

1st Lecture

CARDIAC CONTRACTILITY

Physiology Team - 430

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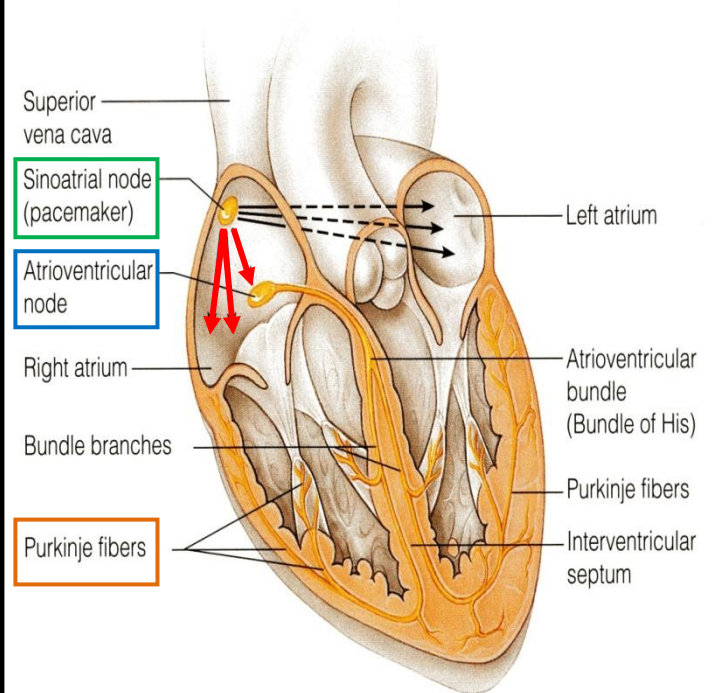
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For You :

- **sinoatrial (SA) node** : located in the right atrium → generate the impulse
- **Atrioventricular (AV) node** is located at the junction of the atria and the ventricles → second generate of the impulse
- the impulse Transmitted from SA to AV by **3 connection** called : **internodal connection**.
- **Purkinje fibers** are located inside the walls of the ventricles (**penetrate myocardium**)

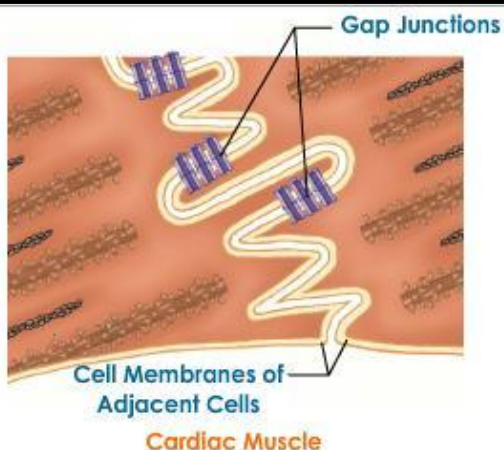


Three major types of cardiac muscle:

- 1- atrial muscle
- 2- ventricular muscle
- 3- specialized excitatory and conductive muscle fibers

Type 1 and 2 : like SK muscle in contract except duration (**Longer** in cardiac muscles)

Type 3 : contract feebly because contain a few contractile fibrils



How do the cardiac muscle contract?

It transmit impulses between muscle fibers though junctions called **gap junction** ,

Heart contractility :

The ability (force) of heart muscle to contract (shortened). It is the main function of cardiac muscle.

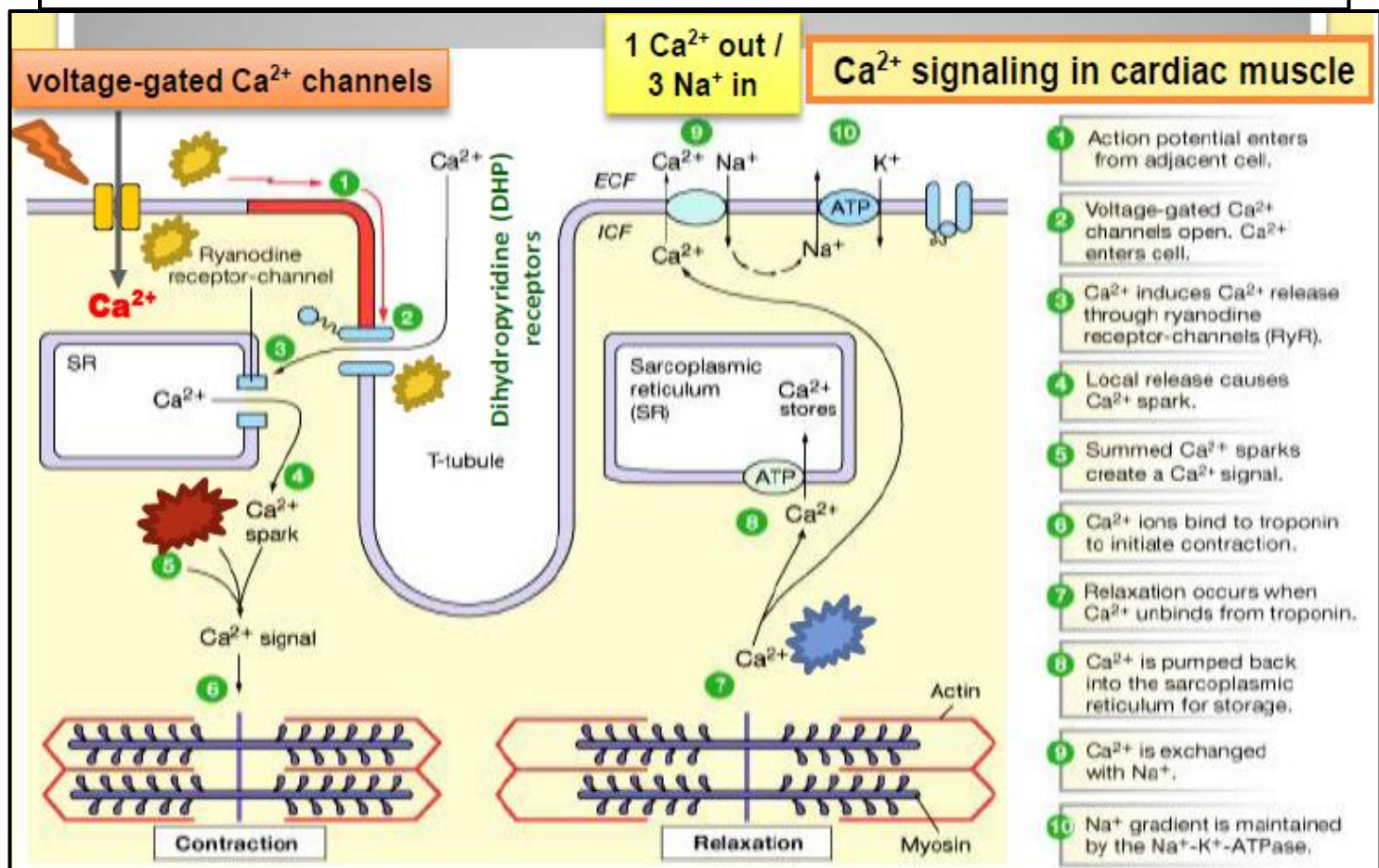
Excitation-contraction coupling :

The mechanism by which action potential (impulse) initiates contraction, cardiac & skeletal muscle have the same mechanism.

This process depends on Ca^{2+} present in extra cellular fluid.

On picture:

The 3 receptors which are the source of Ca^{2+} entry (SR receptor is called RyR, TT's receptor is called DHP, and sarcolemma's receptor is voltage gated Ca^{2+} channels) . **Read the 10 steps.**



In relaxation:

No Ca

3 Na out and 2 K in by ATPase

Cont. Excitation-contraction coupling :

AP passes through TT & myocardium membrane > Ca^{+2} enters through voltage channels and released from SR also TT receptor(DTH) > $\uparrow \text{Ca}^{+2}$ in cytoplasm > binds to troponin > cross bridge occurs > after contraction Ca^{+2} gets back actively to SR.

Factors affecting myocardial contractility (inotropes) :

1- innervation by :

- ANS, look below :

When SNS is stimulated, Noradrenaline & adrenaline levels \uparrow , leading to stimulation of adrenergic receptors that located in the endothelium:

- Alpha1 (α_1), Alpha2 (α_2)
- Beta1 (β_1), Beta2 (β_2), &
- Dopamine (D)

- Action of these receptors :

Alpha Receptors		Beta Receptors		Dopamine
Alpha1 (α_1)	Alpha2 (α_2)	Beta1 (β_1)	Beta2 (β_2)	Dopamine (D)
Cardiac	In peripheral vessels	Heart	<ul style="list-style-type: none"> • Bronchial smooth muscles • Skeletal muscles 	
\uparrow Contractility w/out \uparrow in rate	Mediate vasoconstriction	\uparrow Contractility w \uparrow in HR	<ul style="list-style-type: none"> • Dilation of BSM • Vasodilation in SM • ? Some cardiac effects 	<ul style="list-style-type: none"> • \uparrow Renal & \uparrow Coronary blood flow • Arterial vasodilatation

- ANS actions as following :

- Sympathetic NS \uparrow force of contraction
- Parasympathetic NS (vagus) \downarrow atrial force of contraction w no significant effect on ventricular muscle.

2- Oxygen supply:

Hypoxia contractility

3- $[Ca^{2+}]$ & $[K^+]$ ions in ECF:

- $\uparrow Ca^{2+}$ in extra cellular fluid \uparrow contractility (more Ca^{+2} entrance the more contraction will occur.
- $\uparrow K^+ \downarrow$ contractility

4- Physical factors:

- Warming \uparrow contraction. **How?** Increase substances movement.
- Cooling \downarrow contraction. **How?** Substances freeze \rightarrow no movement of Ca^{+2} or K^+

5- Hormonal & chemical factors:-

Positive (\uparrow Cardiac Contractility)	Negative (\downarrow Cardiac Contractility)
Digoxin, digitalis	Beta blockers (β blockers)
Adrenaline & Noradrenaline	Acetylcholine
Dobutamine	Ether
Dopamine	Some bacterial toxins (e.g. diphtheria toxins)
Isoprenaline	K^+
Alkalosis	Acidosis
Ca^{2+}	Ca^{2+} channel blockers
Caffeine	

We can benefit from these inotropes , however these substances may harm the heart :

Benefits	Risk
Improves cardiac performance	↑ Heart rate, causing further deterioration of failing heart pump
Improves myocardial contractility	↑ Myocardial oxygen requirements
↑ Blood pressure	Potentially arrhythmogenic
	Can ↑ ischemia

-- Mechanical factors:

- Cardiac ms obeys 'all or none law'
- Cardiac ms can perform both **isometric** & **isotonic** types of contractions
- Staircase phenomenon
- Cardiac ms can't be tetanized
- Starling's law of the heart

- Cardiac ms obeys all-or-non-rule :

Cardiac ms respond to threshold stimulus and what's above it, subthreshold can not get the muscle contracted, however during contraction muscle does not respond to any other stimulus.

- **Isometric contraction** : occurs when no work is done by muscle, no change in length.

- **Isotonic contraction** : occurs when work is done by muscle and it's able to shorten itself

Staircase phenomenon: (to understand)

After brief rest, on stimulation at regular frequency, the force of contraction ↑ progressively to a maximum & then is maintained at a plateau

What are the causes of stair case phenomeno

↑ Ca accumulation

↑ Temp.

- Starling's law :

It states that the force of a muscle contraction is proportional to its initial length , initial length depends on diastolic volume
EDV= 130 ml (normally) which is the venous return.

The c.muscle **relaxes** within limits in pre-load state.

-↑ As the pre-load → ↑ the tension on muscle

Length-Tension Relationship :

Passive tension is given by diastolic intraventricular pressure

Active tension is given by systolic intraventricular pressure

Total tension is the **sum** of the parallel elastic tension, i.e.
'passive' & 'active' tension

Good Luck