PASSION ACADEMIC TEAM YU - MEDICINE

Cardiovascular System

Sheet# 4-PHYSIOLOGY

Lec. Date : 25/3/2020

Lec. Title : Cardiac Arrhythmias

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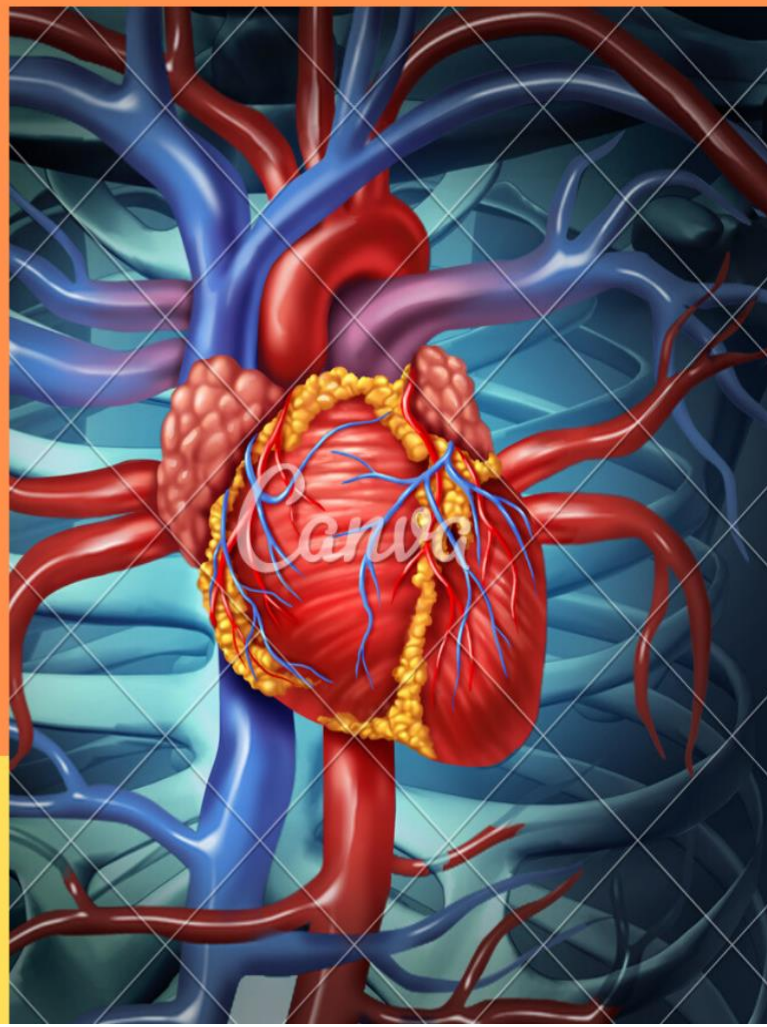

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CARDIAC ARRHYTHMIAS

Cardiac arrhythmias : It's the name given to different conditions that can cause the heart to be *too* fast or too slow or irregularly in beats

Cardiac arrhythmiasc

Cardiac dysrrhythmias can be divided into four categories depending on the functional site affected:

- Disorders of SA node
- Atrial arrhythmias
- Ventricular arrhythmias, and
- Conduction disorders.

Causes of Cardiac Arrhythmias :

1- Abnormal rhythmicity of pace maker

2- Shift of pace maker from Sinus node (SA node) which mean the Sinus node is no longer the pace maker and there is another pace maker in the heart which sick the heart rate

3- Blocking of transmission of cardiac impulse at different point of heart for example : atrial region to ventricular region is blocked

--The blockage Could be complete or incomplete

4- Abnormal pathway of transmission in the heart :
there is conduction but through abnormal or alternative pathways

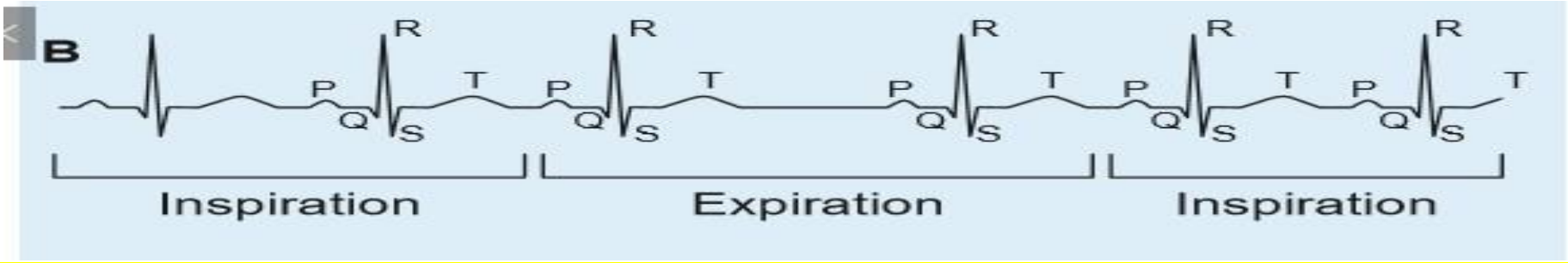
5- Spontaneous generation of abnormal impulses in any part of the heart

Disorders of SA Node

The common disorders of SA node are:

- **sinus arrhythmia**
- **sick sinus syndrome**
- **sinus tachycardia and**
- **sinus bradycardia.**

Disorders of SA Node

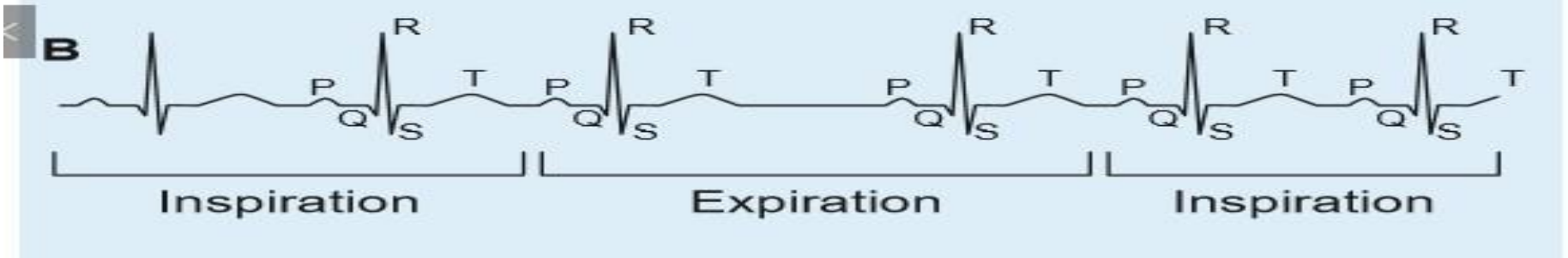


Sinus arrhythmia:

- Is a normal physiological phenomenon referred to the alteration in heart rate in respiratory cycles.
- Heart rate increases in inspiration and decreases in expiration. This is also called respiratory sinus arrhythmia.
- It is explained by different mechanisms:
  - Alteration in autonomic activity
  - Activation of Bainbridge reflex:

Sick Sinus Syndrome : Decrease in heart rate due to disease of SA node is called sick sinus syndrome

Sinus Arrhythmia



- If you notice ECG Heart Rate during inspiration isn't the same as expiration (the interval in inspiration is wider than expiration)
- During inspiration there is increase in Heart Rate
- During expiration there is decreases in Heart Rate

CONT…. Sinus Arrhythmia

Mechanism of Sinus arrhythmia:

1) Alteration in autonomic activity :
   in inspiration, sympathetic activity increases and that will lead to increase SA node Firing which increase Heart Rate
   While in expiration, vagal parasympathetic activity increases which will lead to inhibition SA node firing which will decrease Heart Rate

Q/ What phase in SA node action potential affected by sympathetic and parasympathetic??
(we know SA node action potential is slow response (4,0,3))

ANS : NEXT SLIDE

CONT.... Sinus Arrhythmia

ANS for previous slide question is Phase 4

2)Bainbridge reflex (Atrial Reflex): During inspiration ,reduction in plural cavity pressure occur (more negative) which lead to inflation
-plural pressure reflex the pressure in thorax region-
So during inspiration the decrease in intrathoracic pressure will lead to reduction in right atrium pressure and reduction in SVC and IVC pressure
So Central venous pressure is higher than Right atrial pressure because of reduction in thoracic pressure (because inspiration) this will lead to more venous return -‹more filling and more stretching in atrium which will lead activation of atrial tachycardia producing receptor which is a mechanical receptor (activated because of stretching)
which activate sympathetic innervation which lead to increase SA node activity and Heart Rate.

CONT.... Sinus Arrhythmia

Stretching and filling also increase Heart Rate by Directly stimulate SA node

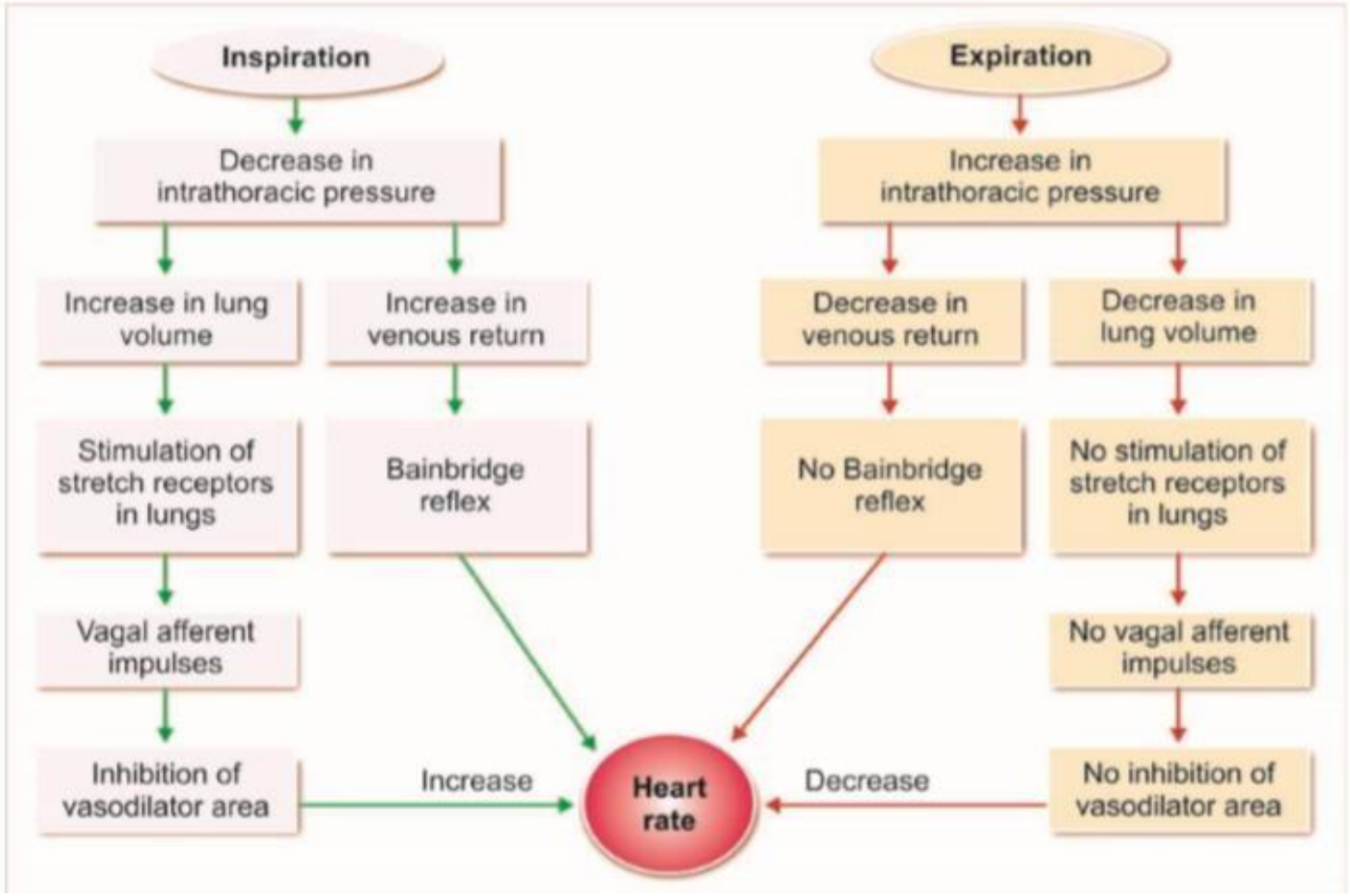
-inspiration cause inflation of lung because of reduction in intrathoracic pressure which will lead to increase in pulmonary pressure and in transmural pressure which lead to stretching inflation.

-Stretching inflation in lung stimulate stretch receptor which send impulses to Cardio inhibitory area (which exist in Cardiovascular centre but it's connected to parasympathetic -so stimulate Cardio inhibitory area will stimulate parasympathetic and vice versa -)through afferent fibbers (Vagus nerve)

so inhibition of Cardio inhibitory area occur which lead to inhibition of Vagus tone (Vagus tone) so Stimulation to SA node (more firing more Heart Rate)

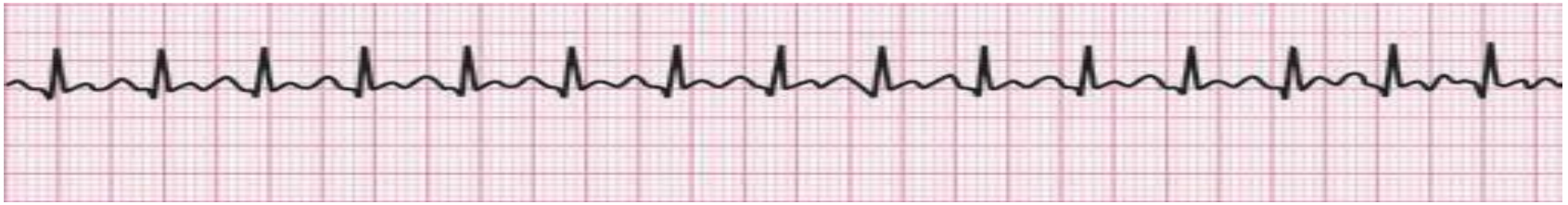
The opposite Mechanism happen during expiration

Bainbridge reflex



Disorders of SA Node

SINUS TACHYCARDIA

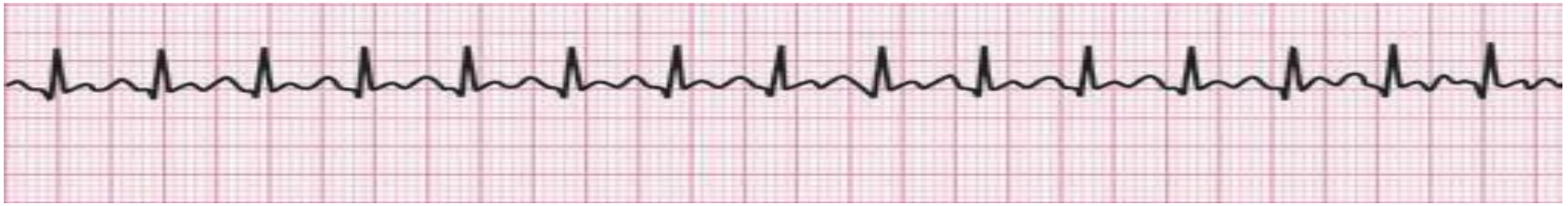


- Sinus tachycardia is the increase in discharge of impulses from SA node, resulting in increase in heart rate; up to 100/minute and sometimes up to 150/minute.
- ECG is normal in sinus tachycardia, except for short R-R intervals because of increased heart rate
- Physiological conditions: 1. Exercise 2. Emotion 3. High altitude 4. Pregnancy.
- Pathological conditions:1. Fever 2. Anemia 3. Hyperthyroidism 4. Hypersecretion of catecholamines 5. Cardiomyopathy 6. Valvular heart disease 7. Hemorrhagic shock.

SINUS TACHYCARDIA

Tachycardia : Fast Heart Rate usually grater than 100/min but rarely 200/min

Etiology : SA node depolarizing faster than normal will lead to increase in Heart Rate



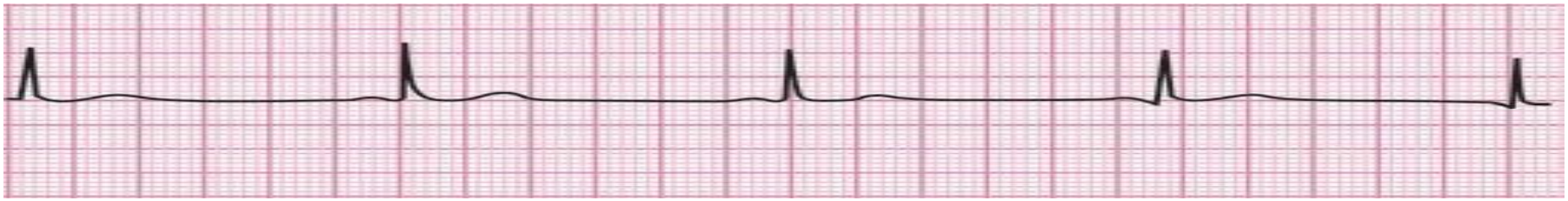
In the figure above Heart Rate increased and interval R-R are very short

The ECG is normal but R-R is short ,Rhythm is normal impulse conduction is normal

SA node is depolarizing faster than normal so phase 4 is shifted upward so the change in membrane voltage to threshold is faster

Disorders of SA Node

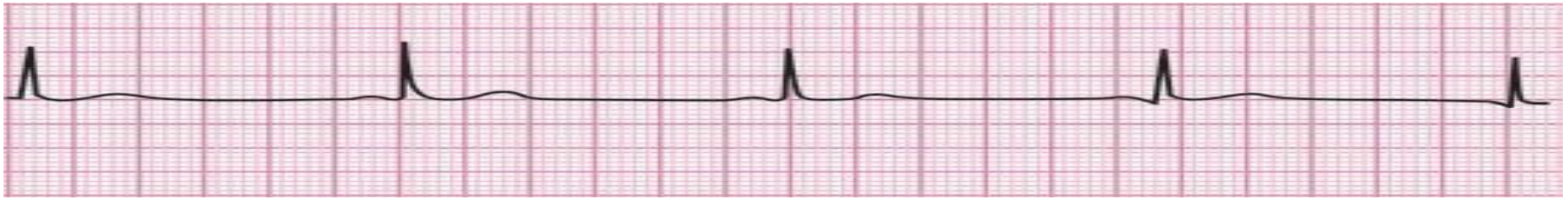
SINUS BRADYCARDIA



- Sinus bradycardia is the reduction in discharge of impulses from SA node resulting in decrease in heart rate. Heart rate is less than 6o/minute.
- ECG shows prolonged waves and prolonged R-R interval.
- occur during sleep, age, secondary to drug (beta blocker, calcium channel blockers) and in athletes (who have a large stroke volume).

SINUS BRADYCARDIA

Bradycardia : A slow Heart Rate usually less than 60/min



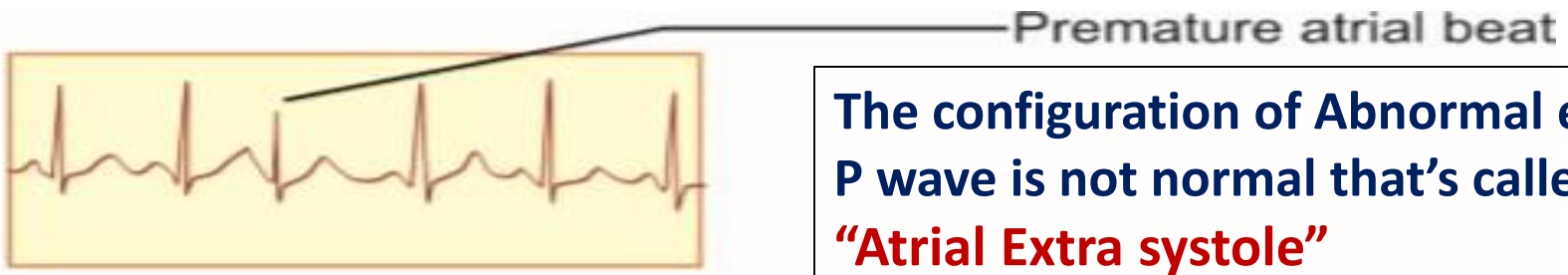
- The heart rate is normal , but R-R interval is Wide (prolonged R-R interval)
- SA node depolarizing slower
- Phase 4 take longer time to reach threshold (Shift Downward)

Atrial Arrhythmias

Atrial Arrhythmias: The common atrial arrhythmias are:

- **atrial premature beats**
- **paroxysmal supraventricular tachycardia**
- **atrial flutter and**
- **atrial fibrillation.**

Atrial Arrhythmias



The configuration of Abnormal ectopic P wave is not normal that's called "Atrial Extra systole"

- Atrial premature beats occur due to premature discharge from an ectopic atrial focus.
- The abnormal P wave of premature atrial ectopic beat appears before the next Normal sinus P wave.
- This is also called atrial extrasystole.
- Atrial ectopics are seen in physiological conditions, like anxiety, consumption of excess tea or coffee, or in heart diseases, like rheumatic heart disease, coronary artery disease, cardiomyopathies or digitalis toxicity

Sinus P wave : P wave generated from SA Node

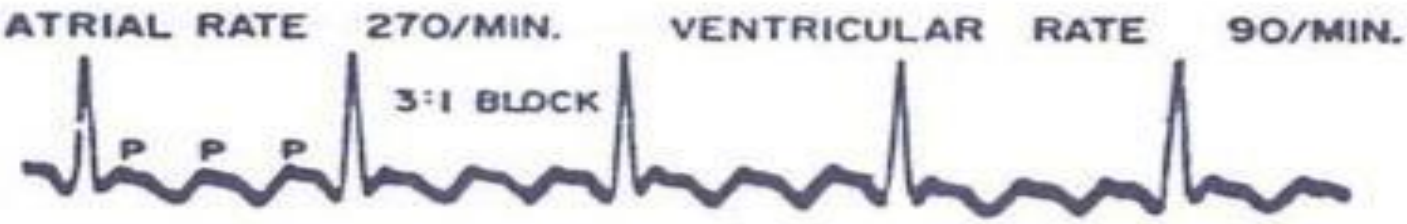
Atrial Premature Beats

It occur due to generation of cardiac impulse in hyper excitable atrial tissues of heart parts other than the normal part (SA Node), and these parts in general are called ectopic foci

As Atrial Premature Beats occurred in atria, so ectopic foci must be in atria regions and it is called "Atrial Ectopic Foci"

Atrial Arrhythmias

Atrial Flutter



- This is a pathologicnl condition in which the atria beat regularly at a rate of 250-350/minute by impulses discharged from hyperexcitable ectopic focus. As the AV node cannot conduct more than 230 impulses minute. incomplete heart block develops, and the ventricles respond once for every 2 or 3 atrial beats.
- So in ECG. each 2 or 3 P waves are followed by one QRST and the P waves are obnormal while the QRS and T waves are normal in shape and occur regularly.
- Atrial flutter is common in patients suffering from cardiovascular diseases such as hypertension and coronary artery disease. ,In a mechanism called Re-Entry

Atrial Flutter

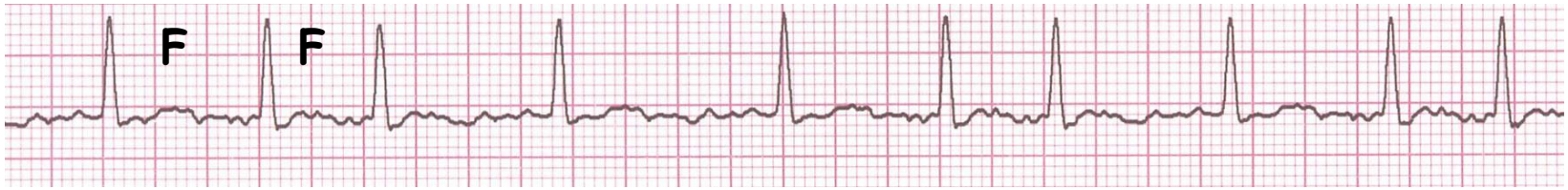
ECG : shape of P waves is normal but the rhythm is not, P waves represent "Saw tooth" .

ECG general rhythm is normal (PR intervals are equal).

Not all atria beats are transmitted to ventricles, because AV node has a limit.

Atrial Arrhythmias

Atrial Fibrillation



- In atrial fibrillation, atria beat rapidly but irregularly in a totally disorganized way. Atria beat at a rate of 300 to 500/min and ventricle beats at 100–180/min.
- It occurs due to the presence of multiple reentrant excitation waves in the atria.
- The atrial fibres contract asynchronously. This leads to inefficient atrial contraction and blood stagnation in the atria which predisposes to intra-atrial thrombosis.
- In ECG there are no P waves (which are replaced by line oscillations called F waves), and the QRS andT waves are normal in shape but irregular in rate

Atrial fibrillation

A difference between atrial flutter and fibrillation is the regularity, Atrial flutter is Regular whereas Atrial fibrillation is Irregular

In ECG: there is no P waves or they are replaced by line oscillation called F (waves), but QRS comp. and t wave are normal in shape but irregular in their rate of occurrence .

Causes:

1- presence of multiple reentrance excitation waves in atria.

2- Impulses are generated by multiple ectopic foci.

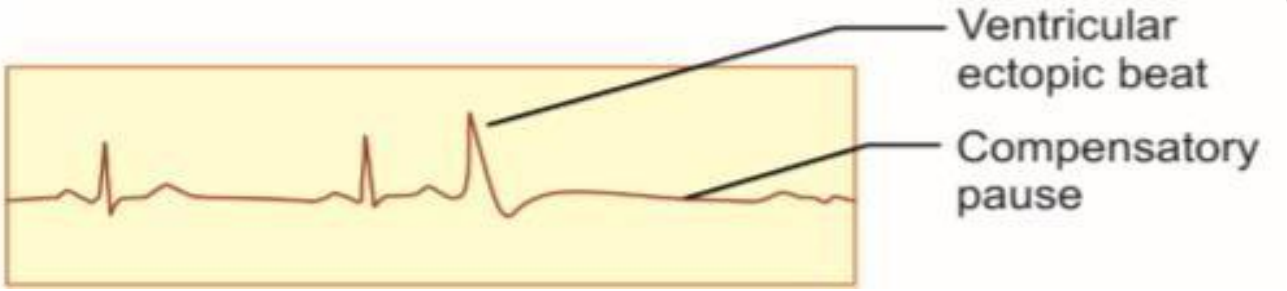
Atria inefficient contraction does not affect Ventricular filling as it's majorly passive process

Ventricular Arrhythmias

Ventricular Arrhythmias: The common ventricular arrhythmias are:

- **ventricular extrasystole**
- **paroxysmal ventricular tachycardia, and**
- **ventricular fibrillation.**

Ventricular Arrhythmias



Ventricular Extrasystole:

- occurs due to premature discharge from a ventricular ectopic focus.
- the QRS complex appears early than anticipated and looks wide, bizarre and slurred or notched.
- the P wave is not seen as it is buried in the QRS of the extrasystole.

Ventricular extrasystole

occurs due to premature discharge from a ventricular ectopic focus. Which means that impulse is generated from hyper excitable area in ventricles is called :
"ventricular ectopic focus"

QRS complex in ECG of ventricular extrasystole appears larger than normal and P wave is merged with it so it won't be seen

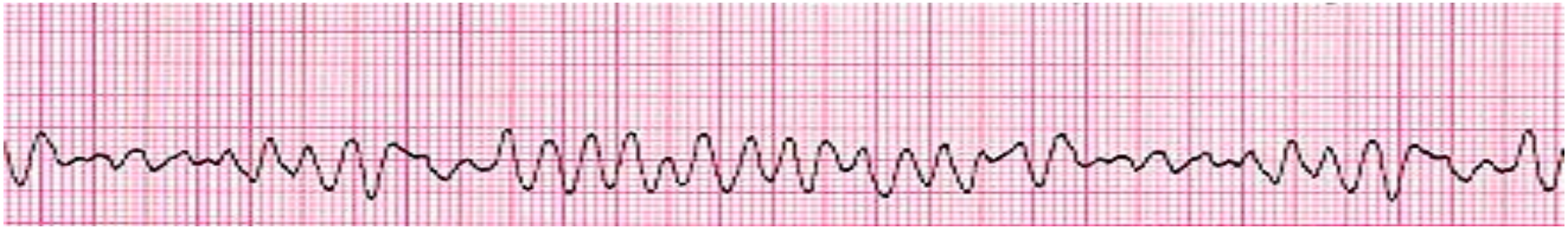
Ventricular premature beats are usually followed by compensatory pause Compensatory pause, it's causes and consequences was explained in the topic of refractory period

Atrial Extrasystole is same of atrial premature beats explained before in this lecture

ventricular extrasystole could be caused by either Physiologic or Pathologic conditions

Ventricular Arrhythmias

Ventricular Fibrillation



- **Occur as a result of:**
  - **Impulses discharged from multiple ectopic loci**
  - **Re-entry or circus movements of impulses**
- **The QRS complexes are quite irregular in shape, rhythm and amplitude and all waves are not clear and cannot be identified**
- **No cardiac output (it is fatal)**

Ventricular Fibrillation

The only difference between Ventricular and Atrial fibrillation is the place of ectopic foci, in ventricles for ventricular fibrillation and in atria for atrial fibrillation.

In ventricular fibrillation, the ventricular rate is very high and irregular and ventricular contraction is totally disorganized and ineffective, because of rapid discharging of impulses from these hyper excitable ectopic foci or Re-entery

Effect on ventricular diastole is more than systole → No filling → No ejection of blood → reduction in force contraction according to Frank-Starling Law

Ventricular fibrillation is fatal because it results in loss of ventricular pumping power and stoppage of circulation
ECG: Irregular QRS Shape, Amplitude, Rhythm , T+P waves are not clear, and you can't find it actually.

Conduction Disorders

- Conduction disorder may be classified as :
  - conduction block or heart block or atrioventricular block are two types:
    - Incomplete heart block, two types
      - 1st degree
      - 2nd degree
    - Complete heart block (3rd degree)
  - conduction acceleration.

Conduction Disorders

Conduction disorder may be classified as :

1- conduction block or heart block or atrioventricular block (3 names) : which is a defect in transmission of impulses from atria to ventricles

- AV Block is classified into 2 types :

A. Incomplete heart block : Conduction of impulses between atria and ventricles is not completely interrupted and that's why are they called Incomplete heart block ,
Its on 2 types : 1st degree and 2nd degree

B. Complete heart block (3rd degree) : atrioventricular conduction of impulses is completely stopped

2- conduction acceleration.

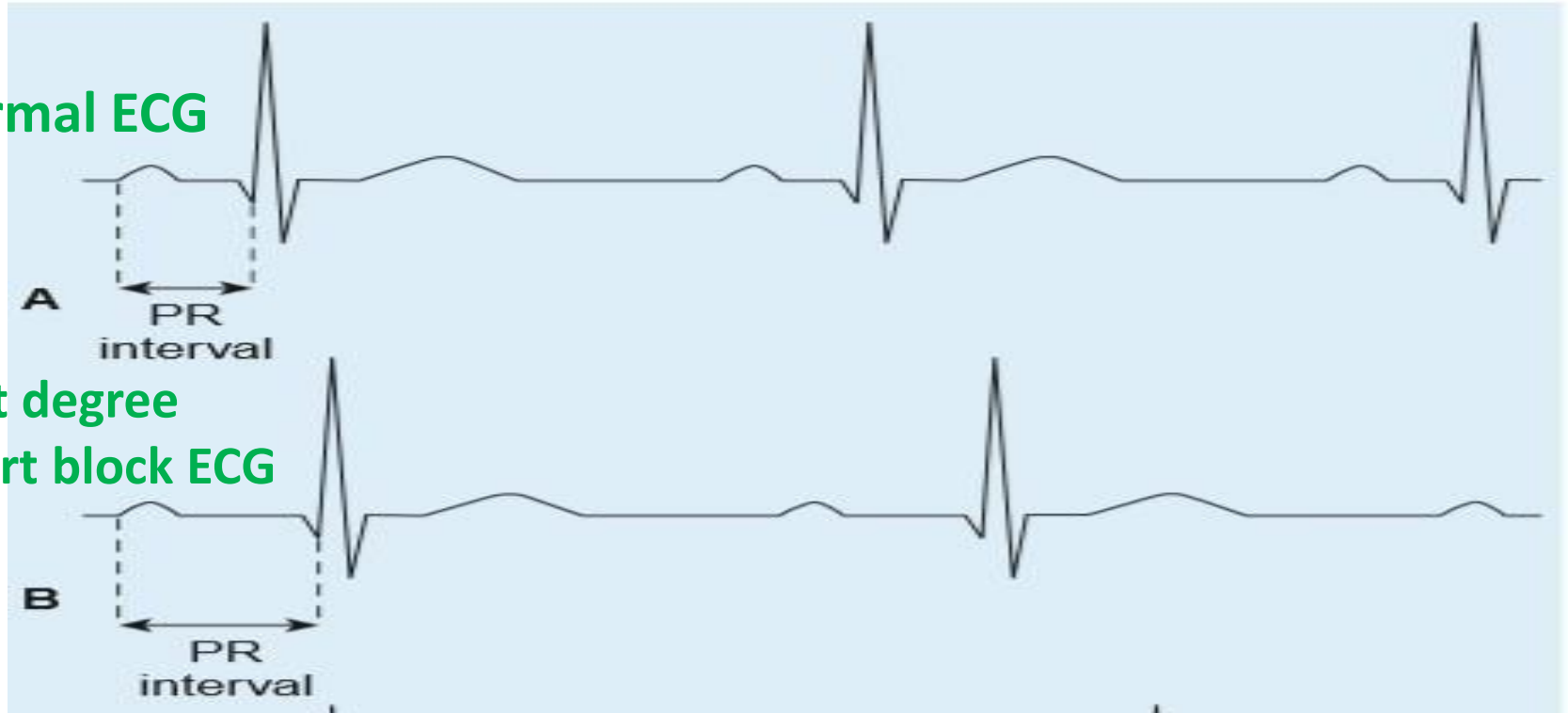
Incomplete heart block

First degree heart block

- All SA node impulses are transmitted to the ventricles but with more delay in the AV node or AV bundle resulting in abnormally prolonged P-R interval in the ECG exceeding 0.20 second.
- In other words: a Delay in AV node slows the conduction of SA node impulses

Normal ECG

First degree Heart block ECG

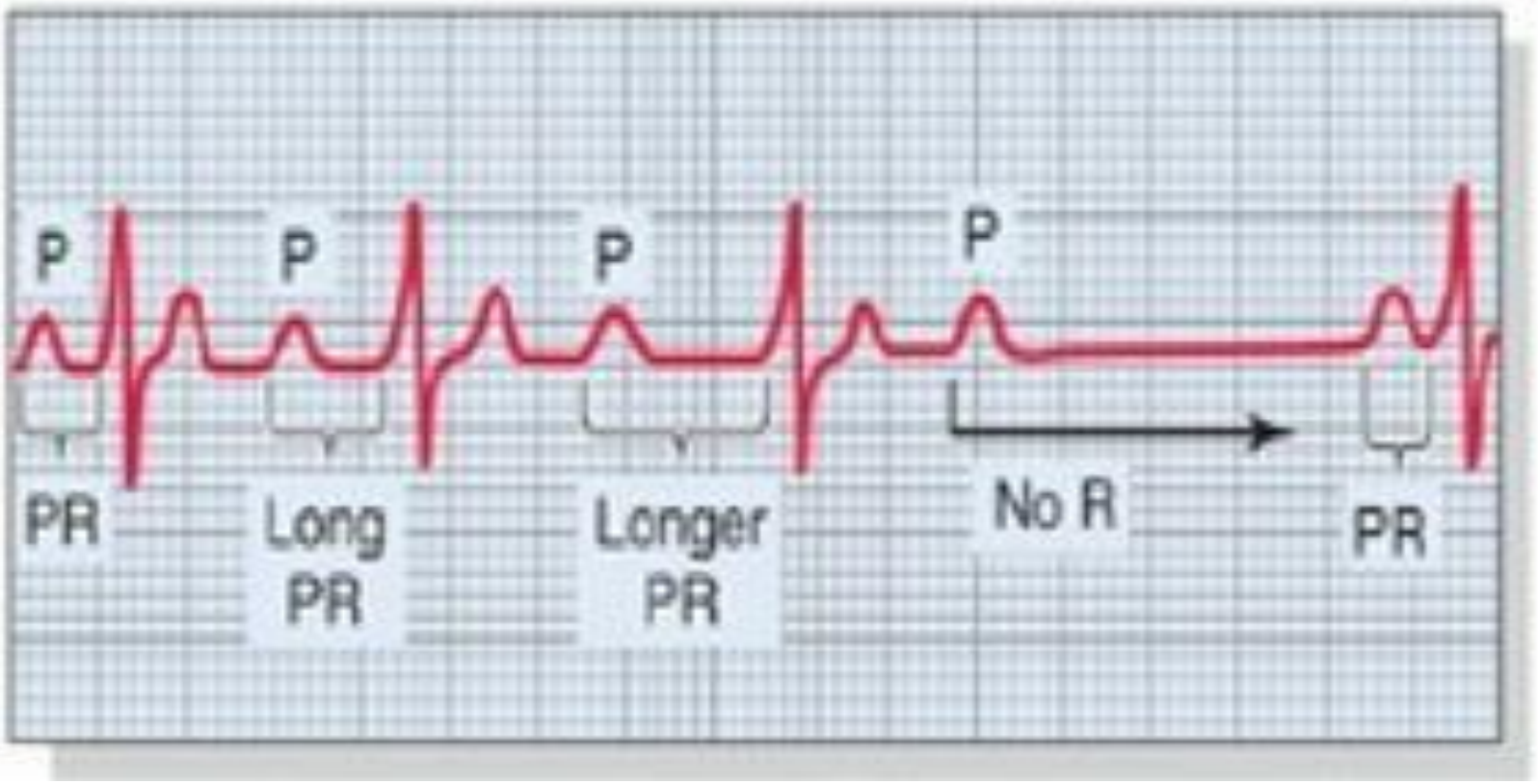


Incomplete heart block

Second degree heart block: This is further subdivided into:

- **Mobitz type I (Wenckebach block) , and**
- **Mobitz type II blocks.**

Mobitz Type I (Wenckebach)



Mobitz type I block (Wenckebach block)

In this type of blocks : the PR interval gradually lengthens from one cycle to the next until the AV node fails completely to conduct QRS wave complex from atria to ventricle which results in elimination of ventricular depolarization or skipping for ventricular depolarization.

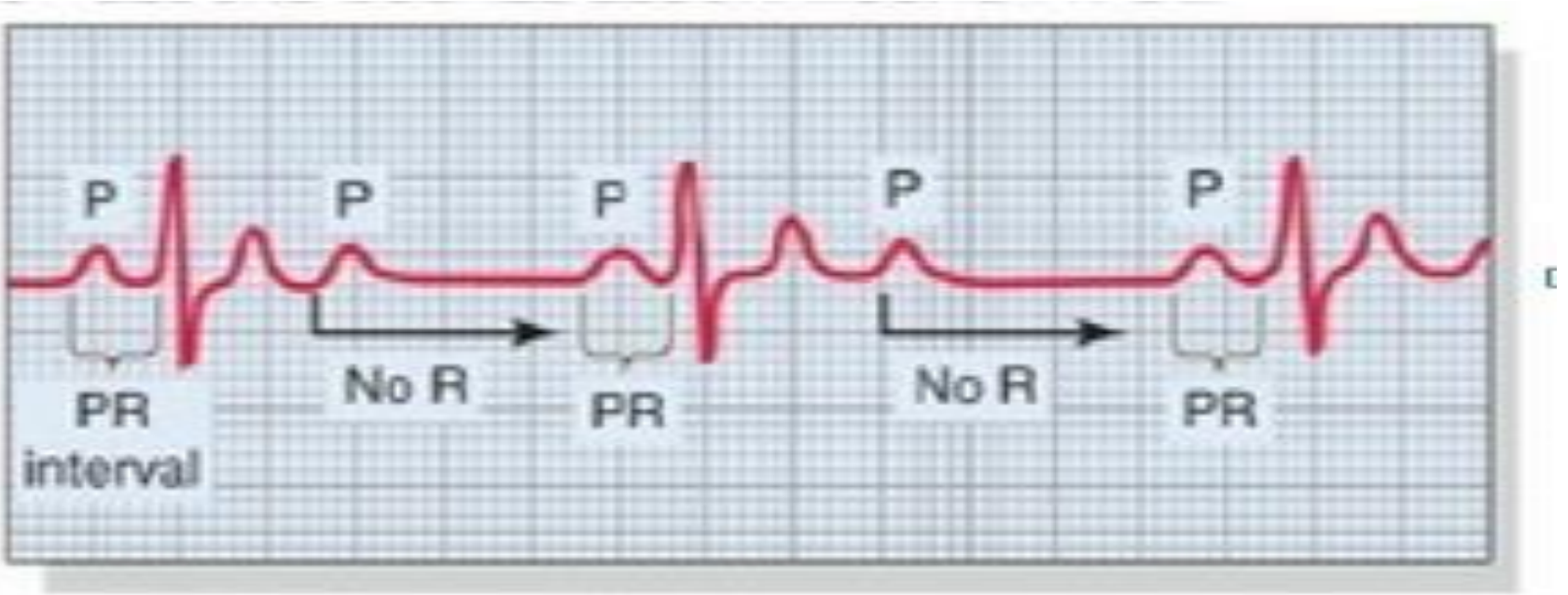
المعنى بالعربي :

في كل دورة قلبية أو Cardiac cycle ال PR interval بتزيد شوي يعني المسافة بين الـ(P) و الـ(R) بتزيد لأنه الـ(AV) بتتأخر لتنقل الإشارة الكهربائية وكل مرة هاي المسافة بتزيد حتى توصل الـ(AV) لدرجة مش انها تتأخر بل حتى تفشل بنقل إشارة الـ(R)

The PR interval following the dropped beat (beat which ventricular depolarization has been skipped) is usually normal ,however in the subsequent beat the PR interval is progressively prolonged until the next beat is dropped.

- It is most common to see every 3rd or 4th atrial depolarization fails to be conduct to the ventricles

Mobitz Type II

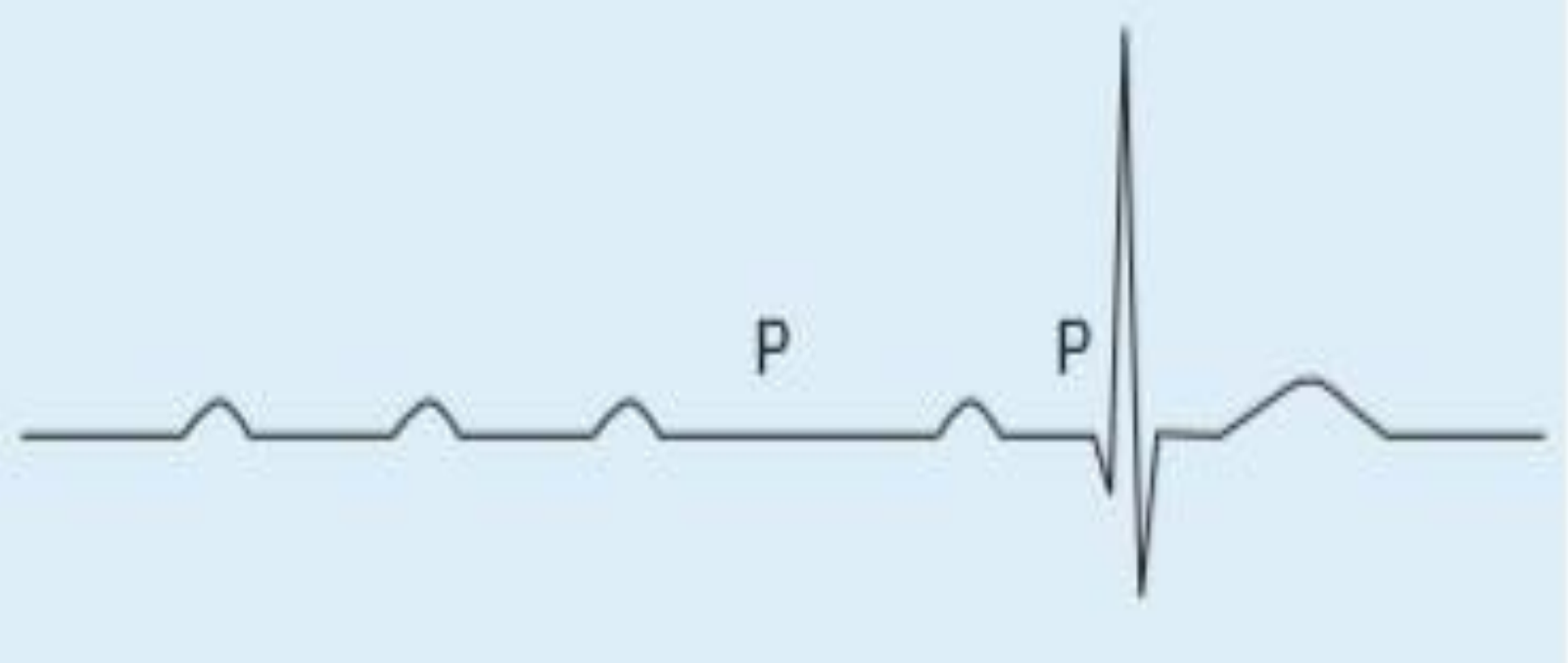


لم يقم الدكتور بشرحها لكن الفكرة الرئيسية فيها كالتالي :

في النوع السابق كانت المسافة تزيد شوي شوي عبين ما تسقط الـ Ventrecular depolarization لكن هون بتسقط فجأة بدون ما المسافة تتأثر

الفرق الثاني إنه في النوع السابق سقوط الموجة كان يحدث كل 3 أو 4 نبضات لكن هون السقوط بصير مرة وراء مرة يعني مرة اه مرة لا

Complete Heart Block (Third degree heart block)



When the heart rate is as low as 15/min, blood circulation decreases that results in cerebral ischemia and causes fainting. This is called Stokes-Adams syndrome.

Complete heart block (Third degree heart block) :

Conduction of impulses from atria to ventricles is completely interrupted or stopped .

- So, if there is no connection between atria and ventricles (as impulses from atria to ventricles is completely blocked)

then atria and ventricles will beat separately and every one of them will be beating alone, that means rate of rhythm of atria will be different from rate of rhythm of ventricles

(for example rate of atria rhythm is 100 or 90 per min. whereas rate of ventricular beat is less than 40),

CONT… Complete heart block (Third degree heart block) :

In the previous slide that's will happen because atria are triggered by SA node while in ventricles a part of them will be a pace maker for ventricles and sets the rhythm for them,

and as SA node is the fastest and ventricles pacemaker is very slower than SA node, there will be a difference in rate of rhythm between atria and ventricles.

Because ventricles are beating at very lower rate than normal, the amount of blood pumped or ejected will be inadequate especially during stress conditions as exercises for example.

CONT… Complete heart block (Third degree heart block):

Because ventricles are beating at very lower rate than normal (because its pacemaker is slower than SA node), the amount of blood pumped or ejected will be inadequate especially during stress conditions as exercises for example.

That's why third degree heart block is associated with Syncope which is caused by insufficient cerebral blood flow which in turn causes Lightheadedness.
(it is Lightheadedness not light headedness).

Nowadays Treatment for third degree heart block is implantation of electrical/electronic pacemaker device.

CONT… Complete heart block (Third degree heart block):

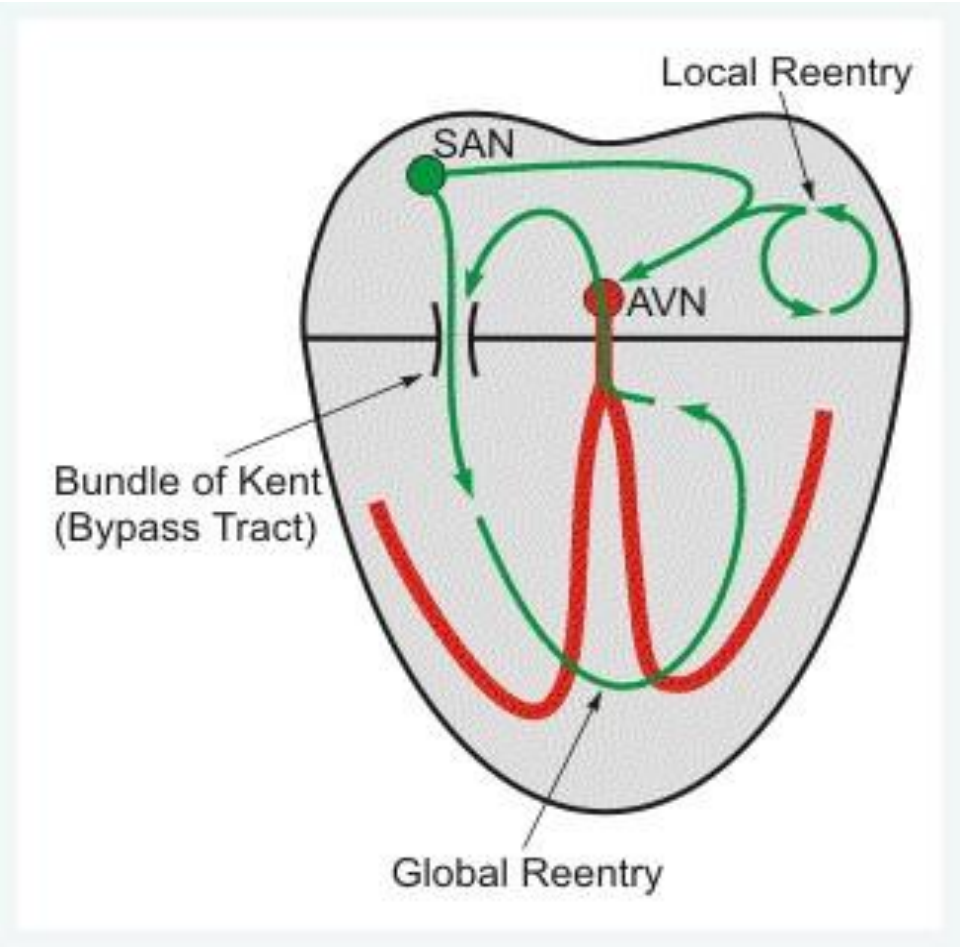
معلومة خارجية :

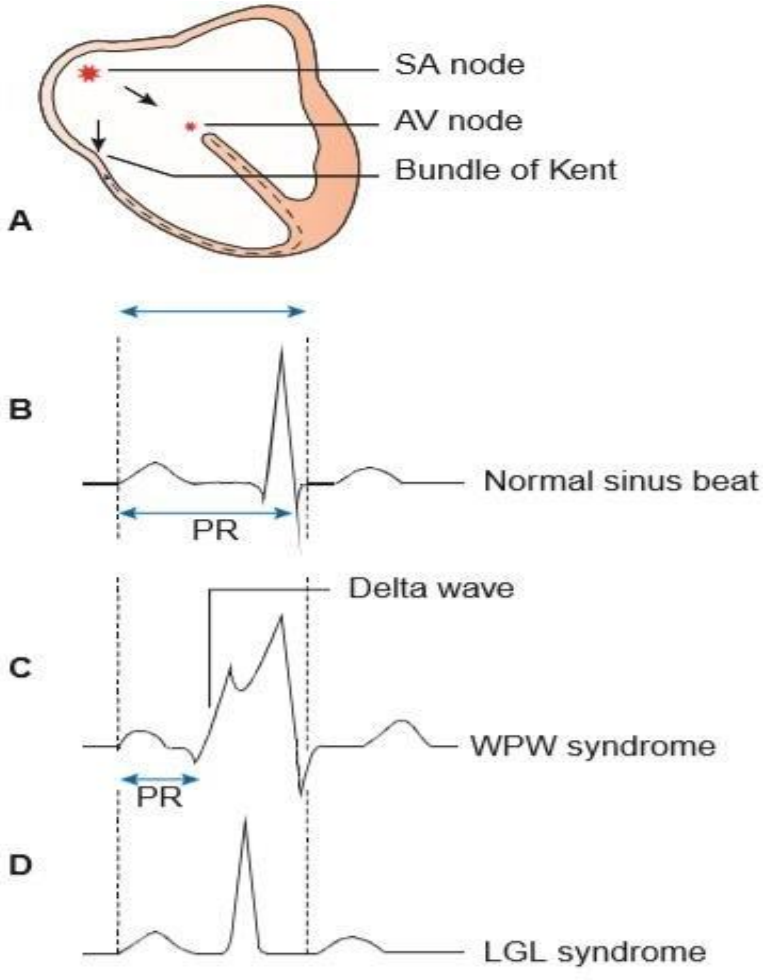
Syncope : temporary loss of consciousness related to insufficient blood flow to brain. It's also called fainting or passing out. It occurs when blood pressure is too low (hypotension) and the heart doesn't pump enough oxygen to the brain.

Lightheadedness : a common and typically unpleasant sensation of dizziness or a feeling that one may faint. The sensation of lightheadedness can be short-lived, prolonged, or, rarely, recurring. In addition to dizziness, the individual may feel as though his or her head is weightless.

Acceleration of Conduction

- **Wolff-Parkinson-White (WPW) Syndrome and**





Acceleration conduction

One example for it is "**Wolff-Parkinson-White (WPW) Syndrome**"

We know that AV bundle is the only electrical connection between atria and ventricles,
But hearts of some individuals have another alternative pathway through which the impulse can be conducted from the atria to the ventricles, (in addition to the AV bundle), usually it is a muscular bundle
An example of the alternative conducting pathway is
"Bundle of Kent" .

So they have multiple pathways through which the impulse can be conducted from atria to ventricles.

CONT… Acceleration conduction

So theses individuals have in their hearts multiple pathways through which the impulse can be conducted from atria to ventricles.

The problem here is that one pathway is faster than another as there is a delay in the AV node and isn't in the alternative one.

Because bundle of Kent is faster in conduction than AV bundle one of the ventricles is going to be excited earlier than the other which in turn shows 2 QRS complexes on ECG (one is earlier than the other) so these 2 QRS will merge together (as seen in the previous slide, the figure on the right, C.) the earlier wave which is excited the fast bundle of Kent is going to merge with the normal one which is excited by the ordinary AV bundle.

CONT… Acceleration conduction

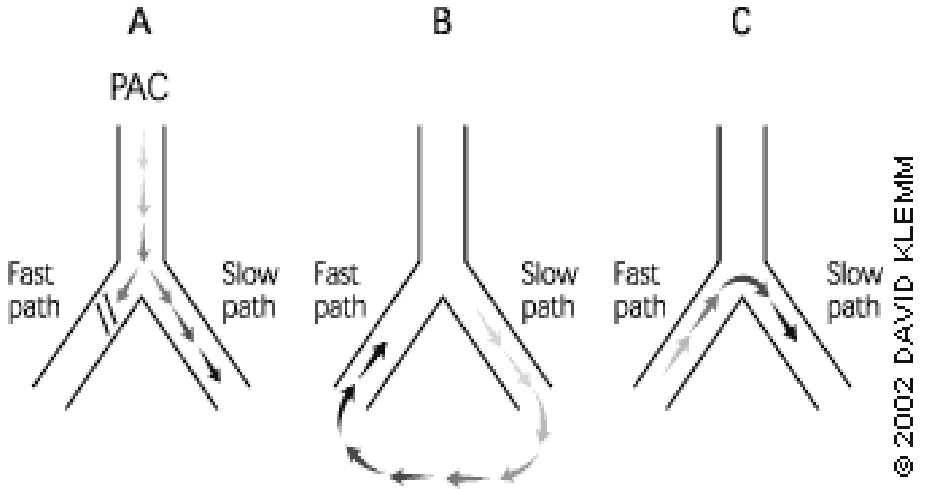
Wolff-Parkinson-White (WPW) Syndrome ECG Characteristics:

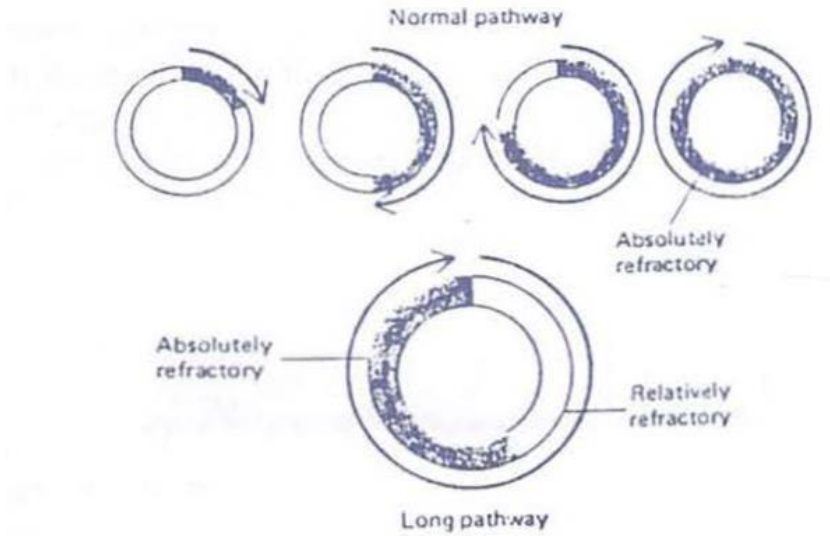
1- P-R interval is shortened
2- P waves still normal
3- QRS complex is widened because of Merging, and is slurred in its up stroke, this wave is called "Delta wave"

From the figure on the Left of slide 27 we notice that:
Impulse is conducted fast through bundle of Kent depolarizing the ventricles, this give a chance for impulse to travel backwards (backward move is called "retrograde") through AV node another time exciting atria and establishing "Reentry" making circus movement of impulses and continuous excitation.

Re-entry mechanism or the circus movement

- Re-entry mechanism or the circus movement:
  - refers to a phenomenon in which the wave of excitation propagates repeatedly (continuously) within a closed circuit
  - Re-entry of excitation wave is known to occur under these situations:
    - (i) in the presence of transient block in the conduction pathway.
    - (ii) The length of the impulse pathway is prolonged and
    - (iii) in the presence of an abnormal extra bundle of conducting tissue called bundle of Kent.





RE-Entry or Circus movement of impulses

A phenomenon in which the wave of excitation Conducted repeatedly (continuously) within a closed circuit.

-The most common mechanism for tachyarrhythmia is re-entry.

-Tachyarrhythmia could happen by other cellular mechanisms:

a. Increase in automaticity: which lead to more rapid phase 4 depolarization and so tachyarrhythmia.

b. Spontaneous depolarization during phase 3 or phase 4.

Spontaneous depolarization during phase3 : is a condition called: "Early After Depolarization".

Spontaneous depolarization during phase 4 : is a condition called: "Late After Depolarization".

CONT… RE-Entry or Circus movement of impulses

A necessary condition for Re-entry is that at some point in the loop or bundle impulse can pass into one direction and not in the other,
which is called: "Unidirectional Block"

For example impulse can't pass through Antegrade direction but it can pass through retrograde direction,
due to pathologic changes

From slide 31, figure on the upper right we notice:

2 parallel pathways, the main impulse bifurcates and moves along the 2 pathways, the slow and the fast, but at the fast pathway there's a unidirectional block so impulse can not continue along it from antegrade direction (but can from retrograde direction)

CONT… RE-Entry or Circus movement of impulses

The conducted impulse will travel down the slow path normally (slowly as it's slow path), but the suitable conditions for re-entry have occurred, so the impulse will travel down the connecting branch that connects the fast and slow paths together, this impulse may then be able to penetrate the unidirectional blocked region in the fast path from retrograde direction.

Remember that when the impulse bifurcate it flowed down the fast pathway a little before it was blocked so that part has depolarized and thus entered the refractory period.

When the conducted impulse through the small pathway reaches the blocked area of fast pathway, the refractory period should have ended, because the normal conduction of impulse was in the slow branch and it took a lot of time move from slow path to fast path, resulting in an another stimulation…. And the cycle repeats.

CONT… RE-Entry or Circus movement of impulses

Remember that when the impulse bifurcate it flowed down the fast pathway a little before it was blocked so that part has depolarized and thus entered the refractory period.

When the conducted impulse through the small pathway reaches the blocked area of fast pathway, the refractory period should have ended, because the normal conduction of impulse was in the slow branch and it took a lot of time move from slow path to fast path, the impulse can be then conducted back through this region and return to the slow bundle so that the impulse will enter the initial area and stat a circus movement or re-entry, which in turn make another stimulation…. And the cycle repeats.

CONT… RE-Entry or Circus movement of impulses

Note: unidirectional block is necessary condition for Re-entry to happen but it can't cause Re-entry alone and there is need for other factors :

1. length of impulse pathway is prolonged as dilated heart for example in slide 31, right lower figure, the heart has both normal and long (not normal) pathways,

In normal pathway, an action potential is formed and conducted and when AP returns to it's original site, this site should be in refractory period, that's why no circus movement could happen and no re-entry.

In long pathway, there's elongation in conduction pathway so impulses are conducted through long pathways, so when impulse returns to it's original site, this site should not be in refractory period and can be simulated again resulting in circus movement and re-entry.

CONT… RE-Entry or Circus movement of impulses

2. Another factor helps in circus movement or re-entry is velocity of conduction of impulse is decreased by having 2 pathways (one is slow and other is fast) as in ischemia.

3. Another factor is that refractory period in the original site should be shortened.

هنا ينتهي تفريغ المحاضرة الرابعة .... دعواتكم ✨🎉🔥