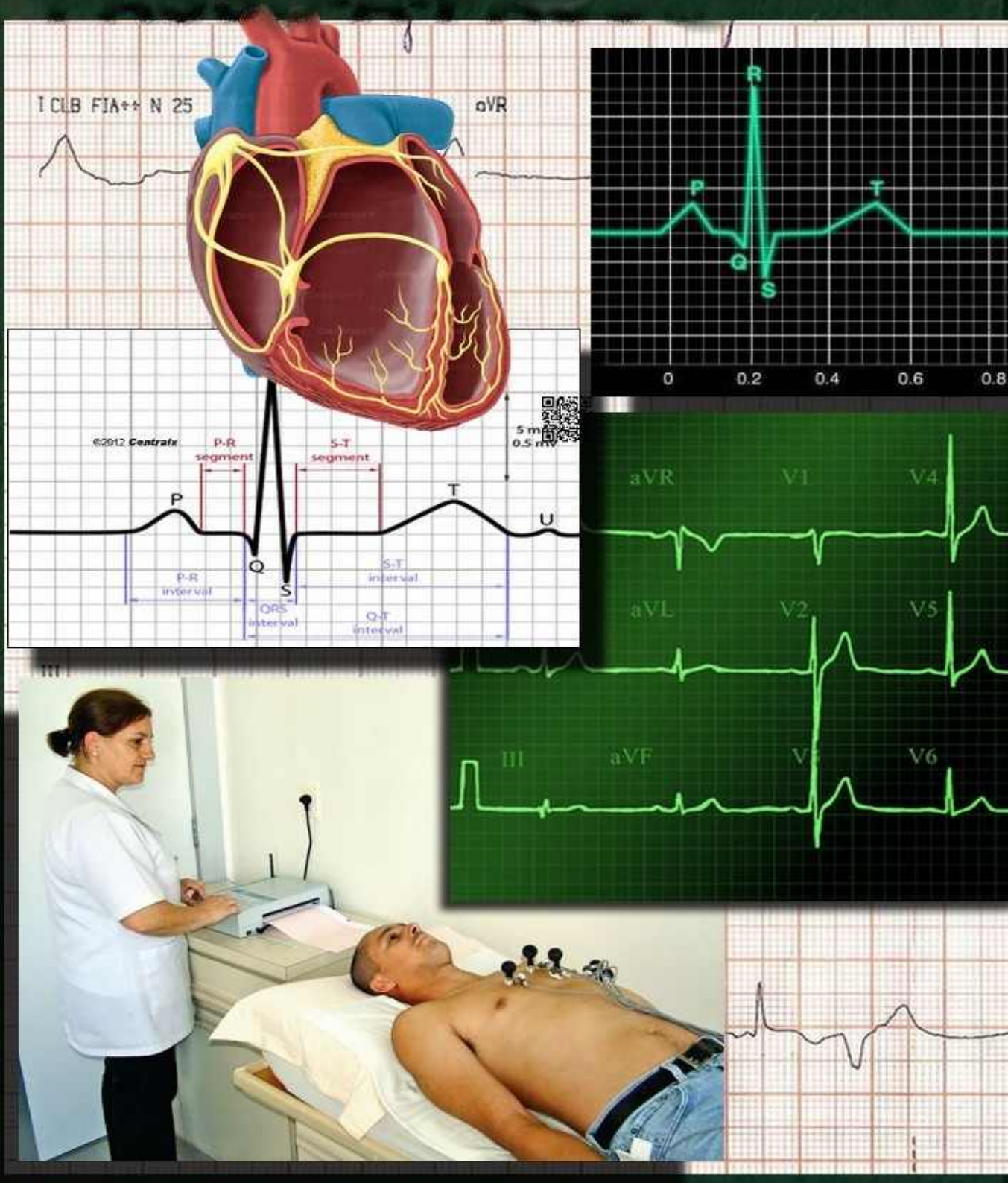
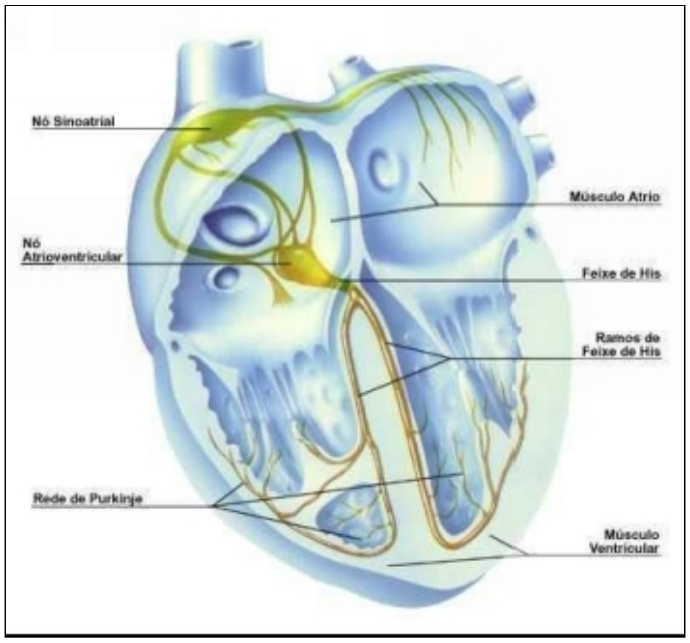
ELETROCARDIOGRAM STEP-BY-STEP

1 ELETROCARDIOGRAM



Rodrigo S. Augusto



ELECTROCARDIOGRAM

HEART LIKE A BOMB

- Heart right- pulmonary circulation
- Heart left-general circulation
- features 2 cameras = atrium (atrium) and ventricle

has a driving propiro system, cardionector system; formed by the sinoatrial node (sinus), internodulares fibers, atrioventricular node, fibrous ring, Hiss bundle, right and left bundle Purkinje cells.

TheHEART syncytium merged cells together by inter-cell membranes as if they were one (unions TIGHT)

has 2 syncytia; headset (atrium) and ventricular, SEPARATE by the fibrous ring

If communicate with the ventricles through HIS beam

- 1ºcontraçao headset (atrial)
- 2 Ventricular contraction - if so dao by retardation of 0,10seg lobby node (atrioventricular) -ventricular

Sistole- contraction - heart contracted and sanguine emptying into the aorta and art. pulmonary

Diastole- relaxation

ventricular systole = atrial diastole / systole atrial ventricular diastole =

FUNCTION CEBADORA = 25% bleed the atrium to the ventricle will by atrial pressure

The blood of the ventricles in diastole is called: VOLUME TELE diastolic blood 120 ml on average

regulatory PUMP

- intrinsic = (own heart) pumps the entire volume without accumulate in the veins = FRANK ACT STARLING
- extrinsic = (SNA) increases sympathetic contraction. Para-sympathetic inhibit contractions

FAILURE heart

failure Ventricular RIGHT AND LEFT

- **RIGHT**= greater preção veins - **CONSEQUENCES =** edema of the lower limbs, ascites and generalized, liver congestion and spleen. Hypoalbuminemia, weight loss
- **LEFT**= decrease of tissue irrigation. **CONSEQUENCES =** easy tiredness / congestion venous circulation pulmonar- lower hematose- hypoxia, cough, dyspnea caused by pulmonary edema, orthopnea

FAILURE BI-VENTRICULAR

dilutionalanemia;hematocrit diluted in H2O. Treatment: Diet / albumin + diuretic (not shown diuretic p / CCT, c / hypotension) EX:

- 90 YEARS OF LADY WITH APPARENT dilutional anemia and edema WITH VOLUME retention and liver FAILURE

Medicines. Digoxin, dopamine, dobutamine increases strength = contraction of the heart.

* Must Not RAISE THE LEGS OF WHY WILL CREATE A GRANDMA congestion LOWER (MEMBER edema INF). HAVE TO USE A POSITION IN 45 °

ELECTROCARDIOGRAPHY

Directed by electrocardiograph machine;is a graphic record of the electricity produced by the heart (small voltage) = systole and diastole

The voltage is measured in millivolts (Mv)

ECG=Records, Extends and writes

PARTICULARS.= electric current changes the heart and nothing else

- You can view: arrhythmias, ischemia (stroke), electric lock, growth of the heart cavities, changes in electrolyte concentrations, over dosage of drugs that affect the heart

The electric current passes through boards / metal electrodes in the following viewpoints:

- MEMBERS: fist and ankle
- TORAX PRECEDING 4 and 5 inter-costal space

electrode cable + = derivation electrocardiographic

lead EKG

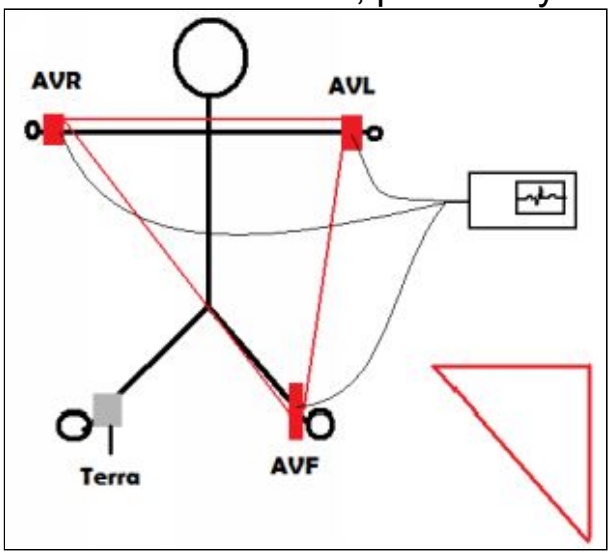
derivation UNIPOLAR (right wrist and left, left ankle)

HANDLE DREITO: AVR (rigth)

HANDLE LEFT: AVL (left)

LEFT ANKLE: AVF (fut)

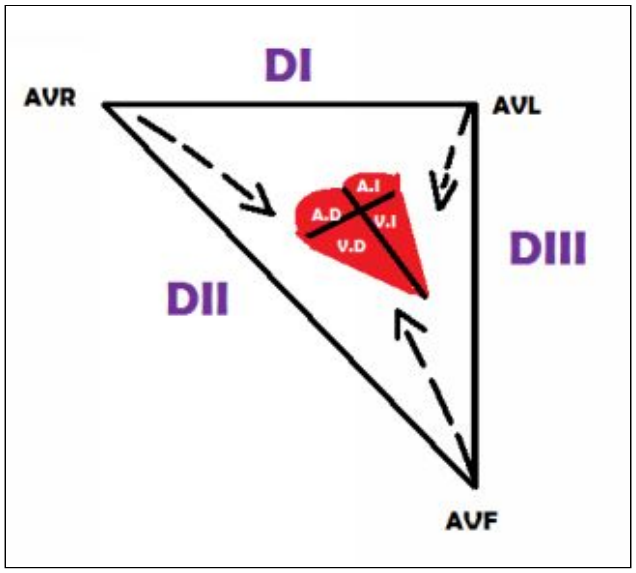
- AVL notes left side, pulmonary face



- AVR notes right, face mediastinal
- AVF points lower face diaphragmatic

Triangle TOB

derivationBIPOLAR: are not essential are only



viewpoint

not have cable, are the leads DI, DII, DIII

and AVL DII- notes left face

AVR and DIII- Notes the right cheek

AVF- Lower panel

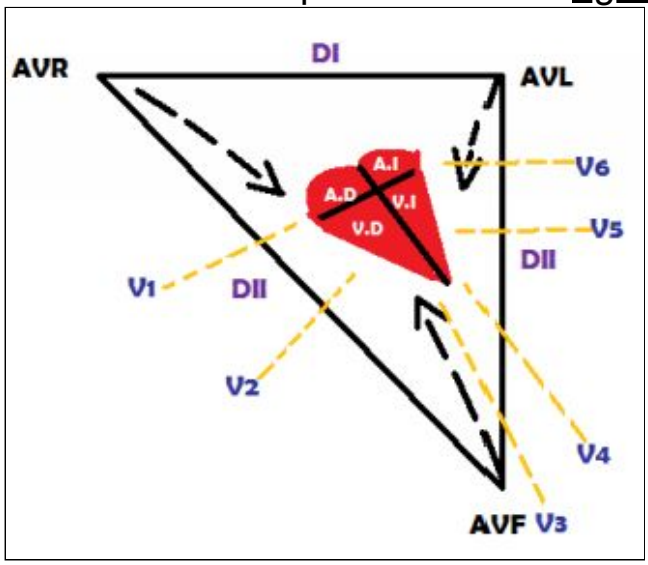
DI, born between avr and avl

IBD, born between avr and avf

IBD, born between avl and avf

leadsPRE-CORDIAL,leads the front of the chest

V1- 4 inter-costal space at theside <u>right</u> of the sternum



V2-4º inter-costal space at the <u>left</u> sternal

V3- the middle of V2 and V4

V4- 5 espacio inter-costal line <u>clavicular average left</u>

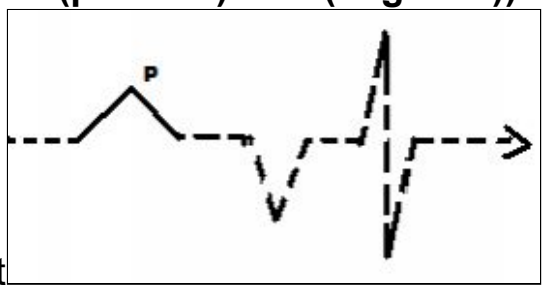
V5- 5 space inter-costal,line <u>left anterior axillary</u>

V6- 5 inter-costal

space,lineV2:.

axillaryaverage left

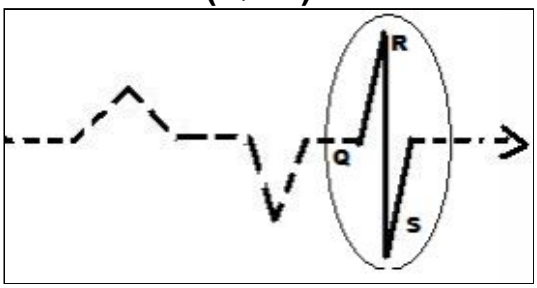
V1 and right ventricle / V3 and V4: tip of the heart / V5 and V6 left ventricle

WAVE (P + (positive) or T (negative)):electrical activity right atrium of the heart 

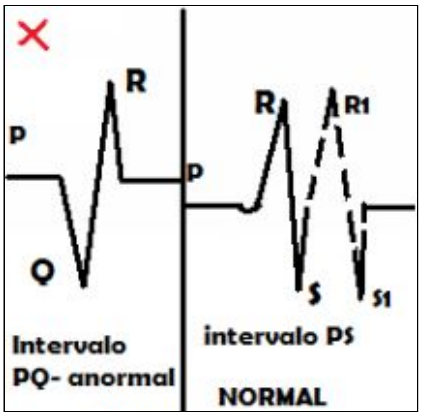
is born ofline (line **isoelectric _____ / \ where there is no electrical activity)**

INTERVAL PQ: Isometric or positive, negative PQ interval (-) is abnormal, it must be very small p / be Physiological

COMPLEX (QRS): waves of Union Q + R + S

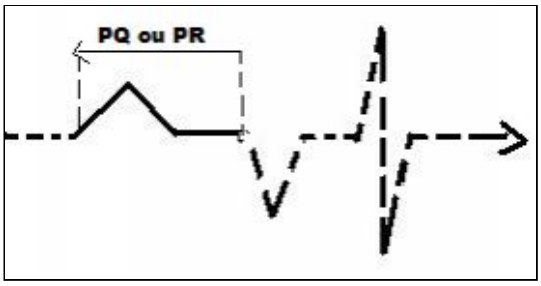


isodiphasic:when has the same size and + (positive) and other - (negative)

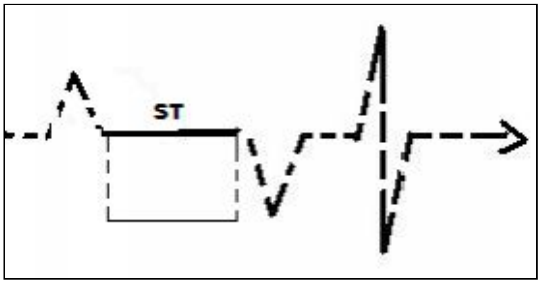


waves: Q negative (-) Large = abnormal, R (+), S (-)

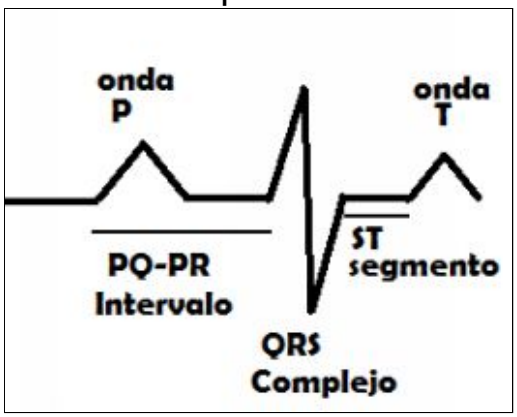
RANGE (PQ or PR): is the separation between a wave and another. Has ACTIVITY ELECTRICAL



SEGMENT (ST): between cycles separation space, NOT HAVE ELECTRICAL ACTIVITY



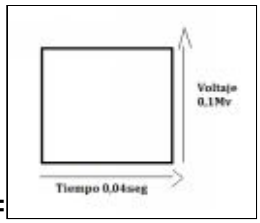
- P-wave depolarization atrial (atrial)



- Complexxo QRS ventricular depolarization
- wave T ventricular repolarization

ELECTROCARDIOGRAPHY PAPER

is a graph paper with squares of 1mm X 1 mm and 5 each small squares becomes large to facilitatereading:

horizontal time 0,04seg / are a large 0,2seg = 

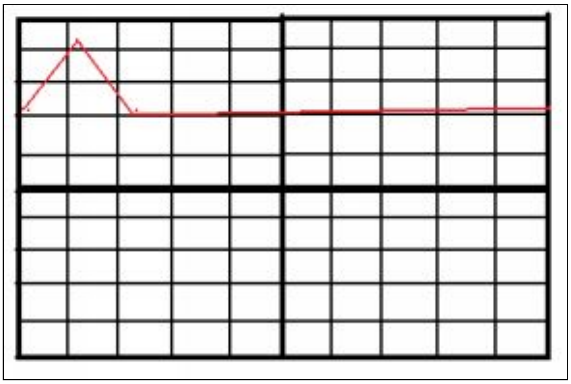
VERTICAL: voltage 0.1mV / and one large valley 0.5mV

VALUES REFERENCE: action potential generated by the sinus node

WAVEQ:depolarization atrial (atrial)

HORIZONTAL; MAX: 2.5 mm 2 and a half squares, time 0,1seg

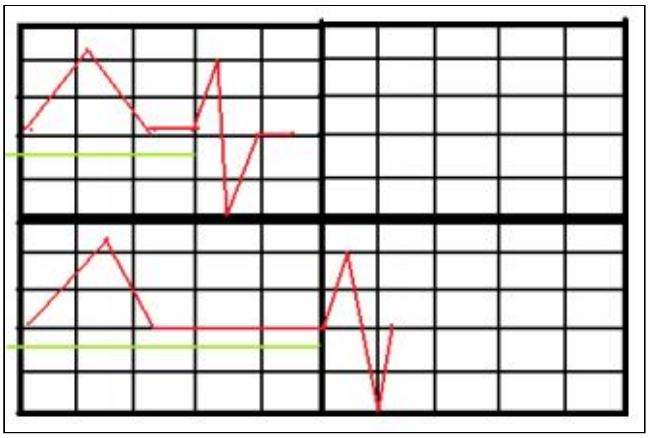
VERTICAL: MAX: 2.5 mm, 2e through squares, voltage 0,25Mv



BREAK OR PQPR,the beginning of the P wave to the beginning of the QRS complex, only interests the time (horizontal). NOTES THIS PERIOD THE LOCKS driving

MINIMUM: 3 squares or 0,12seg

MAXIMO: 5 squares or 0,2seg



QRS COMPLEX: ventricular depolarization,

HORIZONTAL (time) 1-2 and a half squares, 0.04 to 0.1segundos

left ventricular Voltage = SOKOLOF INDEX (index hypertrophy) applies to sum : **R + S wave Wave V5 = V135 mm or**

Exmore:S (8.5 mm) and R (8 mm) = 16.5 mm (NORMAL). /> 35 mm (hypertrophy)

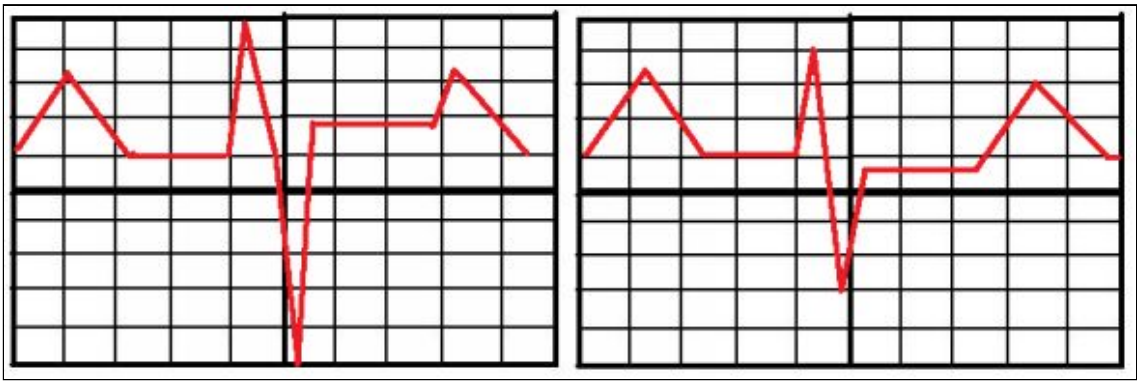


ST SEGMENT the end of the QRS aa beginning of the T wave, ONLY MATTER THE VOLTAGE Serve w / detect HEART

INFARCTION

above- 1 small square or 0.1 mV NOTHING !!

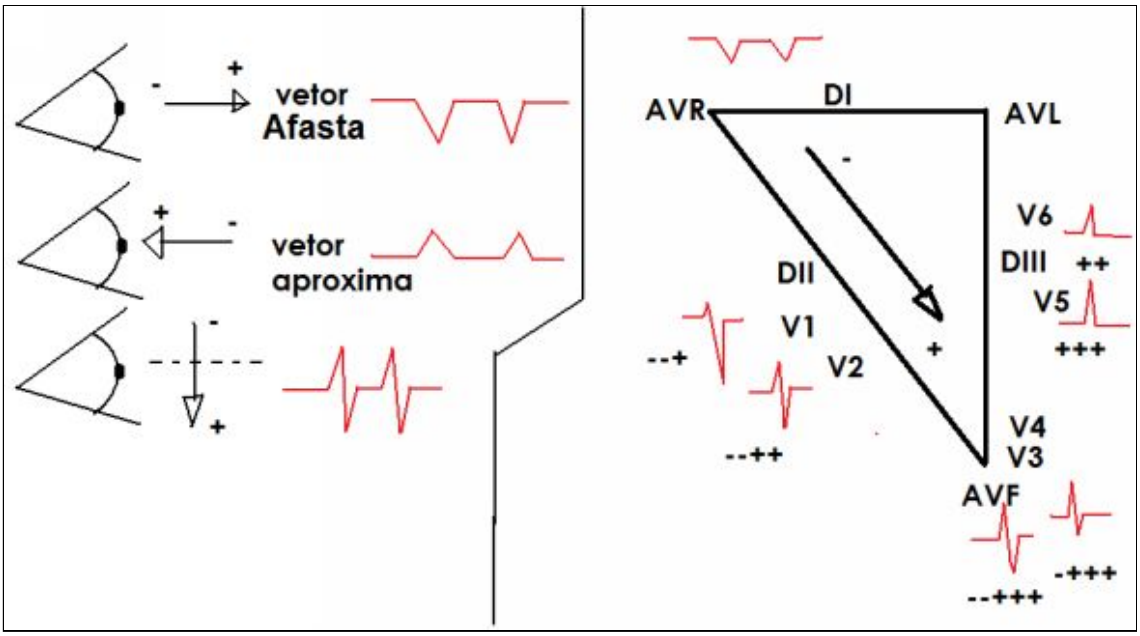
below- means small square or 0, 05Mv NOTHING !!



VECTORS = summed THE POWER STEERING ELECTRIC (HEART OF THE SAME)

Headset (ATRIO) - VECTORP

VECTOR ventriculographyQRS



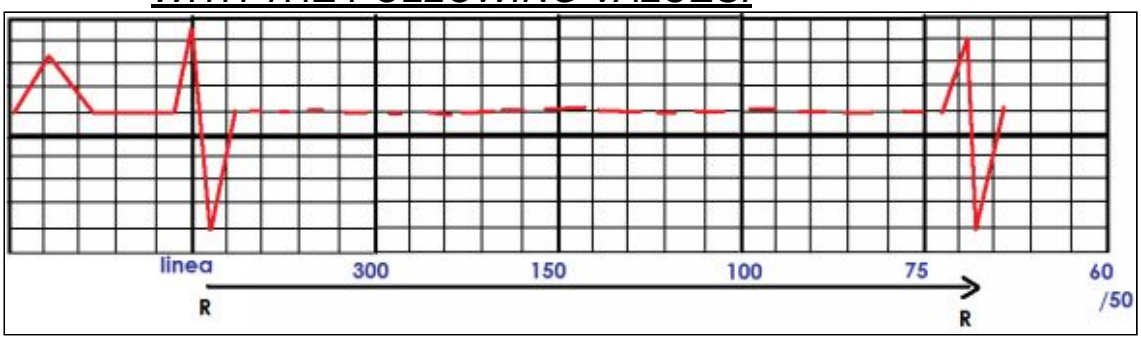
WAY BASIC P / ORDER AND DIAGNOSIS A ELECTROCARDIOGRAM

1) WHEN RUNNING A ELECTROCARDIOGRAM, PUT THE NAME OF THE PATIENT AND . DATE AND TIME

2) **regularity,**is SAY THAT THE COMPLEX recur in the same spaces TIME OR NOTrhythm. THEREFORE ECG regular or irregular

3) **heart**- IF STILL IN THE PRESENCE OF WAVE P, IF THERE IS THE WAVE P START ALL THEN HAVE COMPLEX A rhythm sinuatrial (60-100 contractions P / MIN), IF THERE IS NO WAVE P THEN THERE IS A FREAK rhythm

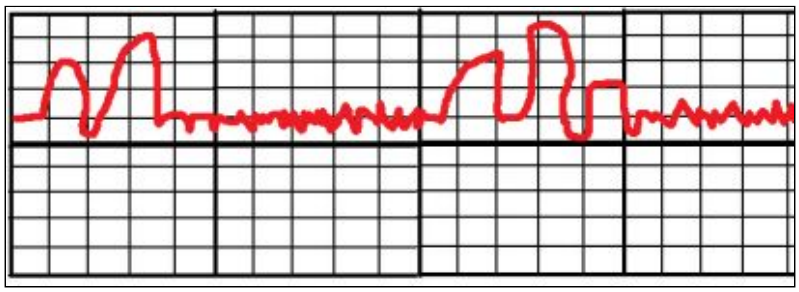
4) **HEART**RATE:60-100 / min, BOTTOM: BRADYCARDIA / TACHYCARDIA TO MAKE QUICK AND SIMPLE IF DOES: *ELECT A WAVE R (OF QRS) that matches A LINE OF LARGE VERTICAL OR THAT APPROACH MORE, APARTIR SE AI ACCOUNT AS THICK LINES TO THE NEXT WAVE R, WITH THE FOLLOWING VALUES:*



NESSTE IF THIS FREQUENCY BETWEEN 75 AND 60 HITS P / MIN

THIS METHOD ONLY APPLIES TO REGULAR ELETROCARDIGRAMAS !!!!

IRREGULAR = NO CAN MEASURE FREQUENCY



fibrillation Atrial

5)***CARDIACO- AXIS*** is the summary of the activity of the heart in a VECTOR with same direction of the heart, if **FLAME;**

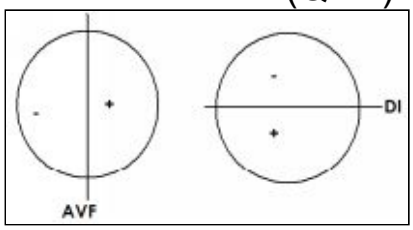
VECTORQRS

qndovector approaches the derivation; print a positive wave

qndo vector away from the derivation; print a negative wave

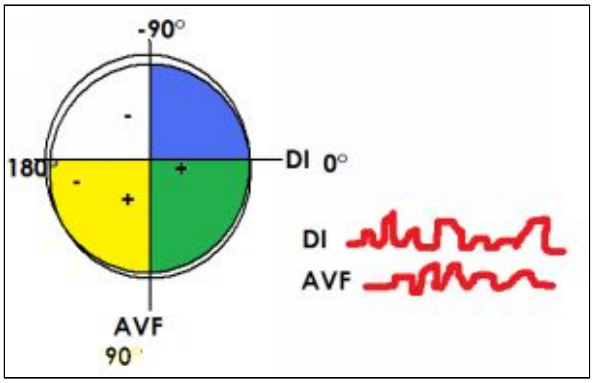
qndo the vector passes at a right angle to get a isoelectricwave

-nowwith 2 leads, only it should be noted the role in AVF derivation and see if the R wave (QRS) is positive (right) or negative (esquera)



- and the derivation DI notes the R wave, negative (above) positive (below)

Add the hemi-circles and FIND the axis:



physiological = 0 ° and 90 ° +

6 ANALASE WAVE P= TIME (0.04SEG) And VOLTAGE (O.1Mv) (2,5QUADRADINHOS)

7-RANGE PR-PQ= 5 squares HORIZONTAL ANALASE TIME, IF THERE change, THERE CONDUCT OF A LOCK AND ELECTRICAL ACTIVITY

8-QRS COMPLEX- HORIZONTAL TIME 0.1sec (2 E HALF squares) AND IMPLEMENTING THE CONTENTS OF SOKOLOF P / VOLTAGE Left Ventricular. <35MM

9-ST SEGMENT- REMOVAL 1 small square UP OR DOWN ½, REMOVAL DIFFERENT: INFARCTION

10-WAVE T- ALWAYS NEGATIVE AVR

VARIABLE IN V1- DIII It SAY, CAN BE POSITIVE OR NEGATIVE

THE REST OF derivations are ALWAYS POSITIVE

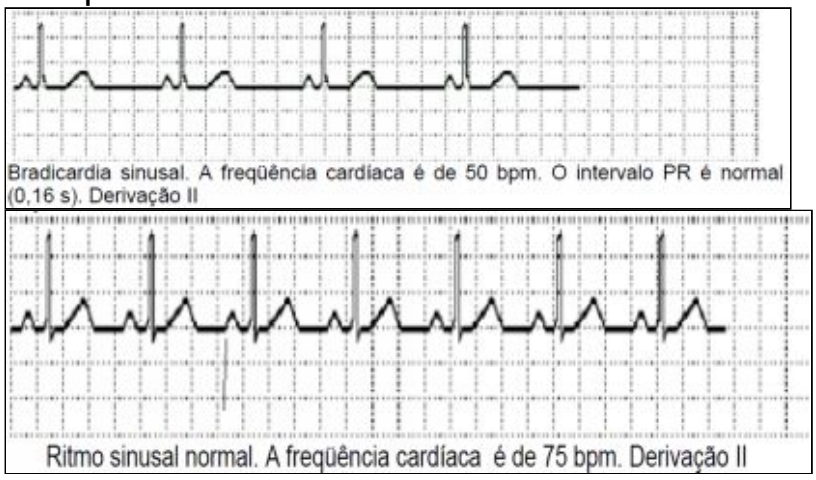
11- SEND ANEWS;DIAGNOSE changes, FOR YOUR TREATMENT PATIENT

ECG AMENDMENTS

ARRHYTHMIAS

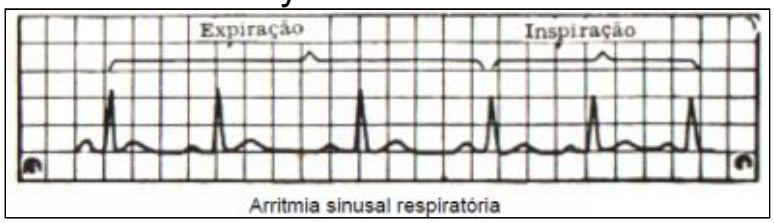
The conduction system of the heart consists of specialized cells that exhibit ability to rhythmic spontaneous depolarization. The cells located in the sinus or sino-atrial node are those that have the inherent rhythm faster, in such a way that the sinus node
acts as the pacemaker of the heart, beginning the wave of depolarization that spreads through the atria to the junction atrioventricular.

From the atrio-ventricular junction, the pulse enters the His bundle and reaches the ventricles through the right and left bundle branches, and the Purkinje fibers. The sinus node is under sympathetic and parasympathetic control, as well as, to a lesser extent, the atrio-ventricular junction. The decrease in impulse formation in the sinus node leads to pacemaker function to be assumed by cells located below the conduction system in slower inherent frequency. The normal heart rate is 120 beats per minute in children, and about 70 to 100 beats per minute in adults.



The heart rate accelerates during inspiration and slows during expiration. Not yet fully known reflex pathways by which breathing acts on the discharge frequency of the sinus node. Sinus arrhythmia is normal finding, being particularly noticeable in young people and people of high vagal tone.

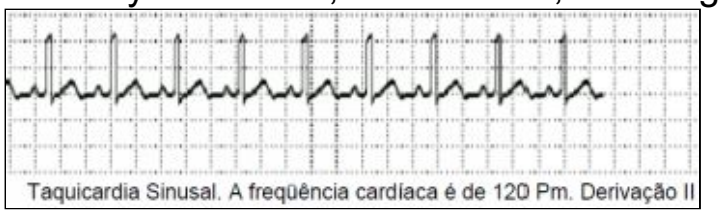
Sinus arrhythmia is absent in ASD which is large pulmonary blood flow.



Arritmia sinusal respiratória

There is talk of sinus tachycardia when the heart rate is above 100 beats per minute, under the control of the sinus node. The causes include: exercise, emotion,

thyrotoxicosis, heart failure, bleeding and feverbradycardia..


Taquicardia Sinusal. A freqüência cardíaca é de 120 Pm. Derivação II

When the heart rate is below 60 beats per minute, remaining under the command of the sinus node, speaks with sinus The causes include: high vagal tone (as in trained athletes) myxedema, intracranial hypertension and obstructive jaundicenode..

is present an arrhythmia when the impulse that initiates cardiac contraction originates outside the sinus Cardiac revolutions are replaced unequal length, or are, arrhythmias themselves. According to the location of the cardiac pacemaker, arrhythmias are classified as supraventricular (pacemaker in the atria or the AV junction) and ventricular (pacemaker in the ventricles).

I. Sinus Arrhythmia

In sinus arrhythmia, the track is normally, but arrhythmic. It is benign disorder in which the irregularity is more often influenced by breathing movements.

II. ARRHYTHMIA EXTRASSISTÓLICA

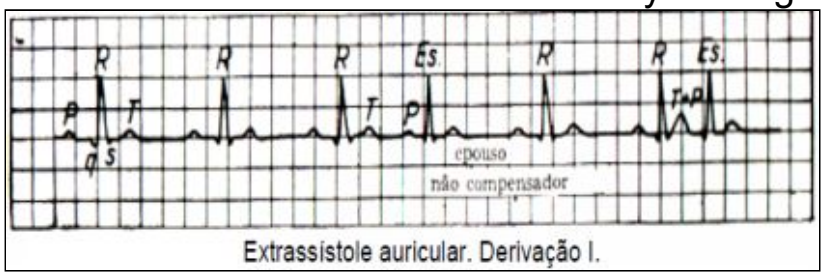
premature contractions are caused by an impulse originated in a focus located somewhere in the atria or the AV junction, which triggers the early sinus node and spreads to the atria and down to the ventricles, causingheart

prematurecontraction.The next sinus impulse is delayed until the sinus node to recharge. Occasional beats are common in healthy people. When very frequent, can be harbinger of atrial fibrillation.

The P wave extrasystole is deformed, once the impulse originates in abnormal location and spreads also abnormal in direction through the courts. The extrasystole occurs prematurely, the QRS complex is normal, unlessbundle branch block, and a pause after the extrasystole is not fully compensating, ie the distance between the normal sinus rate is less than twice the basic cycle length



Extrassistole auricular bloqueada. A excitação não é transmitida aos ventrículos. Observa-se, então, uma onda P habitualmente anormal, não seguida de QRS.



Extrassistole auricular. Derivação I.

..If the path is nearly normal morphology, which is the only anomaly shorter PR interval, it is an atrial extrasystole

Sometimes they add up other 1.

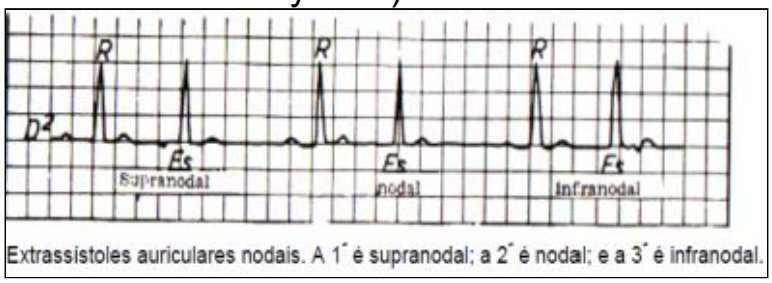
abnormalities:**normal or inverted form P-wave or diphasic:** the more pronounced the anomaly, the more the starting point of atrial excitation is far sinusventricles;.

2- **Sometimes, atrial excitation can follow an unusual path in the** so exceptionally may create a QRS deformation, something widened and thickened, followed by normal or pathologic T.

3 **An atrial extrasystolemay be** blocked,or not followed by a ventricular systole. To diagnose the premature atrial extrasystoles P-wave and may have abnormal morphology.

If premature contraction of the QRST morphology is rigorously equal to the fundamental contractions, but the QRST not being preceded by a P-wave, has the typical appearance the nodal extrasystole.

Sometimes the QRST nodal extrasystole is preceded by a negative P wave or biphasic very rough (supranodal extrasystole) or followed by a strictly negative or biphasic P
(infra-nodal extrasystole)



Extrassistoles auriculares nodais. A 1ª é supranodal; a 2ª é nodal; e a 3ª é infranodal.

is not required treatment when the beats are accidental finding . Frequent uncomfortable palpitations can be lessened by avoiding factors known to trigger premature such as:. Coffee, mate tea, alcohol and nicotine

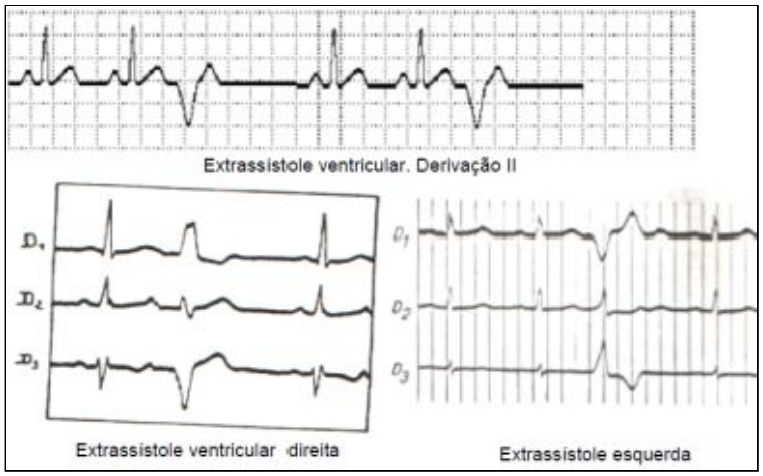
When a focus located somewhere in the ventricles triggers a boost early and takes the pacemaker function in sinus node for a beat, there is a premature ventricular contraction called ventricular premature. The next sinus impulse will find the ventricles in refractory status, and causes a rewarding break that immediately follows the ventricular premature. The frequency of the pulse is interrupted by a beat occurs earlier than normal.

The morphology of the premature contraction is shown very unusual, presenting no accident P, QRS complex, a hook, and followed by a thickened line ST uneven and an inverted T wave.

Its exact configuration depends on the location irritable focus. The distance between normal QRS complexes that line the ventricular extrasystole is exactly twice the normal cycle length, ie the pause after the extrasystole is fully
compensatory. The premature polyfocal produce variable forms of abnormal QRS complex.

It is important to note that the morphology of the ventricular stroke is provided by the ventricle contracts last. Therefore, the QRS complex has a left electric axis, it is a right ventricular extrasystole. And vice versa, if the QRS has a right axis, this is a left ventricular premature.

If the main QRS accident is negative in the three standard leads to premature complex originated in the apical region.
Conversely, if there is a R great in the three standard leads to premature complex comes from the region of the heart base.



occasional ventricular extrasystoles may occur in normal heart. Frequent ventricular premature indicate organic disease, and may precede ventricular tachycardia or ventricular fibrillation.

Ventricular extrasystoles originating in more of a focus in the ventricles (ventricular premature polyfocal) are particularly dangerous.

Coupled ventricular extrasystoles (bigeminism) a premature complex following each sinus beat often are digitalis intoxication signal.

III. ATRIAL FLUTTER

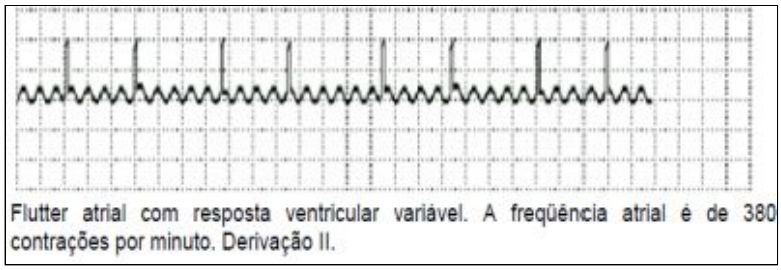
Atrial Flutter occurs when the atria are fast and regularly stimulated, about 300 times per minute, for atrial ectopic pacemaker. The AV junction can not drive all the impulses to the ventricles, and these therefore beat frequency that is half or a quarter of atrial. The most common type is the type. 2: 1, that is, the number of atrial contractions is double the ventricular contractions

The pulse is usually regular and quick, and atrial flutter can be transient or persist for months. The vagal stimulation maneuvers increase the degree of atrioventricular block, ventricular rate slowing sharply, only during the stimulation periodASD)..

Ninety percent of cases have underlying heart disease (rheumatic, ischemic, hypertensive, or congenital - particularly The high ventricular rate is uncomfortable and may precipitate heart failure.

The rapid and repetitive atrial stimulation, from a single focus, results in F waves on the electrocardiogram, all identically and completely regular way. The F are generally more prominent waves in lead II, giving the baseline electrocardiogram aspect of saw teeth.

IV. ATRIAL FIBRILLATION



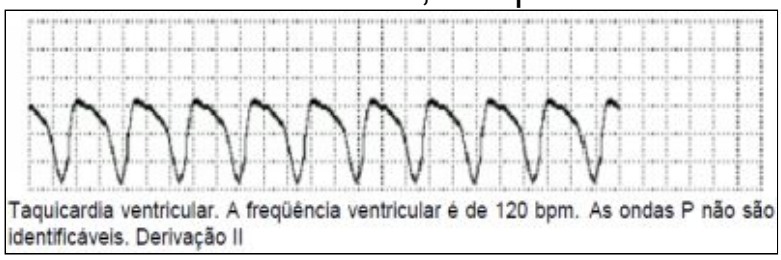
Flutter atrial com resposta ventricular variável. A freqüência atrial é de 380 contrações por minuto. Derivação II.

Small areas of atrial muscle are stimulated at different times, and the lobby has uncoordinated contractions.

Although there are approximately 600 stimuli per minute acting in the atria, resulting in irregular and ineffective contractions of the atrial muscle small areas, and only part of these pulses is conducted through the AV junction into the ventricle.

The ventricular rate is rapidly and completely irregular.

In most cases no disease present It is particularly common rheumatic mitral valve disease, the thyroid related heart disease and ischemic heart disease. Less commonly, other diseases are responsible for fibrillation, such as atrial septal defect, cardiomyopathy and chronic lung disease. Occasionally atrial fibrillation following thoracotomy, electric shock or acute viral infection. . There are cases of atrial fibrillation that appears in some adults without underlying disease, and no identifiable reason, compatible with normal life



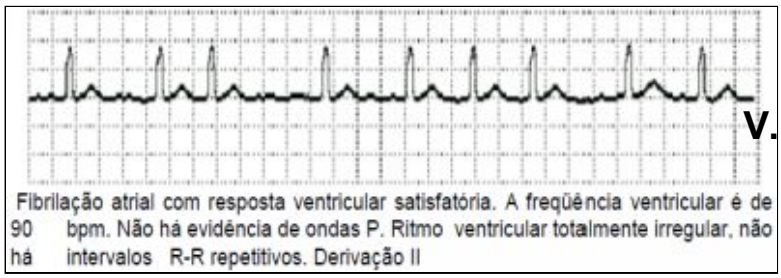
Taquicardia ventricular. A freqüência ventricular é de 120 bpm. As ondas P não são identificáveis. Derivação II

Two factors are very important in determining the onset and maintenance of atrial fibrillation in any patient: age or presence of increased lobby.

The electrocardiogram in atrial fibrillation appears as small, irregular and fast waves that vary in size and shape.

QRS complexes are normal configuration, but irregular rhythm.

Atrial fibrillation is visible in three standard leads V1 and V2 and especially in V1.



Fibrilação atrial com resposta ventricular satisfatória. A freqüência ventricular é de 90 bpm. Não há evidência de ondas P. Ritmo ventricular totalmente irregular, não há intervalos R-R repetitivos. Derivação II

V. Tachycardia Ventricular

tachycardia ventricular occurs when an irritable focus in the ventricle triggers impulses quickly and

takes the heart pacemaker function. These impulses are usually not conducted in retrograde fashion to the courts, which, therefore, continue to beat normal frequency under the control of the sinus node.

The frequency of the pulse is regular and fast, usually 120 to 200 beats per minute.

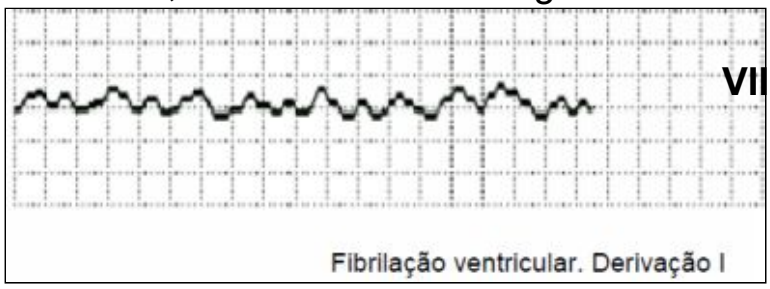
Ventricular tachycardia means the ventricular muscle advanced disease, ischemic or generally severe digitalis intoxication.

The electrocardiogram exhibits unusual and large QRS complexes, but identical to each other. P waves can be identified at a slower rate, with no fixed relationship with the QRS complexes.

VI. Ventricular

In ventricular fibrillationFibrillation,the depolarization of the ventricles becomes irregular and focal way, for quick and wandering impulses. There is no coordinated contraction of the ventricles, or output, and not peripheral pulse.

Ventricular fibrillation is often terminal event after serious heart damage. Occasionally appears when it is not so intense the underlying myocardial disease (myocardial infarction, aortic stenosis, complete heart block, electric shock or drug intoxication.



Fibrilação ventricular. Derivação I

VII. DRIVING LOCKS

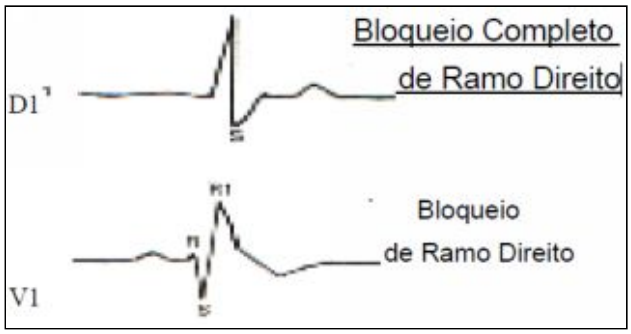
From the atrioventricular junction, located in the right atrium, just above the opening of the coronary sinus, the specialized conducting tissue of the heart flows through the interventricular septum, initially in single tract (His bundle), and then divided into a right branch, which supplies the right ventricle, and left branch, which supplies the left ventricle.

It installs branch block or when the beam right or left are unable to conduct the electrical impulse.

In the branch blocks, the QRS shown prolonged (0.12 s or more) due to slower diffusion of depolarization wave through the myocardium distal to the blockage. The QRS complex is shown deformed because

the depolarization wave takes an unusual way, and the electrical forces generated by the heart, therefore, are abnormal in magnitude and direction.

When a terminal branch of the His bundle does not show more permeable to motor excitations, it is said that there is complete blockage. When the excitation pass with noticeable delay, the lock is called incomplete. In this, the QRS increase is smaller, lasting between 0.08 to 0.11 s. The deflection intrinsecoid is little delayed.



It is called deflection intrinsecoid the wide and sudden drop following line to the apex A. Your delay is considered physiological up to 0.03 s for the left precordial. It may be found, for example, a delay of 0.04 s in V2 for a right block; and a delay of 0.09 s in V6 for a left block.

In the standard leads, have been usually in D1, an R followed by an S wave something deep, broad and slurred, often hook, followed by a T positive. In D3, R is often predominant and slurried.

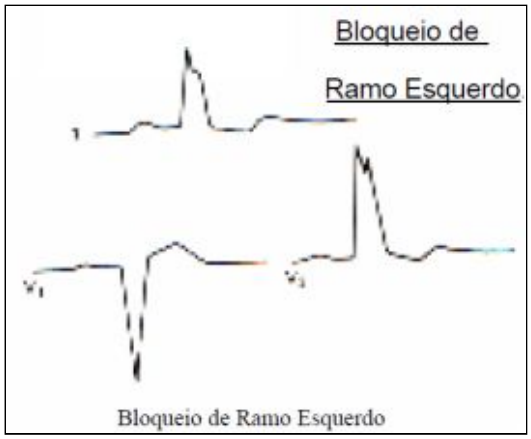
In unipolar derivations of the members, it should be noted a peculiarity in aVR, where the negative Q wave can be followed by a large R wave and slurred.

In the right precordial leads, it is observed, often , three-phase aspect (S wave surrounded by two waves R). Sometimes there is no trifasismo, but R wave shown enlarged and hook. In the left precordial leads, R wave is normal and then a deep and S hook.

The right ventricle is depolarized last in right bundle branch block, because a delayed depolarization impulse that reaches the right ventricle. This is located above the left, and the forces of the QRS terminals, therefore, are directed forward, producing R 'in right precordial. The right ventricle is also located to the right of the left ventricle and the terminal QRS forces are thus directed to the right producing terminal S wave in D1.

In 15% of patients with right bundle branch block is not cardiac abnormality.

The main causes of blockage
1 dilatation and hypertrophy of the right ventricle, eg, pulmonary embolism, atrial septal
are:...2- ischemic heart disease, affecting the right beam
3- Subsequently surgical ventriculotomy



Bloqueio de Ramo Esquerdo

Prolonged QRS results intramyocardial diffusion slow depolarization beyond the block.

Typically the interventricular septum is activated before, and depolarizes from the left side, producing Q waves in V4, V5 and V6 and V1 small R-wave.

In left bundle branch block, the septum interventricular is launched from the right beam, and the depolarization spreads from the right side. This initial force is directed back and to the left, and therefore can not subscribe R waves in the anterior precordial leads V1 and V2, or Q waves in V4, V5 and V6.

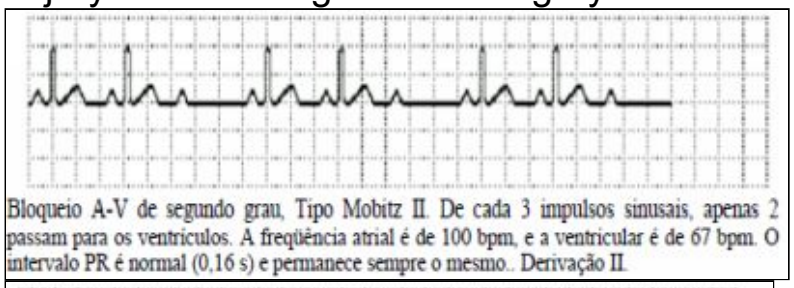
The main Left Bundle Branch Block causes are
1-Myocardial ischemia
:...2-cardiomyopathy
3- myocardial fibrosis

VIII. AV LOCK

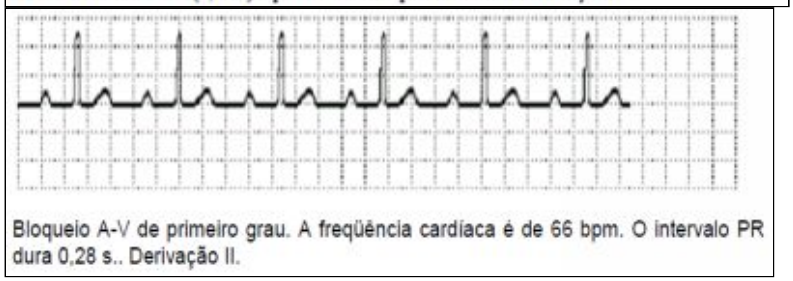
There atrioventricular heart block whenever there is delay or complete obstruction of the sinus passage impulse to the ventriclesare:.

The main causes
1-degeneration and fibrosis the atrioventricular bundle of unknown cause.
2-ischemic heart disease.
Depression 3-conductor system for drugs.

4-Commitment beam by chronic disease processes (syphilitic aortopathy, calcific aortic stenosis, ankylosing spondylitis)
5-acute myocarditis
6-Injury beam during cardiac surgery.



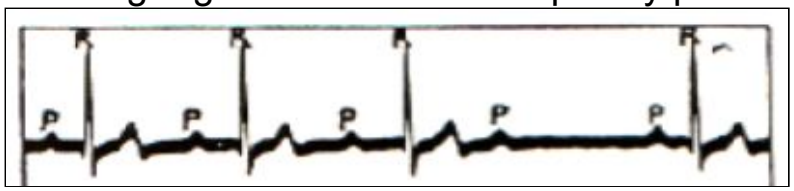
Bloqueio A-V de segundo grau, Tipo Mobitz II. De cada 3 impulsos sinusais, apenas 2 passam para os ventrículos. A freqüência atrial é de 100 bpm, e a ventricular é de 67 bpm. O intervalo PR é normal (0,16 s) e permanece sempre o mesmo.. Derivação II.



Bloqueio A-V de primeiro grau. A freqüência cardíaca é de 66 bpm. O intervalo PR dura 0,28 s.. Derivação II.

7-congenital heart block.

In the first degree AV block, every impulse atriaissão conducted to the ventricles after abnormal delay in conducting tissues. The cardiac output does not change. This type of lock can be caused by digitalis. The electrocardiographic diagnosis is established when the PR interval is greater than 0.20 s, remaining regular and normal frequency pulse.



SECOND DEGREE AV BLOCK

There are right bundle branch block on the ECG indicated by S wave in D1, and R wave in V1.

There is also disruption of the anterior division of the left bundle branch. This causes axis deviation to the left. Now, the subsequent division of the left fails to conduct, appear suddenly complete blockage. The PR interval does not appear appreciably elongated at any stage before it appears complete blockage.

It is said that there is AV block second degree when some of the atrial impulses are not conducted to the ventricles. For example, if only one of every two pulses is conducted, there is AV block 2: 1, and the pulse frequency is half of the atrial rate. Like the figure below, only two out of three impulses pass to the ventricles, thus, there is AV block 3:. 2, and the pulse rate is two-thirds of the atrial rate

The heart block second degree is divided into heart block Mobitz Type I and Type II Mobitz.

Mobitz Type I In the blockage occurs in the upper AV junction, usually reversible condition, such as lower myocardial infarction or digitalis intoxication. The electrocardiogram shows normal QRS complexes and Wenckebach phenomenon.

This phenomenon is characterized by the appearance, from time to time, a QRS complex preceded by two elevations P. Progression to Stokes Adams crises is not customary in block Mobitz type I.

Phenomenon Wenckebach. Between each cardiac revolution and the following is seen to increase PR interval duration, until P is produced at the beginning of diastole
and, therefore, no ventricular stay home. Results paused, then everything starts.

In Type II Mobitz the lock is on a branch, and beat failures occur by intermittent blockage in another branch, for example, the ECG shows right bundle branch block with axis deviation to the left and range Normal PR (axis shift to the left means that the anterior division of the left bundle branch is blocked). Rate failures occur without stretching and without PR Wenckebach phenomenon, when driving the rear ends of the left branch division.

Type II is a form of bilateral bundle branch block, and usually progresses to full lock, with slow QRS complex and bizarre and Stokes Adams crisesCORONARY:.

T coronary OT is perfectly clear when the ST segment is not uneven, seeing himself then a T inverted and deep, of pointed apex and the two sides are symmetrical, contrary to normal Tsegment.

When the ST is uneven, the T wave is usually the opposite direction of the deflection. It is therefore negative if the ST is positive, and vice versa. In case of ST elevation, not infrequently T is biphasic.

The direction of the wave of repolarization in ventricular muscle is reversed, both by the presence of a muscle area that does not take part in this process (the infarcted area) and by the abnormal repolarization of ischemic muscle around the infarcted area. It is observed T wave inversion in the same leads that have Q waves

II MYOCARDIAL INFARCTION

In practice, myocardial infarction confined to the left ventricle.

Only the presence of a pathological Q (QS or QR) to suggest that there or there was a myocardial necrosis, i.e., a myocardial infarction itself.

In the absence of a pathological Q it follows that one can not speak properly, a myocardial infarction. Faced with a similar clinical picture of myocardial infarction, but without the presence of pathological Q (QS or QR), it is proposed for such cases the designation of **large acute coronary insufficiency.**

This distinction has practical interest because the prognosis is more serious in myocardial infarction itself, in which there is a higher risk of subsequent ventricular aneurysm

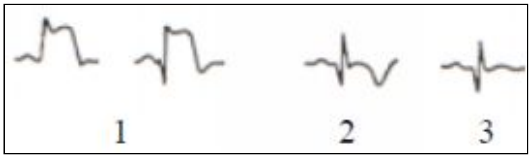
..In the absence of pathological Q, can be, at most, find microscopic infarcts

The electrocardiogram shows:

1-ST segment elevation - minutes after changes in the coronary flow.

2-Q Waves of 0.04 seconds in duration and increased amplitude - usualmente appear within hours.

4-wave inversion T. The T waves are inverted in leads showing ST elevation, and within a few weeks, the ST segment becomes isoelectricinfarction:...

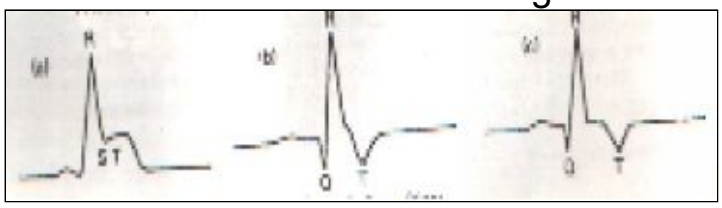


Evolution of electrical abnormalities of myocardial

1-At first, 2 types of waves dome

2-ST-elevation disappears and the crown T becomes very sharp

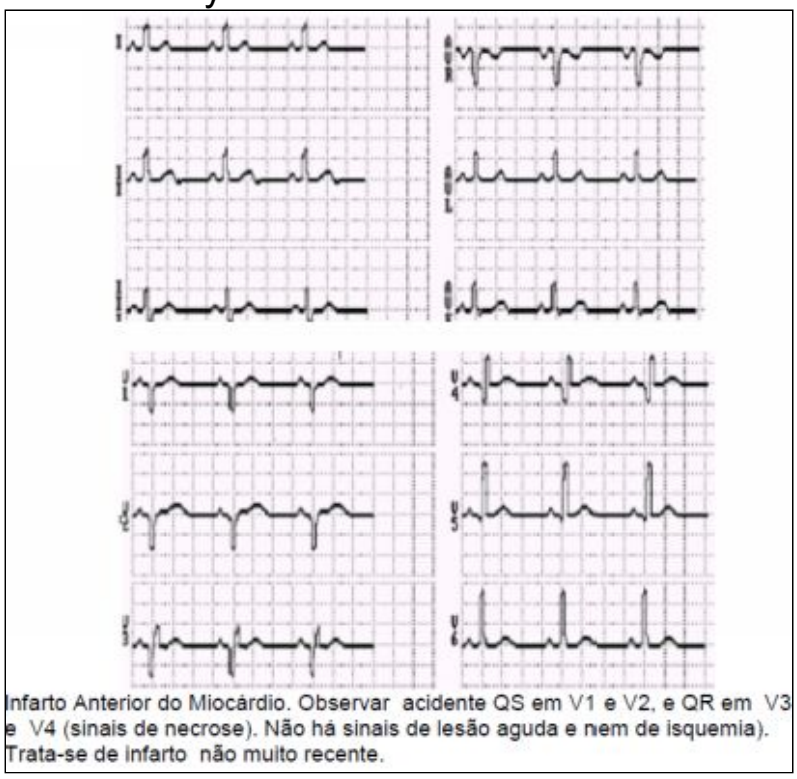
3- Finally, only persists the accident Q. There are ST depression or inversion of T Sometimes the negative coronary T persists indefinitely.



Myocardial infarction diaphragmatic in D2. (A) - Two hours after infarction.

(B) -Doze hours after onset of pain. (C) Two weeks after the onset of pain

-..The derivations in which these changes can be observed, are determined by the location of the infarction



Infarto Anterior do Miocárdio. Observar acidente QS em V1 e V2, e QR em V3 e V4 (sinais de necrose). Não há sinais de lesão aguda e nem de isquemia). Trata-se de infarto não muito recente.

In the figures below, the arrows represent the electric forces during the 0.04 s inciais of ventricular depolarization (the ventricle depolarizes the endocardial surface out). The large arrow is the direction and magnitude averages of all these forces, and usually does not vary much from the direction shown. Consider the figure showing the injury, myocardial infarction diaphragmatic. Myocardial infarction has committed the bottom face (or diaphragm) of the left ventricle, and does not produce electrical forces in thearea.
necrotic The big arrow represents the average of the remaining forces of the initial 0.04 s. This force will produce negative deflections in leads II and III during the first 0.04 s of the QRS complex. Therefore, myocardial infarction diaphragm produces Q waves of 0.04 s duration in leads II and III. Similarly, one anterior myocardial infarction abolish all forces directed forward during the initial 0.04 s. Resultam ondas Q em V1, V2 e V3, pois agora não há forças iniciais anteriores que produzem ondas R nestas derivações.

X. FORMER INFARCTION MYOCARDIAL

In infarction of the anterior wall myocardial appear V1 to V4 strain, consisting of QS or QR with ST elevation and negative T. Few modifications to the standard derivations, at most, a minor D1 T T than D3, and in unipolar members, flat or inverted T aVL

X. SIDE INFARCTION MYOCARDIAL

As shown below, deformations are observed in the left precordial V5 and V6, consisting of QS or QR, with ST elevation and negative T. Analogous aspect in D1 and aVL.



Infarto Agudo Lateral do Miocárdio. Supradesnivelamento de ST (sinal de lesão aguda) em D1, aVL, V4, V5 e V6. Acidente QS em D1, aVL, V5 e V6 (Sinal de necrose). Não há sinais de isquemia.

IX. INFARCTION diaphragmatic OR LOWER MYOCARDIAL WALL

As shown in the pictures below, there is no deformation in precordial at most accident wide T. in leads standard, are observed QS or QR in D2 and D3, with ST elevation and negative T in D2 and especially in D3. In aVF observe same aspect of D3.

Previous Acute Myocardial with ischemia Side. Coronary T in D1, aVL, V5 and V6 (inverted T, symmetrical and sharp) - ischemia signal.

ST elevation in V1, V2, V3 and V4 (necrosis signal).

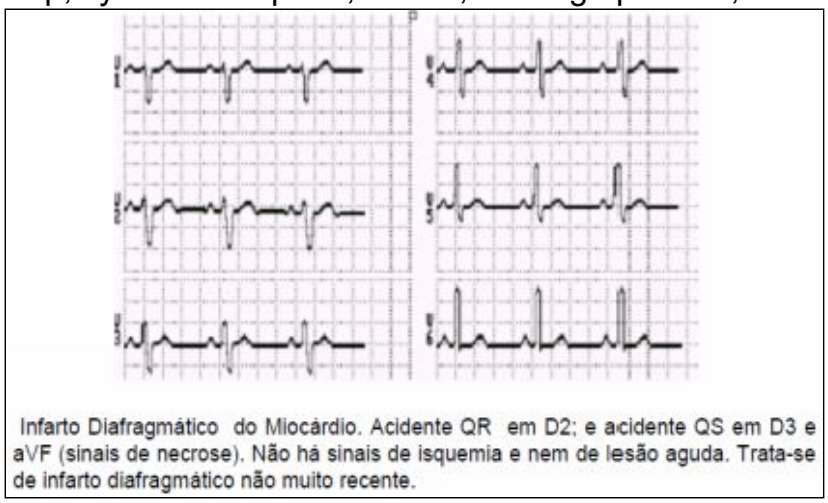


II ANGINAL BREAST

The angina pectoris is the result of a chronic coronary insufficiency of mild or medium grade.

The T-wave inversion, deep in sharp apex and symmetrical parts, is the most common accident in the range of angina attacks.

It can be observed in standard derivations, or at D1 or D3. As D1 and D3 have, often, the opposite aspect, there will be therefore cases where simple T, sharp, symmetrical parts, will be, although positive, a coronary T.



Infarto Diafragmático do Miocárdio. Acidente QR em D2; e acidente QS em D3 e aVF (sinais de necrose). Não há sinais de isquemia e nem de lesão aguda. Trata-se de infarto diafragmático não muito recente.

The frequent disruption this may associar-se um acidente Q, seqüela de um infracto mais ou menos antigo do miocárdio, ou um desnivelamento do segmento ST, particularmente se, algumas semanas ou alguns meses, antes, sobreveio um infarto do miocárdio..

Às vezes, um desnivelamento pode tornar-se definitivo, se a insuficiência coronária for acentuada or even if a ventricular aneurysm.

Finally, any failure can inexistence of the ECG, as occurs in about a third of cases, or also may be an electrical fault which is not dependent angina pectoris and therefore, is purely coincidental. The lack of ECG abnormality is variable frequency phenomenon, according to the authors. Therefore, when the

anamnesis leads to the diagnosis of angina pectoris, a normal ECG is not moved safe clinical diagnosis.

This is the case whether to use the exercise ECG (gymnastic step LIAN, MASTER testing, etc. It will be drawn at the end of the stress, then after 4 minutes after 7 and 10 minutes after the end of the



effort. This can result in anginal an angina crisis and electrical anomalies. There are, often, an ST elevation in the inferior standard leads and in the left precordial with biphasic or negative T. the effort causes no accident Q. Fifteen minutes after the effort, the path regains its usual appearance.

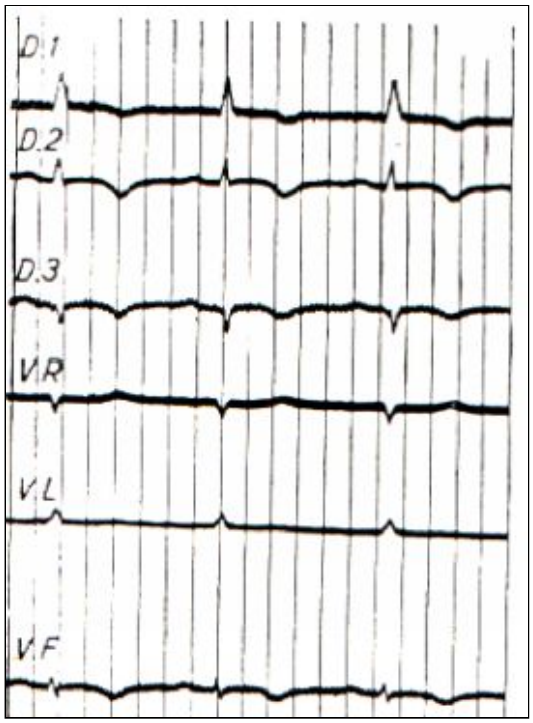
Very exceptionally been reported sudden death during exertion so provoked. Therefore, only rarely necessary to resort to the test effort, reserving it for cases where it is almost certain that the pains are not anginal and where it is considered useful to confirm this good clinical impression.

It is believed that, more often, angina attacks depend on a subendocardial ischemia, sometimes subepicardial.

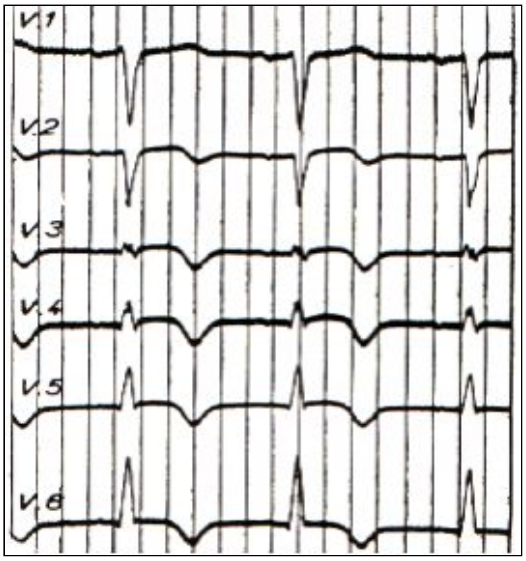
Diagnostica is a subendocardial transient or chronic ischemia of the following signs: subdesnivelamento ST, flattening

or difasismo t, J point depression by more than one millimeter.

It is important not to make it too hastily the diagnosis of coronary impairment based on a modification of isolated T, since T is a negative



coronary not necessarily.



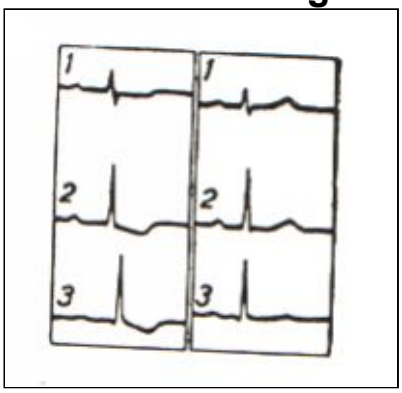
POISONING digitalis: Under the influence of digitalis preparations, the heart beats more slowly, driving stretches (PR elongation) sometimes adds to the excitement, appearing premature; it is known that it is important bigeminado pulse signal digitalis intoxication. In deep digitalis intoxication can be observed atrioventricular dissociation.

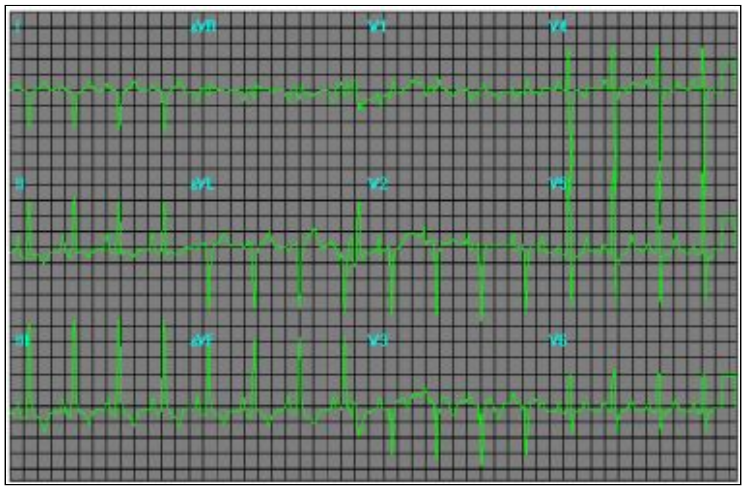
How influence on the ventricular complex, there is very often a ST subdesnivelamento in D1 and D2, sometimes in D3, and in precordial leads. This lowering of TS is concave upward and leads to T biphasic or negative.

One can also notice a shortening of the electrical ventricular systole (QT space).

These different anomalies disappear very quickly after a digitalis treatment alone. There remain, however, in **patients undergoing**

treatments digitalis numerous and close together.





IV - HIPOPOTASSEMIA

Are observed, sometimes QT elongation, and flattening or inversion of T, especially in the precordial leads; possible lowering of the ST line, lengthening the time of atrioventricular conduction and QRS enlargement, which may cause branch block.

V- hyperkalemia

Shortening of the QT interval with a pointed T, coronary type.

VI -TETANIA-HYPOCALCEMIA

Lengthening of the QT interval under the influence of hypocalcemia.

VII -MIXEDEMA

Low voltage QRS and T. It is considered the flattened T wave when its amplitude is less than one millimeter in classical

derivations.

VIII -COR-pulmonale

In cor-pulmonale, the transmission of the electrical activity of the heart to the body surface is considerably disturbed by the presence of the sick lungs.

A proportion of cases fulfills the criteria for electrocardiographic diagnosis of right ventricular hypertrophy.

Cor-pulmonale. QRS deviated to the right. S larger than R, D1 and aVL.

P wave larger than 3 mm, D2 and D3.

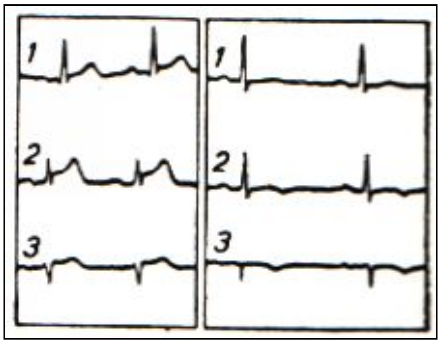
IX - PERICARDITIS

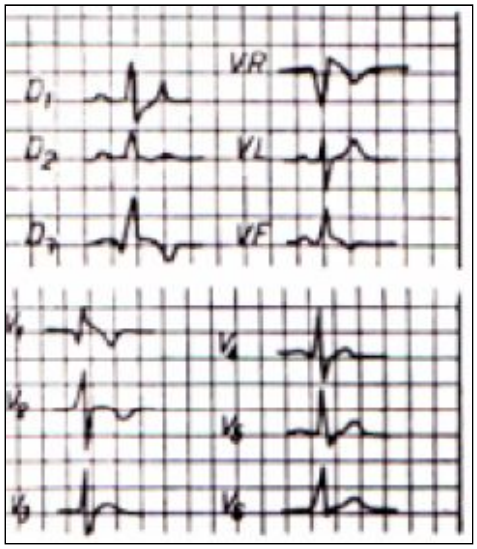
In general, in the beginning, is present in many lead ST segment elevation. Later T waves in leads are reversed previously showed ST elevation. However, contrary to what is seen in myocardial infarction, the ST segment, if it occurs in the three standard leads, has the same meaning here, rather than in the opposite direction in D1 and D3. Furthermore, there is the Q wave myocardial necrosis.

The ECG abnormalities disappear after a few days, but sometimes only after a few weeks.

Pericarditis rheumatic dry. The original route accuses ST superdesnivelamento in standard leads. Three weeks later disappeared but the unevenness of

ST. OT is flattened in D1, D2 and D2 negative.





X - PULMONARY EMBOLISM

In pulmonary embolism, large deviation occurs in the electrical axis to the right ventricular complexes are of the type S D1, and Q D3. The T wave is negative in the right precordial. The drawing is of right ventricular overload, sometimes with incomplete right bundle branch block.

These deformations disappear, often within a few days, unless the emboli are not the starting point of a permanent and progressive right heart failure.