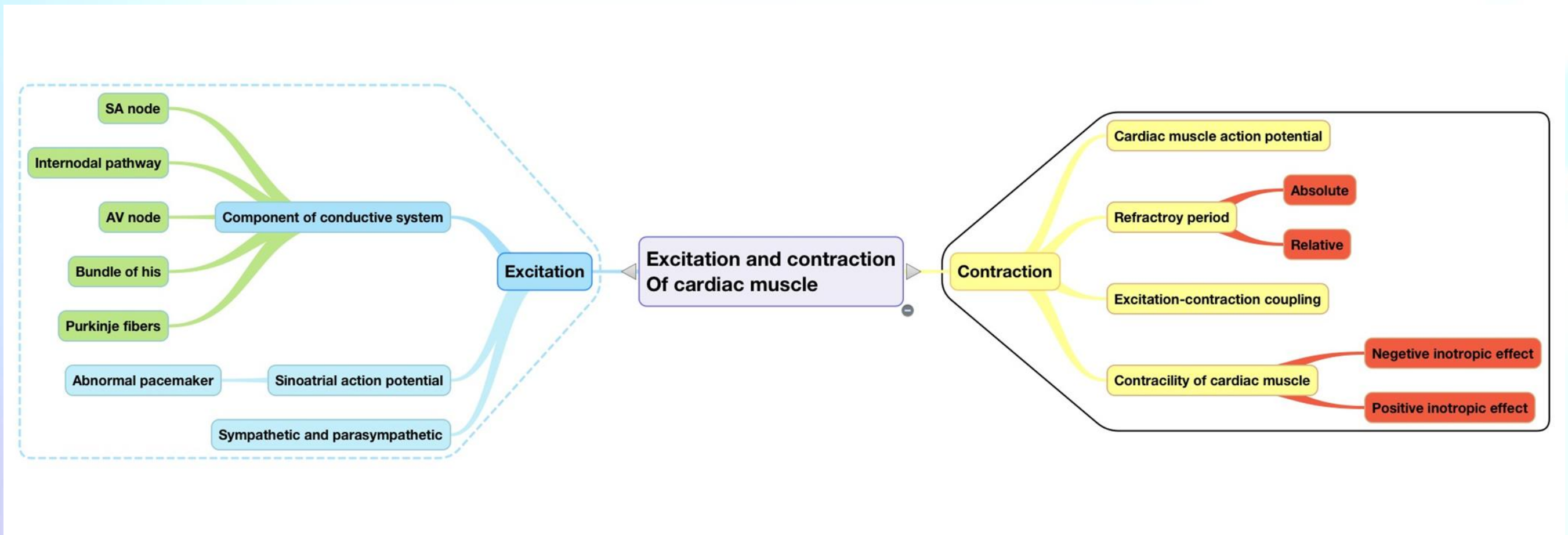


@PhysiologyTeam



Pht433@gmail.com

Mind Map



Objectives

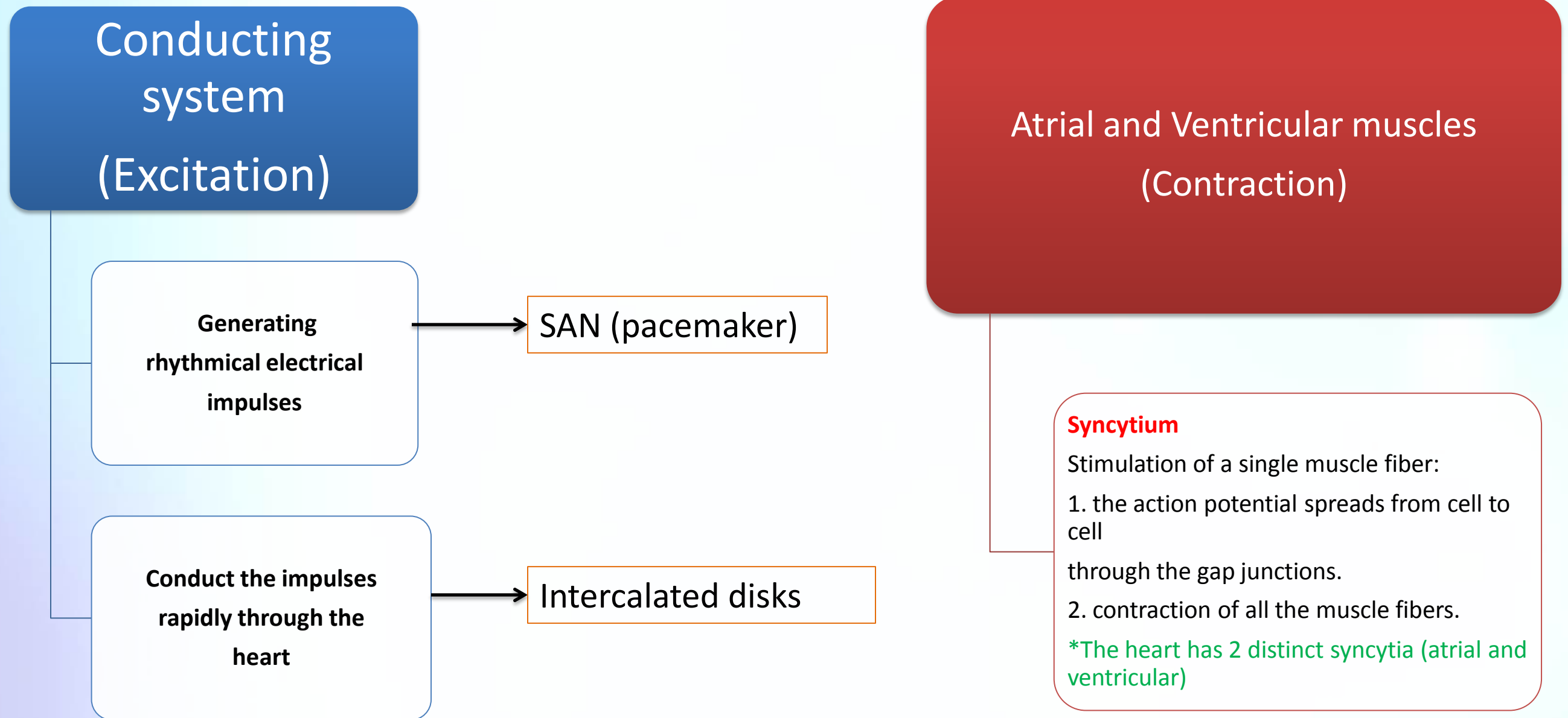
First : Excitation

- Discuss the cardiac conductive system and its function.
- Describe the action potential of the cardiac muscle and its components.
- Define the refractory period and the excitation-contraction coupling
- Discuss the control of excitation and conduction of the heart.

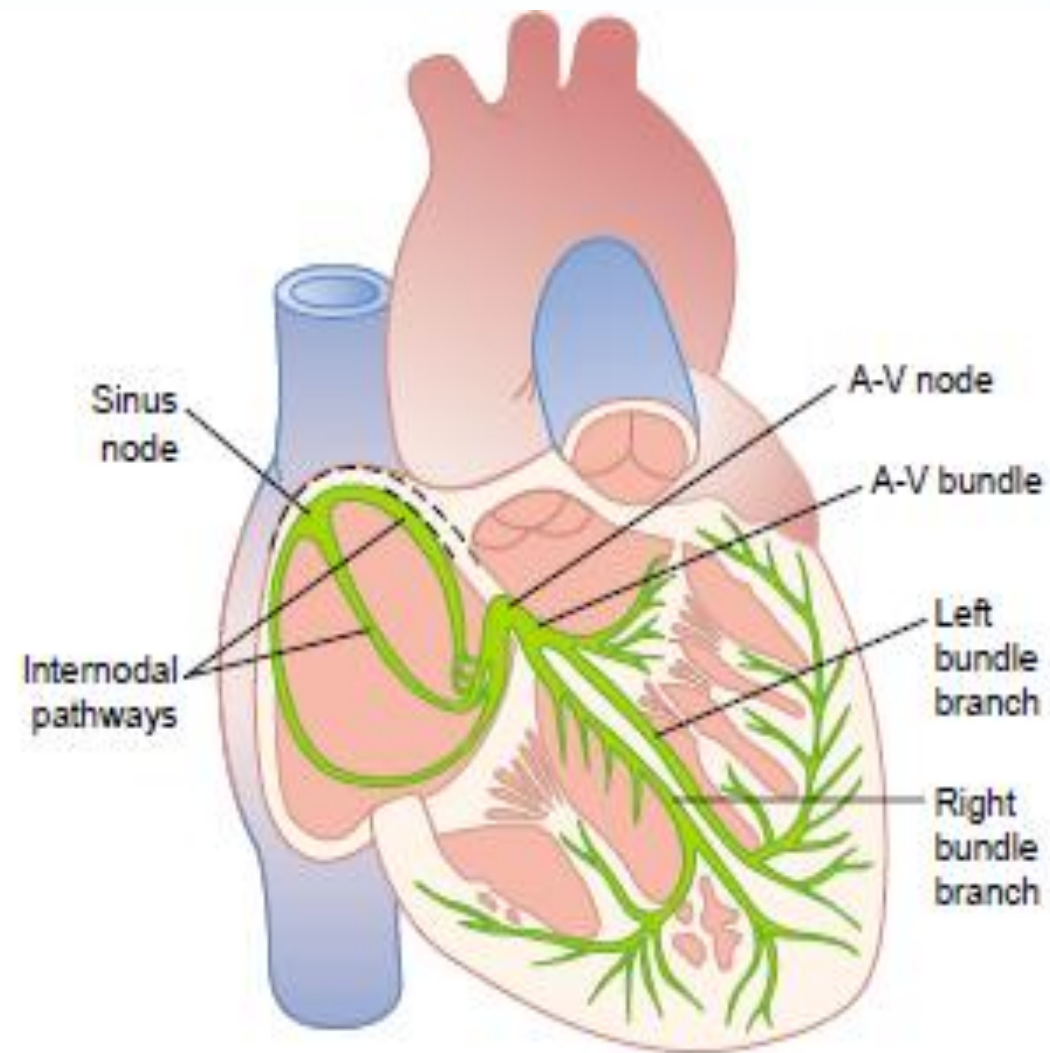
Second : Contraction

- Define cardiac muscle contractility
- Understand the phases of cardiac action potential and the ionic bases
- Discuss the role of calcium ions in the regulation of cardiac muscle function
- Describe the mechanism of excitation contraction coupling
- Factors affecting cardiac contractility

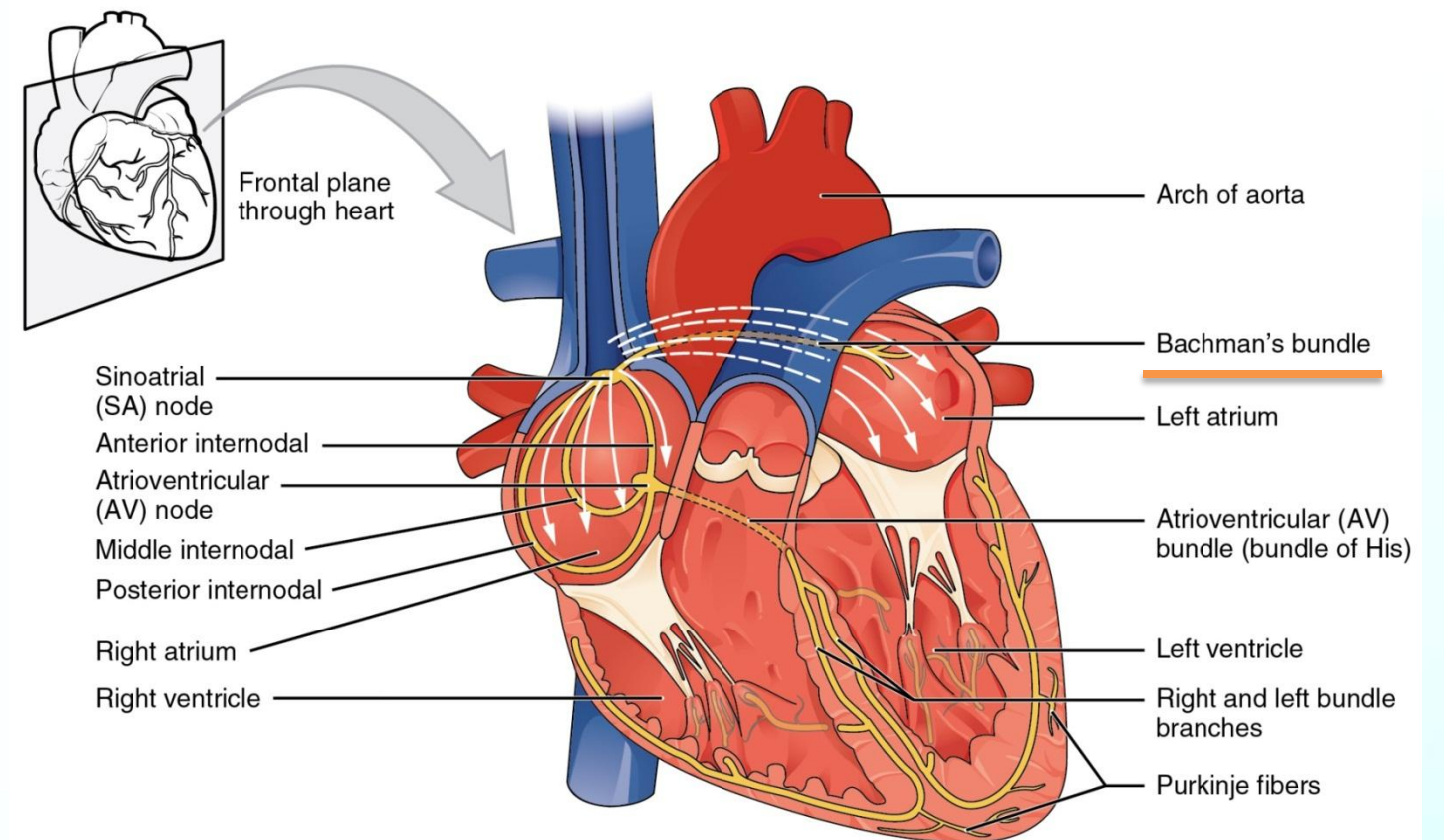
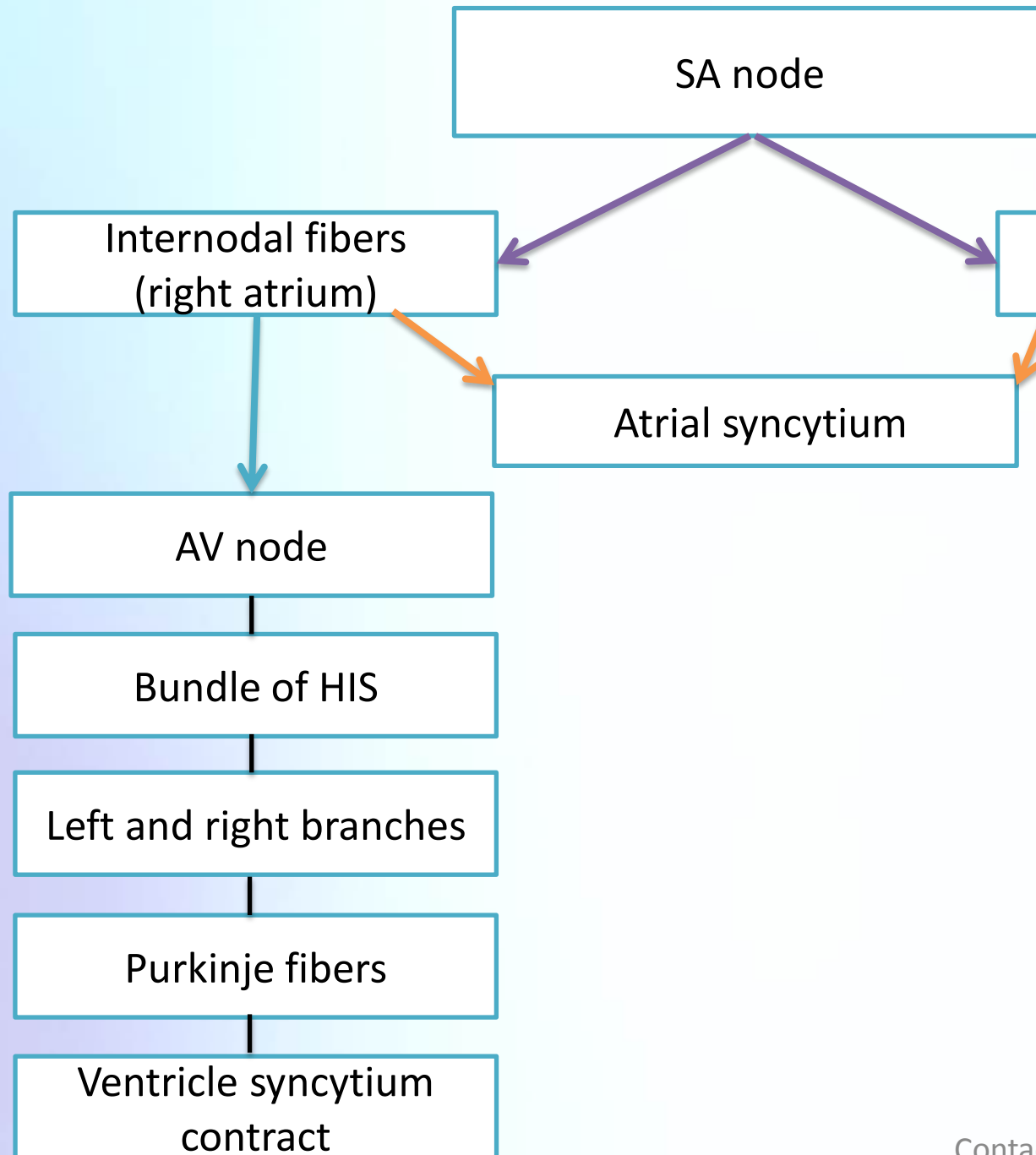
Excitation and Contraction of heart muscles



First : Excitation (Conductive system or Autorhythmic cells)



Sequence of excitation



Anterior view of frontal section

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Component of Conductive System of the Heart

structure	location	function
Sinoatrial node (S-A node)	superior lateral wall of the right atrium	<ul style="list-style-type: none"> ❖ Pacemaker of the heart. Why? Because Its <u>rate of rhythmic discharge</u> is greater than any other part in the heart and it has the Highest frequency ❖ originating action potentials by itself (Auto
internodal pathway	_____	conduct the impulses at a faster rate than atrial muscle fibers , because of specialized conduction fibers
Atrioventricular node (A-V node)	posterior wall of the right atrium	<ul style="list-style-type: none"> ❖ Delay in the conduction of impulses (0.1 sec). Why? To allows time for the atria to empty the blood into the ventricles before ventricular contraction begin ❖ It is slow conduction because diminished number of gap junctions
Atrioventricular bundle(Bundle of His)	Sides of the ventricular septum	<ul style="list-style-type: none"> ❖ inability of action potentials to travel backward from the ventricles to the atria. ❖ Fibrous barrier surrounding the bundles prevents re-entry of cardiac impulse by this route from the ventricles to the atria.
Purkinje fibers	Penetrate Atrioventricular fibrous tissue	<ul style="list-style-type: none"> ❖ It has very high permeability of gap junctions so , ions are transmitted easily from one cell to the next that will lead to enhance the velocity of transmission



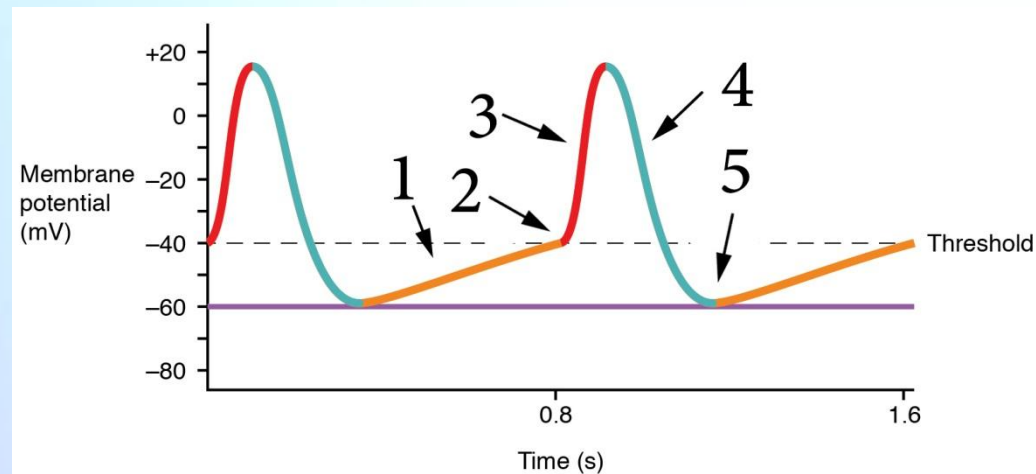
<http://www.youtube.com/watch?v=7K2icszdxQc>
<http://www.youtube.com/watch?v=2ZCbDI2V41Q>

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Sinoatrial Node APs

It's controlled by **Voltage-Gated channels (Na⁺,Ca²⁺,K⁺)^{*}**:

Sinoatrial Node APs



- 1- Slow sodium channels opened (**Na influx**)
- 2- At threshold level, the sodium and calcium channels become activated causing the action potential
- 3- Depolarization
- 4- potassium channels open (**K efflux**)
- 5- Rest membrane potential (**-55 mV**)

Note:

- Voltage-Gated channels are controlled by changing in charges.
- This cycle will never stop (sodium channels always open)

Abnormal pacemakers

1- Ectopic pacemaker:

Any other part of the heart develops a rhythmical discharge rate that is more rapid than that of the sinus node

Example: the **A-V node** or in the **Purkinje fibers**

2-Blockage of transmission of the cardiac impulse from the sinus node to the other parts of the heart

Example: A-V block

- cardiac impulses fails to pass from atria into the ventricles
- the atria continues to beat at the normal rate of rhythm of the S-A node
- a new pacemaker develops in the Purkinje system with a new rate

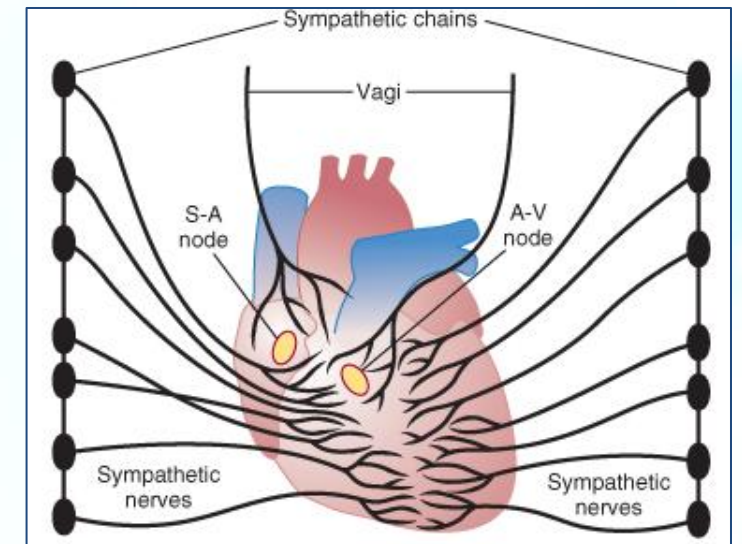
****there are 2 pacemakers here (one in atria and another in the Purkinje system)**

<http://www.youtube.com/watch?v=OQpFFiLdE0E>

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Sympathetic and parasympathetic

	Sympathetic	Parasympathetic
Supply	all parts of the heart with strong supply to the ventricles	mainly to the S-A and A-V nodes
Rate of rhythm	↑ rate of rhythm of the S-A node.	↓ rate of rhythm of the S-A node
Transmission of impulses	↑ transmission of impulses to the A-V node.	↓ transmission of impulses to the A-V node
Force of contraction	↑ force of contraction.	-----
Inotropic effect*	Positive	negative



Note:

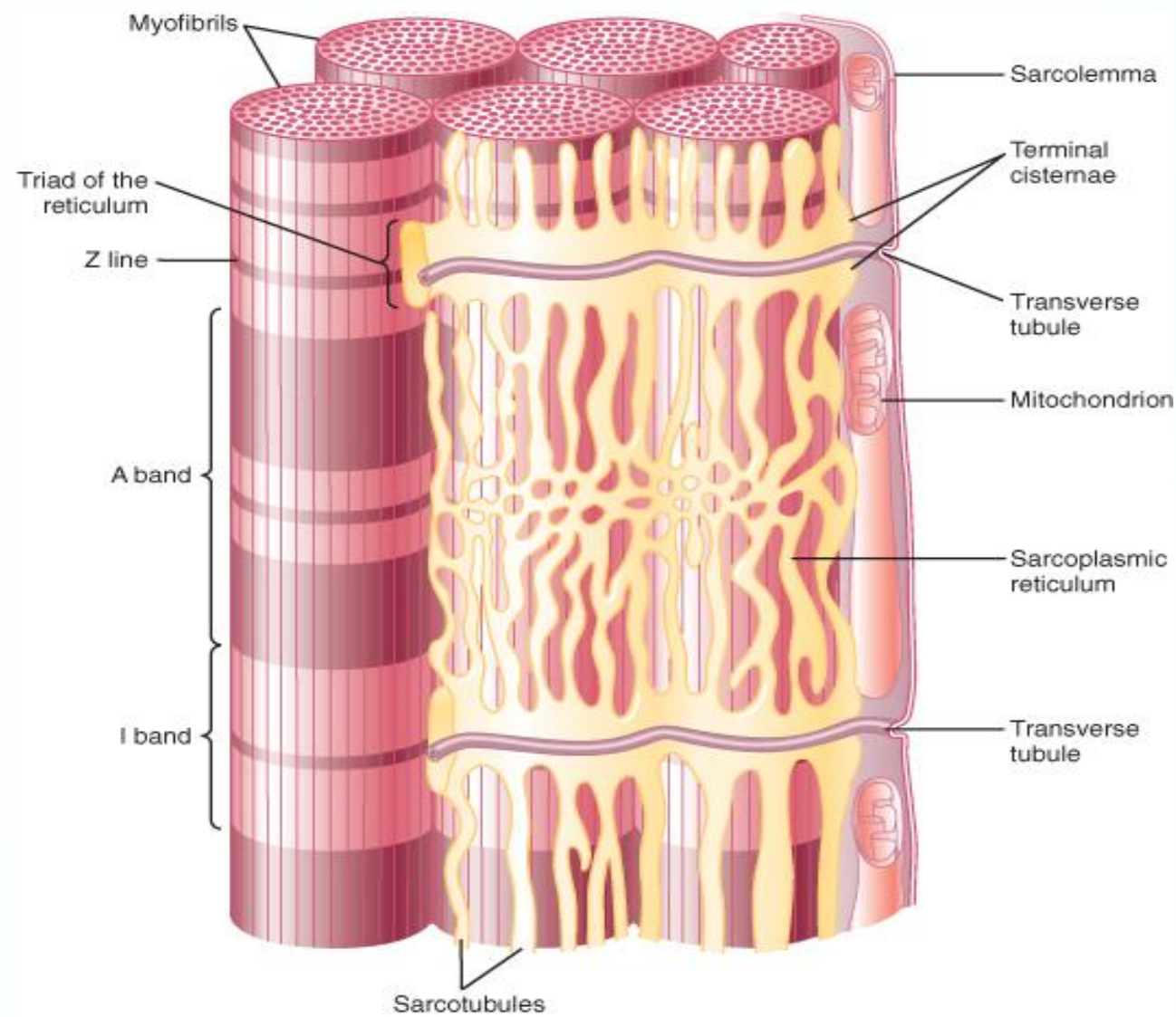
Inotropic effect means can produce a contraction

Strong stimulation of the vagi:

- Stop completely the rhythmical excitation by the S-A node
- Block completely transmission of cardiac impulses from the atria to the ventricle
- Some point in the Purkinje fibers develops a rhythm of its own (**Ventricular Escape**)

Second : Contraction

(Contractile cells or Myocytes or cardiac cells)



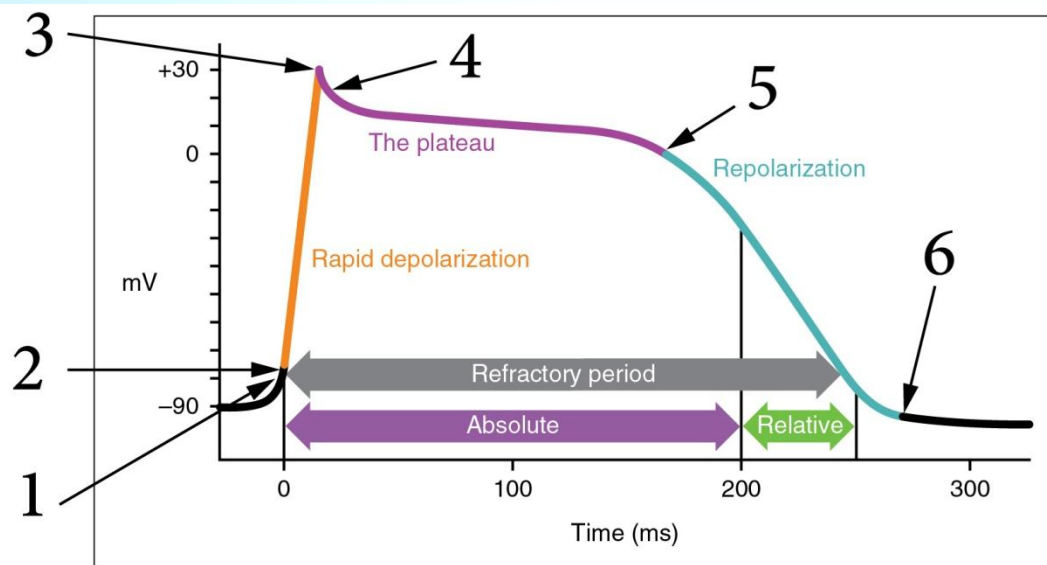
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Cardiac Muscle APs

It's controlled by **Voltage-Gated channels (Na⁺,Ca²⁺,K⁺)^{*}**:

Cardiac Muscle APs



- 1- Na and Ca influx while K efflux through gap junction (-70 mV)
- 2- Fast sodium channels opened (Na influx) and cause **rapid depolarization (+20 mV)**
- 3- Sodium channels closed and K efflux which cause **partial repolarization (5-10 mV)**
- 4- Slow calcium channels opened (Ca influx) and K remain efflux which cause **plateau (0 mV)**
- 5- Slow calcium channels closed and K efflux which cause **repolarization.**
- 6- Rest membrane potential (-85 mV)

Note:

Duration of cardiac muscle action potential is = **0.4 sec**

What causes the Plateau (state of little or no change of the curve) in the Action Potential?

1. Slow calcium channels: (MAIN CAUSE)

-Large quantity of calcium ions flow to the interior of the cardiac muscle fiber. (0.2 sec)

-Maintains prolonged period of depolarization (because permeability of membrane to K⁺ is reduced -> delay repolarization) .

2. Decreased permeability of the cardiac muscle membrane for potassium ions

that result from the excess calcium influx:

-decrease outflux of potassium ions during the action potential plateau.



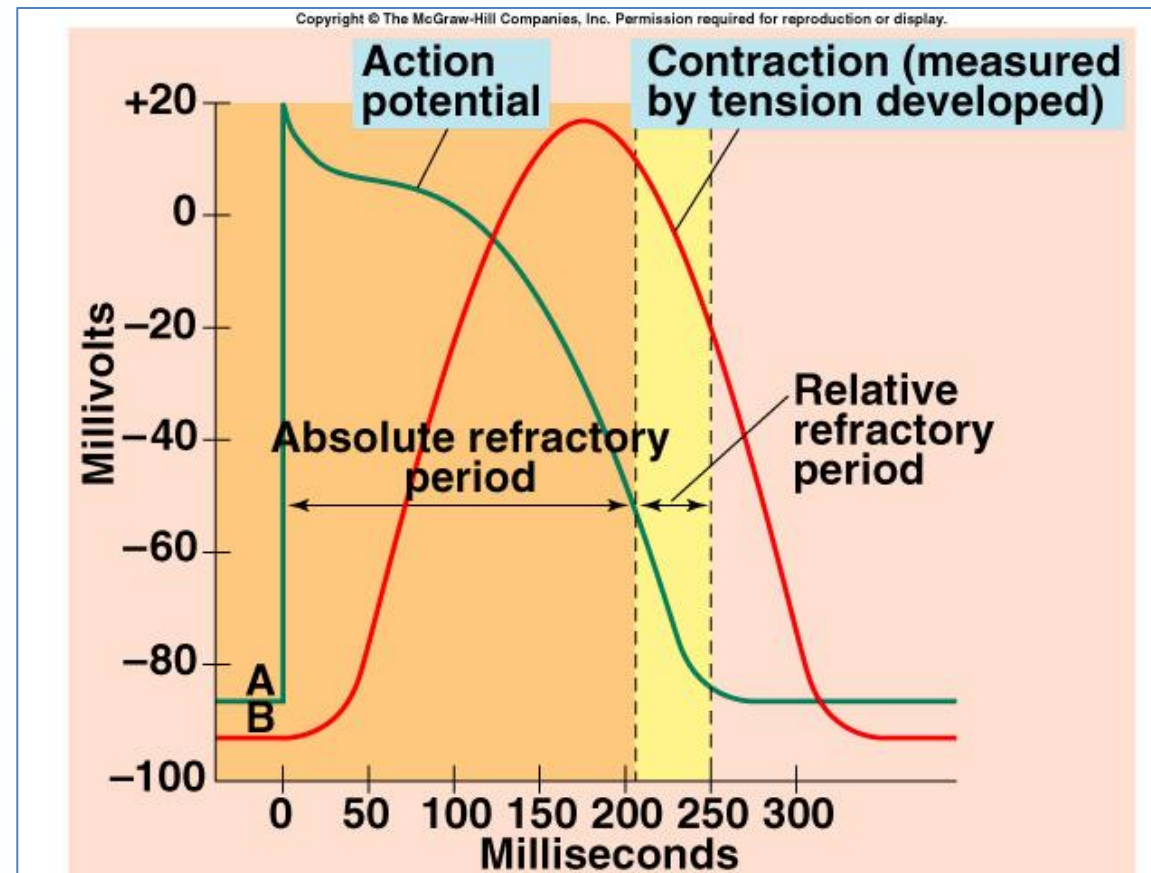
<http://www.youtube.com/watch?v=rIVCuC-Etc0>
<http://www.youtube.com/watch?v=5SsPyjatG4U>

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Refractory period for cardiac muscle

When is the cardiac muscle refractory to stimulation?

During the AP



Absolute refractory period

- Cardiac muscle cannot be excited while it is contracting, Why?
To prevent **summation and tetanus**, via repeated stimulation
- Time: depolarization and 2/3 repolarization

Relative refractory period

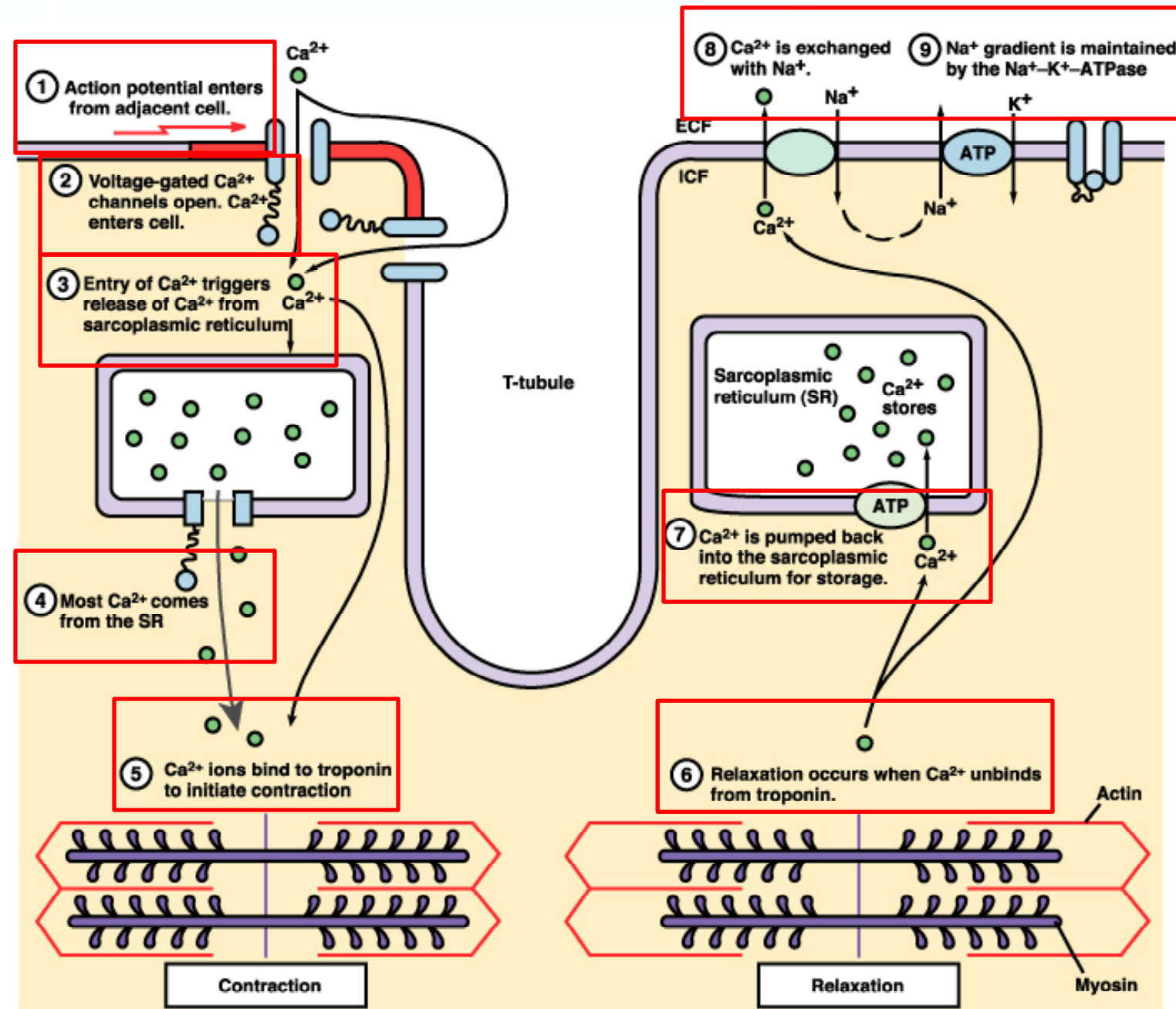
- Cardiac muscle can be excited by strong stimulus
- Time: 1/3 repolarization

Note:

Summation and tetanus = a lot of Action potentials at same time will lead to tetanus.

Excitation-contraction coupling

It is the mechanism by which the action potential causes muscle contraction



Excitation-contraction coupling

(How the AP causes contraction of the cardiac muscle)

1. The AP spreads to the **interior** of the **Myocytes** by transverse-tubules
2. These T-tubules attach to the **terminal cisternae** of the sarcoplasmic reticulum (forming the T-tubule-sarcoplasmic reticulum system)
3. AP travels along T-tubules: Ca^{2+} is released from T-tubules and SR into the sarcoplasm
4. Ca^{2+} diffuses into **myofibrils**
5. Binds to **troponin** → sliding of actin & myosin filaments over one another
6. **Contraction** of cardiac muscle

When the slow Ca^{2+} channels close, Ca^{2+} is pumped back into T-tubules and SR ending the contraction (this coincides with repolarization)

Excitation-contraction coupling

The T tubules of cardiac muscle have a diameter **5 times as great as** that of the skeletal muscle tubules.

The strength of contraction of cardiac muscle depends to a great extent **on the concentration of calcium ions in the extracellular fluids**

Each contraction involves the **hydrolysis of an ATP** molecule for **the process of contraction and sliding mechanism**

Cardiac muscle are continually contracting and require **substantial amounts of energy**

The energy is derived from ATP generated by **oxidative phosphorylation** in the **mitochondria**

The myocytes contain **large numbers of mitochondria**

The Contractility of the Cardiac Muscle

Contractility is the force of contraction of the heart. It is essential for the pumping action of the heart

INOTROPIC EFFECT: MECHANISM THAT AFFECT THE CONTRACTILITY



Positive Inotropic Effects: factors that increase the cardiac contractility

1. Sympathetic stimulation
2. Calcium ions
3. Catecholamine



Negative Inotropic Effects: factors that decrease the cardiac contractility

1. Parasympathetic stimulation
2. Acetylcholine
3. Vagal stimulation

Questions

Q1: Why resting state of SA node is -55 mV instead of -85 mV ? And what is the result ?

The fast sodium channels are inactivated **ONLY** the slow sodium channels can be activated

Q2: Why does The atria contract about one sixth of a second ahead of ventricular contraction ?

To allow filling of the ventricles before they pump the blood into the circulation.

Q3: Why do we call SA node "Pacemaker of the heart" ?

1-Its rate of rhythmic discharge is **greater** than any other part in the heart

2-Highest frequency

Q4: Why Positive sodium ions leak from the outside of the fibers to the inside in SA node?

1. High sodium ion concentration in the extracellular fluid outside the nodal fibers

2. Already open sodium channels

Q5: Why Atrioventricular node is slower than Sinoatrial node?

because **diminished** number of **gap junctions**

Q6: What is the cause of Self-Excitation of S-A node?

Sodium and calcium leakage to interior of the cell.

Q7: Purkinje fibers transmit action potentials at a very high velocity (0.1-4.0 m/sec) , what's the reason behind that ?

It has **very high permeability of gap junctions** so , ions are transmitted easily from one cell to the next that will lead to enhance the velocity of transmission

Q8: What is the definition of *Ectopic pacemaker* ?

a pacemaker elsewhere than the sinus node .

Q9: Parasympathetic nerves (vagi) mainly supply ?

SA node & AV node

Q10: Sympathetic nerves strongly supply ?

Ventricles

Questions

Q11: Strong stimulation of the Vagus nerve will lead to ?

Ventricular Escape (Purkinje fibers develops a rhythm of its own)

Q12: Work as a membrane separates the cardiac muscle cells ?

Intercalated disks

Q13: AP can travel from one cardiac muscle to the adjacent cardiac muscle through ?

Gap junctions which is part of intercalated disk

Q14: Fast diffusion of sodium ions can be observed as which phase ?

Rapid depolarization

Q15: Diffusion of calcium ions occurs at which phase in cardiac muscle? And why?

Plateau. To prolonged period of depolarization.

Q16: Repolarization is triggered by?

Potassium outflux

Q17: Cardiac muscle can be excited by strong stimulus at/in which period ?

Relative refractory period "Repolarization period"

Q18: Action potential spreads to the interior of the cardiac muscle fiber along ?

T – tubules

Q19: Release of calcium ions from sarcoplasmic reticulum triggered by?

The diffused calcium from extracellular fluids.

Q20: What is contractility means? And what is the benefit of it?

the force of contraction of the heart. It is essential for the pumping action of the heart

Q21: Why the heart has Automaticity ?

Because SA node can produce AP by itself **(Without stimulus)**

Q22: Why refractory period in cardiac muscles is longer than skeletal muscles?

To prevent summation and tetanus, via repeated stimulation

MCQs

Q1: According to cardiac muscle, which one is separate cell from one another:

- A. Intercalated discs
- B. Gap Junctions
- C. Sarcoplasmic reticulum
- D. Cytoplasm

Q2: Duration of cardiac action potential in cardiac muscle:

- A. 0.05 sec
- B. 0.5 sec
- C. 0.4 sec
- D. 0.04 sec

Q3: The action potential plateau phase of the cardiac muscle occurs when:

- A. voltage-gated Ca²⁺ ion channels open
- B. voltage-gated K⁺ ion channels open
- C. voltage-gated Na⁺ ion channels open
- D. both b and c

Q4: At rapid depolarization phase of the cardiac muscle occurs when:

- A. voltage-gated Ca²⁺ ion channels open
- B. voltage-gated K⁺ ion channels open
- C. voltage-gated Na⁺ ion channels open
- D. voltage-gated Na⁺ ion channels close

Q5: In Repolarization phase of the cardiac muscle:

- A. voltage-gated Ca²⁺ ion channels open
- B. voltage-gated K⁺ ion channels open
- C. Back to RMP
- D. Both b and c

Q6: Ones an Ectopic pacemaker happen where a rhythmical discharge rate develops?

- A-sinus node
- B-A-V node
- C-Purkinje fibers
- D-Both B&C

Q7: During absolute refractory period, the normal cardiac impulse :

- A. Re-excite
- B. Cannot Re-excite
- C. Inhibition of Action potential phase
- D. Both B & C

Q8: During Excitation – Contraction of cardiac muscle, which one of these well happen firstly:

- A. Ca²⁺ binds to troponin
- B. Calcium ions diffuse into the myofibrils
- C. Sliding of actin and myosin filaments
- D. Release of calcium ions from sarcoplasmic reticulum

Q9: Mechanism that increase the contractility:

- A. Parasympathetic stimulation
- B. Acetylcholine
- C. Sympathetic stimulation
- D. Vagal stimulation

Q10: One of Specialized Excitatory and Conductive System of the Heart is Highest frequency:

- A-The Atrioventricular node
- B-Purkinje fibers
- C-Sinoatrial node
- D-The internodal pathway

Q11: What a function of purkinje fibers?

- A-Stop completely the rhythmical excitation
- B-Transmit action potentials at high velocity
- C-Pacemaker_of the Heart
- D-Blockage of transmission of the cardiac impulse

**Answers: 1-A 2-C 3-A 4-C 5-D
6-D 7-B 8-D 9-C 10-C 11-B**

WORK HAS DONE BY :

- 1- Mojahed Otayf
- 2- Ahmed Al Hussein
- 3- Mohaned Al wabel
- 4- Abdulmalek Al Qhtani
- 5- Khalid al Nasser
- 6- Hamad Al Dossari
- 7- Suliman Al Suliman

REVISED BY :

Dahna Alkahtani