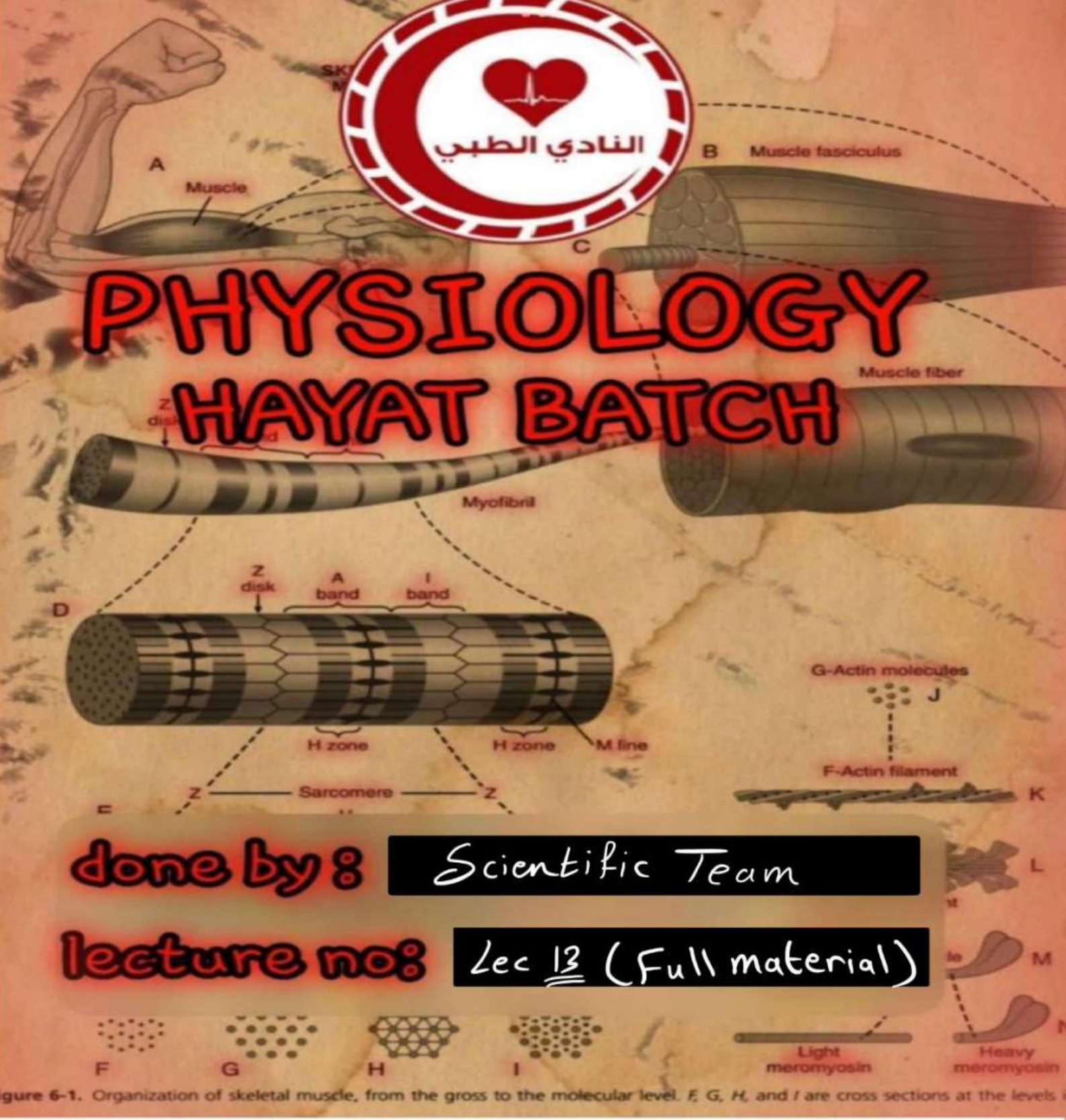


PHYSIOLOGY
HAYAT BATCH

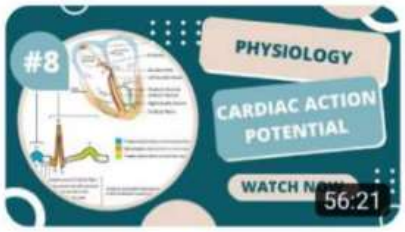
done by : Scientific Team

lecture no: Lec 13 (Full material)

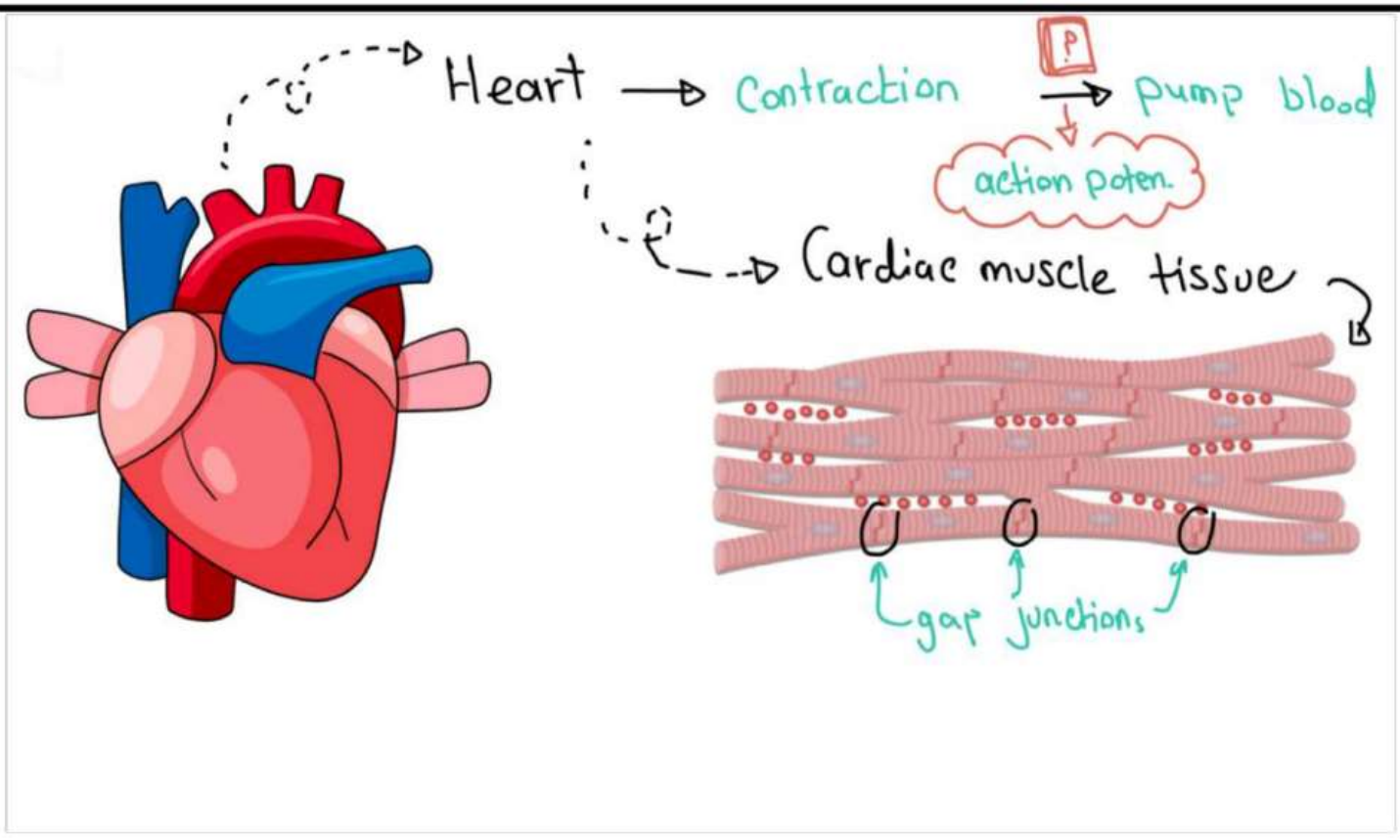
igure 6-1. Organization of skeletal muscle, from the gross to the molecular level. *F*, *G*, *H*, and *I* are cross sections at the levels

Electrical Properties of Cardiac Cells and Cardiac Cells Action Potential

رح نبدأ بداية بسلايدات
عملتهم المبدعة جهينة
ورح تلاقوا لينك فيديو
شرح هاي المحاضرة
بالبوست
موفقين ✨



Physiology || Cardiac Action Potential || By Johainah Taha
Medical Club •



* The Skeletal muscle contraction is stimulated by nervous system

* However, the heart can generate its own electrical stimulation.

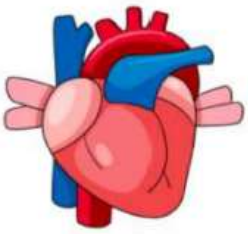
* So, the nervous system can't generate AP in Cardiac cells.

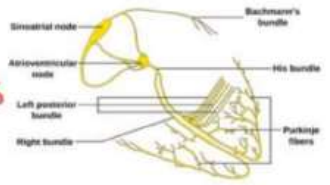
لهيك الأشخاص اللي بموتوا دماغياً قلبهم ما بتوقف عن النبض بل و بتقدر تتبرع منه.

Types of Cardiac cells

NOTE
الـ CNS هو مسؤول فقط عن + الـ - الـ heart beat

- Contrictile
  - Arterial
  - Ventrical
- Autorhythmic ↓ Pacemakers



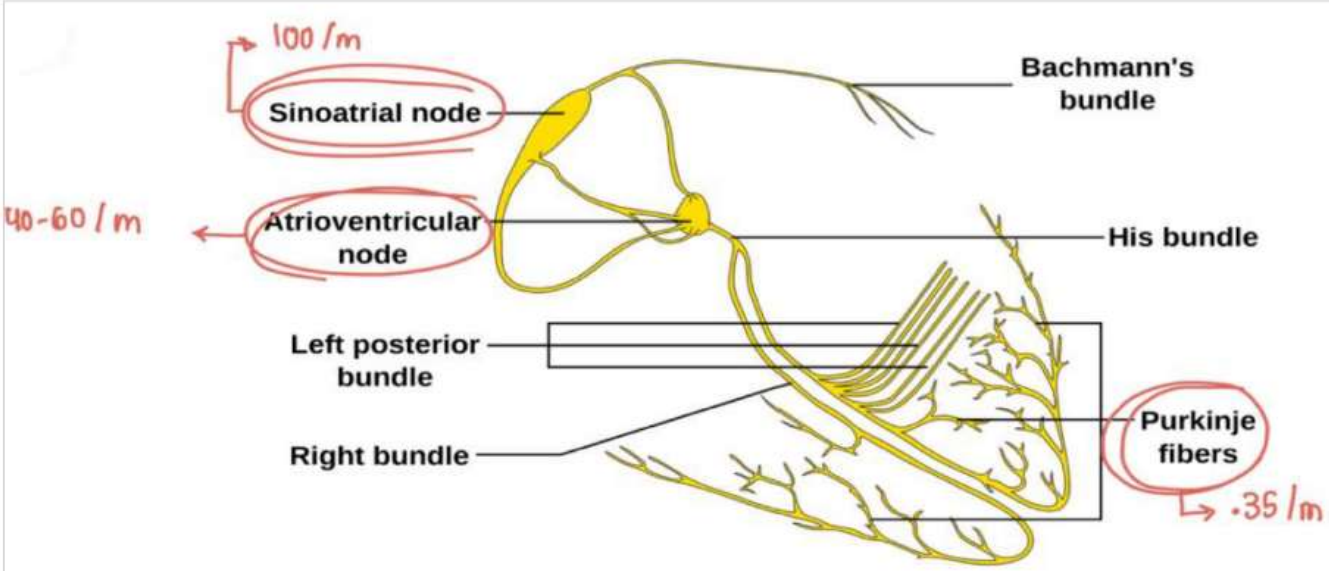


* There is a small group of myocyte called → Pacemaker cell

Impulses من هنا بيلشو تتكون

Pacemaker cell ↓ Conductive System of the heart

* Pacemaker Cells: are modified myocyte, that loose its ability to contract, and become specialized for initiating and conducting A.P.



* SA + AV produce Slow AP.

* Purkinje fibers + other myocyte produce Rapid AP.

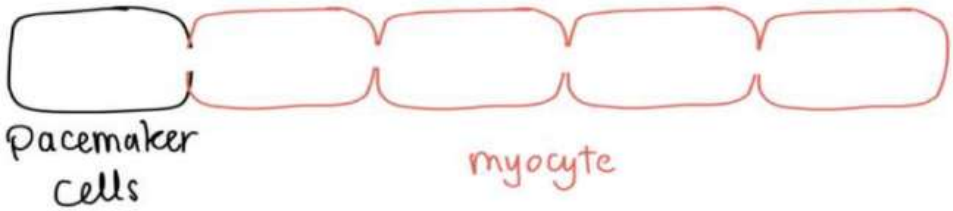
in normal heart :-

SA Node is the pacemaker → arterial muscle
SA Node is the pacemaker → internodal fibers → AV nodes → Purkinje fibers → ventricular muscle

---

* the primary pacemaker of the heart ?? SA nodes
  - initiate all heart beats
  - control heart rate
* if SA nodes are damaged → other parts of the conduction System will take over this rule

---



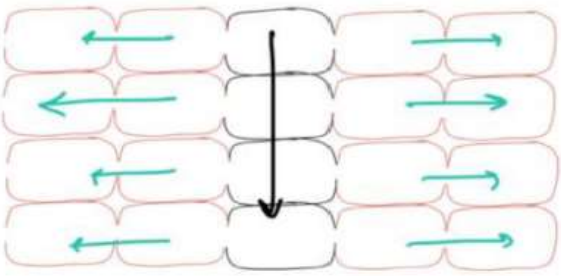
* the pacemakers generate AP → spread through contrictile myocyte of artium
* the myocyte are connected by gap junctions
  - allow ions to flow from one cell to another

rapidly بتنتشر Signals وهيك <=

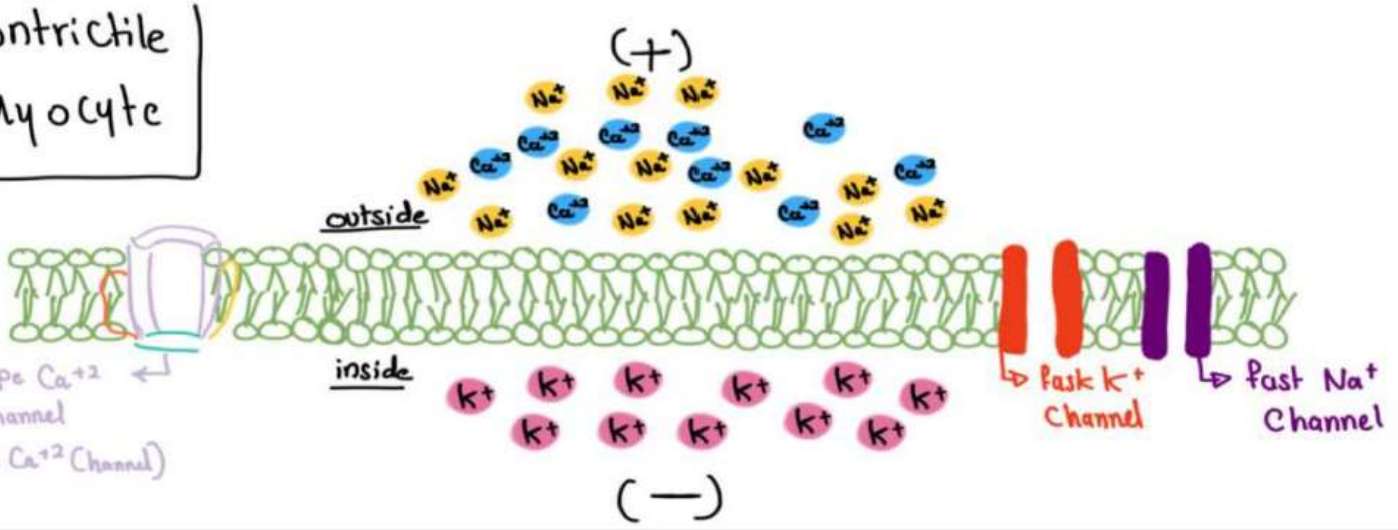
---

* Action Potenial generation and Conduction are essential for all myocyte to act in Synchrony تزامن .
* there are 2 forms of AP.
  - By contrictile myocyte
  - By Pacemaker cells

Next Slides



Contrichile Myocyte



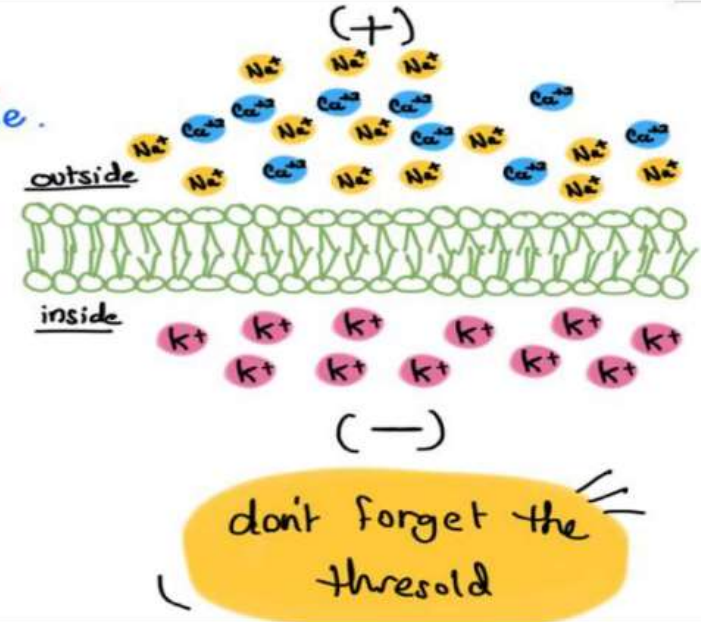
* Resting Phase (4) جهد الراحة

- it is usually negative = ↑(–) inside.

how?

* more Na^+, Ca^{+2} outside.
* K^+ inside.

- average ≈ −90 mv

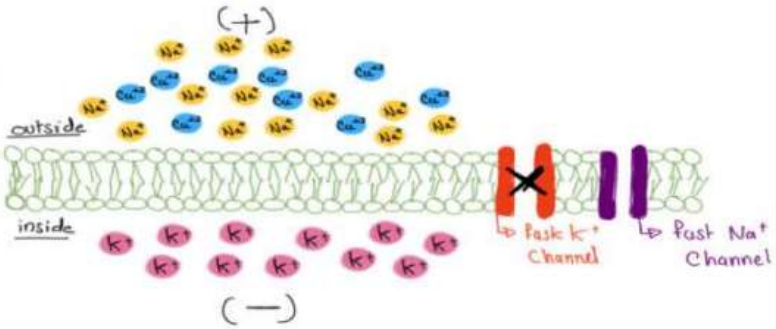


don't forget the threshold

* Phase 0 (depolarization)

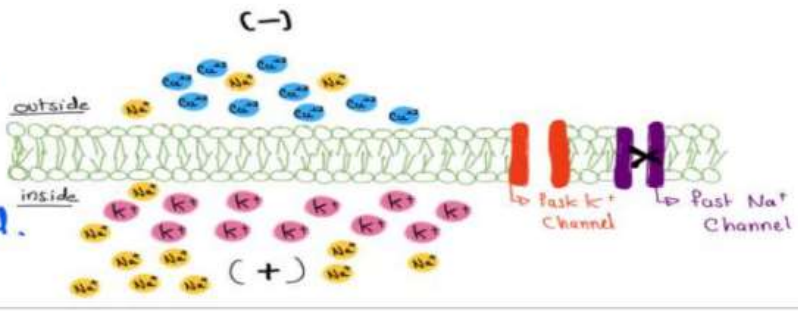
- Stimulation + depolarising (from pace maker cells) ⟶ (+) membrane potential ≈ 20 mv
- opening fast Na^+ Channel (rapid)
  - ↳ rapid flow of Na^+
- when we reach +20 mv

Na^+ fast Channels Closes



* Phase 1 (initial repolarization)

- fast Na^+ Channels close
- the cell begin to repolarize by opening K^+ channels. (slow)
- K^+ starts to leave the cell.



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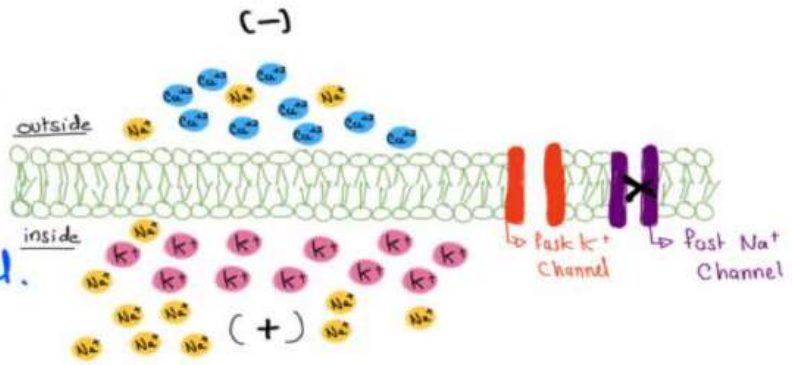
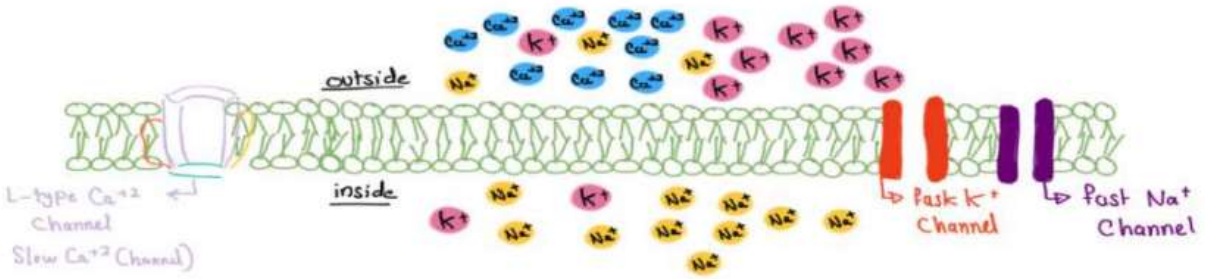


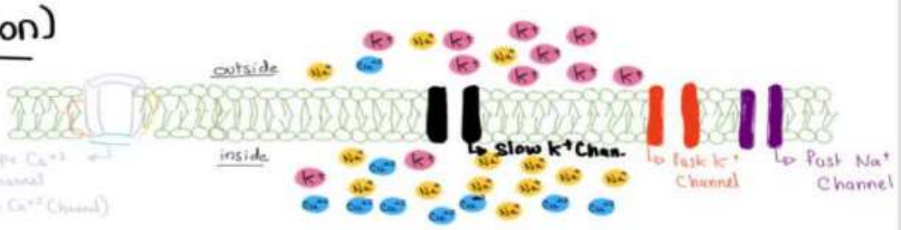
* Phase 2 (Plateau) ≈ Balance stage

- due to opening of L-type Calcium channels (Slow Ca^{+2} channels)
- fast K^+ Channels closes.



* Phase 3 (rapid repolarization)

- Ca^{+2} Channel → Close
- K^+ Channels → open
  - ↳ rapidly K^+ exit the cell
  - ↳ end plateau
  - ↳ return membrane potential to the rest level ≈ −90mv.



* Action Potenial generation and Conduction are essential for all myocyte to act in Synchrony.

* there are 2 forms of AP.

By contrictile myocyte ✓

↳ 1. Phase 0
2. Phase 1
3. Phase 2
4. Phase 3
5. Phase 4

Rapid AP

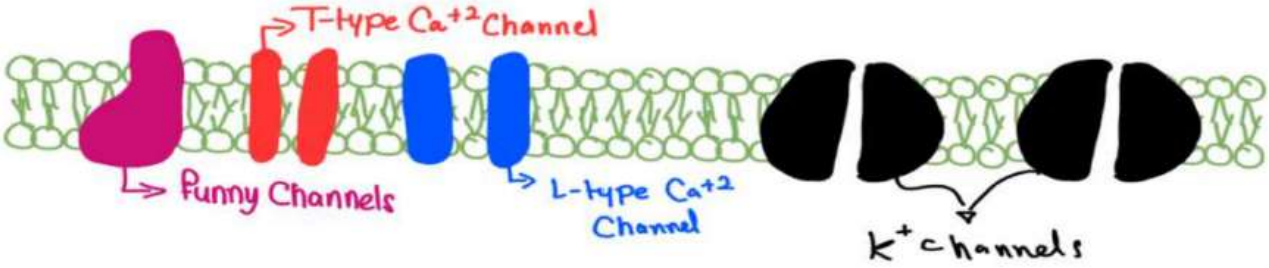
By Pacemaker cells

↳ next slides

* Pacemaker cells don't have a true Resting Potential.

* The pacemaker cells of SA node spont. fire 80 AP/minute

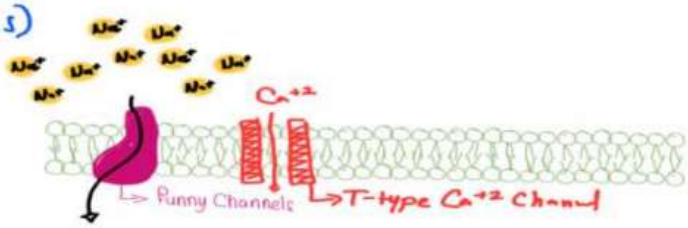
that's why the normal H.R = 80-100 beat/min.



* Pacemaker potential (Phase 4) ≈ -65 mv

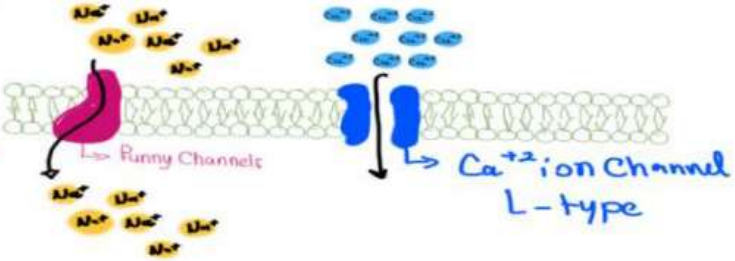
- only in pacemakers.
- opening of funny Channels (f-channels)
- allow slow influx of Na^+
- this depolarization is called Pacemaker potential.

+ T-type Ca^{+2} ion Channel



* depolarization (Phase 0)

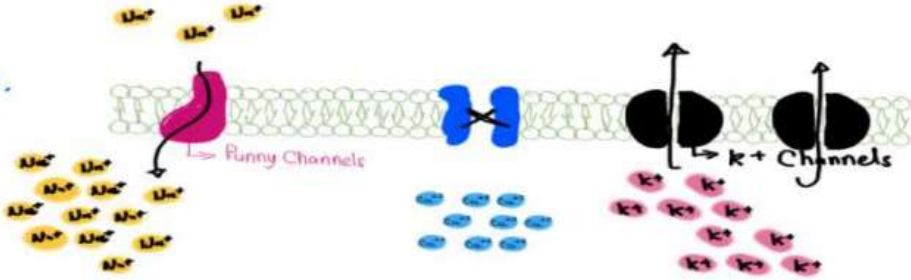
- once the cell's membrane reaches ≈ -50mv
- opening of L-type Ca ion Channel
  - ↳ Ca^{+2} start to flow into the cell.
  - ↳ peak of depolarization
- the voltage will reach +10mv
  - ↳ this happen so fast in milli seconds



2 + 1 lecture *

* Repolarization: (Phase 3)

- starts when we reach ≈ +10 m.v
- Ca^{+2} Channels will be closed.
- K+ Channels open
  - ↳ k+ get out of the cell.



Note

Because there are so many K^+ ion channels relative to funny channels, there is a net outward positive current ions are leaving the cell faster

So the membrane potential will go down again ⇝ −65mv

one heart beat

---

* Action Potenial generation and Conduction are essential for all myocyte to act in Synchrony (ஒத்திசைவு).

* there are 2 forms of AP.

By contrictile myocyte ✓

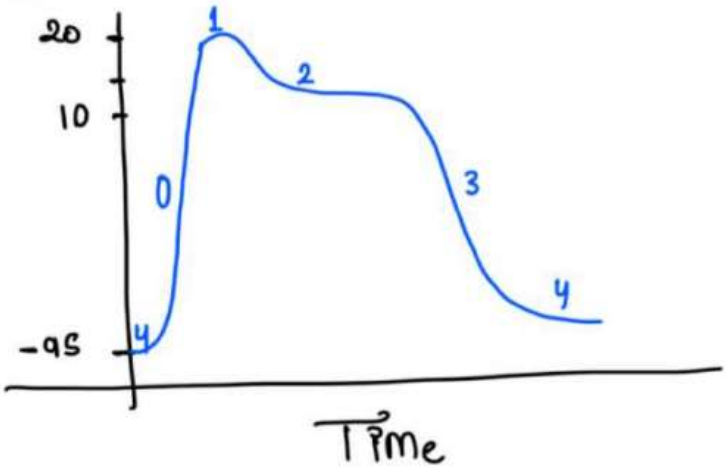
↳ 1. Phase 0
2. Phase 1
3. Phase 2
4. Phase 3
5. Phase 4

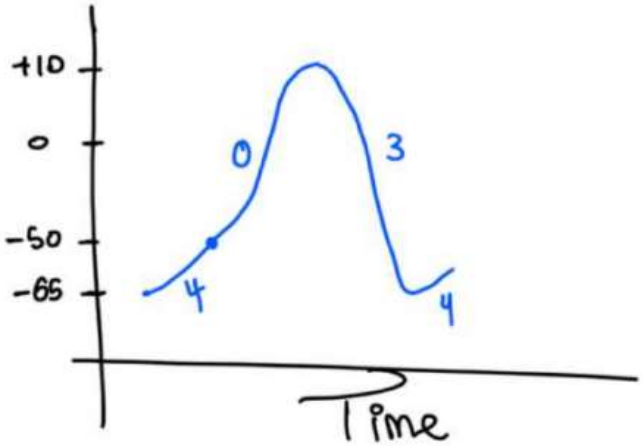
Rapid AP

By Pacemaker cells ✓

↳ 1. phase 4
2. Phase 0
3. phase 3

Slow AP



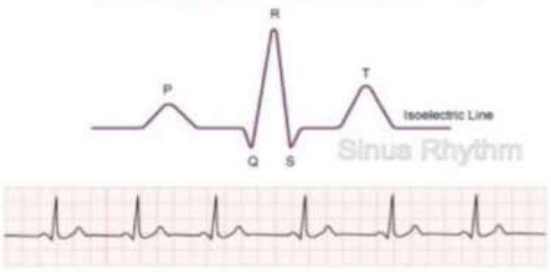


تفريغ الزملاء
لكلام دكتور زهير ✍🏻

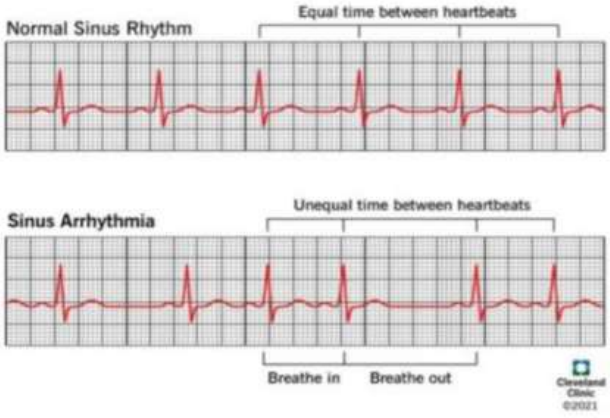
Lecture Objectives

- Outline the component and function of the conduction system of the heart
- Describe the spread of excitation through the conduction system of the heart
- Describe the characteristics of cardiac resting potentials and "fast" and "slow" response cardiac action potentials.
- Identify the refractory periods of the cardiac cell electrical cycle.
- Define threshold potential and describe the interaction between ion channel
- conditions and membrane potential during the depolarization phase of the action potential.
- Define pacemaker potential and describe the basis for rhythmic electrical activity of cardiac cells.
- List the phases of the cardiac cell electrical cycle and state the membrane permeability alterations responsible for each phase.
- Identify the refractory periods of the cardiac cell electrical cycle.
- Describe gap junctions and their role in cardiac excitation
- Compare the action potential of skeletal muscles and cardiac muscle

normal rhythm of the heart هاي الصورة



هاي الصورة توضح الـ arrhythmia



بهاي المحاضرة هي إعادة للمحاضرات السابقة لكن هون الكالسيوم Ca^{2}+ رح يكون اله تأثير في الـ repolarization phase و تكون مدة المرحلة هاي أطول شوي يعني الـ action potential في الـ skeletal muscles بتكون 3mv ، هون تصل 300 و هذا مهم لعدة اسباب ذكرها الدكتور :

١. زيادة سرعة القلب ، ما رح ينبض و يضل ينقبض : المقصود فيها انه القلب عضلة يعني تنقبض و تنبسط و هكذا ، لما يصير tachycardia هون يضل منقبض لفترة زمنية لا يمكن للإنسان انه يلاحظون و سببها اختلال السرعة عن العضلات الأخرى مثل الهيكلية و هون نسميها tetanization

٢. دخول الكالسيوم أثناء الـ action potential للقلب يعمل على enhancing the contractily of heart

الدكتور جاب سيرة الـ arrhythmia : irregular heart beat بس قبل هيك شو يعني rhythm

شو يعني arrhythmia؟؟

لما تاخذ ECG (تخطيط القلب electro cardio gram) و لما تاخذ treatment of anti arrhythmic drugs للناس الي بصير عندهم arrhythmia رح تشوفوا انه كل نوع من الـ anti arrhythmic يشتغل على احد الـ phases تاعون الـ action potential و على منطقة معينة في القلب

Electrical Properties of Cardiac Cells and Cardiac Cell Action Potential

يوضوح

- Cardiac action potentials differ sharply from those of skeletal muscle or nerve in three important ways that promote synchronous rhythmic excitation of the heart

متزامن

بحكيلك انه خلايا القلب تختلف عن خلايا العضلات الهيكلية بمئات الأمور، و هي مسؤولة عن التزامن المنتظم او التحفيز المزمن لخلايا القلب

الله لما خلق الإنسان ، ميز عضلة القلب عن غيرها انها تنقبض من غير محفز خارجي (لو القلب يشتغل بالتحفيز لمات الإنسان من اول دقائق حياته)

القلب يختلف عن باقي الأعضاء خلاياه تولد انقباضه لحاله

- They can be self-generating
- They can be conducted directly from cell to cell via gap junctions

شرحها آخر سلايد

- They have long durations.

جهد القلب 9 300ms 3ms ليس

- Slower conduction velocity (السرعة)

↳ Speed at which depolarization wave spread among myocardial cells.
→ measured in meter/second.

Arterial and ventricle cardiomyocytes form the myocardium جدار عضلة القلب

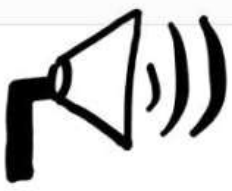
Types of Cardiac Muscle Cell

1. Contractile cells (Atrial and Ventricular)
   - 99% of cardiac muscle cells
   - Contract during the cardiac cycle and pump blood
2. Autorhythmic cells: Pacemaker cells
   - Exhibits spontaneous depolarization
   - Do not contract. Known as (conductive system of the heart).
   - Specialized for initiating and conducting action potentials responsible for contraction of atria and ventricle

مشكلتها slow depolarization

هاي الخلايا عندها intrinsicability لعمل depolarization rhythmically and initiate an action potential

Pacemaker cells : tell the heart to pump



* Cardiac conduction Velocity

→ speed at which depolarization wave spreads among myocardial cells.

→ measured in meter/sec.

* each myocardial structure has a different conduction speed related to it purpose
  - ↳ slowest → AV node [.01 - .05 m/s] → Blood flow from artria
  - ↳ fastest → Purkinje fibers. [2-4 m/s] to ventricles to ventricles

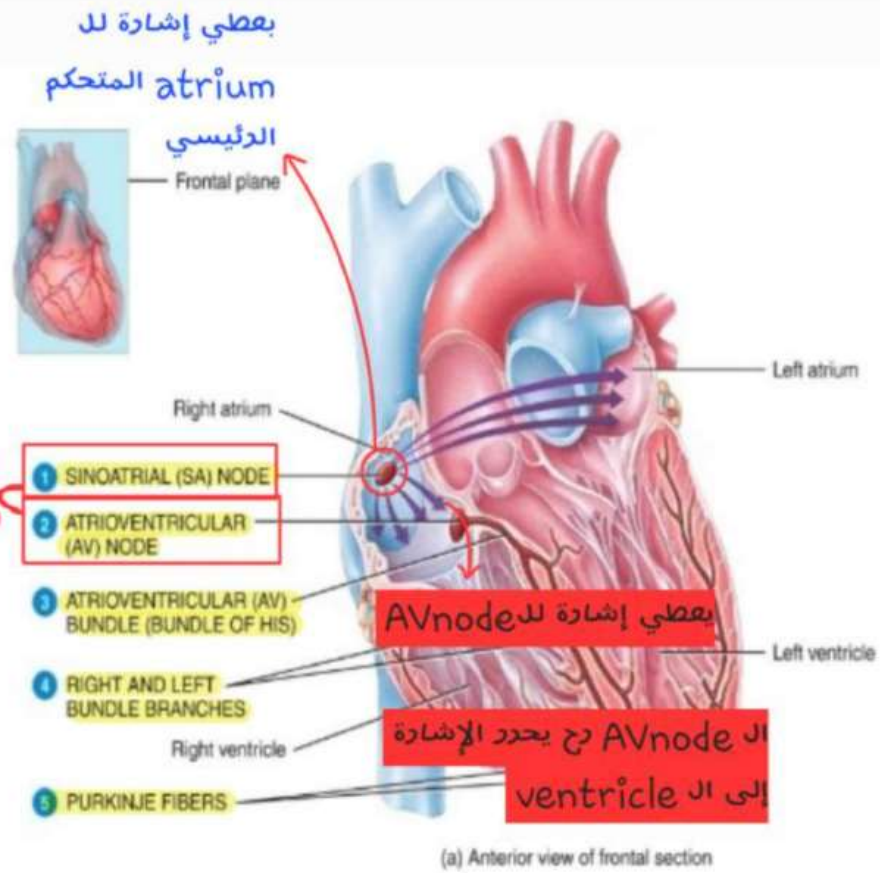
---

هون تشريح القلب موضوع سابق لاوانه لانه رح نوخذه بال (CVS) في ال anatomy لكن مطالبين بحفظ أجزاء القلب ما دام ذكرت معنا

هذا نظام يجعل القلب ينتظم و يدق بسرعة معينة يعني controls the activity of myocardic عضلة القلب , ventricle and atrium

Conduction system of the heart and spread of electrical activity

لما يصير خلل في ال SAnode بصير اسمها ectopic pacemaker بصير القلب غير منتظم و السبب انه يحدث blocks لل signal الي تيجي من SAnode و تدوح لل AVnode ساعتها بصير ال atrium يشتفل لحال و ال ventricle يشتفل لحال و السبب في ذلك هو ventricle يستلم signal من SAnode و لكن AVnode ما رح يستلم اي إشارة من SAnode عشان يمررها إلى ال ventricle لانه صار block و بالتالي AVnode رح يخلي ال ventricle يشتفل على كيفه

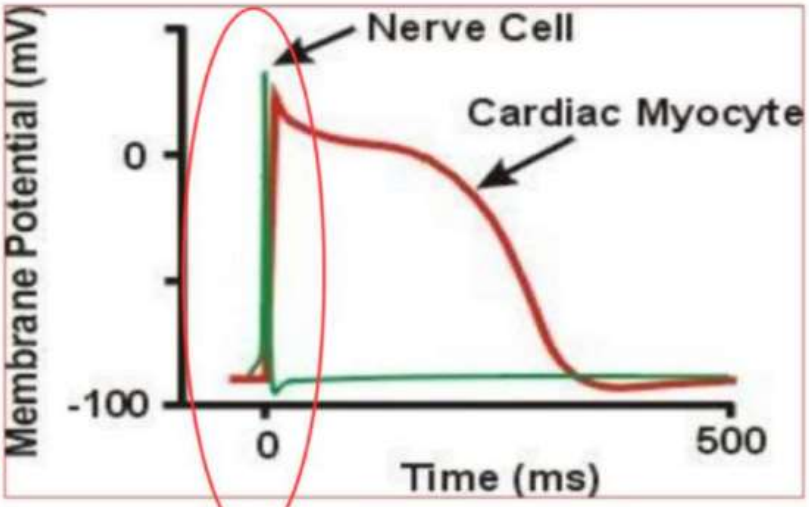


highlight الي عليهم هذول مكونات ال conducting كلهم يظهروا self- excitation لكن SAnode اسرع واحد الي يصير اله excitation عشان هيك هو يتحكم في نشاط ال excitation لباقي المكونات

Conduction velocity of action potentials in cardiac cells

- Action potential conduction velocity in the atrial and ventricular muscle fibers is slow (0.3-0.5 m/s) compared with velocity of conduction in the heart conductive system (4 m/s in Purkinje fibers).
- It is also much slower than in nerve fibers and skeletal muscle fibers.

Action potential in nerve cells and cardiac cells



fast depolarization
الثنين عندهم
لانه عندهم قنوات
صوديوم

هون بحكيلك انه عندنا action potential سريع
يسبب سرعة ال depolarization و هذا تم
شرحه تحت

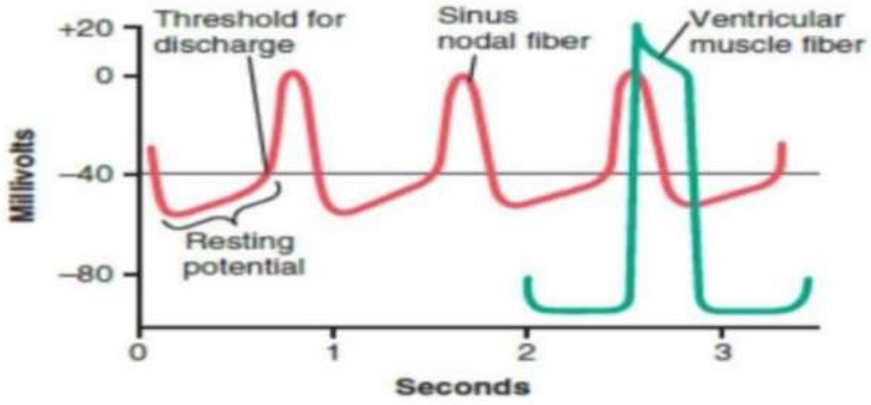
Cardiac Muscles AP

There are two cardiac action potentials produced by myocytes, (خلية عصبية)

1- Fast AP: is characterized by containing a plateau phase. Usually seen in the normal contractile cardiomyocytes.

2- The Slow AP: As seen in the conductive system (for example SA node and Av node cells

هون يكون بطيء لانه ذي
ما حكينا ال pace
بكون ال mackers
depolarization
الهم بطيء بسبب ال
slow current of
Na

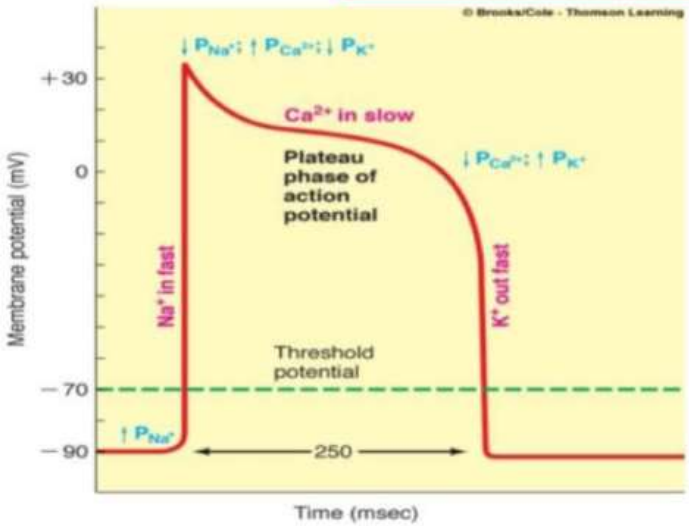


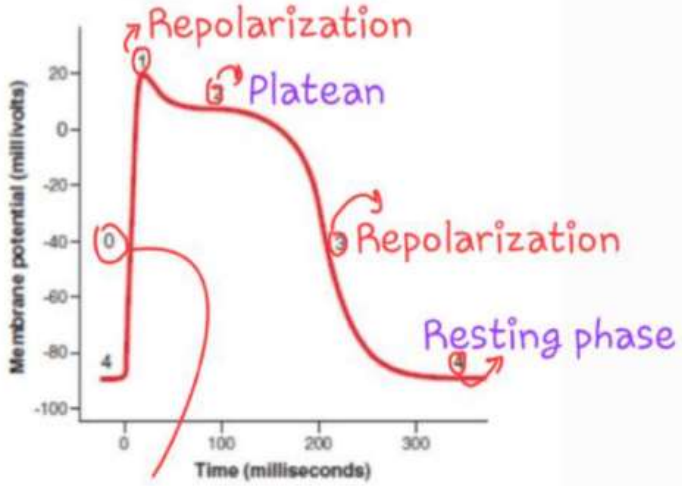
هذا السلايد بس تخلص المحاضرة ارجع اله لانه مراجعة

Cardiac action potentials Ventricular and Purkinje system (Intracellular recording)

Phases of action potentials and its ionic basis of action potential

انتبه للتغيرات الي بتحدث في كل مرحلة





pace macker هو

Cardiac Action Potentials of Ventricular Cells

- *Phase 0 (depolarization)*

طبعا يكون عن طريق ال SA ثم إلى ال AV

When the cardiac cell is stimulated and depolarizes, the membrane potential becomes more positive. Voltage gated sodium channels (fast sodium channels) open and permit sodium to rapidly flow into the cell and depolarize it. The membrane potential reaches about +20 millivolts before the sodium channels close

صون ال membrane potential قريب لل nernst potential تاع ال Na+ بسبب زيادة conductance ال

- *Phase 1 (initial repolarization)fast sodium channels close.*
  - The fast sodium channels close, the cell begins to repolarize, and potassium ions leave the cell through the activation fast potassium channels.

نفاذية الصوديوم تقل و يزيد ال K+

- *Phase 2 (plateau)*

موجودات في ال conducting system في ال myocardic و في ال pace macker (بطيء)

Due to the opening or activation of L-type calcium channels (slow calcium channels), which are also called calcium-sodium channels open and fast potassium channels close. prolong repolarization فتح قنوات L-type عشان تعمل

- *Phase 3 (rapid repolarization)*
  - *calcium channels close and slow potassium channels open.* The closure of calcium ion channels and increased potassium ion permeability, permitting potassium ions to rapidly exit the cell, ends the plateau and returns the cell membrane potential to its resting level.

- *Phase 4 : (resting membrane potential) averages about −90 millivolts.*

فائدة ال prolong phase شفلتين:
١. ال fast activity of SAnode (يعني يبجي متها كثير signals)
وقتها ال heart ما رح يعمل conduction يعني رح يعمل tetanization
٢. في فترة هاي المرحلة يعمل دخول كمية كافية من ال Ca^{2}+ و هذا بدوره
يعمل انه يصير contraction قوي و جيد للقلب
اذا تتذكروا ال digitalis شرح الدكتور وليد كيف دخول الكالسيوم للسيتوبلازم
يحفز القلب للإنقباض

---

* All or None Law

* The refractory period : - when the nerve is refractory to the stimulation
  - to protect the nerve from extremely rapid repitive stimulation.
  - 2 types

Absolute (effective) → firing → early of repolarization

Relative → last part of repolarization phase

Absolute Refractory Period (ARP), Effective Refractory Period (ERP) , and Relative Refractory Period (RRP) In The Ventricle.

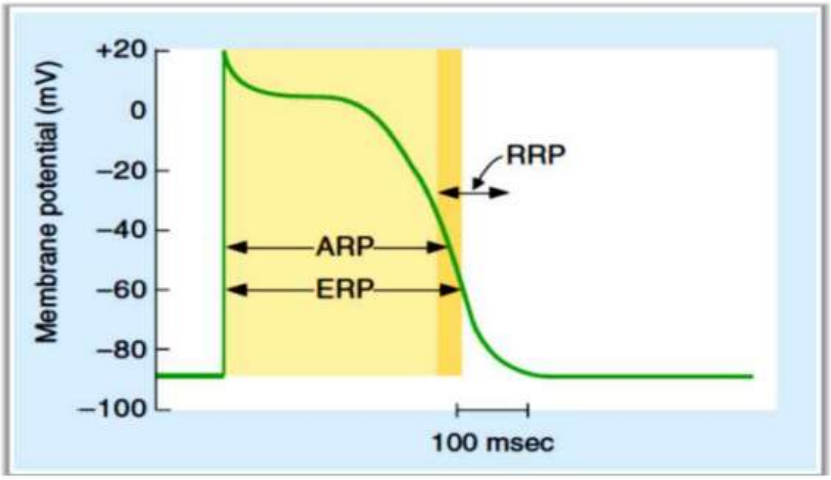
Absolute refractory period (ARP). For most of the duration of the action potential, the ventricular cell is completely refractory to fire another action potential.

– no matter how the Stimulous is

Effective refractory period ERF : means that a conducted action potential cannot be generated (i.e., there is not enough inward current to conduct to the next site)

Relative refractory period (RRP). The RRP begins at the end of the ARP and continues until the cell membrane has almost fully repolarized

- Strong Stimulation



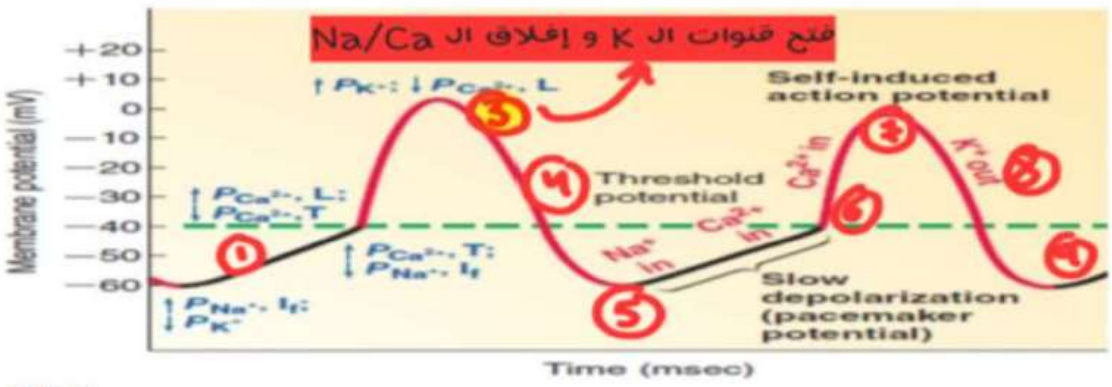
تذكروا الـ Ca^{2+} channels الـ L-type تاعها ممكن يفوت +Na
فبحكوا الهم Na-Ca channel لكن كمية Na و CA لاذم تطلع في
مرحلة الـ plateau عشان هيك في عنا Ca-pump و صوديوم يخرج
من الـ Na-K-ATPase pump

حكى الدكتور انه ما عنده هون phase 0 و لا phase 2 بد يذهب إلى 3 و 4 (يعني من ١-٣)
potential of pace macker (action potential of ventricular) ➡ mainly we have depolarization and repolarization
، في اختلافات بينهم يعني repolarization و depolarization بد نقول يا phase يعني هون لا نتعامل مع

من ١-٣ يكون depolarization بطيء بسبب بطء الـ Ca channel
حتى تزيد من الـ depolarization بدها ايون ثاني

Pacemaker Activity Of Cardiac Autorhythmic Cells

pacemaker potential (prepotential) and action potential of SA node cells



KEY

I_f = Funny channels
T = Transient-type Ca^{2+} channels
L = Long-lasting Ca^{2+} channels

من 5 إلى 9 تكرار لنفس العملية بانتظام و هذا سبب دقات القلب المنتظمة

انشرح فوق

Pacemaker Electrical Activity of Sinoatrial Node (pacemaker potential and action potential)

- **Pacemaker potential**
- The first half of the pacemaker potential is the result of simultaneous opening of unique funny channels, which permits inward Na current, and closure of K channels, which reduces outward K current.
- The second half of the pacemaker potential is the result of opening of T-type Ca ions channels.
- **Action potential**
- Once threshold is reached, the rising phase of the action potential is the result of opening of L-type Ca ions channels, whereas the falling phase is the result of opening voltage gated of K channels

Pacemaker Potential and Action Potential of SA Node Cells Summary

resting يعني ما بعد ال phase

Na channel المقصود هنا

- After hyperpolarization of SA node cells that is caused by activation of K channels , a different channel open that can pass both K and Na is activated
- Because this channel is activated following hyperpolarization, it is referred to as an "h" channel; however, because of its unusual (funny) activation, it has been of this has also been given a nick name (funny channel , f channel)
- As the depolarizing current moves through the h channels increases, the membrane begins to depolarize, forming the first part of the prepotential. Then Transit Ca channels (T Channels are activated
- and completes the prepotential, and the cell reaches the threshold
- At this point L type Ca channels are opened and cause the second depolarization phase of action potential
- Finally, the L type Ca channels close and Voltage gated K channels are activated causing repolarization and slight hyperpolarization

بعد ال hyperpolarization رح تفتح H-channels و من ثم تعاد العملية مرة أخرى؛ هذا هو السبب انه القلب ينبض لوحده و يتحفز لوحده دون مؤثر خارجي حيث أن ال Na channels تفتح بسبب ال hyperpolarization بدل ما تبقى مغلقة و بتدرج تعمل depolarization و كأنه اجا محفز خارجي

pace هون ما بكون فيها macker، يكون بطيء depolarization بسبب ال Na channels تكون بطيئة

في المراحل ما قبل ال plateau يعني phase 0،1 تكون rapid depolarization

Action Potential in SA and AV node

- The action potentials in the SA and AV nodes are largely due to Ca 2+ , with no contribution by Na + influx. Consequently, there is no sharp, rapid depolarizing spike before the plateau, as there is in other parts of the conduction system and the atrial and ventricular fibers.
- In addition, prepotentials are normally prominent only in the SA and AV nodes.
- However, "latent pacemakers" are present in other portions of the conduction system that can take over when the SA and AV nodes are depressed or conduction from them is blocked.
- Atrial and ventricular muscle fibers do not have prepotentials, and they discharge spontaneously only when injured or abnormal

pacemaker will form prepotential before action potential to ال activate it in conduction system

لما يصير عنا block في عنا pacemaker احتياطيين تشتغل في conduction system يتم تفعيلهم

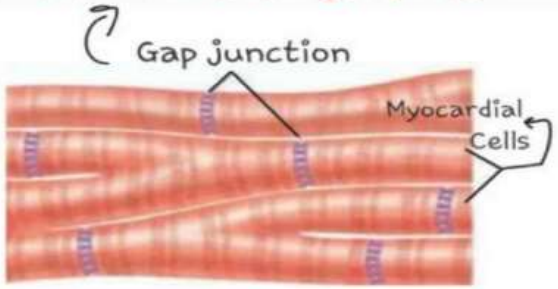
Ventricular and atrial muscle fibers do not have prepotential

يعني مو pacemaker لكن إذا abnormal للقلب رح يضطروا يقوموا لوحدهم و هذا الاشي رح يكون مدمر لانه رح يكون في اختلاف في rhythm كل وحدة

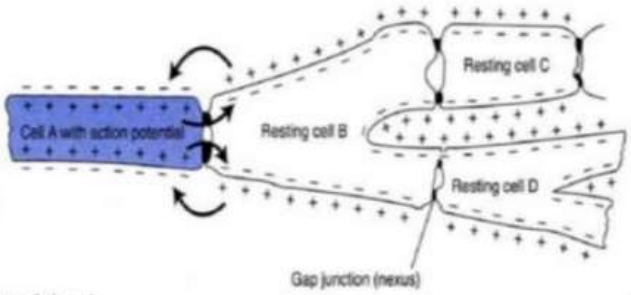
هون الموضوع سهل لانه أخذناه بالهستولوجي

Spread of AP through cardiac muscle

- Spreads very rapidly because of the presence of **gap junctions** between cardiac muscle fibers.
- Gap junctions are permeable junctions and allow free movement of **ions**→ AP spreads rapidly from one muscle fiber to another fiber.

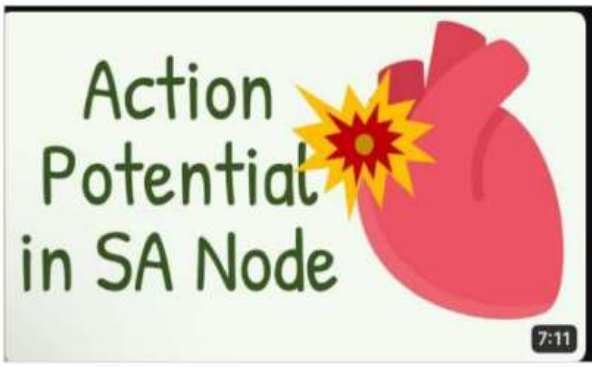


Cardiac muscle is a *syncytium* of many heart muscle cells in which the cardiac cells are so interconnected that when one cell becomes excited, the action potential rapidly spreads to all of them. The rapid transmission occurs via *intercalated discs; which* are actually cell membranes that separate individual cardiac muscle cells from one another.
Atrial syncytium, which constitutes the walls of the two atria, and the *ventricular syncytium of the walls of the two ventricles*



المقصود هون انه خلايا القلب من كثر تدابطهم و توافقهم و كأنهم خلية وحدة بعدة انوية

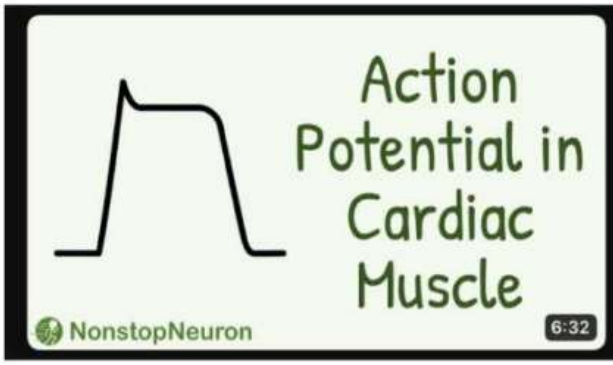
Another Notes from Videos



Action Potential In SA Node || Primary Pacemaker of the Heart

8.3K views · 10 months ago

Nonstop Neuron



Action Potential In Cardiac Muscle || Heart || Cardiovascular Physiology

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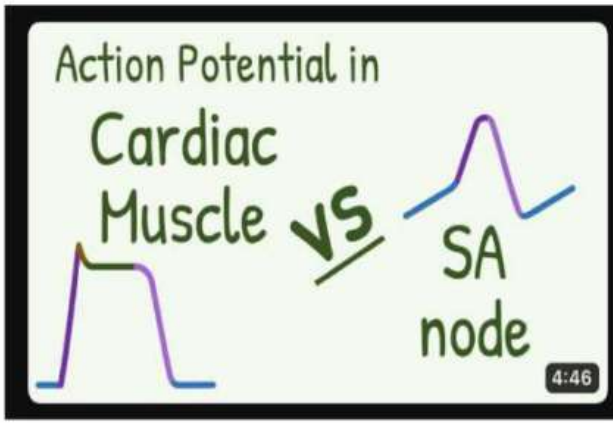
Nonstop Neuron



Action Potential in Cardiac Muscle vs SA Node a Comparison

6.9K views · 10 months ago

Nonstop Neuron

Full Videos

Physiology Chapter 13

Action potential differs vastly in cardiac tissue compared to neural tissue.

Firstly, the pacemaker nodes of the heart are responsible for generating the action-potential stimulus that is used to allow the heart cells to contract. There are 3 sites where AP is generated; SA (sinoatrial) node, AV (atrioventricular) node, and the Purkinje fibres. The SA node is the fastest node in generating signals, and the other nodes generate signals at the same speed as the SA node due to stimulation from the SA node to do so. If the SA node becomes faulty for any reason, the other nodes will generate the signal at their slower rate.

Starting from resting membrane potential of -60mV: The nodes have a special type of sodium channel that allows the sodium ions to slowly enter the cell, without the need for any stimulus. This is known as the "funny channel" (or "h" channel) and is responsible for the autonomy in generating an action potential. The funny channels alongside some T-type calcium channels (transient channels) increase the positivity of the membrane from rest (phase 4, more on the irregular numbering later), and upon reaching the threshold, more calcium channels open resulting in a massive influx of calcium ions in the cell, resulting in the depolarization of the cell (phase 0). Note how this differs than the neurons where the influx is in sodium ions.
Once enough calcium ions enter the cell, the calcium channels close and potassium channels open, resulting in the massive efflux of potassium ions. This is the repolarization phase (phase 3). Once the cell returns to RMP, the cycle repeats again at phase 4.
The signal generated from the SA node (or any other node) is then propagated to the other cardiac cells via gated channels.

Cardiac contractile cells work slightly differently, since they do not have funny channels nor is the influx caused by calcium ions. Instead, upon being stimulated, the sodium channels open leading to a massive influx of sodium ions (phase 0, just like the nodes), causing the membrane potential to rise from rest (-90mV) to +30mV. Upon reaching +30mV, the sodium channels close and the fast potassium channels open for a limited time resulting in a very small initial repolarization (phase 1).
Next up, L-type calcium channels (L is for long-lasting) open resulting in very slow and long-lasting influx of calcium (phase 2, or plateau phase). This plateau phase allows the heart cells to contract for longer. After the L-type calcium channels close, the fast potassium channels open resulting in the efflux of potassium ions, the stage known as final repolarisation (phase 3). Finally, we have phase 4, which is where the cell is at RMP.
As we can see, the reason why SA nodes have the order of 0à3à4 is because of the lack of a first repolarisation (only one repolarisation instance which is phase 3) and no plateau phase.
Important notes:
The absolute refractory period in cardiac cells is much greater than that of neuron cells due to the longer cycles caused by the plateau phase.
The signals spread very quickly between cells due to the gap junctions.

Another Questions

1. **Which is not true regarding the difference between neuronal and cardiac action potentials?**
   A. Cardiac action potentials are much longer in duration
   B. The ions responsible for the depolarizing upstroke is Na+ and Ca2+ in both types of action potentials
   C. Na+ channel inactivation plays a major role in repolarization of neuronal action potentials but not SA nodal action potentials
   D. Slow-activation cardiac action potentials display automaticity while neuronal action potentials do not

Answer : B

2) ONE OF THE FOLLOWING IS NOT TRUE ABOUT THE DIFFERENCES BETWEEN NERVE CELL AND SA NODE CELL IN THE CARDIAC MUSCLE?

A) NA+ CHANNELS ARE OPENED BEFORE THRESHOLD POTENTIAL IN SA NODE , BUT AFTER IT IN NERVE MUSCLE
B) SA NODE POTENTIAL DOESN'T NEED STIMULUS ,BUT NERVE CELL NEEDS
C) THE DPOLARISATION OF TWO TYPES OF CELLS SHOULD BE SLOW FLOWING
D) THE REPOLARISATION OF TWO TYPES OF CELLS SHOULD BE SLOW FLOWING

Answer : C

بالتوفيق

#النادي_الطبي

#معكم_خطوة_بخطوة