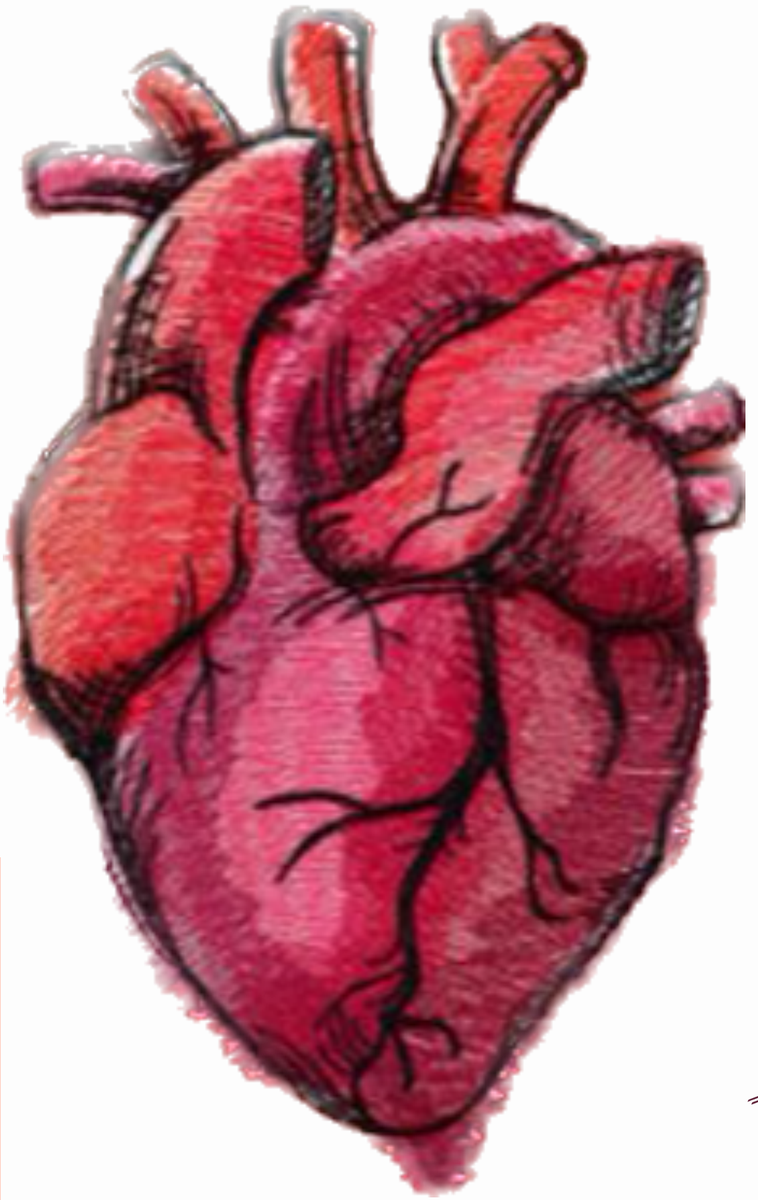


النادي الطبي

CARDIOVASCULAR SYSTEM

SUBJECT : physiology

LEC NO. : 5

DONE BY : Abdullah Bseni Mustafa

وَقُل رَّبِّ زِدْنِي عِلْمًا

SCAN ME!

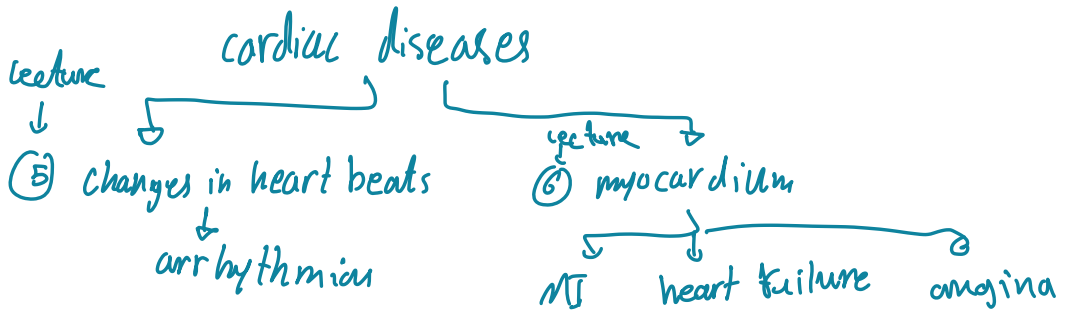
THE ELECTROCARDIOGRAPHY (ECG) III

CARDIAC ARRHYTHMIAS

Dr. Waleed R. Ezzat

LECTURE OBJECTIVES:

1. Explain the changes in the function of the sinus node.
2. Understand the disturbances in the conduction of impulses.
3. Describe the ectopic foci



ARRHYTHMIAS

1. Cardiac arrhythmia is the ***abnormal rhythm of the heart***. This abnormality is either due to disturbances in impulse initiation or impulse propagation.
2. Disturbances in impulse initiation include those that arise from the SA node and those that originate from various ectopic foci.
3. The principal disturbances in impulse propagation are re-entrant rhythms and conduction blocks.

CAUSES OF CARDIAC ARRHYTHMIAS

↳ common

The causes of the cardiac arrhythmias are usually one or a combination of any of the following abnormalities

① increase thyroxin production
↳ increase metabolic rate in SAN → Faster / Slower

- Abnormal rhythmicity of the pacemaker
- Shift of the pacemaker from the sinus node to another place in the heart

might be caused due to decrease in blood prefusion in SA node in case of atherosclerosis

↳ might be caused by surgery

- Blocks at different points in the spread of the impulse through the heart

SA → AV → bundle of his → branches → Purkinje fibers

increase delay in O_2 ↓ or subs funcers ↓

- Abnormal pathways of impulse transmission through the heart

in young children → eg. loss of isolation between atria & ventricles causing episodes of fast heart rate

- Spontaneous generation of false (unauthentic) impulses in almost any part of the heart

common

after an MI → area of the myocardium might suffer from hypoxia → cause the K^+-Na^+ pump to be weaker result in decrease in negativity of resting membrane potential → so without reaching the original potential the fast Na^+ channels won't be able to open so slow Ca^{2+} channels will work making it like pacemaker cells causing arrhythmias

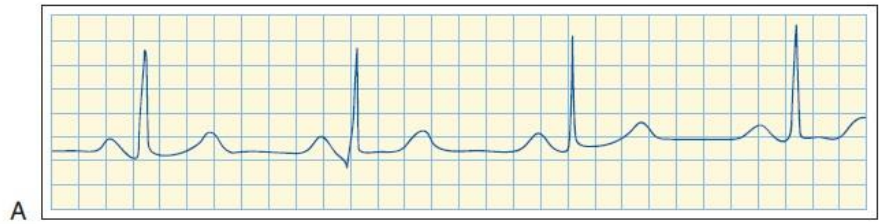
ALTERED SINOATRIAL RHYTHMS

tachycardia might be normal in case of fever or sympathetic stimulation

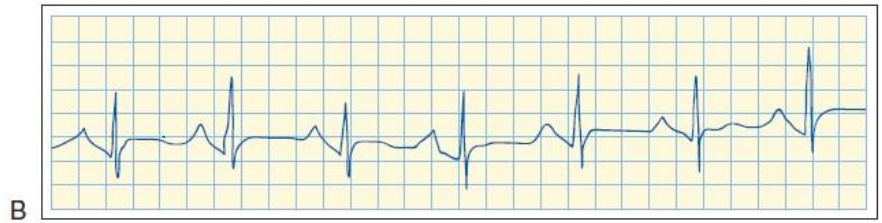
Abnormal sinus rhythms:

increase in heart rate >100

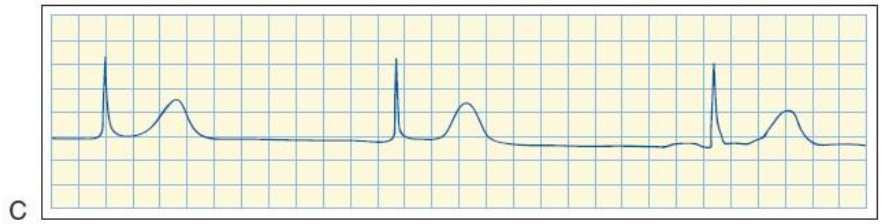
1. Sinus tachycardia: when heart rate exceeds 100 beats/min in an adult. (example; fever, sympathetic stimulation, toxic conditions of the heart, etc.) 60-90 normal heart rate
2. Sinus bradycardia: the heart slows below 60 beats/min. This can be a normal physiological finding in well-trained athletes. Abnormal bradycardia can be due to over stimulation of the vagus in patients with *carotid sinus syndrome.*



Normal sinus rhythm



Sinus tachycardia



Sinus bradycardia

RESPIRATORY SINUS ARRHYTHMIA (RSA)

remember → decrease distance between the R-R and increase distance between R-R can be caused by inspiration & expiration with age this difference decrease and might vanish

Respiratory sinus arrhythmia (RSA) is typically a benign, normal variation in heart rate that occurs during each breathing cycle. *The heart rate increases during inspiration and decreases during expiration*. Normally the heart rate increases and decreases no more than 5 percent during quiet respiration.

There are **TWO** physiological explanations for the RSA;

1. During inspiration blood flow to the heart increases, this in turn triggers atrial baroreceptors which act to diminish vagal tone. This causes an increase in heart rate.

   atrial baroreceptors: are receptors found within atria which can sense the pressure inside the atria when pressure increase it sends signals to the vagal centers in the brain to decrease the vagal tone → inspiration → more blood in atria → more pressure → ↑ heart beat by ↓ vagal tone

   During expiration the diaphragm relaxes, moving upward, causing an increase in intrathoracic pressure. This increase in pressure inhibits venous return to the heart resulting in both reduced atrial expansion and reduced activation of baroreceptors. This relieves the suppression of vagal tone and leads to a decreased heart rate.

   diaphragm moves up → decrease thoracic volume → ↓ venous drainage to the atria → ↓ activation of baroreceptors.

2. Respiratory sinus arrhythmia may result from "spillover" of signals from the medullary respiratory center into the adjacent vasomotor center during inspiratory and expiratory cycles of respiration. The spillover signals cause an alternate increase and decrease in the number of impulses transmitted through the sympathetic and vagus nerves to the heart.

   remember in RS → when recepiratory centers are activated they carry impulses to the diaphragm to contract throug the phrenic nerve so it's belived part of the AP (that should be only in the respiratory centers) "spillover" (get out) & effect the vasomotor center which mean it effect the sympathetic region that works on the heart results on increase in heart beat

ATRIOVENTRICULAR CONDUCTION BLOCKS

First-Degree Heart Block:

The PR interval increases to greater than 0.20 second.

increase AV delay causing an increase in PR interval

Second-Degree Heart Block:

There are two types of second-degree AV block:

Some AP can pass from AVN but other can't

1. Type I (also known as *Wenckebach periodicity*) is characterized by progressive prolongation of the PR interval until a ventricular beat is dropped. In most cases, this type of block is benign and no specific treatment is needed.

gradual increase in PR interval until it can't pass the AVN

2. Type II. There is usually a fixed number of nonconducted P waves for every QRS complex. For example a 2 : 1 block. Patient may require implantation of an *artificial pacemaker.*

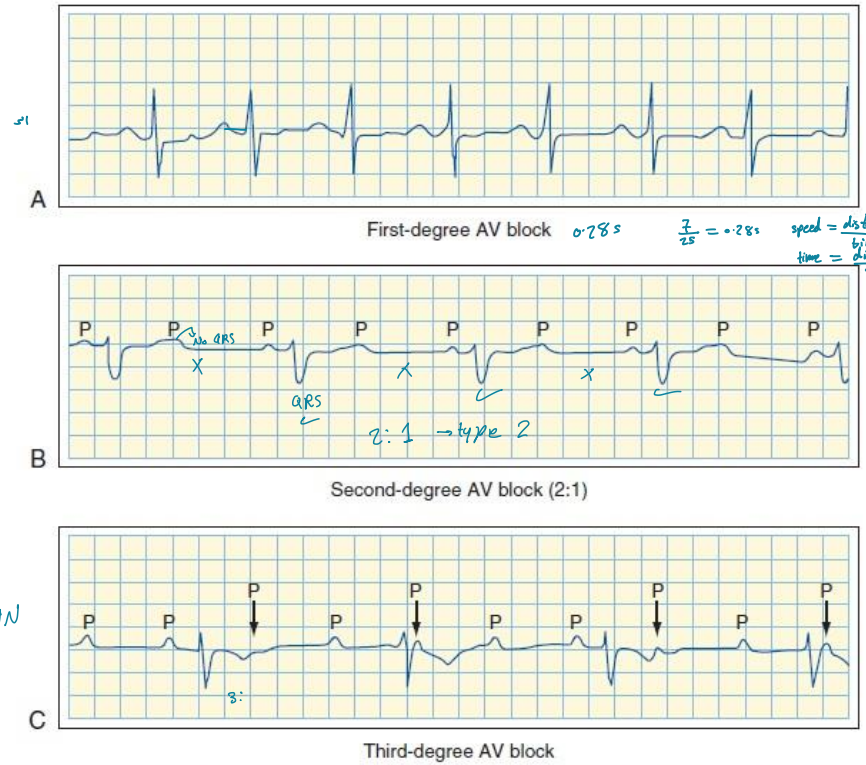
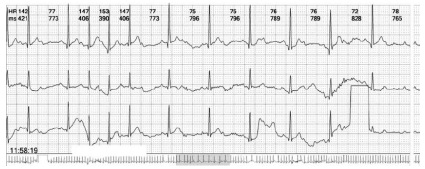


Fig Atrioventricular (AV) Blocks. **A,** First-degree block; the PR interval is 0.28 second (normal, <0.20 sec). **B,** Second-degree block (ratio of P waves to QRS complexes, 2:1). **C,** Third-degree block; note the dissociation between the P waves and the QRS complexes.



ATRIOVENTRICULAR CONDUCTION BLOCKS (CONT.)

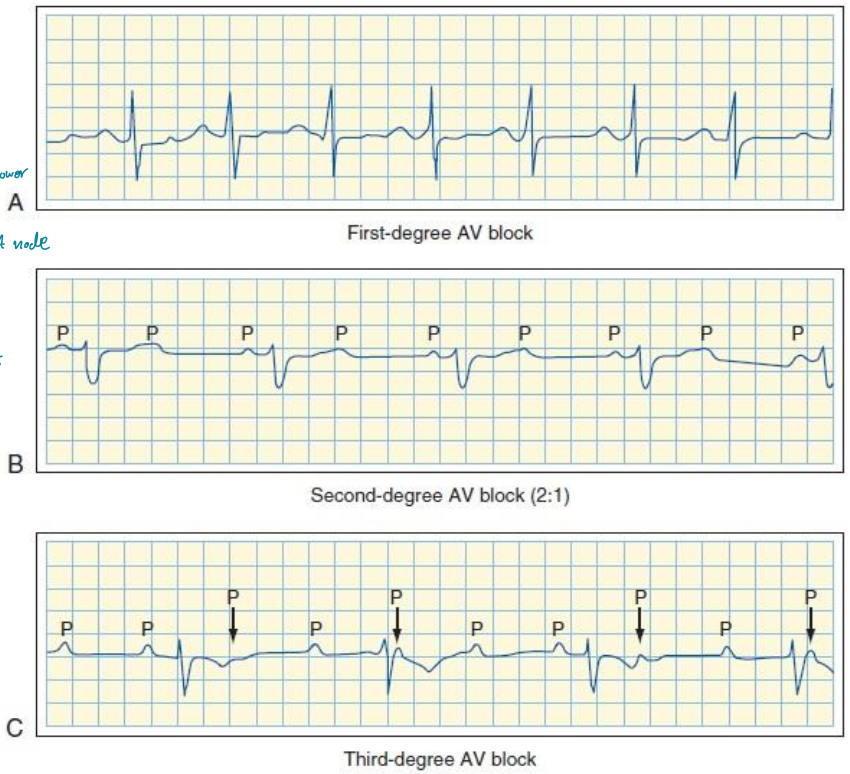
Complete AV Block (Third-Degree Heart Block):

There is a complete block of the impulse from the atria into the ventricles. In this case, the ventricles spontaneously establish their own signal, usually originating in the AV node or AV bundle distal to the block.

more distant more slower
4
slower than SA node
4
secondary
other pacemakers cells in bundel of his & Purkinje fibers

There is no relation between the rhythm of the P waves and that of the QRS-T complexes because the ventricles have "escaped" from control by the atria and are beating at their own natural rate.

Most of these patients need to implant an *artificial pacemaker*. The pacemaker provides continued rhythmical impulses to the ventricles.



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ventricles heart rate is down

artificial pacemaker :- is a long lasting battery planted under the skin in the left upper side of the chest close to the clavical bone, by surgery → we extract the subclavine vein → we push the wire of the battery through it → SVC → RA → RV → and the end of the wire touch the apex of RV → which overall 80 impulse/min → the heart follow the faster impulse so it will ignore the other scattered pacemaker cells & follow the battery

this is important to understan the MOF of anti-arrhythmatic drugs

REENTRY MECHANISM

how other points generate AP other than pacemaker (SAN) ? → this cause arrhythmias

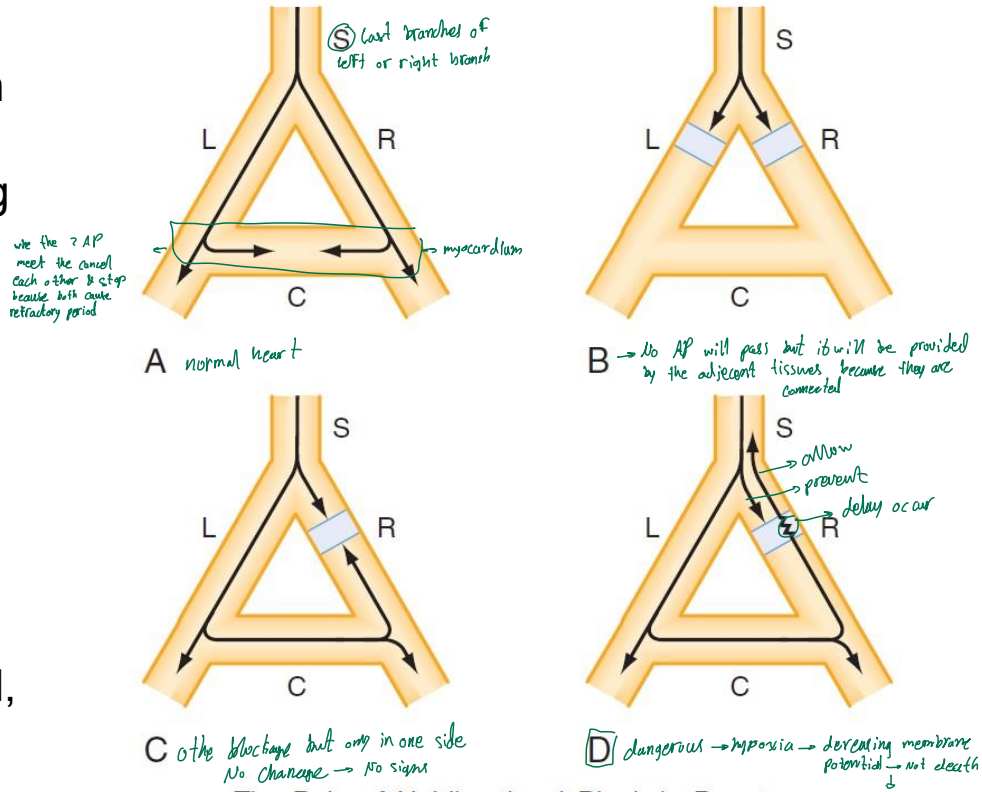
* assume there is shortage in blood perfusion in area of the myocardium, what will happen ?

A, An excitation wave traveling down a single bundle (S) of fibers continues down the left (L) and right (R) branches. The depolarization wave enters the connecting branch (C) from both ends and is extinguished at the zone of collision.

B, The wave is blocked in the L and R branches.

C, A bidirectional block exists in the R branch.

D, A unidirectional block exists in the R branch. The antegrade impulse is blocked, but the retrograde impulse is conducted through and reenters the S bundle.



The Role of Unidirectional Block in Reentry.

we the ? AP meet the cancel each other & stop because both cause refractory period

S last branches of left or right branch

B → No AP will pass but it will be provided by the adjacent tissues, because they are connected

C the blockage but only in one side
No change → no signs

D dangerous → hypoxia → decreasing membrane potential → not electrically

allow
prevent
delay occur

might not work as pacemaker but it will become like AV allowing the AP in one direction & prevent it in the other direction, the AP in the opposite direction will pass after a delay but it won't pass to the atria due to the AV node but it will go to the same area again causing second stimulation, to happen 2 ways
1) this area have short refractory period
2) Long loops so it will have the time to be out of refractory period before the 2nd potential arrive to the area

Note:
For reentry to occur, the effective refractory period of the reentered region must be shorter than the conduction time around the loop. Therefore, the conditions that promote reentry are those that prolong the conduction time or shorten the effective refractory period.

this is caused due the production of AP in other places than SAN

PREMATURE DEPOLARIZATIONS

if its source from the atria → normal QRS complex

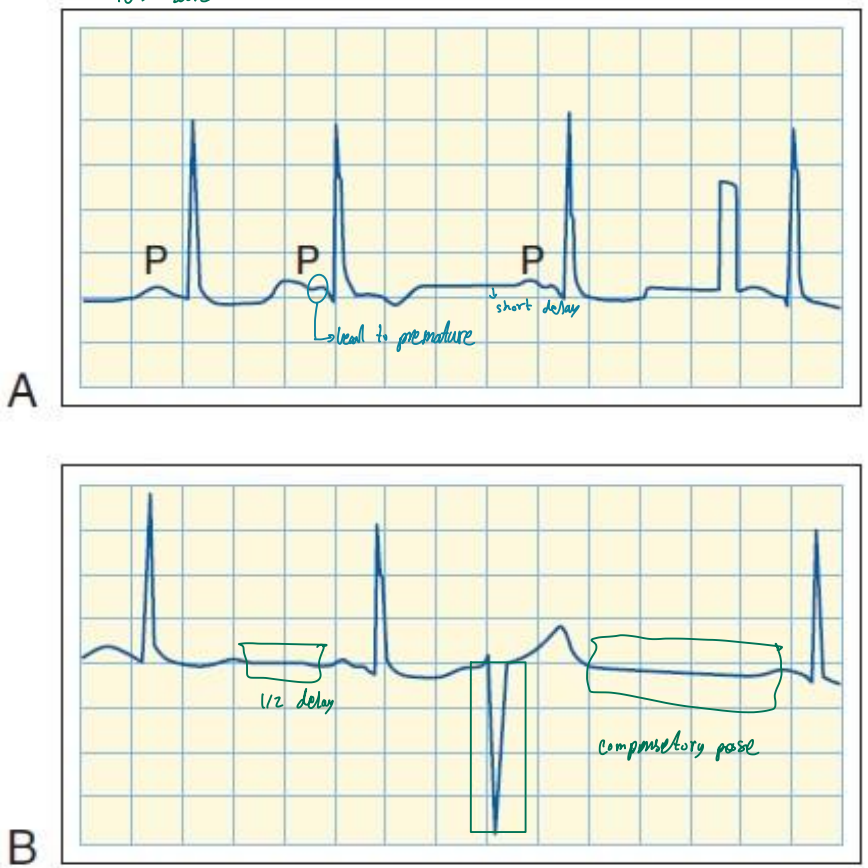
(**A**) Premature Atrial Depolarization. The premature atrial depolarization (second beat) is characterized by an inverted P wave (just below the second "P") and normal QRS complexes and T waves. The interval after the premature atrial depolarization is not much longer than the usual interval between beats. The brief rectangular deflection just before the last atrial depolarization is a standardization signal.

(**B**) The premature ventricular depolarization is characterized by bizarre, inverted QRS complexes and elevated T waves and is followed by a compensatory pause.

if its source from ventricle

this short delay caused by the affecting the SAN by the premature depolarization wave "preset" → like restart the SAN causing delay

it's safe because it has normal direction atria → ventricles



it will transmit on its own way not by the conducting system, producing abnormal QRS and the AP from SAN after the premature depolarization wave won't effect the ventricle because the ventricle is in refractory period this distance x double the original distance, dangerous if its more than 9 beats/min

more complicated than premature causing tachycardia

shorter AP → shorter refractory period

ECTOPIC TACHYCARDIA:

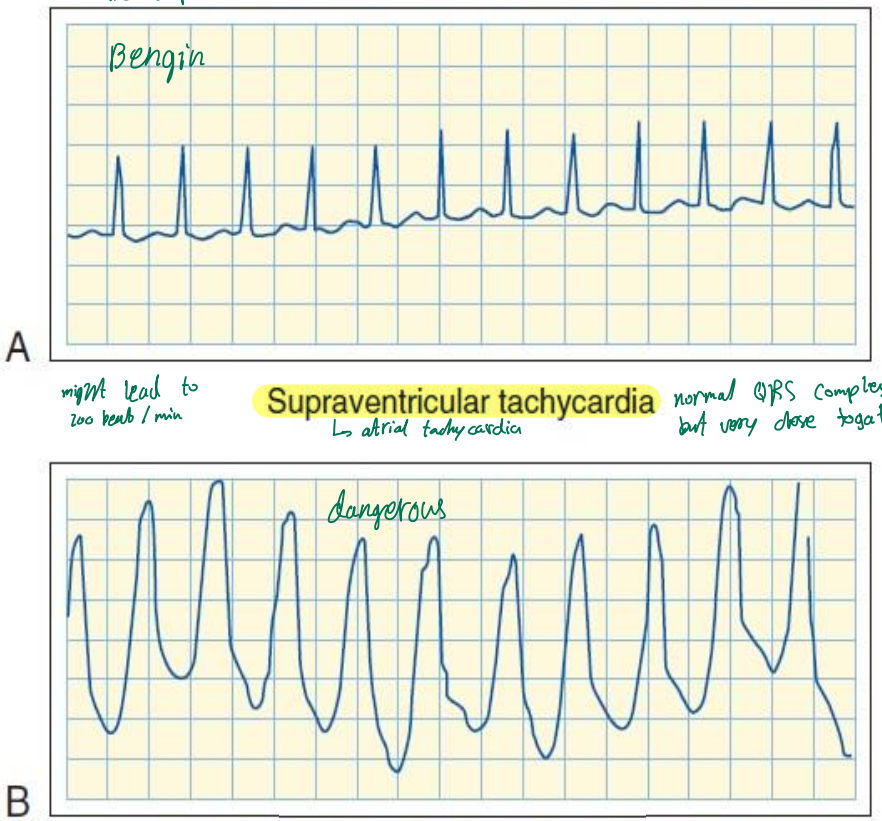
→ in stress conditions → sympathetic stimulation
→ shorter AP → shorter refractory period
→ circus current longer than refractory period
→ attach happens

This phenomenon is believed to be caused most frequently by re-entrant "**circus movement**". Because of the rapid rhythm in the irritable focus, *this focus becomes the pacemaker of the heart*.

The attack of tachycardia comes in the form of **paroxysm** (i.e. sudden attack). The paroxysm usually ends as suddenly as it began, with the pacemaker of the heart instantly shifting back to the sinus node. The paroxysm may last for a few seconds, a few minutes, a few hours, or even much longer.

sudden attach

if the loop of re current mechanism is longer than refractory period



might lead to 200 beat / min

Supraventricular tachycardia
↳ atrial tachycardia

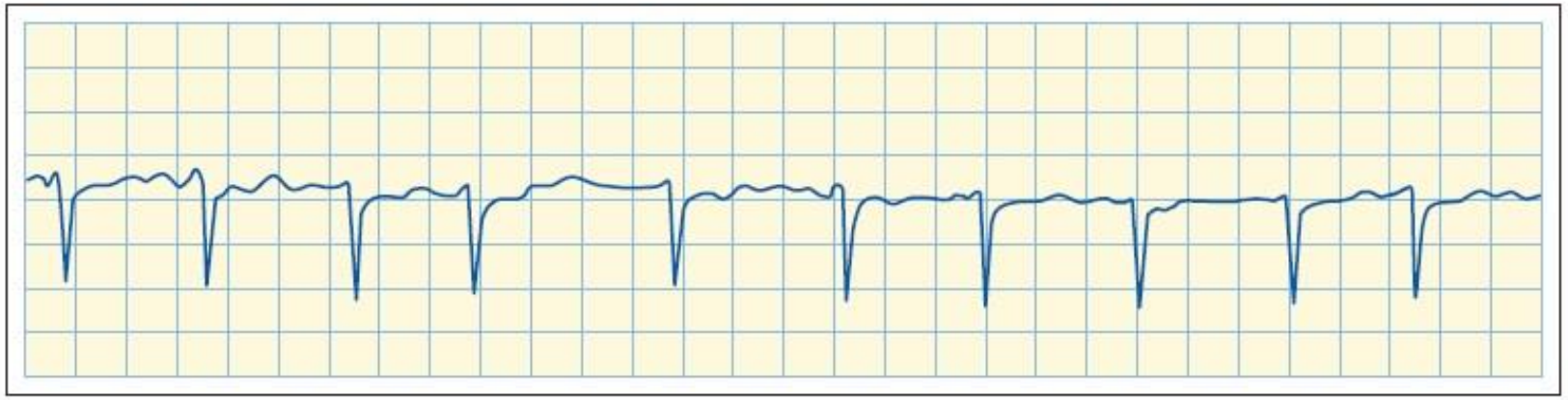
normal QRS complex but very close together

Ventricular tachycardia

abnormal QRS complex and sever tachycardia
very dangerous because it might become ventricle fibrillation
→ the fastest treatment is to give calcium ions channels blockers

FIBRILLATION:

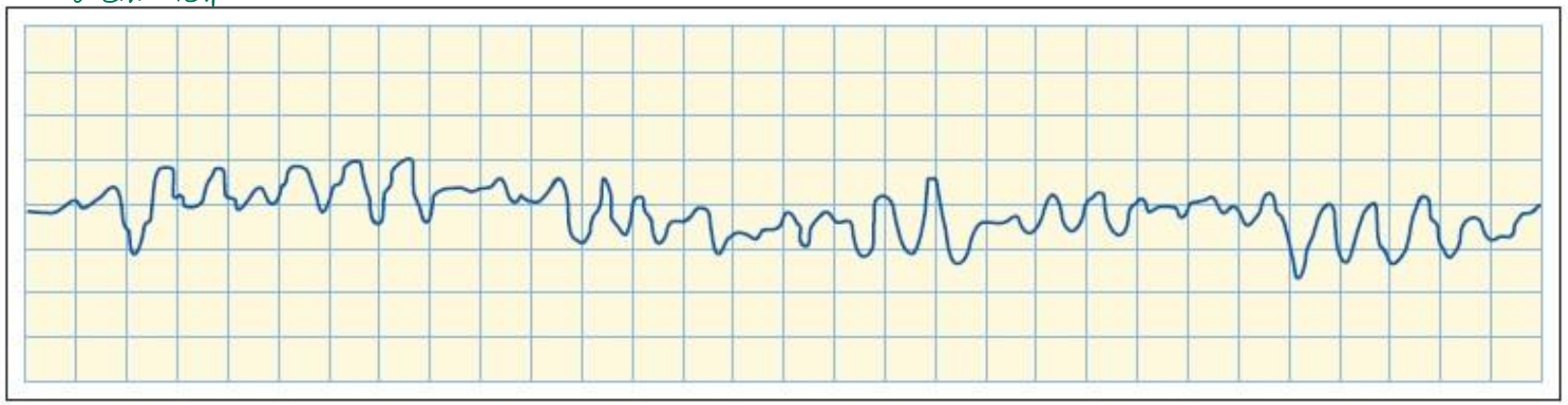
Loss of synchronization in myocardium
Part of the fibers are contracted and other part in the same mass of myocardium are relaxed → can't hold blood



A

we try to convert circus current in the atria to fibrillation in case the patient doesn't responed to drugs

Atrial fibrillation → no death because to fill the ventricles we don't need atrial contraction



B

in larger MI
main cause of death after MI

← **Ventricular fibrillation** → clear → No blood is being pumped to the ventricles within minutes =death

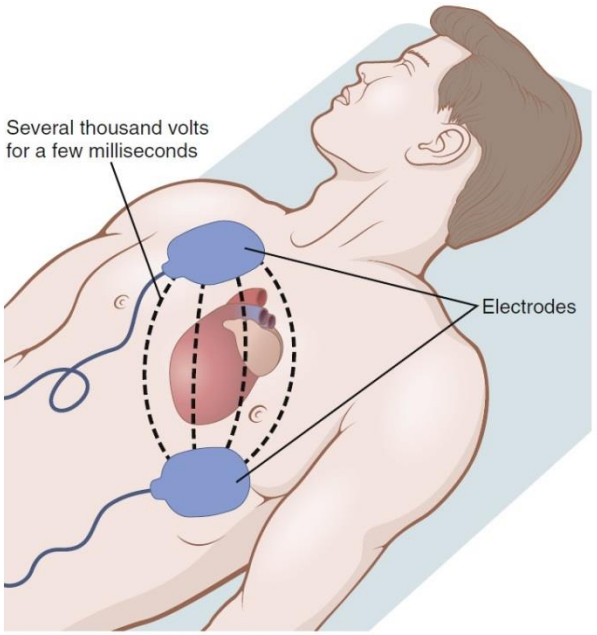
not all cardiac cells have the same refractory period → abnormal beat from the ventricle at the time where some of the cells are out of refractory period and other are still in refractory period → so some fibers will contract while other won't → DC shock

VENTRICULAR DEFIBRILLATION:

- A procedure in which a strong high-voltage electrical current is passed through the ventricles.
- A **direct current** with 1000 volts is applied for a few thousandths of a second.
- In most cases, defibrillation current is delivered to the heart in **biphasic waveforms** (alternating the direction of the current pulse through the heart).
- Such current can stop fibrillation by throwing all the ventricular muscle into simultaneous refractoriness.
- The biphasic waveforms current reduces the threshold needed for successful defibrillation, thereby decreasing the risk for burns and cardiac damage.
- All action potentials stop, and the heart remains quiescent for 3 to 5 seconds, after which it begins to beat again, usually with the SA node or some other part of the heart becoming the pacemaker.

the AP need to pass through Purkinje fiber to reach the myocardium ←

Purkinje fibres prevent the ventricular fibrillation because it has the longest refractory period



Application of electrical current to the chest to stop ventricular fibrillation.

TEST QUESTION

Q. P wave of ECG is absent in?

A. Atrial hypertrophy.

B. Ventricular extrasystole.

C. Heart failure.

D. Supraventricular tachycardia.

E. First degree heart block.