CVS MODULE
PHYSIOLOGY (LECTURE 2)
Physiology of Cardiac Muscle II

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2024-2025

2. Excitability

Excitability: It means the ability of the cardiac muscle to **respond** to **stimulation**.

The cardiac muscle is **self-excited** by signals **generated** in specific **pacemaker cells** and **conducted** via an **excitatory conductive system** to **generate** an action potential **called (i.e. Cardiac muscle or FAST response Action Potential).**

The **RMP** of the cardiac muscle is ~ **(-90 mV)**.
When the cardiac muscle is **stimulated** → an **Action Potential** is generated which is **responsible** for **initiating** cardiac muscle **contraction**.

Phases of cardiac muscle (FAST response) Action Potential

Phase 0 (i.e. Depolarization phase):

Caused by **rapid depolarization** (i.e. from -90 to +20 mV) and it is **due to** rapid **sodium influx** (***via*** **voltage-gated fast Na^+ channels**).

Phase 1 (i.e. Rapid initial partial repolarization):

A small rapid repolarization, **due to** inactivation (i.e. **closure)** of voltage-gated Na^+ channels **along with** limited **K^+ efflux** due to opening of **transient K^+ channels**.

Phase 2 (Plateau):

In which **repolarization slows down, and membrane potential is nearly sustained about zero mV.**

It is caused by a **BALANCE** between:

Ca^{2+} inflow (i.e. depolarizing Ca^{2+}) due to **opening** of long lasting Ca^{2+} channels **(L-type Ca^{++} Channels)**.

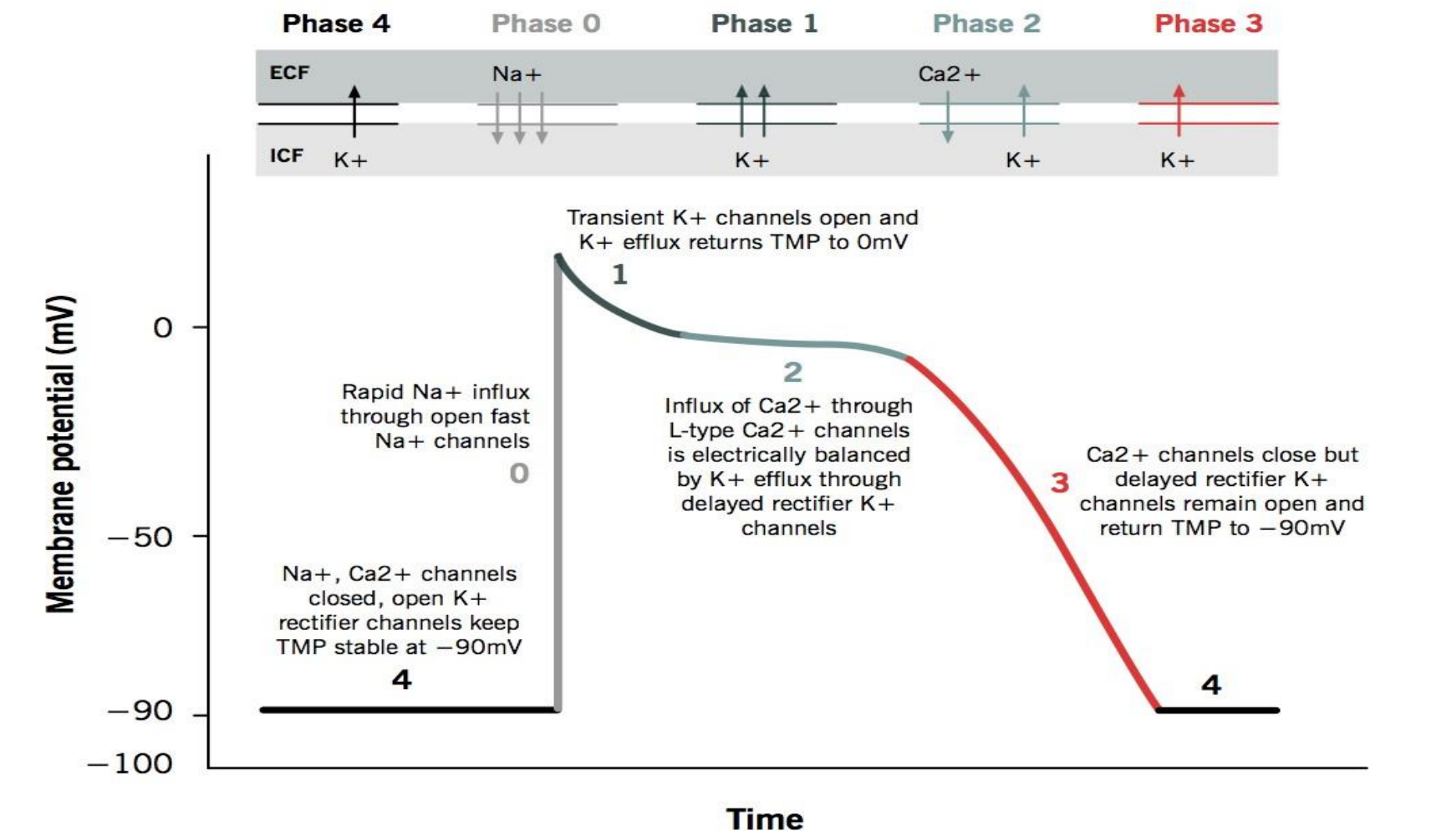
K^+ outflow through K^+ channels.

Phase 3 (Rapid repolarization):

Due to inactivation (i.e. **closure)** of **L-type Ca^{2+}** channels while **K^+ channels** become maximally activated→ ***K^+ efflux*** → **repolarization.**

Phase 4 (complete repolarization and Returning to RMP): This is achieved by increased K^+ efflux.

Cardiac muscle (i.e. FAST response) AP.



Pacemaker potential (slow response)Versus Action potential of the ventricular muscle (Fast response)

Cardiac muscle AP (i.e. Fast Response)	Pacemaker AP (i.e. Slow Response)
• The RMP is ~ -90 mv. • Constant (i.e. stable).	• The RMP is - 55 to - 60 mv. • Unstable (i.e. self-excitation or prepotential).
• The upstroke (i.e. ascending limb; depolarization) is rapid. • It is due to rapid Na+ influx and reaches up • Amplitude: to ~ +20 mV.	• The upstroke (depolarization) is slow. • It is due to slow Ca^{2+} influx (L-type Ca^{2+} channels) and reaches up • Amplitude: to ~ +10 mV.
• There is a prominent plateau (AP is longer 300-400 ms).	• There is NO plateau (AP is shorter 200-250 ms).
• Repolarization is triphasic.	• Repolarization is one phase only

Excitation-Contraction Relationship:

1. The mechanical response (i.e. contraction) of the cardiac muscle **starts** just **after** the beginning of depolarization **(i.e. ~ 0.02 sec.)** and takes **longer time** than the AP **(~ 1.5 time)** as long as the duration of AP.

2. The **systole** reaches its **maximum** at the **end of the plateau** (i.e. **phase 2**).

3. The **diastole starts** with the rapid phase of repolarization (**phase 3**), which is completed at about the **mid-diastole**.

4. The **second half of diastole** coincides with **Phase 4** (i.e. RMP is reached).

Excitability Changes during Cardiac Activity:

I. Absolute Refractory Period (ARP):

The excitability is **completely lost** (= **zero**).

It extends from the **start of phase 0** → **Phase 3** of the AP (i.e. phases 0, 1, 2 till the middle of phase 3).

It occupies the **whole systole and early part of diastole = long ARP.**

Significance of Long ARP:

1. Prevents the heart from being **tetanized which is fatal.**
2. Prevents cardiac **fatigue**.

II. Relative Refractory Period (RRP):

The excitability starts to be **restored gradually** but still **less than normal**.

It extends from the **middle of Phase 3** till the membrane potential repolarizes to **about -75mV.**

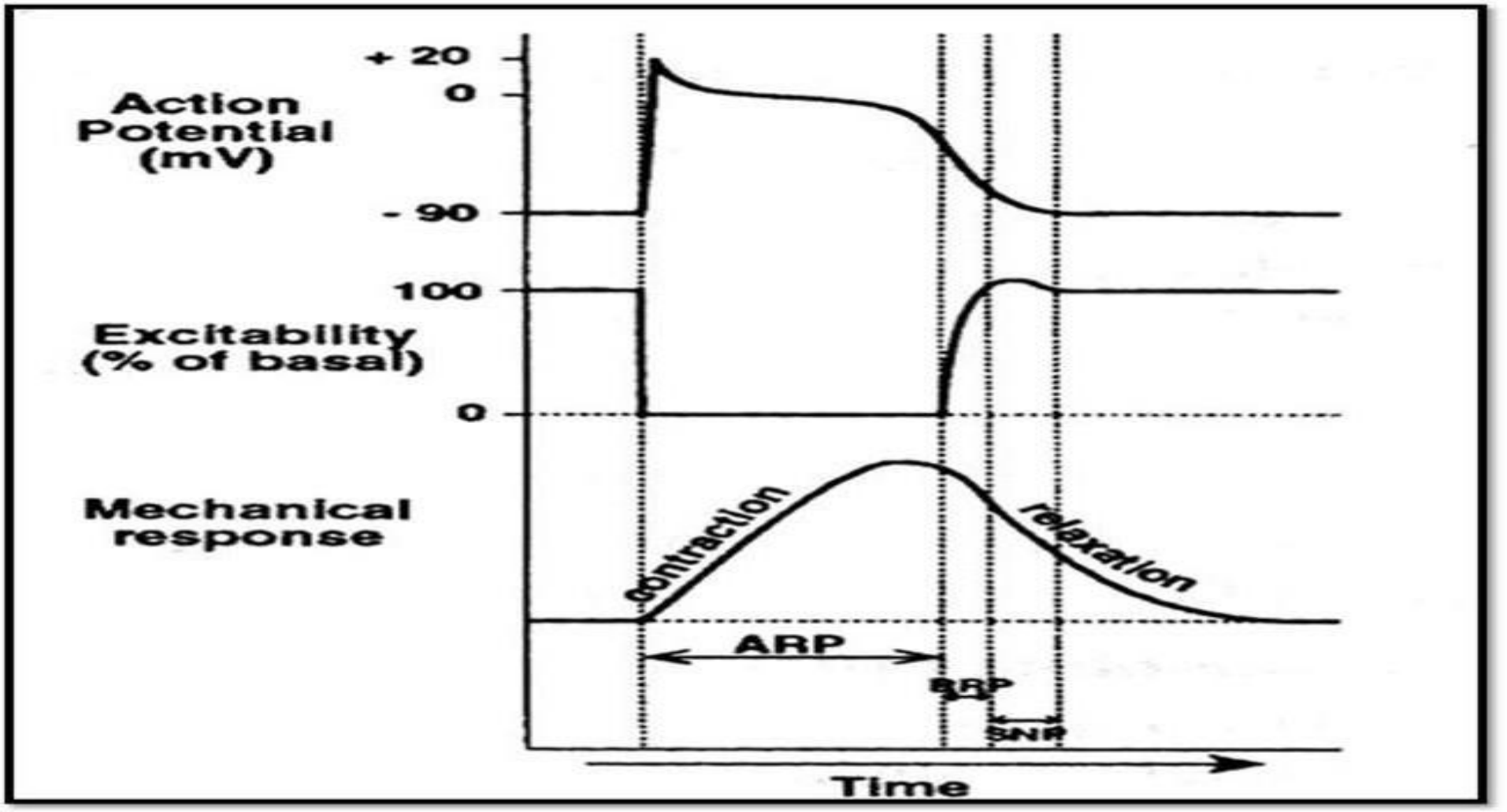
III. Super normal Phase of Excitability:

The excitability is **higher than normal**.

It occurs during **the late part of phase 3.**

Early in this phase, the ventricular muscle is in **vulnerable period of the heart** (i.e. a **dangerous** period in which the **excitation wave** may lead to **cardiac arrhythmia** as paroxysmal ventricular tachycardia or ventricular fibrillation).

Relation between electric response, mechanical response, and excitability changes in the heart



Factors that affect myocardial excitability (Bathmotropism)

1. Nervous factors:

Sympathetic stimulation increases the excitability.

Parasympathetic stimulation decreases the excitability.

2. Physical factors:

An increase in body temperature increases cardiac excitability and vice versa.

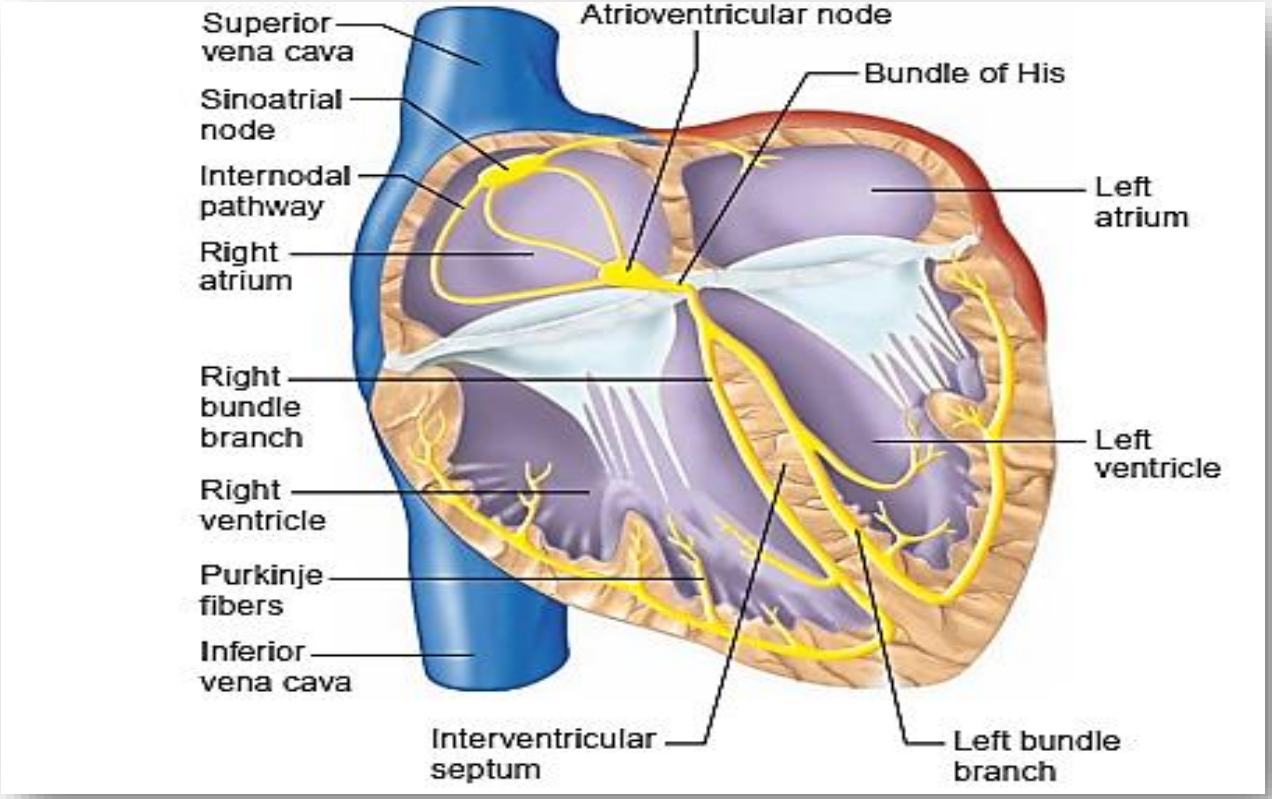
3. Chemical factors:

- Hormones: catecholamines and thyroxine increase the myocardial excitability and may activate ectopic foci.
- Hypoxia and ischemia: decrease the myocardial excitability.
- Drugs: xanthines (e.g. caffeine and theophylline) increase the myocardial excitability, while cholinergic drugs decrease it.
- **Inorganic ions:**

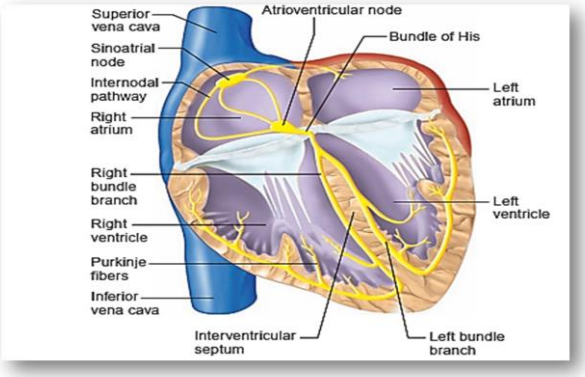
Calcium: hypercalcemia decreases the myocardial excitability and can cause cardiac arrest in systole.

3. Conductivity

It **means** the ability of the cardiac muscle to **transmit** the excitation wave (action potentials) originating in SAN **from** one part of the heart to another **through a highly-specialized conduction system.**



The Cardiac Conduction System



It consists of the following 3 parts

1. The nodal system:

This includes 2 nodes present in the right atrium.
The SAN.
The AVN.

2. The internodal system:

It includes the following 3 tracts (or bundles), which are located in the right atrial wall and consist of:
The anterior internodal tract: this gives an interatrial bundle to the left atrium (Bachmann's bundle.
The middle internodal tract.
The posterior internodal tract.

3. The His-Purkinje System:

It includes the following 3 structures:

- The AV bundle (AVB; bundle of His): It arises from AVN and passes to the ventricles.

It is the only normal muscular connection between the atria and the ventricles.

- The right and left bundle branches.
- The Purkinje fibers: These are fine fibers that arise from both right and left bundle branches.

They convey excitation to the ventricular muscle.

Normal Spread of Cardiac Excitation

1. Sinoatrial (SA) node (NORMAL pacemaker):

Here the initial impulses start → then conducted to the atrial muscle mass **through** the gap junction and to the left atrium **through** the anterior inter-atrial bundle(Bachmann's bundle).

and to → the AVN **through** anterior, middle, and posterior inter-nodal pathways.

The average velocity of conduction in the internodal pathways → one meter/second.

2. Atrioventricular (AV) node (SLOWEST conduction):

The electrical impulses **CANNOT** be conducted **directly** from the atria to the ventricles, because of the **fibrous skeleton**, which is an **electrical isolator**, located between the atria and ventricles.

But there is a DELAY in the conduction occurs in the AV node due to:

- Fewer gap junctions.
- The smaller size of the nodal fiber.

The average velocity of conduction in the AVN → 0.05 meter/second.

Characters of AV nodal conduction

1. One way conduction: the conduction from AVN is a one-way conduction only.

2. AV nodal delay:

Significance:

a. **Allows** atria to empty blood into ventricles during the cardiac cycle **before** the beginning of ventricular contraction.

b. **Protects** the ventricles from the **pathological high atrial rhythm** (to prevent ventricular fibrillation).

N.B.

The **maximum rate of transmission** of impulses through **AV node is ~ 230 impulse/min.**

3. AV bundle (Bundle of His)

It arises from AVN and passes to the ventricles.

It is **subdivided** into: **Right** and **left** bundle: They start at the top of interventricular septum.

4.Purkinje`s fibers (FASTEST conduction):

It is formed of fibers that arise from both right and left bundle branches and spread to all parts of ventricular myocardium.

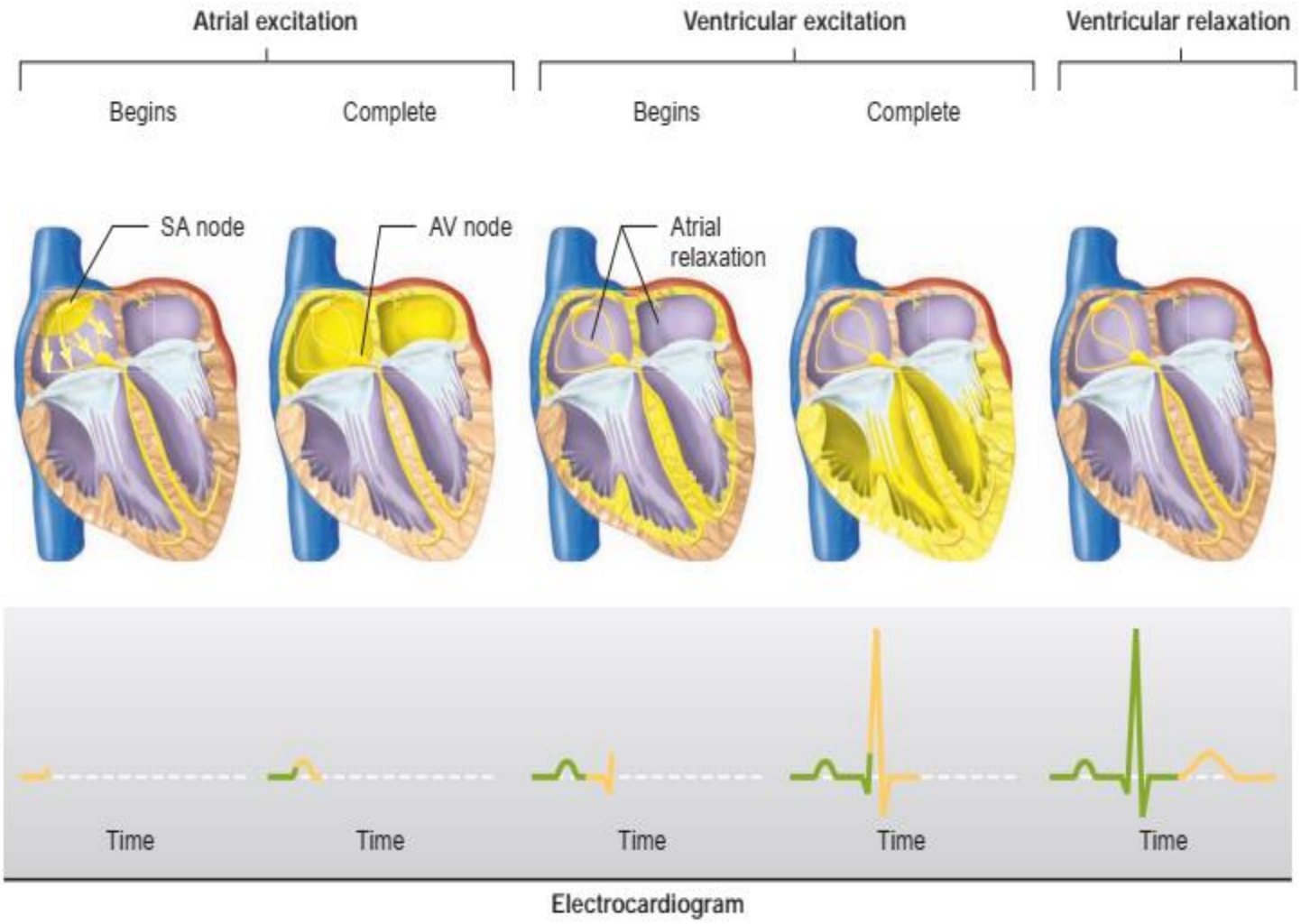
Large fibers with velocity of conduction → **4 meter/second.**

It **allows** spread of excitation wave to the whole ventricles simultaneously and thus contraction of the both ventricles as **one unit**)

The high conduction velocity of these fibers is due to:

The abundant gap junctions.

Their nature as very large fibers.


Atrial excitation
Ventricular excitation
Ventricular relaxation
Begins
Complete
Begins
Complete
SA node
AV node
Atrial relaxation
Time
Time
Time
Time
Time
Electrocardiogram

Factors affecting cardiac conductivity (i.e. Dromotropism):

I. Positive (+ve) dromotropic factors:

1. Nervous:

Sympathetic stimulation: it accelerates conduction and decreases AV delay.

2. Chemical:

- **Hormones:** e.g. Catecholamines & Thyroxine.
- **Alkalosis.**
- **Drugs**: e.g. Sympathomimetic.

3. Physical: rise of body temperature accelerates conductivity.

II. Negative (-ve) dromotropic factors:

1. Nervous:

Parasympathetic stimulation (vagal): it decreases conduction (atria) and ↑AV delay and may cause heart block.

2. Chemical:

- **Most of electrolyte disturbances** → ↓ conductivity (especially K^+)
- **Acidosis.**
- **Severe ischemia.**
- **Drugs: e.g.** cholinergic drugs, Digitalis.

3. Physical: decreased body temperature.

Myocardial Conductivity Disturbances

Myocardial conduction can be accelerated or decreased.

Decreased conduction:

A) Sinoatrial block.

B) Atrioventricular block (= Heart Block; HB):

According to severity, there are **3 degrees:**

1. First Degree (1st degree HB).
2. Second Degree (2nd degree HB).
3. Third Degree (3rd degree HB; complete HB).

C) Bundle branch block(BBB):

In this case, impulses cannot be conducted through the left branch **(LBBB)** or the right branch **(RBBB)** of the AVB.

It causes activation of one ventricle before the other.

The ventricle with the normal branch will beat **earlier** than the ventricle with the blocked branch. In the latter ventricle, conduction of impulse occurs directly through the ventricular muscle.

Thank You