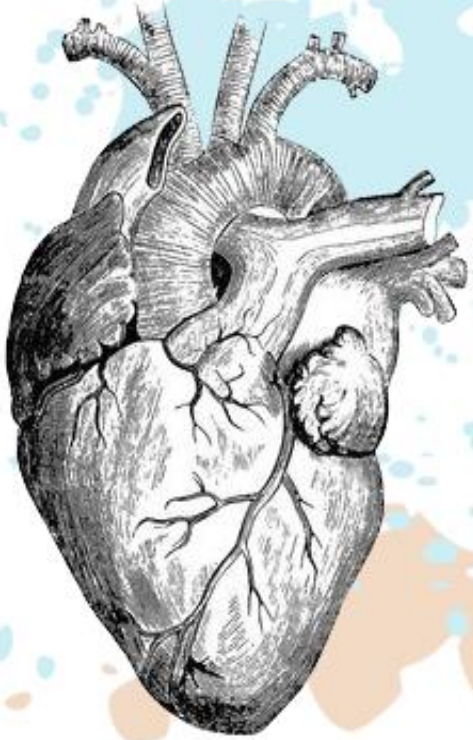


Very important

Extra information

Physiology

OF THE CARDIOVASCULAR SYSTEM

* Guyton corners, anything that is colored with grey is EXTRA explanation

Cardiac Arrhythmias

Objectives :

- Describe sinus arrhythmias.
- Describe the main pathophysiological causes of cardiac arrhythmias.
- Explain the mechanism of cardiac block.
- Explain the origin of an ectopic foci.
- Enumerate the common arrhythmias and describe the basic ECG changes.

Contact us : Physiology435@gmail.com

The Normal Conduction System

- **Guyton corner:**

Figure 10-1 shows the specialized excitatory and conductive system of the heart that controls cardiac contractions. The figure shows the sinus node (also called sinoatrial or S-A node) in which the normal rhythmical impulses are **generated**; the internodal pathways that conduct impulses from the sinus node to the atrioventricular (A-V) node; the A-V node in which impulses from the atria are delayed before passing into the ventricles; the A-V bundle, which conducts impulses from the atria into the ventricles; and the left and right bundle branches of Purkinje fibers, which conduct the cardiac impulses to all parts of the ventricles

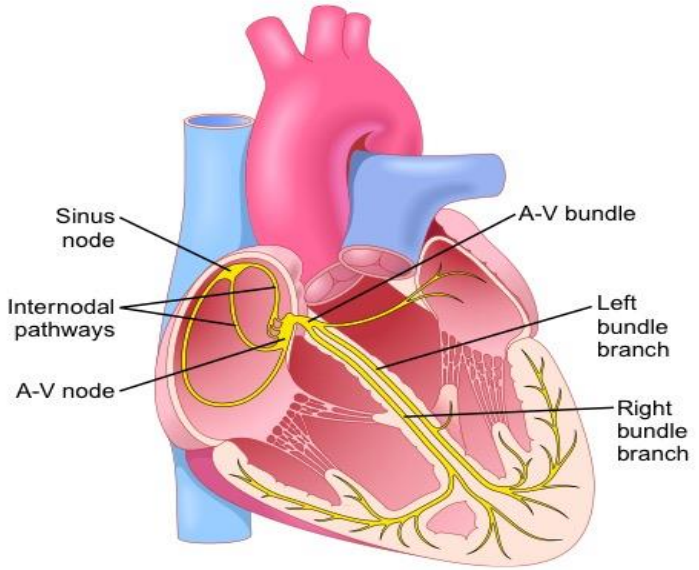


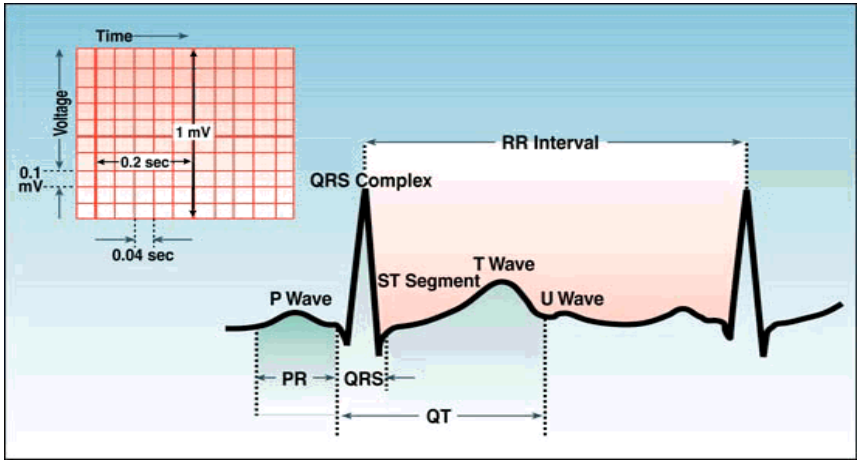
Figure 10-1. Sinus node and the Purkinje system of the heart, showing also the atrioventricular *(A-V)* node, atrial internodal pathways, and ventricular bundle branches.

Normal conduction system starts from the SA node "**SA node ,, its located in the superior part of the right atrium**" and it has normal automaticity, the impulse originate there and then travels to another node called AV node, "**AV node ,, its located in the junction between the Right atrium and Right ventricle**" the electrical system is available in the **right** side of the heart starts from SA node and goes to AV node through the atrium "**you can't see the SA node activity in the ECG because it's too small, you're going to see what's happening in the atrium** "so the atrial activity is what you will see in the ECG", so after that of course the impulse has to go from atrium to ventricles, the atria has to talk to the ventricle by bundle of his, and from this bundle it'll spread to the right ventricle "**right bundle**" & the left ventricle "**left bundle**" through purkinje fibers, left ventricle "bundle" will divide into two bundle's because the left ventricle is bigger than the right. This will give us anterior fascicle "smaller and supply small area of the left ventricle" & posterior fascicle "the main fascicle and it'll supply most of the septum & the apex & the lateral wall of the left ventricle".

Rhythm

Sinus:

* Originating from **SA node**.
* P wave **before** every QRS.
* P wave in same direction as QRS.

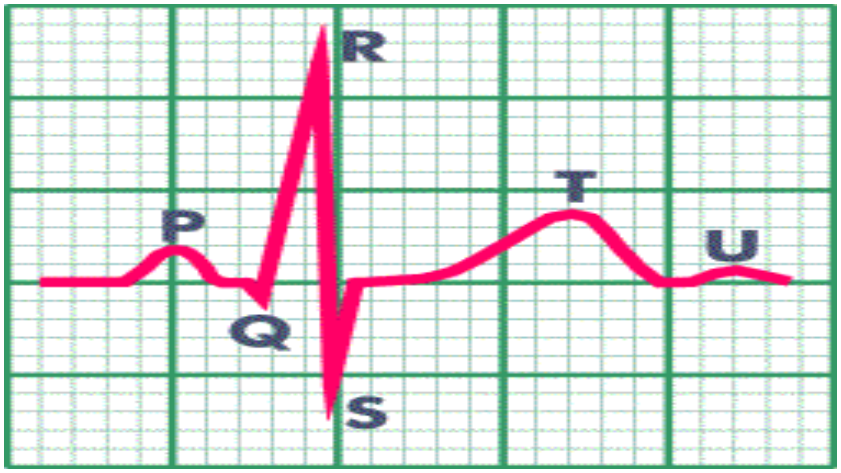


- **Guyton corner:**

The sinus node (also called sinoatrial node) is a small, flattened, ellipsoid strip of specialized cardiac muscle about 3 millimeters wide, 15 millimeters long, and 1 millimeter thick. It is located in the superior posterolateral wall of the right atrium immediately below and slightly lateral to the opening of the superior vena cava. The fibers of this node have almost no contractile muscle filaments and are each only 3 to 5 micrometers in diameter, in contrast to a diameter of 10 to 15 micrometers for the surrounding atrial muscle fibers. However, the sinus nodal fibers connect directly with the atrial muscle fibers so that any action potential that begins in the sinus node spreads immediately into the atrial muscle wall. **-Page 123-**

- The most important part of ECG is the **rhythm**, the normal rhythm called **sinus rhythm**, the impulse goes from SA node to the atrium and then it'll give us **P wave** "this wave is a result of the impulse firing from SA node to the atrium", so it means atrial activity, then there will be **PR interval**, when this impulse conducts to the ventricle it gives me what is called **QRS complex** "this complex indicates ventricular activity", then **ST segment** and **T wave**
- "T wave represent what we call repolarization (= relaxation) of the ventricle

Waveforms



- Some people have **U wave** and it disappears when they get older and start to be seen in pathological cases,
- **"U wave is not important"**

Interpretation

- Develop a systematic approach to reading EKGs and use it every time.
- **<u>The system we will practice is:</u>**
  - ✓ Rate.
  - ✓ Rhythm (including intervals and blocks).
  - ✓ Axis.
  - ✓ Hypertrophy.
  - ✓ Ischemia.

Interpretation-EXTRA NOTES

when you look at ECG it is like when you look at an elephant, there's a lot of things to see.

- if the SA node stops working for some reason the other part of the heart will take over,
- *also you have to know the approach, what's approach? Approach is systematic way of how to read ECG, if you have this approach you'll not miss anything,*

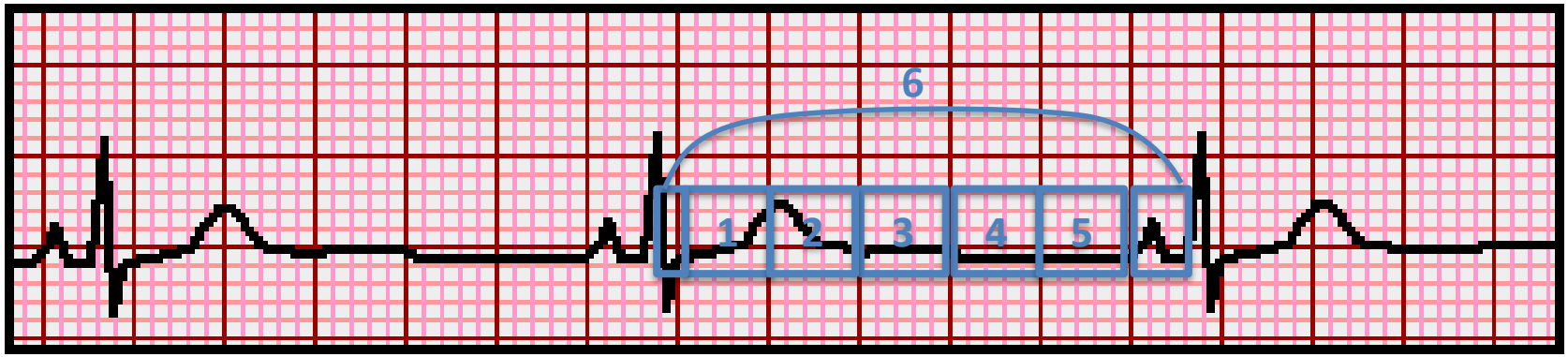
<u>The approach we follow :</u>

- **Rate** (rapid-normal-slow)
- **Rhythm** (coming from SA node or not **"this is very important"**)
- **Axis** (to tell me how is the heart sitting in the chest? ''it *is sitting in the left side towards the middle'',* so the force of the activity should go to the left side "downward and a little bit towards the left", otherwise it will be abnormal)
- **Hypertrophy** (from axis you can tell me if there's hypertrophy or not, means there's enlargement of the heart or hypertrophy of the wall)
- **Ischemia** (means one part of the muscles won't get enough blood, what's going to happen? I'll have problems, scarring, damaged part of the muscles, and electrical system around that part won't work anymore, it'll be irritable, this irritation "زي الالتماس الكهربائي بالضبط" will cause what we call arrhythmia either ventricular arrhythmia or atrial arrhythmia)

Rate

Rule of 300

Rate= divide **300** by the number of boxes between each QRS e.g. :



Rate= 300/6= 50 bpm

We calculate the rate by the **rule of 300**, what is the rule of 300?

في الاي سي جي فيه مربعات "زي دفتر الرياضيات" كل مربع كبير فيه خمس مربعات صغيرة ، طيب كيف نحسب ؟ نعد كم فيه مربع بين كل ‘‘كيو أر إس’’ والثاني وناخذ هالعدد ونقسم 300 عليه

اذا كان العدد واحد ف300 تقسيم واحد يساوي 300 ,, اذا العدد كان ثنين نقسم ال300 على ثنين يطلع الناتج 150 وهكذا ,

ايش هو المعدل الطبيعي ؟ هو ” Range” من 60-100

Rate

Number of big boxes	Heart Rate
1	300
2	150
3	100
4	75
5	60
6	50

>100 bpm is tachycardia

60-100 bpm is normal

<60 bpm is bradycardia

Heart rate variable depends on:

- Age
- Activity
- Time of the day

Normal sinus rhythm

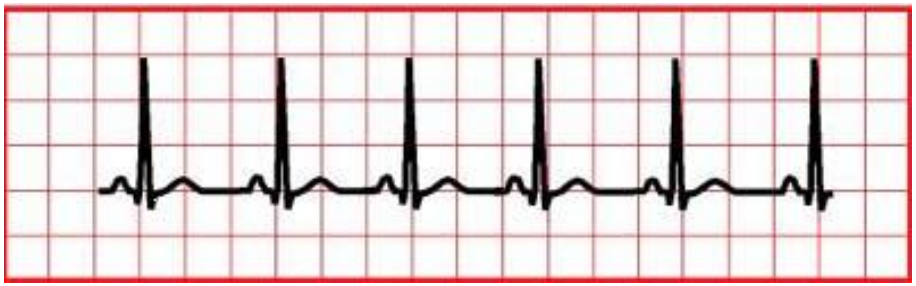
Single P-wave precedes (before) each QRS complex

P-R interval is constant and within normal range

regular

P-P interval is constant

Normal sinus rhythm



What is normal sinus rhythm? **Regular** 3 squares between each rhythm.
Normal P waves followed by normal QRS and it's regular and a normal PR interval "this is important"

Common arrhythmias

rate / location	Bradyarrhythmia	Tachyarrhythmia
SA node	Sinus bradycardia Sick sinus syndrome	Sinus tachycardia
Atria	---	Atrial premature beats Atrial flutter Atrial fibrillation Paroxysmal SVT (supraventricular tachycardia) Multifocal atrial tachycardia
Av node	Conduction blocks (Three Types: Degree 1, Degree 2, Degree 3) junctional escape rhythm	---
ventricles	Ventricular escape rhythm	Ventricular premature beats Ventricular tachycardia Torsade's de pointes Ventricular fibrillation

Differential diagnosis of tachycardia

aberrancy means there's bundle" branch blocked"

Tachycardia	Narrow Complex	Wide Complex
Regular	**ST** (sinus tachycardia) **SVT** (supraventricular tachycardia) **Atrial flutter**	**ST** w/ **aberrancy** **SVT** w/ **aberrancy** **VT** (ventricular tachycardia)
Irregular	**A-fib** (atrial fibrillation) **A-flutter** w/ variable conduction **MAT** (multifocal atrial tachycardia)	**A-fib** w/ **aberrancy** **A-fib** w/ **WPW** **(Wolff-Parkinson-White Syndrome)** **VT**

PHYSIOLOGY TEAM 435

Causes of Cardiac Arrhythmias

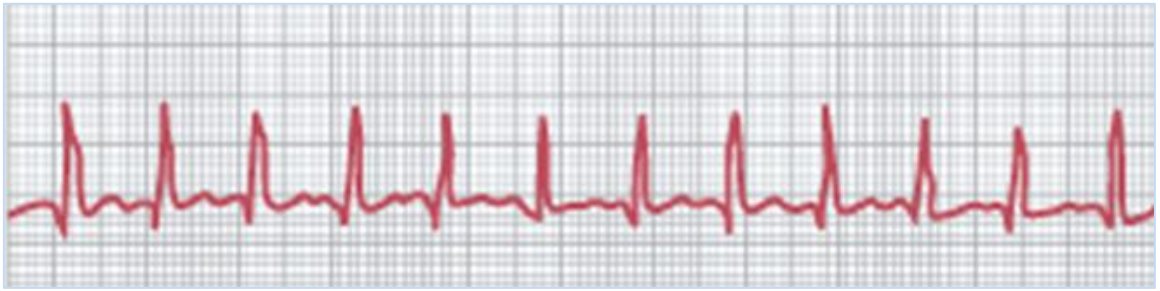
- Spontaneous generation of impulses at any part of the heart
- Abnormal rhythmicity of the pacemaker *(means SA node stops working)*
- Shift of the pacemaker from the sinus node to another place in the heart
- Blocks at different points during the spread of the impulse through the heart
- Abnormal pathways of impulse transmission through the heart
- Rate above or below normal
- Regular or irregular rhythm
- Narrow or broad QRS complex
- Relation to P waves

The causes of arrhythmias:

- **if the SA node doesn't work this will lead to shift of the pacemaker to another place (either the AV node or the ventricle.) Or there will be problem with the AV node , so the AV node will be a blocked. This block will lead to a problems in communication between the ventricle and the atrium.**
- **the most important sign to know if the ECG is normal or not Is the P waves because it's the normal Sinus Rhythm , if there's no P waves now the problem will start**

Abnormal Sinus Rhythm

- **Tachycardia:** an increase in the heart rate
- **Heart rate : more than100 beats per minute**
- **Causes:**
  - Increased body temperature
  - Sympathetic stimulation
  - Drugs such as :digitalis
  - Inspiration

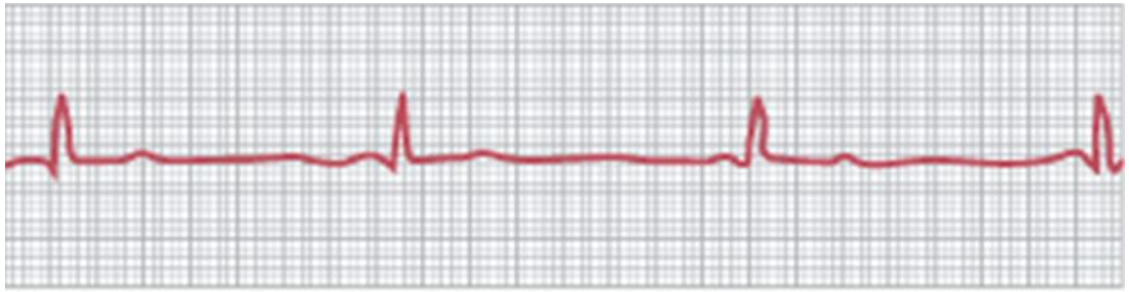


Guyton corner:

page 143 : an electro diagram recorded from a patient with tachycardia will be normal except that the heart rate ,as determined from the time intervals between QRS complexes, is about 150 per min instead of normal 72 per minute .

Abnormal Sinus Rhythm

- **Bradycardia**: means a decrease in heart rate
- **Heart rate** below **60** beats per minute
- **Causes:**
  - Parasympathetic (vagal) stimulation
  - Expiration



Guyton corner:
:PAGE144: bradycardia in athletes :the athlete heart is larger and considerably stronger than that of a normal person which allows the athletes to pump a large stroke of volume output per beat even during period of rest so in rest excessive quantities of blood pumped into the arterial tree each beat initiate feedback circulatory reflexes or other effects to cause bradycardia
-Vagal stimulation : any circulatory reflex that stimulate vagus nerves causes release of acetylcholine at vagal ending giving the parasympathetic effect

Abnormal Sinus Rhythm

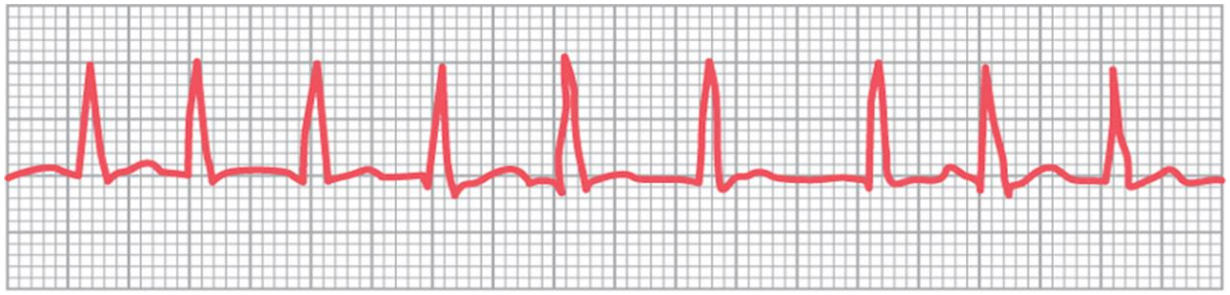
Abnormal Cardiac Rhythms that Result from Impulse Conduction Block :

- 1- **Sinoatrial Block :**

  -Blockade of the S-A node impulse before entering atrial muscle

  -Cessation of P wave *(No SA node activity)*

- **Causes:**
  - Ischemia of the A-V node
  - Compression of the A-V node by scar formation
  - Inflammation of the A-V node
  - Strong vagal stimulation



- **Guyton corner:**
- Page 144 : in rare cases the impulses from SA node is blocker before it enters the atrial muscle this shows cessation of P waves however the ventricles picks up a new rhythm the impulses usually origination spontaneously in AV node so the rate of ventricular QRS-T is slowed but not otherwise altered

Abnormal Sinus Rhythm

Abnormal Cardiac Rhythms that Result from Impulse Conduction Block :

- 2- **A-V Block**

  When impulse from the S-A node is blocked

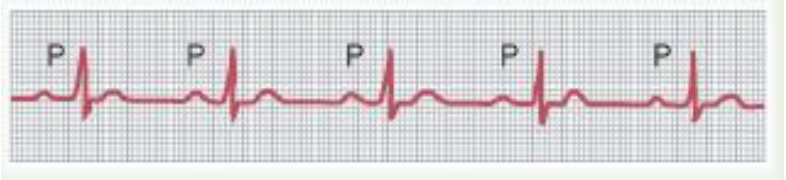
- **Causes**:
  - Ischemia of the A-V node *this often delay or blocks conduction from atria to the ventricles .coronary insufficiency can cause ischemia of the AV nodes and bundle of His in the same way that it can cause ischemia to the myocardium.*
  - Compression of the A-V node by scar formation *or by calcified portions of the heart can depress or block conduction from atria to the ventricles*
  - Inflammation of the A-V node *can depress the conductivity from the atria to the ventricles .Inflammation results frequently from different types of myocarditis caused for example by diphtheria or rheumatic fever*
  - Strong vagal stimulation *this in rare instance block impulses conduction through the AV nodes such vagal excitation occasionally results with carotid sinus syndrome*

- **Guyton corner:**
- The only means by which impulses ordinarily can pass from atria into ventricles is through the AV bundle(bundle of his),condition that can either decrease the rate of impulse conduction in this bundle or block the impulse entirely

Types of the A-V Block

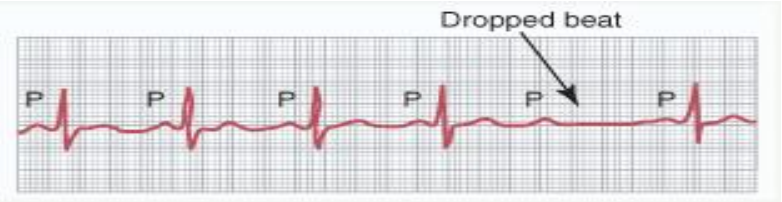
First degree block

- Prolong P-R interval (0.2 seconds).
- "NO" beats Drop



Second degree block

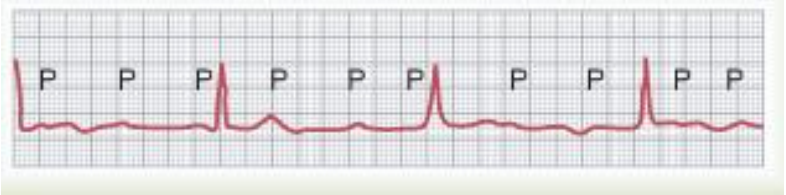
- P-R interval > **0.25** second.
- Only few impulses pass to the ventricles.
- → atria beat faster than ventricles.
- →"dropped beats" of the ventricles.



what happens in second degree block? Whatever happen in the atrium it's not going to the ventricle, because AV node has a problem, usually those patients will have prolonged PR as well and then prolonged more and more and then dropped beat.

Third degree block (complete)

- Complete dissociation of P wave and QRS waves.
- Ventricle escape from the influence of S-A node.
- Atrial rate is **100** beats/min.
- Ventricular rate is **40** beats/min.
- *Stokes-Adams Syndrome*: AV block comes and goes.



Types of the A-V Block- Extra

Just read it; you will understand the idea

Guyton corner : P 145, 12TH edition

- first-degree block is defined as a delay of conduction from

the atria to the ventricles but not actual blockage of conduction. The P-R interval seldom increases above 0.35 to 0.45 second because, by that time, conduction through the A-V bundle is depressed so much that conduction stops entirely. One means for determining the severity of some heart diseases—acute rheumatic heart disease, for instance—is to measure the P-R interval.

- Second-Degree Block. When conduction through the A-V bundle is slowed enough to increase the P-R interval to 0.25 to 0.45 second, the action potential is sometimes strong enough to pass through the bundle into the ventricles and sometimes not strong enough. In this instance, there will be an atrial P wave but no QRS-T wave, and it is said that there are "dropped beats" of the ventricles. This condition is called second-degree heart block.
- Complete A-V Block (Third-Degree Block). When the condition causing poor conduction in the A-V node or A-V bundle becomes severe, complete block of the impulse from the atria into the ventricles occurs. In this instance, the ventricles spontaneously establish their own signal, usually originating in the A-V node or A-V bundle. Therefore, the P waves become dissociated from the QRS-T complexes. Furthermore, 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 they are beating at their own natural rate, controlled most often by rhythmical signals generated in the A-V node or A-V bundle.
- Stokes-Adams Syndrome—Ventricular Escape.

In some patients with A-V block, the total block comes and goes; that is, impulses are conducted from the atria into the ventricles for a period of time and then suddenly impulses are not conducted. The duration of block may be a few seconds, a few minutes, a few hours, or even weeks or longer before conduction returns. This condition occurs in hearts with borderline ischemia of the conductive system. Each time A-V conduction ceases, the ventricles often do not start their own beating until after a delay of 5 to 30 seconds. This results from the phenomenon called overdrive suppression. This means that ventricular excitability is at first in a suppressed state because the ventricles have been driven by the atria at a rate greater than their natural rate of rhythm. However, after a few seconds, some part of the Purkinje system beyond the block, usually in the distal part of the A-V node beyond the blocked point in the node, or in the A-V bundle, begins discharging rhythmically at a rate of 15 to 40 times per minute and acting as the pacemaker of the ventricles. This is called ventricular escape Because the brain cannot remain active for more than 4 to 7 seconds without blood supply, most patients faint a few Seconds after complete block occurs because the heart does not pump any blood for 5 to 30 seconds, until

the ventricles "escape." After escape, however, the slowly beating ventricles usually pump enough blood to allow rapid recovery from the faint and then to sustain the person. These periodic fainting spells are known as the Stokes-Adams syndrome.

Premature contractions

- **Premature contractions, *extrasystoles, or* ectopic beat:** result's from *ectopic foci* that generate abnormal cardiac impulses (pulse deficit).
- Ectopic foci are abnormal pacemaker sites within the heart (outside of the SA node)
- The premature contractions can originate in **atria**, **A-V** junctions or **ventricles**.

Caused by

- Drugs. E.g. caffeine
- Myocardium irritations (caused by calcified foci)
- Ischemia

- premature contraction "إلتماس"
- normal conduction: SA node > atria > AV node > bundles

Guyton corner : P 127, 13TH edition

A pacemaker elsewhere than the sinus node is called an *"ectopic"* pacemaker.

Occasionally some other part of the heart develops a rhythmical discharge rate that is more rapid than that of the sinus node.

For instance, this development sometimes occurs in the A-V node or in the Purkinje fibers when one of these becomes abnormal. In either case, the

pacemaker of the heart shifts from the sinus node to the A-V node or to the excited Purkinje fibers.

Under rarer conditions, a place in the atrial or ventricular muscle develops excessive excitability and becomes the pacemaker.

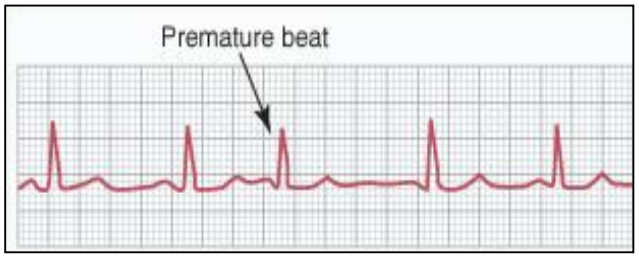
A pacemaker elsewhere than the sinus node is called an "ectopic" pacemaker. An ectopic pacemaker causes an abnormal sequence of contraction of the different parts of the heart and can cause significant debility of heart

pumping.

Premature contractions

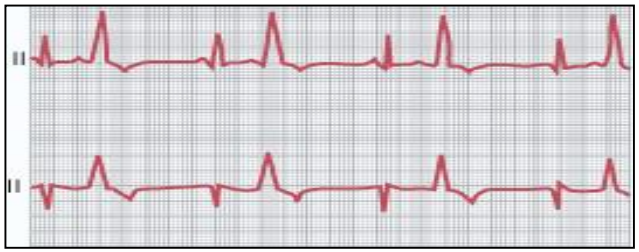
Atrial

- **Signs**:
  1- Short P-R interval
  (depends on how far the ectopic foci from the AV node).
  2- Pulse deficit
  if there is no enough time for ventricles to fill with blood.
  3- The time between the premature contraction and the next beat is increased (compensatory pause).



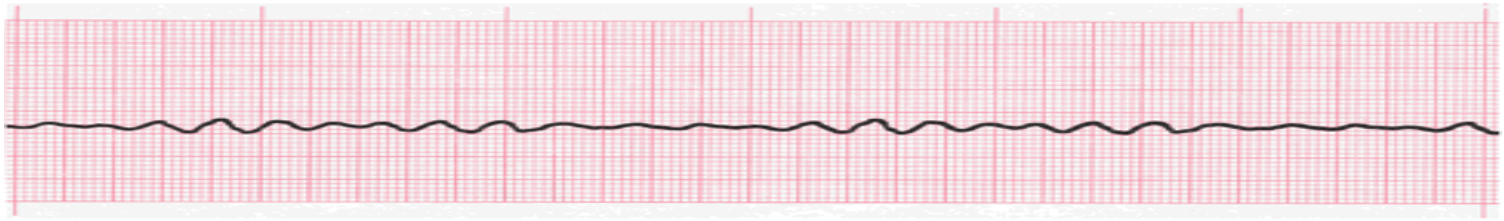
Ventricular (PVC)

- **Signs:**
  1- Prolonged QRS; because the impulses are carried out with myocardial fibers with slower conduction rate than purkinje fibers.
  2- Increased QRS voltage; QRS from a ventricle cannot neutralize the one from other ventricle.
  3- after PVCs, T wave has an electrical potential of opposite polarity of that of QRS because of the slow conduction in myocardial fibers; the fibers that depolarize first will repolarize first.
- **Causes**:
  Drugs, Caffeine, Smoking. Lack of sleep, Emotional irritations.



- **Guyton corner :** If the cardiac impulse should travel through the ventricles slowly, much of the ventricular mass would contract before contraction of the remainder, in which case the overall pumping effect would be greatly depressed.
  Guyton 13th- p.128

Ventricular fibrillation (V-fib)



What is it ?

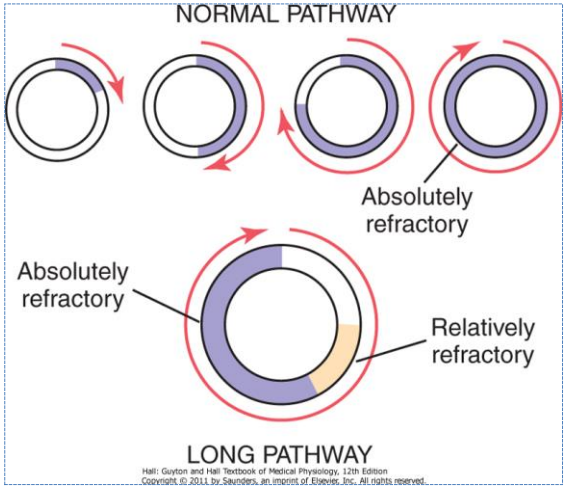
It is the uncoordinated contraction of the cardiac muscle of the ventricles in the heart.

As if the ventricles are quivering not pumping.

The most serious form of arrhythmias.

Causes:

- Sudden electric shock or Ischemia.
- impulses stimulate one part of the ventricles, then another, then itself. Many part contracts at the same time while other parts relax (Circus movement).



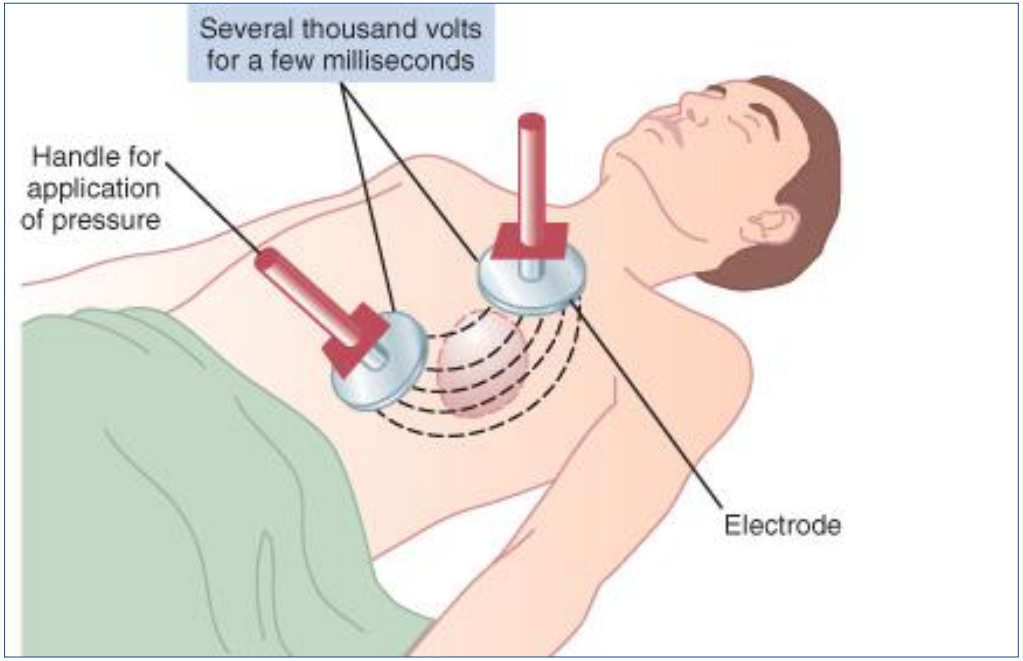
Circus movement, Many parts contract at the same time while other parts relax

Manifestations:

- Tachycardia
- Irregular rhythm
- Broad QRS complex
- No P wave

V-fib continued

- Treatment: Direct current shock (DC).



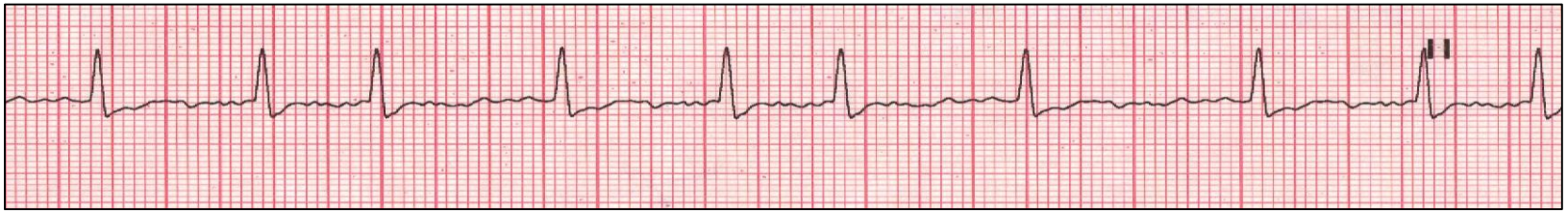
- **Guyton corner :**
- If ventricular fibrillation is not stopped within 1 to 3 minutes, it's almost invariably FATAL.

After fibrillation begins, unconsciousness occurs within 4 to 5 seconds for lack of blood flow to the brain.

VIDEO1

VIDEO2

Atrial fibrillation (A-fib)



- Same mechanism as ventricular fibrillation, but as the name suggests it affects **ATRIA.**
- Frequent in patients with enlarged hearts
- Atria do not pump if they are fibrillating.
- The efficiency of ventricular filling is decreased 20 to 30%.
- As seen in the ECG above you either see no P wave or high frequency of low voltage P wave.

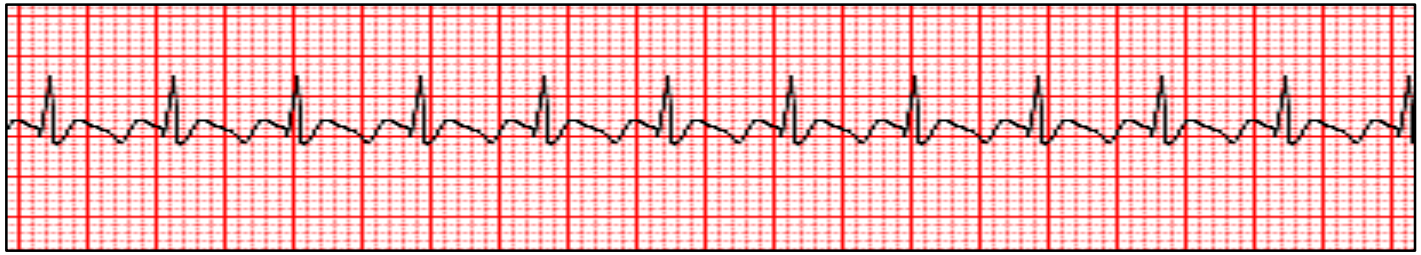
- Treatment:

Direct current shock (DC).

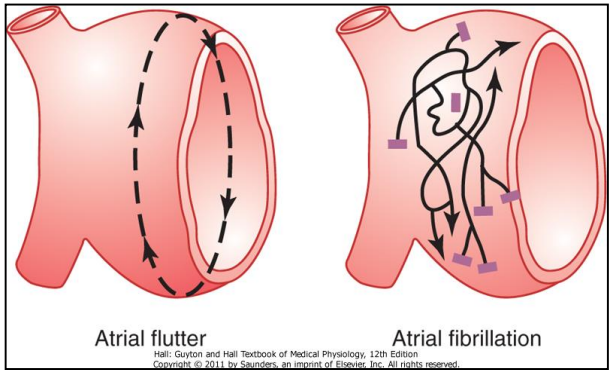
- **Guyton corner :**
- Atrial fibrillation often occurs without ventricular fibrillation since they are separated by fibrous tissue

VIDEO

Atrial flutter



- A single large wave travels around and around in the **atria**, which results in abnormal heart rhythm.
- Contraction of atria occurs at **250** Beats/minute. (high rate)
  However, the amount of blood being pumped by the atria is slight since some areas of them are contracting while others are relaxed.
- The refractory period of the AV node causes 2-3 beats of atria for one single ventricular beat
  2:1 or **2:3** rhythm.



- **Guyton corner :**

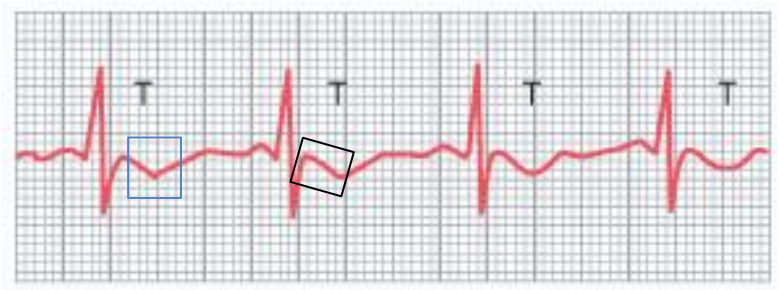
Atrial flutter it is also caused by circus movement in atria

Ischemia and the ECG

- One of the common uses of the ECG is in acute assessment of chest pain
- **Cause**: restriction of blood flow to the myocardium, either:
  - **Reversible**: angina pectoris
  - **Irreversible**: myocardial infarction
- Ischemia → injury → infarction

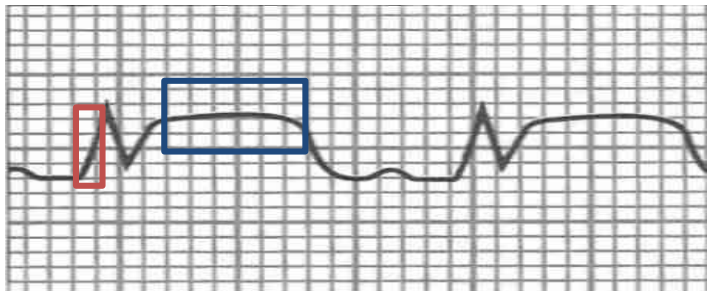
Reversible ischemia

- Inverted T wave as shown in blue box
- ST segment depression as shown in black box



Irreversible: Myocardial Infarction

- **Cause :**Complete loss of blood supply to the myocardium resulting in necrosis or death of tissue
- ST segment elevation as shown in blue box
- Deep Q wave as shown in red box



Ischemia and the ECG- Extra

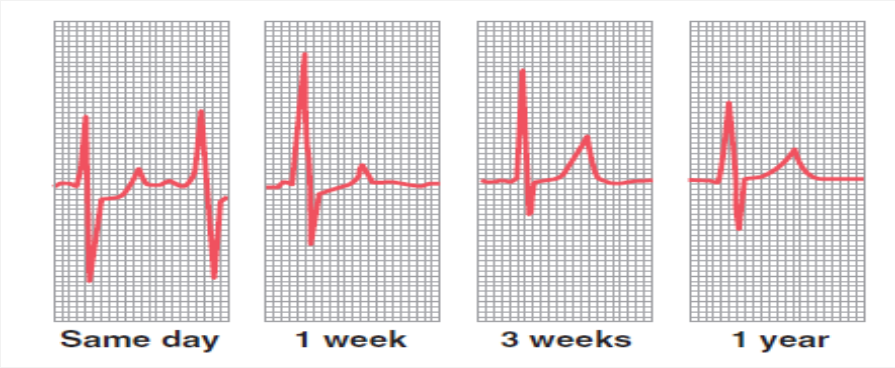
- **Guyton corner :**

Current of Injury in Angina Pectoris. "Angina pectoris" means pain from the heart felt in the pectoral regions of the upper chest. This pain usually also radiates into the left neck area and down the left arm. The pain is typically caused by moderate ischemia of the heart. Usually, no pain is felt as long as the person is quiet, but as soon as he or she overworks the heart, the pain appears.
An injury potential sometimes appears in the ECG during an attack of severe angina pectoris because the coronary insufficiency becomes great enough to prevent adequate repolarization of some areas of the heart during diastole.

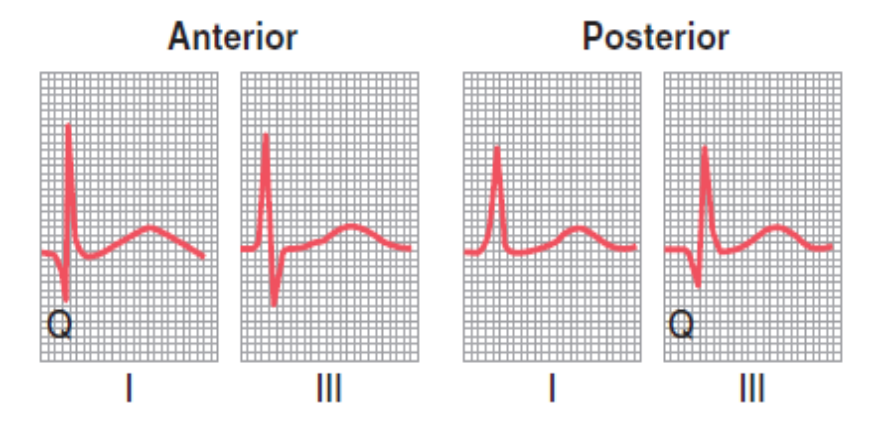
Recovery from Acute Coronary Thrombosis. Demonstrating changes in the ECG from the day of the attack to 1 week later, 3 weeks later, and finally 1 year later. From this ECG, one can see that the injury potential is strong immediately after the acute attack (the T-P segment is displaced positively from the S-T segment). However, after about 1 week, the injury potential has diminished considerably, and after 3 weeks, it is gone. After that, the ECG does not change greatly during the next year. This is the usual recovery pattern after acute myocardial infarction of moderate degree, showing that the *new collateral coronary blood flow* develops enough to re-establish appropriate nutrition to most of the infarcted area. In some patients who experience myocardial infarction, the infarcted area never redevelops adequate coronary blood supply. Often, some of the heart muscle dies, but if the muscle does not die, it will continue to show an injury potential as long as the ischemia exists, particularly during bouts of exercise when the heart is overloaded. **Old Recovered Myocardial Infarction. Figure 12-22** shows leads I and III after *anterior infarction* and leads I and III after *posterior infarction* about 1 year after the acute heart attack. The records show what might be called the "ideal" configurations of the QRS complex in these types of recovered myocardial infarction. Usually a Q wave has developed at the beginning of the QRS complex in lead I in anterior infarction because of loss of muscle mass in the anterior wall of the left ventricle, but in posterior infarction, a Q wave has developed at the beginning of the QRS complex in lead III because of loss of muscle in the posterior apical part of the ventricle.
These configurations are certainly not found in all cases of old cardiac infarction. Local loss of muscle and local points of cardiac signal conduction block can cause very bizarre QRS patterns (especially prominent Q waves, for instance), decreased voltage, and QRS prolongation.

Ischemia and the ECG- Extra

- **Guyton corner : Figure 12-21.** Recovery of the myocardium after *moderate posterior wall infarction*, demonstrating disappearance of the injury potential that is present on the first day after the infarction and still slightly present at 1 week.



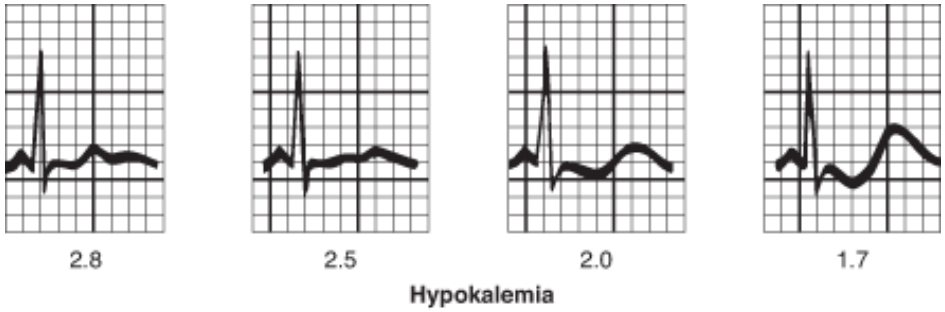
- **Figure 12-22.** Electrocardiograms of anterior and posterior wall infarctions that occurred about 1 year previously, showing a Q wave in lead I in anterior wall infarction and a Q wave in lead III in *posterior wall infarction.*



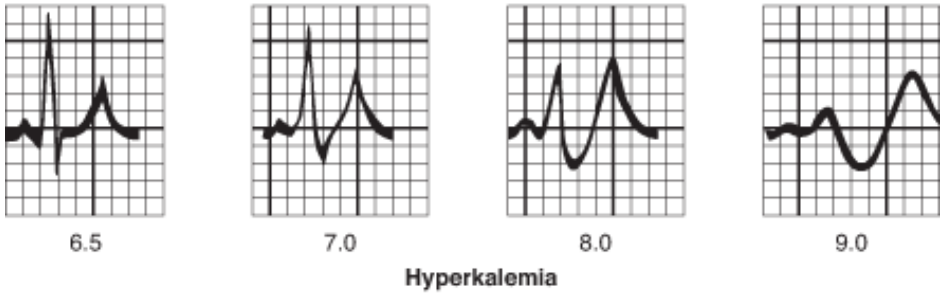
K+ and the ECG

- **Guyton corner :**

More common are the acquired forms of LQTS that are associated with plasma electrolyte disturbances, such as hypomagnesemia, hypokalemia, or hypocalcemia, or with administration of excess amounts of antiarrhythmic drugs such as quinidine or some antibiotics such as fluoroquinolones or erythromycin that prolong the Q-T interval.



Hypokalemia: Flat T wave



Hyperkalemia: Tall peaked T wave

Physiology
OF THE CARDIOVASCULAR SYSTEM

Physiology Leaders :

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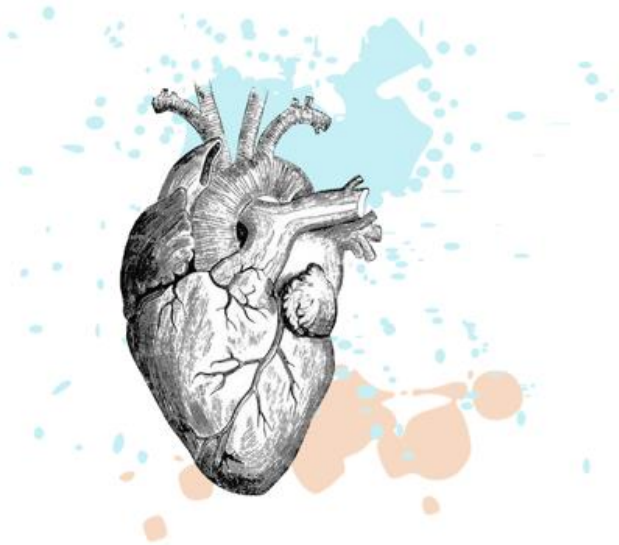
Nojood Alhaidri
Rawaf Alrawaf



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- Deema AlFaris
- Elham Alzahrani
- Johara Almalki
- Lojain alsiwat
- Malak Alsharif
- Monirah Alsalouli
- Nora AlRomaih
- Nurah Alqahtani
- Nouf Alabdulkarim
- Nora Albusayes
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- Norah Alakeel
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- Abdulrahman Albarakah
- Adel Alshehri
- Abdulaziz Alghanaym
- Abdulmajeed Alotaibi
- Khalil Alduraibi
- Hassan Albeladi
- Omar Alshehri
- Saleh Alshawi
- Abdulaziz Alhammad
- Faisal Alabdulatif
- Abdulnasser Alwabel
- Saad Almutairy