|| ISSN(online): 2589-8698 || ISSN(print): 2589-868X ||
International Journal of Medical and Biomedical Studies
Available Online at www.ijmbs.info
NLM (National Library of Medicine ID: 101738825)
Index Copernicus Value 2020: 79.44
Review Article
Volume 6, Issue 01; January: 2022; Page No. 122-144

CARDIOVASCULAR DISEASE IN WORKERS -EARLY DETECTION AND EARLY TREATMENT--ROLE OF HEALTH CAMPS FOR INDUSTRIAL /ESTABLISHMENTS WORKERS AND BENEFICIARIES

Dr K Padma

Joint Director, IMS, Hyd

Article Info: Received 02 January 2022; Accepted 25 January 2022
DOI: https://doi.org/10.32553/ijmbs.v6i1.2465
Corresponding author: Dr K Padma
Conflict of interest: No conflict of interest.

Abstract

Identifying coronary artery disease in itsearliest stages could mean the difference between lifeand death for thousands ofpeople who don't realize they are at risk from this potentially life-threatening condition.

Keywords: CVD, CAD

Introduction

Cardiovascular disease (CVD) still remains the main cause of morbidity and mortality and consequently early diagnosis is of paramount importance.

Working conditions can be regarded as an additional risk factor for CVD. Since different aspects of the job may affect vascular health differently, it is important to consider occupation from multiple perspectives to better assess occupational impacts on health many features of the environment exert has an important influence on CVD risk, progression, and severity. Numerous epidemiological and experimental studies showed that air pollution, causes a systemic vascular oxidative stress reaction. Increased cardiovascular mortality was also related to long-term and short-term exposure to nitrogen dioxide. Exposure to air pollution and road traffic was associated with an increased risk of arteriosclerosis with premature aortic and coronary calcification and increased risk of systemic hypertension myocardial infarction, stroke, and acute heart failure.

Atherosclerosis, the basis of coronary artery disease (CAD), develops into a complex process CAD is recognized as a Para occupational disease; thus, working conditions could be regarded as a possible risk factor for disease onset, development, or deterioration .The relationships between psychosocial work load and CAD, as well as interactions among neuropsychological and immunological systems, have also attracted attention, although stress-related disorders comprise only a small fraction of occupational injuries and illnesses, overall, the median time away from work is more than four times greater for stress-related conditions than for all other diseases.

Work-related stress (WRS) is thought to contribute to several occupational illnesses including cardiovascular disease.

There is always a relationship between occupational stress and early atherosclerotic changes Differences in body mass index, systemic hypertension, and smoking may show conflicting results actually, workers in high strain jobs may have bad health behaviors that cannot be adjusted adequately for statistical models and act as confounding factors.

Additionally, workers with known heart disease could choose jobs with lesser degrees of stress or leave overall their work, and people who work in high strain occupations who develop heart disease may potentially change their positions with new ones with lower degrees of stress.

However, the triad of occupational exposures, such as shift work, noise, and physical workload emerged as significant risk factor of CAD.

A deleterious effect of shift work was also shown on lipid metabolism Women working in a rapid forward rotating shift pattern had poorer sleep quality according to self-reported indicators of the validated Pittsburgh Sleep Quality Index and they had a higher prevalence of the metabolic syndrome compared with women working during the day

only working shift was, independently of lifestyle or BMI, significantly related to more elevated plasma triglycerides and rate of hypertriglyceridemia, lower plasma HDL-C levels, and hypertension compared to nonshift daytime work The prevalence of lifestyle- related risk factors including hypertension, diabetes, dyslipidemia, metabolic syndrome, and obesity was higher in men than women and higher overall in workers aged 60–69 years, reinforcing the importance of developing effective strategies for the prevention of cardiovascular disease among middle-aged and older workers, especially in men.

Many features of the environment exert an important influence on CVD risk, progression, and severity. Numerous epidemiological and experimental studies showed that air pollution, causes a systemic vascular oxidative stress reaction. Increased cardiovascular mortality was also related to long-term and short-term exposure to nitrogen dioxide. Exposure to air pollution and road traffic was associated with an increased risk of arteriosclerosis with premature aortic and coronary calcification and increased risk of systemic hypertension, myocardial infarction, stroke, and acute heart failure.

The Global Burden of Disease Study 2015 ranked ambient exposure to fine particulate matter with an aerodynamic diameter of

<2.5 μm (PM2.5) as the fifth most important risk factor for mortality worldwide and the burden from air pollution was comparable to that from hypertension and diabetes mellitus ,thus it should be considered an important modifiable environmental cardiovascular risk factor.

Whereas the link between occupation and cardiovascular disease has been recognized, it is unclear which role occupation plays in the progression of subclinical CVD measured as carotid intima-media thickness (CIMT). Occupation is only an indicator of the person's socioeconomic position or a source of potentially health-compromising exposures. Sometimes

Occupation represents additional damage to the risk profile. Health Camps for workers for early diagnosis and early treatment

Top benefits of conducting health camps and medicine distribution programs is timely health Check-ups, early diagnosis, prompt treatment because most often, people who work in industrial area/establishment suffer from several health complications.

Medical camps are conducted by health professionals to carry out a health intervention, check- up, investigation and treatment. Getting the appropriate kind of health checkup is vital for every human being and while considering it, some important factors like age, lifestyle, family background, and risks are taken into account.

Health examinations and tests at the early stages of the illness can help to cure it faster and save a life before it can cause any damage.

One can live longer and healthier only when the individual gets the right kind of health check-up, screening, and treatments.

Even the most basic checkups can identify underlying illnesses.

These provide overall physical examinations which include eyes and health check- ups, assessment of the functioning of vital organs like the heart, lungs, digestive system, liver, kidneys, and immune system. These are extremely helpful for the workers who earn a meager income and cannot afford expensive healthcare services.

These can be considered as a life-saving program that aims to provide medical and surgical intervention by a mobile team with varieties of medical specialized services. The main objective is to provide initial care to people in life-threatening conditions which reflect the unique strengths and goals of medical ethics

We obviously need to improve our failing healthcare system where costs are skyrocketing for the care they need.

Healthcare is a basic right of a human being that should be available for everyone and shouldn't be treated as a commodity only for those who can spend money to buy the services.

The physicians are obliged to act in the best interest of the patients. They inform the whole truth about the diseases and treatments of the patients. Every patient attending the camps are treated similarly and cared for evenly.

Other objectives are as stated below:

Raise health awareness among the community and teach them to deal with communicable and non- communicable diseases.

Register rare and severe cases and refer them to specialized hospitals of ESI. Evaluate the living conditions and determine the obstacles and challenges to work on solving their problems.

Refer medical cases towards surgeries if required.

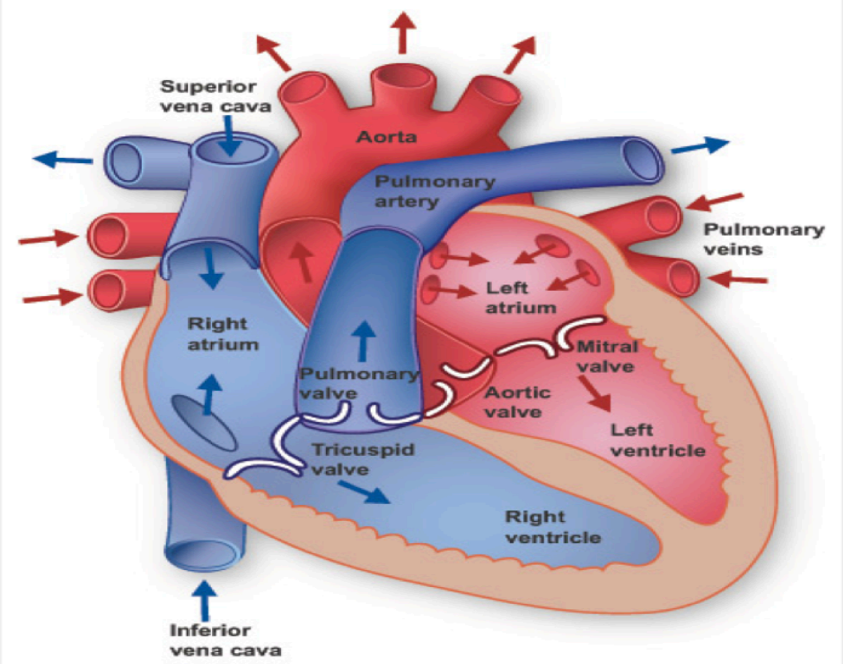
Free-Of-Cost Medicines:

People who work in various industries /establishment cannot afford the rising medical costs .These are set up with a sacred aim to bring awareness among them to basic healthcare services or knowledge about the diseases they are suffering from, and do timely and early investigations, Give medical advice, medicine and refer them for specialized treatment or surgery whenever it is required. These camps make sure people are getting the healthcare at the right time, early diagnosis and seeing the doctor early enough before a small health problem turns serious.

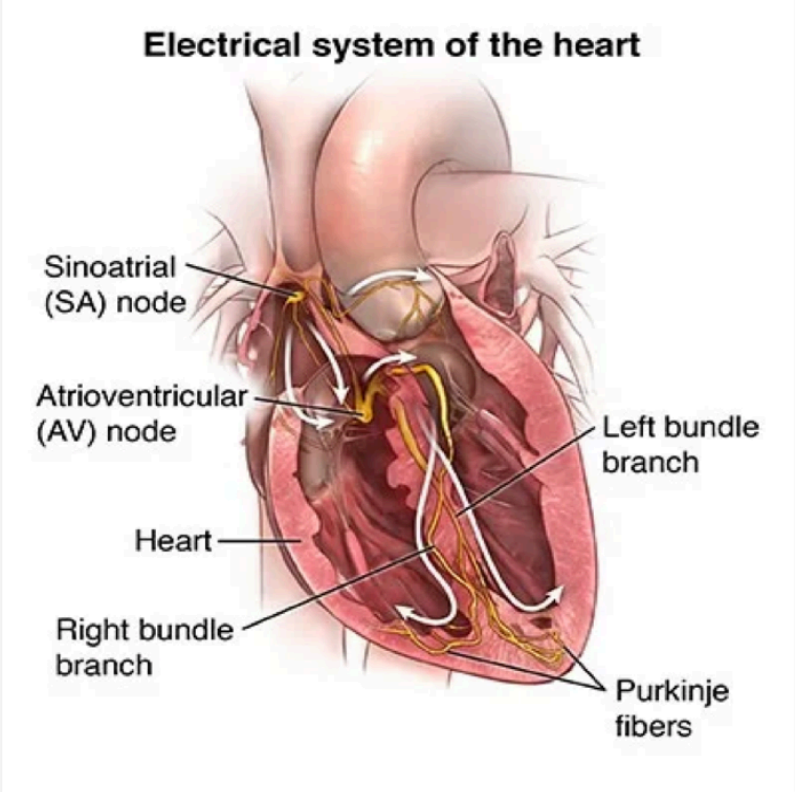
Several camps were done by the IMS team and JD IMS Hyd, the team consisted of doctors of various specialties of various dispensaries, staff nurses, pharmacist, lab-technician, and other staff of various dispensaries worked in this camps /dispensaries and study and screening for cardiac diseases were done on more than 25 thousand workers /beneficiaries from various industries / establishment.

Heart

Cardiac muscle (also called heart muscle or myocardium) is one of threetypes of vertebrate muscle tissue, with the other two being skeletal muscle and smooth muscle. It is involuntary, striated muscle that constitutes the main tissue of the wall of the heart.

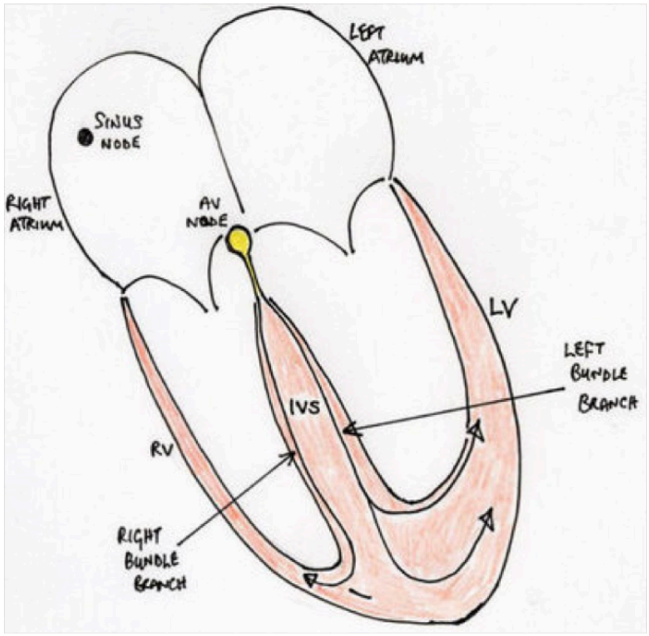


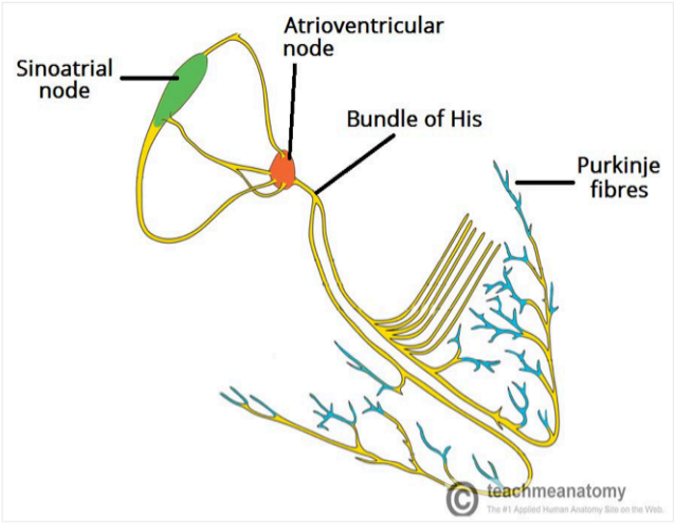
The sequence of electrical events during one full contraction of the heart muscle:



- An excitation signal (an action potential) is created by the sinoatrial (SA)node.

- The wave of excitation spreads across the atria, causing them tocontract.
- Upon reaching the atrioventricular (AV) node, the signal is delayed.
- It is then conducted into the bundle of His, down the interventricular septum.
- The bundle of His and the Purkinje fibres spread the wave impulses along the ventricles, causing them to contract.





ECG used for cardiac investigation

The electrocardiogram (ECG) is one of the simplest and oldest cardiac investigations available, yet it can provide a wealth of useful information and remains an essential part of the assessment of cardiac patients.

An ECG is simply a representation of the electrical activity of the heart muscle as it changes with time, usually printed on paper for easier analysis.

Like other muscles, cardiac muscle contracts in response to electrical depolarisation of the muscle cells. It is the sum of this electrical activity, when amplified and recorded for just a few seconds that we know as an ECG.

- To look for the cause of chest pain
- To evaluate problems which may be heart- related, such as severe tiredness, shortness of breath, dizziness, or fainting
- To identify irregular heartbeats
- To help determine the overall health of the heart before procedures such as surgery; or after treatment for conditions such as a heart attack(myocardial infarction, or MI), endocarditis (inflammation or infection ofone or more of the heart valves); or after heart surgery or cardiac catheterization
- To see how an implanted pacemaker is working
- To determine how well certain heart medicines are working
- To get a baseline

tracing of the heart's function during a physical exam; this may be used as a comparison with future ECGs, to determine if there have been any changes

Basic Electrophysiology of the Heart

The normal cardiac cycle begins with spontaneous depolarisation of the sinus node, an area of specialised tissue situated in the high right atrium (RA). A wave of electrical depolarisation then spreads through the RA and across the inter-atrial septum into the left atrium (LA).

The atria are separated from the ventricles by an electrically inert fibrous ring, so that in the normal heart the only route of transmission of electrical depolarization from atria to ventricles is through the atrioventricular (AV) node. The AV node delays the electrical signal for a short time, and then the wave of depolarisationspreads down the interventricular septum (IVS), via the bundle of His and the right and left bundle branches, into the right (RV) and left (LV) ventricles.

Hence with normal conduction the two ventricles contract simultaneously, which is important in maximising cardiac efficiency.

After complete depolarisation of the heart, the myocardium must thenrepolarise, before it can be ready to depolarise again for the next cardiaccycle.

Basic electrophysiology of the heart

Electrical axis and recording lead vectors

The ECG is measured by placing a series of electrodes on the patient's skin– so it is known as the 'surface' ECG.

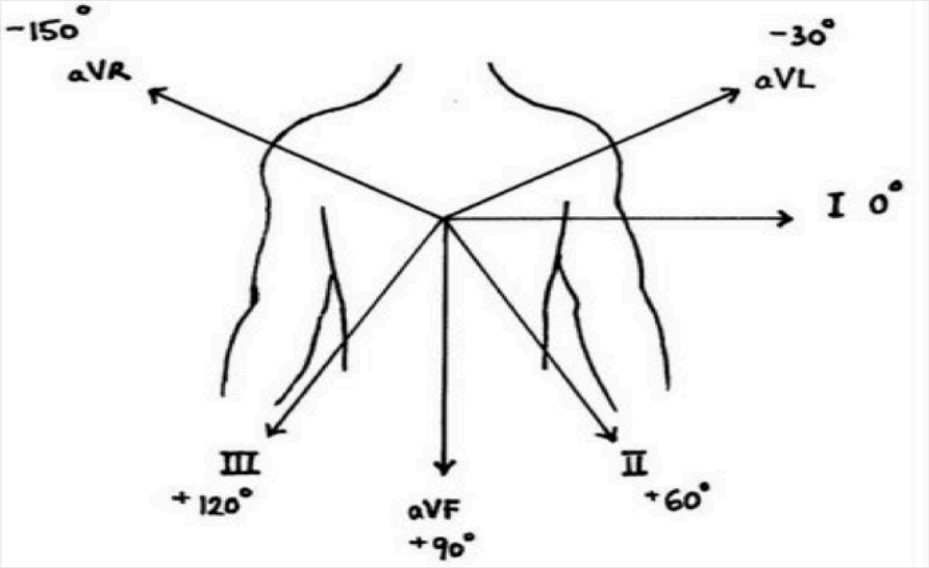
The wave of electrical depolarisation spreads from the atria down though the IVS to the ventricles. So the direction of this depolarisation is usuallyfrom the superior to the inferior aspect of the heart.

The direction of the wave of depolarisation is normally towards the left due to the leftward orientation of the heart in the chest and the greater musclemass of the left ventricle than the right. This overall direction of travel of the electrical depolarisation through the heart is known as the electricalaxis.

A fundamental principle of ECG recording is that when the wave of depolarisation travels toward a recording lead this results in a positive or upward deflection. When it travels away from a recording lead this results ina negative or downward deflection.

The electrical axis is normally downward and to the left but we can estimateit more accurately in individual patients if we understand from which'direction' each recording lead measures the ECG-

Orientation of the limb leads showing the direction from which each lead 'looks' at the heart.



By convention, we record the standard surface ECG using 12 differentrecording lead 'directions,' though rather confusingly only 10 recordingelectrodes on the skin are required to achieve this. Six of these are recorded from the chest overlying the heart – the chest or precordial leads. Four are recorded from the limbs – the limb leads. It is essential that each of the 10 recording electrodes is placed in itscorrect position, otherwise the appearance of the ECG will be changedsignificantly, preventing correct interpretation.

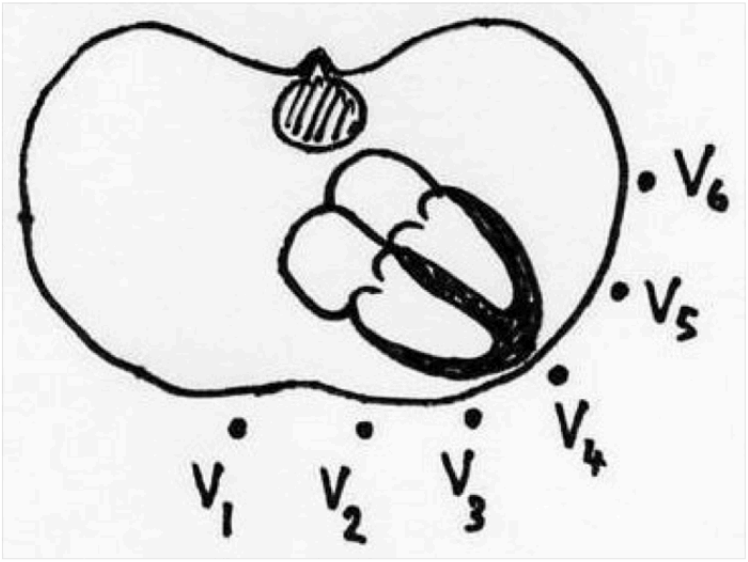
The limb leads record the ECG in the coronal plane, and so can be used to determine the electrical axis (which is usually measured only in thecoronal plane).

The limb leads are called leads I, II, III, AVR, AVL and AVF.

A horizontal line through the heart and directed to the left (exactly in the direction of lead I) is conventionally labelled as the reference point of 0degrees (0 o). The directions from which other leads 'look' at the heart aredescribed in terms of the angle in degrees from this baseline.

The electrical axis of depolarisation is also expressed in degrees and isnormally in the range from -30 0 to + 90 0. A detailed explanation of how to determine the axis isbeyond the scope of this article but the principles mentioned here should help readers to understand the concepts involved.

The chest leads record the ECG in the transverse or horizontal plane, and are called V1, V2, V3, V4, V5 and V6



Transverse section of the chest showing the orientation of the six chest leads in relation to the heart

Voltage and timing intervals

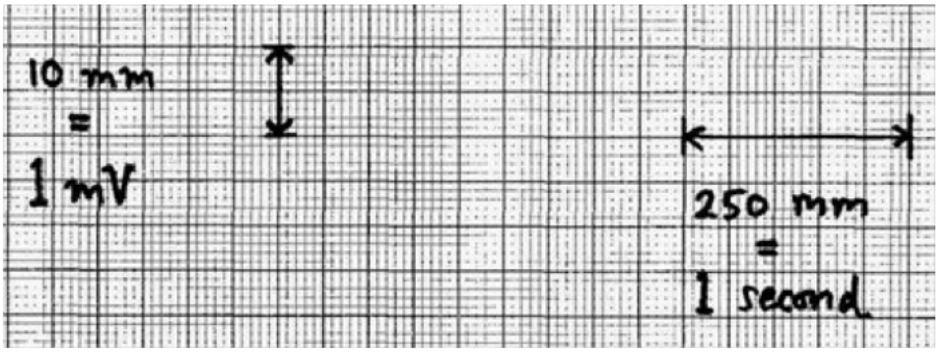
It is conventional to record the ECG using standard measures for amplitude of the electrical signal and for the speed at which the paper moves during the recording. This allows:

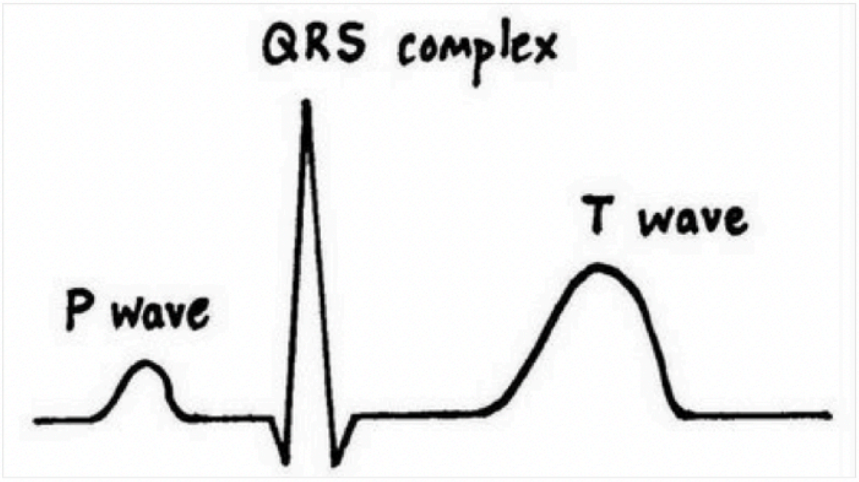
- Easy appreciation of heart rates and cardiac intervals and
- Meaningful comparison to be made between ECGs recorded on different occasions or by different ECG machines.

The amplitude, or voltage, of the recorded electrical signal is expressed on an ECG in the vertical dimension and is measured in millivolts (mV). Onstandard ECG paper 1mV is represented by a deflection of 10 mm. Anincrease in the amount of muscle mass, such as with left ventricular hypertrophy (LVH), usually results in a larger electrical depolarisation signal, and so a larger amplitude of vertical deflectionon the ECG.

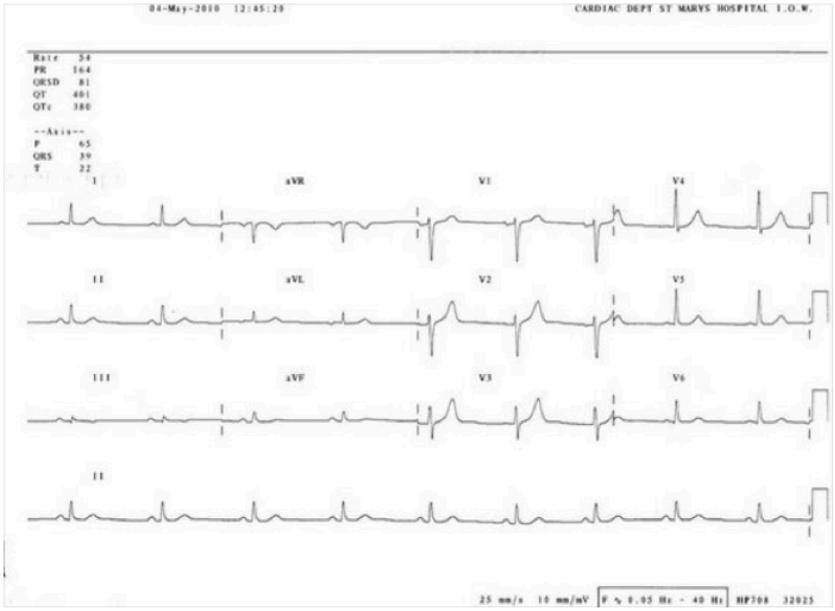
An essential feature of the ECG is that the electrical activity of the heart isshown as it varies with time. In other words we can think of the ECG as agraph, plotting electrical activity on the vertical axis against time on thehorizontal axis. Standard ECG paper moves at 25 mm per second during real-time recording. This means that when looking at the printed ECG a distance of 25 mm along the horizontal axis represents 1 second in time.ECG paper is marked with a grid of small and large squares. Each small square represents 40 milliseconds (ms) in time along the horizontal axis and each larger square contains 5 small squares, thus representing 200 ms.

Standard paper speeds and square markings allow easy measurement of cardiac timing intervals. This enables calculation of heart rates and identification of abnormal electrical conduction within the heart





Sample of standard ECG paper showing the scale of voltage, measured on the vertical axis, against time on the horizontal axis
The major waves of a single normal ECG pattern



Example of a normal 12 lead ECG; notice the downward deflection of all signals recorded from lead aVR. This is normal, as the electrical axis is directly away from that lead
It will be clear from above that the first structure to be depolarised during normal sinus rhythm is the right atrium, closely followed by the left atrium. So the first electrical signal on a normal ECG originates from the atria and isknown as the P wave. Although there is usually only one P wave in mostleads of an ECG, the P wave is in fact the sum of the electrical signals from the two atria, which are usually superimposed.
There is then a short, physiological delay as the atrioventricular (AV) nodeslows the electrical depolarisation before it proceeds to the ventricles. Thisdelay is responsible for the PR interval, a short period where no electricalactivity is seen on the ECG, represented by a straight horizontal or 'isoelectric' line.
Depolarisation of the ventricles results in usually the largest part of the ECG signal (because of the greater muscle mass in the ventricles) and this isknown as the QRS complex.

- The Q wave is the first initial downward or 'negative' deflection
- The R wave is then the next upward deflection (provided it crosses theisoelectric line and becomes 'positive')
- The S wave is then the next deflection

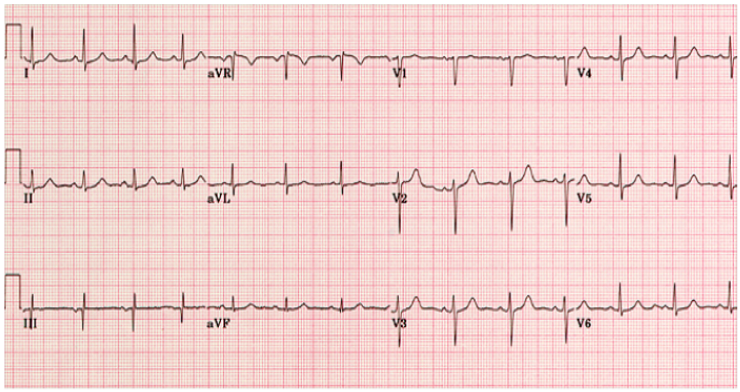
downwards, provided it crosses the isoelectric line to become brieflynegative before returning to the isoelectric baseline
Ventricular depolarization (activation) is depicted by the QRS complex, whereas ventricular repolarization is defined by the interval from the beginning of the QRS complex to the end of the T- or U-wave.
On the surface ECG, ventricular repolarization components include the
J-wave, ST-segment, and T- and U-waves. . The ST segment is normallyisoelectric, and the T wave in most leads is an upright deflection of variableamplitude and duration
Normal interval
The recording of an ECG on standard paper allows the time taken for the various phases of electrical depolarisation to be measured, usually inmilliseconds. There is a recognised normal range for such 'intervals':

- PR interval (measured from the beginning of the P wave to the firstdeflection of the QRS complex). Normal range 120 – 200 ms (3 – 5small squares on ECG paper).
- QRS duration (measured from first deflection of QRS complex to end of QRS complex at isoelectric line). Normal range up to 120 ms (3 smallsquares on ECG paper).
- QT interval (measured from first deflection of QRS complex to end of T wave at isoelectric line). Normal range up to 440 ms (though varies with heart rate and may be slightly longer in females)

Heart rate estimation from the ECG
Standard ECG paper allows an approximate estimation of the heart rate (HR) from an ECG recording. Each second of time is represented by 250 mm (5 large squares) along the horizontal axis. So if the number of large squares between each QRS complex is:

- 5 - the HR is 60 beats per minute.
- 3 - the HR is 100 per minute.
- 2 - the HR is 150 per minute.

Normal ecg



In normal ECG, the heart is beating in a regular sinus rhythm between 60 - 100 beats per minute (specifically 82 bpm). All the important intervals on thisrecording are within normal ranges.
1. P wave:

- upright in leads I, aVF and V3 - V6
- normal duration of less than or equal to 0.11 seconds
- polarity is positive in leads I, II, aVF and V4 - V6; diphasic in leads V1 andV3; negative in aVR
- shape is generally smooth, not notched or peaked 2. PR interval:
- Normally between 0.12 and 0.20 seconds.

3. QRS complex:

- Duration less than or equal to 0.12 seconds, amplitude greater than 0.5 mV in at least one standard lead, and

greater than 1.0 mV in at least one precordial lead. Upper limit of normal amplitude is 2.5- 3.0 mV.

- small septal Q waves in I, aVL, V5 and V6 (duration less than or equal to0.04 seconds; amplitude less than 1/3 of the amplitude of the R wave in the same lead).
- represented by a positive deflection with a large, upright R in leads I, II,V4 - V6 and a negative deflection with a large, deep S in aVR, V1 and V2
- in general, proceeding from V1 to V6, the R waves get taller while the Swaves get smaller. At V3 or V4, these waves are usually equal. This is called the transitional zone.

4. ST segment:

- isoelectric, slanting upwards to the T wave in the normal ECG
- can be slightly elevated (up to 2.0 mm in some precordial leads)
- never normally depressed greater than0.5 mm in any lead 5. T wave:
- T wave deflection should be in the same direction as the QRS complexin at least 5 of the 6 limb leads
- normally rounded and asymmetrical, with a more gradual ascent thandescent
- should be upright in leads V2 - V6, inverted in aVR
- amplitude of at least 0.2 mV in leads V3 and V4 and at least 0.1 mV inleads V5 and V6
- isolated T wave inversion in an asymptomatic adult is generally a normal variant

6. QT interval:

- Durations normally less than or equal to 0.40 seconds for males and 0.44 seconds

ECG Disease Patterns

The electrocardiogram can be used to diagnose a wide variety of cardiacand non- cardiac conditions.

PRINCIPLE OF THE ECG DIAGNOSIS

the possibilities to solve the cardiac inverse problem -no unique solution exists for the inverse problem. From clinical practice it is possible to make accurate ECG diagnoses in some diseases and to estimate other diseases with an acceptable probability, the inverse solution is impossible ifmeasurements cannot be made inside the source and if no additional information about the nature of the source isavailable. There is, however, much knowledge of the electrophysiological behaviorof the heart. This limits the degrees of freedom of the source and reduces the degree of uncertainty in reaching a diagnosis.

The following are examples of these helpful constraints:

1. The size, location, and orientation of the heart are well known and theirvariabilities are limited.
2. The action impulse of individual muscle cells can be approximated ashaving only two electrophysiological states: (re)polarization anddepolarization.
3. Each muscle cell exhibits a specific form of activation; depolarization is followed by repolarization after approximately 0.2-0.4 seconds.
4. The atria and the ventricles form temporarily separate regions ofactivation.
5. The propagation velocity of the activation front in various parts of the heart muscle is known.
6. The conduction system has a dominant effect on initiation of theactivation front.
7. The relationship between muscle load and muscle hypertrophy is well understood.
8. There are a limited number of causes of muscular overload.
9. The electrophysiological effect of ischemia on heart muscle is known.
10. The location of ischemia or infarction is governed by the anatomy of the coronaryarteries.
11. There are a limited number of congenital cardiac abnormalities.

These anatomical and physiological constraints limit the degrees of freedomof the inverse solution and usually make it possible to obtain solutions.However, in most cases the cardiac diagnosis must be made more accurately. The diagnosis often needs to be verified or completely made with other diagnostic methods like auscultation, x- ray, coronary angiography,radiocardiographic imaging, clinical chemistry, ultrasound, and so on.

Bioelectric principles in ECG diagnosis

This discussion of ECG diagnosis is based on the following three principles:

First, the propagating activation front is characterized by its resultant vector. This signal can be detected and estimated through the lead vectoraccording to Equation

When the heart's electric activity is considered a vector, it is usually easierfirst to examine the path (trajectory) of the vector's tip (the vectorcardiogram). Then the signals in the 12- lead ECG may be regardedas projections of the electric heart vector on the respective lead vectors as afunction of time (multiplied by the absolute value of the lead vector).

Second, the sensitivity of the lead may be considered distributed accordingto lead field theory. In this case the propagating activation front contributes to the ECG signal of the lead according to Equation namely

In this formulation the dipole sources are not reduced to a single resultant dipole, but are considered as spatially distributed. Furthermore, the volume conductor inhomogeneities are taken into account.

Third, the solid angle theorem offers substantial help for understanding theformation of the ECG signal, especially in the diagnosis of myocardial infarctionaccording to Equation In arriving at Equation one assumes the double layer sources to be uniform, but otherwise takes intoaccount their spatial distribution.

However, the volume conductor is assumed to be infinite in extent anduniform.

This leads to the solution of the inverse problem through the empiricalapproach.

THE APPLICATION AREAS OF ECG DIAGNOSIS

- The main applications of the ECG to cardiological diagnosis include thefollowing
- The electric axis of the heart
- Heart rate monitoring
- Arrhythmias
- Supraventricular arrhythmias
- Ventricular arrhythmias
- Disorders in the activation sequence
- Atrioventricular conduction defects (blocks)
- Bundle-branch block
- Wolff-Parkinson- White syndrome
- Increase in wall thickness or size of the atria and ventricles
- Atrial enlargement (hypertrophy)
- Ventricular enlargement (hypertrophy)
- Myocardial ischemia and infarction
- Ischemia
- Infarction
- Drug effect
- Digitalis
- Quinidine
- Electrolyte imbalance
- Potassium
- Calcium
- Carditis
- Pericarditis
- Myocarditis
- Pacemaker monitoring

In cardiac diseases where cardiac muscle changes is due to drug effect,electrolyte imbalance, and carditis - their effects on the ECG signal cannotreadily be explained.

Application areas of ECG diagnosis

DETERMINATION OF THE ELECTRIC AXIS OF THE HEART

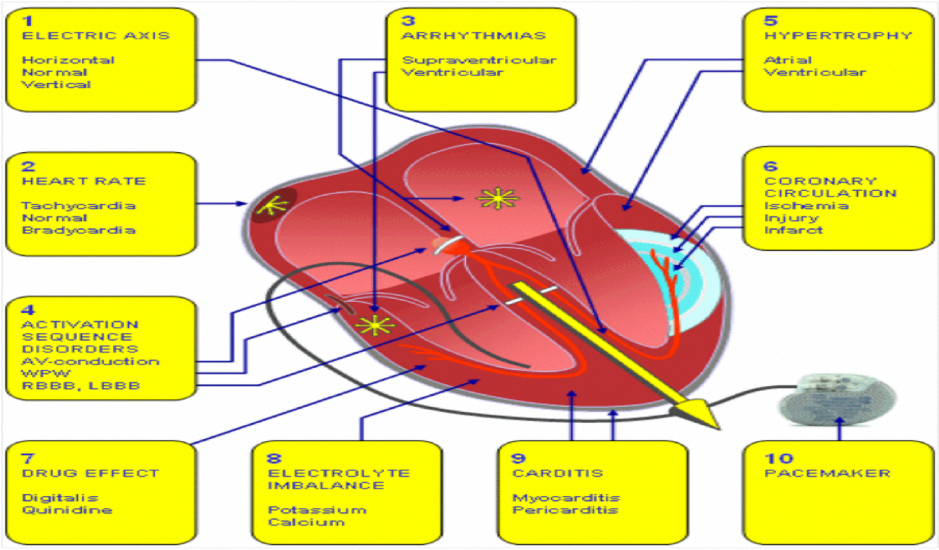
There are five main electrical axis classifications:

1. Normal axis
2. Left axis deviation (LAD)
3. Right axis deviation (RAD)
4. Extreme axis deviation, and
5. Indeterminate axis

The concept of the electric axis of the heart usually denotes the averagedirection of the electric activity throughout ventricular (or sometimes atrial) activation. The term mean vector is frequently used instead of "electricaxis." The direction of the electric axis may also denote the instantaneous direction of the electric heart vector.

This is shown in vector cardiography as a function of time. The normal range of the electric axis lies between+30° and -110° in the frontal plane and between +30° and -30° in the transverse plane.

The direction of the electric axis may be approximated from the 12-lead ECG by finding the lead in the frontal plane, where the QRS- complex has largest positive deflection. The direction of the electric axis is in the direction of this lead vector. The result can be checked by observing that the QRS-complex is symmetrically biphasic in the lead that is normal to the electricaxis.(In the evaluation of the ECG it is beneficial to use the lead-aVR instead of the lead aVR)

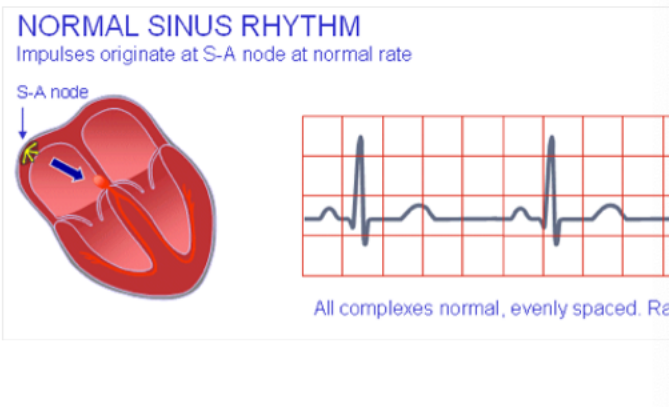


Deviation of the electric axis to the right is an indication of increased electricactivity in the right ventricle due to increased right ventricular mass. This is usually a consequence of chronic obstructive lung disease, pulmonary emboli, certain types of congenital heart disease, or other disorders causing severe pulmonary hypertension and corpulmonale.

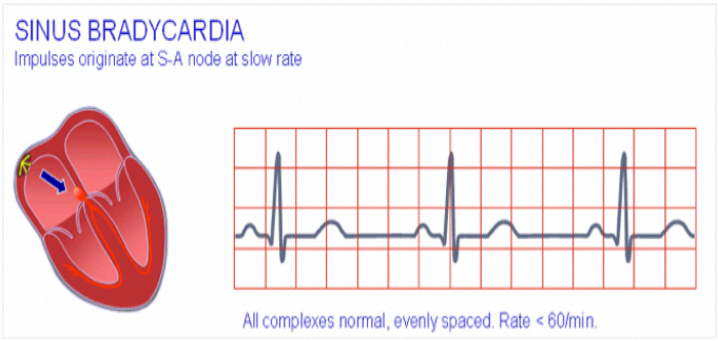
Deviation of the electric axis to the left is an indication of increased electricactivity in the left ventricle due to increased left ventricular mass. This is usually a

consequence of hypertension, aortic stenosis, ischemic heartdisease, or some intraventricular conduction defects
The ventricular axis is typically used in common clinical practice, although the atrial axis can be quite useful in clinical situations. Since theleft
ventricle makes up most of the heart muscle under normal circumstances; thus, it generates the most electrical force visible on the ECG The normal ventricular axis is thus directed downward and slightly towards the left.
The ventricular axis can be determined by looking at the QRS complex, which represents ventricular depolarization. Because the QRS complex is used to determine the ventricular axis, it is also referred to as the QRS axis.The ventricular (QRS) axis signifies the sum of all individual vectorsgenerated by the depolarization waves of ventricular myocytes egventricular hypertrophy.
CARDIAC RHYTHM DIAGNOSIS
Differentiating the P-, QRS- and T-waves
Because of the anatomical difference of the atria and the ventricles, their sequential activation,depolarization, and repolarization produce clearly differentiabledeflections. This may be possible even when they do not follow one another in the correct sequence: P-QRS-T.
Identification of the normal QRS-complex from the P- and T-waves does notcreate difficulties because it has a characteristic waveform and dominating amplitude. This amplitude is about 1 mV in a normal heart and can be much greater in ventricular hypertrophy. The normal duration of the QRS is
0.08-0.09 s.
If the heart does not exhibit atrial hypertrophy, the P- wave has an amplitude of about 0.1 mV and duration of
0.1 s. For the T-wave both of these numbers are about double. The T-wave can be differentiated from the P- wave by observing that the T-wave follows the QRS- complex after about 0.2 s.
Supra-ventricular rhythms
-Definition
Cardiac rhythms may be divided into two categories: supraventricular(above the ventricles) and ventricular rhythms.
The origin of supraventricular rhythms (a single pulse or a continuousrhythm) is in the atria or AV junction, and the activation proceeds to the ventricles along the conduction system in a normal way.
Normal sinus rhythm
Normal sinus rhythm is the rhythm of a healthy normal heart, where the sinus node triggers the cardiac activation. This is easily diagnosed by notingthat the three deflections, P-QRS-T, follow in this order and are differentiable. The sinus rhythm is normal if its frequency is between 60 and 100/min.<

- Normal sinus rhythm.

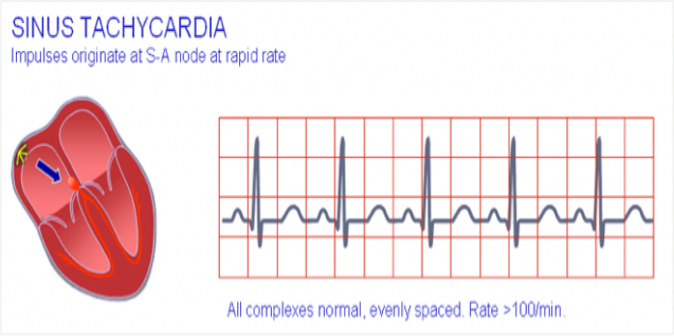


Sinus bradycardia
A sinus rhythm of less than 60/min is called sinus bradycardia. This may bea consequence of increased vagal or parasympathetic tone.



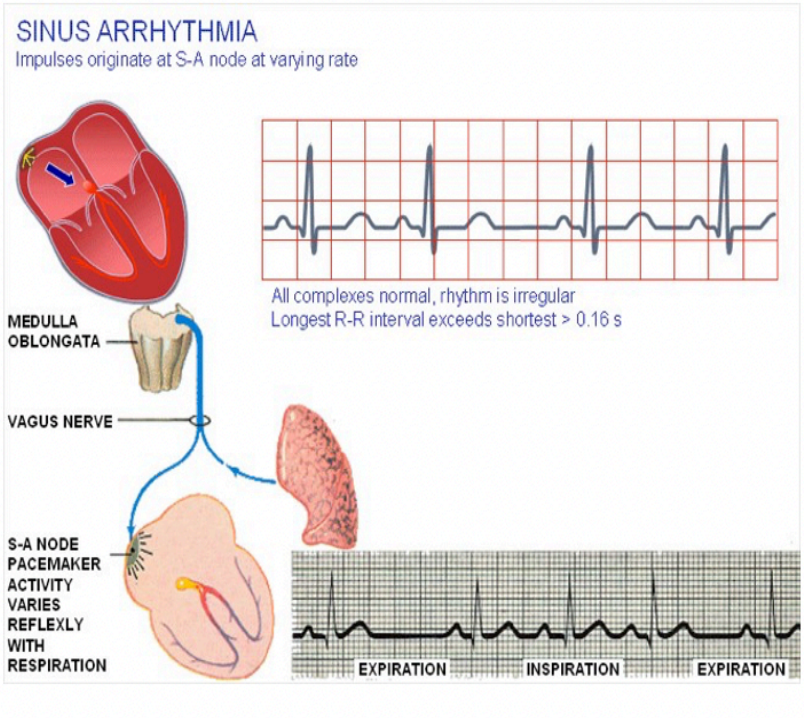
- Sinus bradycardia.

Sinus tachycardia



A sinus rhythm of higher than 100/min is called sinus tachycardia. It occurs most often as a physiological response to physical exercise or psychicalstress, but may also result from congestive heart failure.

- Sinus tachycardia.



Sinus arrhythmia

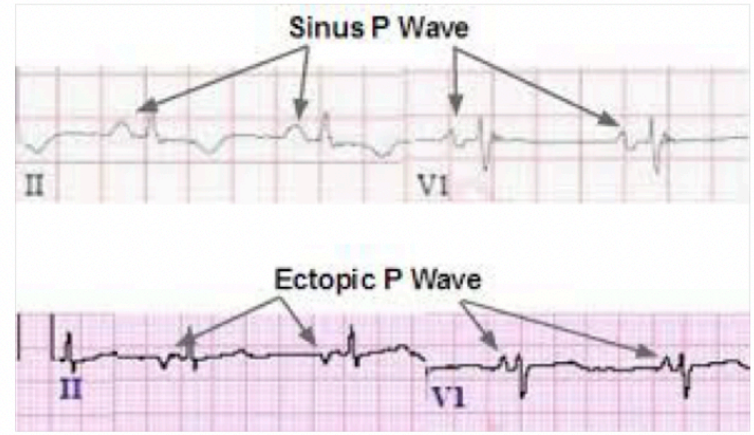
If the sinus rhythm is irregular such that the longest PP- or RR-intervalexceeds the shortest interval by 0.16 s, the situation is called sinusarrhythmia. This situation is very common in all age groups. This arrhythmia is so common in young people that it is not considered a heart disease.One origin for the sinus arrhythmia may be the vagus nerve which mediatesrespiration as well as heart rhythm. The nerve is active during respiration and, through its effect on the sinus node, causes an increase in heart rate during inspiration and a decrease during expiration. The effect is particularly pronounced in children.

Note, that in all of the preceding rhythms the length of the cardiacactivation cycle (the P-QRS- T-waves together) is less than directly proportional to the PP-time. The main time interval change is between the T-wave and the next P-wave. This is easy to understand since the pulserate of the sinus node is controlled mainly by factors external to the heart while the cardiac conduction velocity is controlled by conditions internal to the heart.

- Sinus arrhythmia.

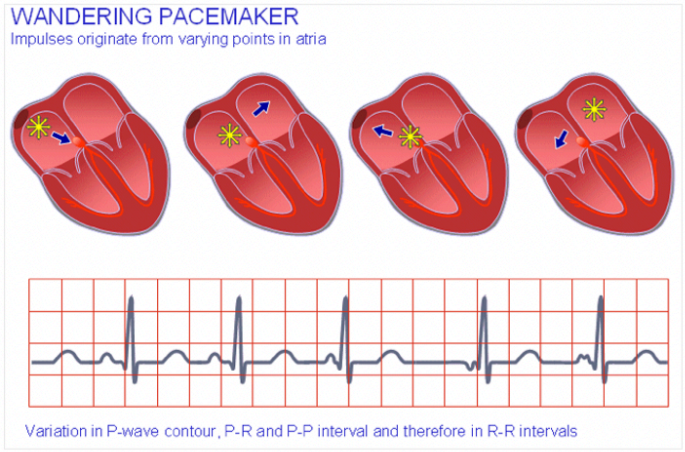
Non-sinus atrial rhythm

The origin of atrial contraction may be located somewhere else in the atriaother than the sinus node. If it is located close to the AV node, the atrialdepolarization occurs in a direction that is opposite the normal one. An obvious consequence is that in the ECG the P-wave has opposite polarity..



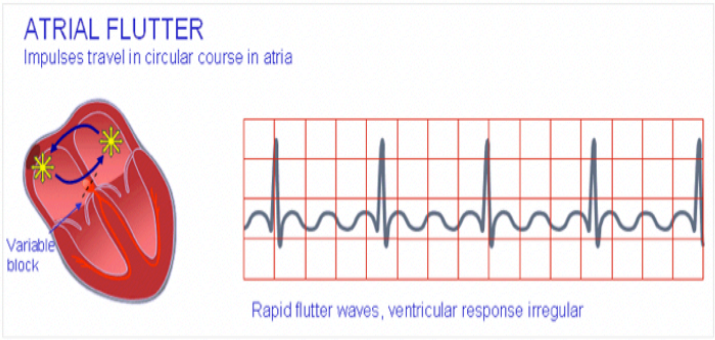
Wandering pacemaker

The origin of the atrial contraction may also vary or wander. Consequently, the P-waves will vary in polarity, and the PQ-interval will also vary.



- Wandering pacemaker.

Paroxysmal atrial tachycardia (PAT) Paroxysmal atrial tachycardia (PAT) describes the condition when the P- waves are a result of a reentrant activation front (circus movement) in the atria, usually involving the AVnode. This leads to a high rate of activation, usually between 160 and220/min. In the ECG the P-wave is regularly followed by the QRS-complex. The isoelectric baseline may be seen between the T-wave and the next P-wave.



Atrial flutter

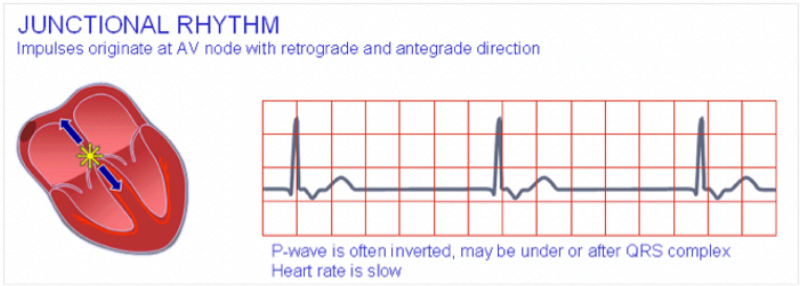
When the heart rate is sufficiently elevated so that the isoelectric intervalbetween the end of T and beginning of P disappears, the arrhythmia iscalled atrial flutter. The origin is also believed to involve a reentrant atrialpathway. The frequency of these fluctuations is between 220 and 300/min. The AV-node and, thereafter, the ventricles are generally activated by everysecond or every third atrial impulse (2:1 or 3:1 heart block).

- Atrial flutter.

Atrial fibrillation

The activation in the atria may also be fully irregular and chaotic, producing irregular fluctuations in the baseline. A consequence is that the ventricular rate is rapid and irregular, though the QRS contour is usually normal. Atrial fibrillation occurs as aconsequence of rheumatic disease, atherosclerotic disease, hyperthyroidism,and pericarditis. (It may also occur in healthy subjects as a result of strongsympathetic activation.)

- Atrial fibrillation.



Junctional rhythm

If the heart rate is slow (40-55/min), the QRS- complex is normal, theP- waves are possibly not seen, then the origin of the cardiac rhythm is in the AVnode.

Because the origin is in the juction between atria and ventricles, this is called junctional rhythm. Therefore, the activation of the atria occurs retrograde(i.e., in the opposite direction).

Depending on whether the AV-nodal impulse reaches the atria before,simultaneously, or after the ventricles, an opposite polarity P-wave will beproduced before, during, or after the QRS-complex, respectively. In the second case the P-wave will be superimposed on the QRS- complex and will not be seen.

- Junctional rhythm.

Ventricular arrhythmias

Definition

In ventricular arrhythmias ventricular activation does not originate from the AV node and/or does not proceed in the ventricles in a normal way. If the activation proceeds to the ventricles along the conduction system, the inner walls of the ventricles are activated almost simultaneously and the activation front proceeds mainly radially toward the outer walls. As a result, the QRS-complex is of relativelyshort duration. If the ventricular conduction system is broken or theventricular activation starts far from the AV node, it takes a longer time for the activation front to proceed throughout the ventricular mass.

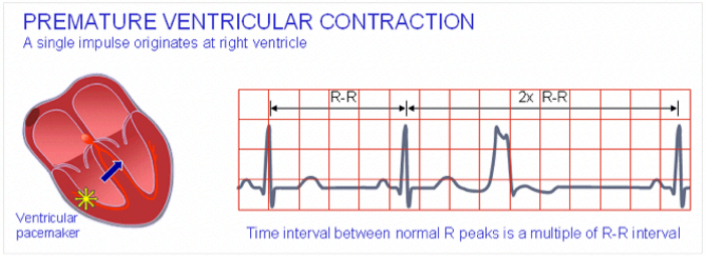
The criterion for normal ventricular activation is a QRS-interval shorter than0.1 s. A QRS-interval lasting longer than 0.1 s indicates abnormal ventricularactivation.

Premature ventricular contraction

A premature ventricular contraction is one that occurs abnormally early. Ifits origin is in the atrium or in the AV node, it has a supraventricular origin. The complex produced by this supraventricular arrhythmia lasts less than 0.1 s. If the origin is in the ventricular muscle, the QRS-complex has a very abnormal form and lasts longer than 0.1 s.

Usually the P-wave is not associated with it.

- Premature ventricular contraction.



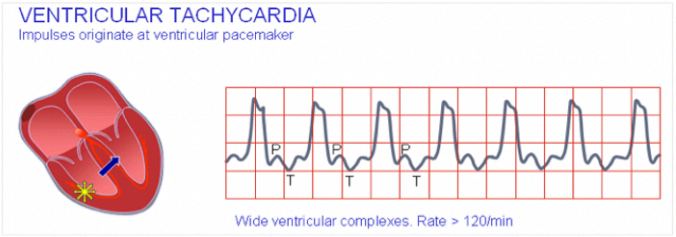
Idioventricular rhythm

If the ventricles are continuously activated by a ventricular focus whoserhythm is under 40/min, the rhythm is called idioventricular rhythm. The ventricular activity may also be formed from short (less than 20 s) bursts ofventricular activity at higher rates (between 40 and 120/ min). This situationis called accelerated idioventricular rhythm.

The origin of the ventricular rhythm may be located by observing the polarity in various leads. The direction of the activation front is, of course, the direction of the lead vector in that lead where the deflection is mostpositive. The origin of the activation is, of course, on the opposite side of the heart when one is looking from this electrode.

Ventricular tachycardia A rhythm of ventricular origin may also be aconsequence of a slower conduction in ischemic ventricular muscle that leads to circular activation (re-entry). The result is activation of the ventricular muscle at a high rate (over 120/min), causing rapid, bizarre, and wide QRS- complexes; the arrythmia is called ventriculartachycardia. As noted, ventricular tachycardia is often a consequence ofischemia and myocardial infarction.

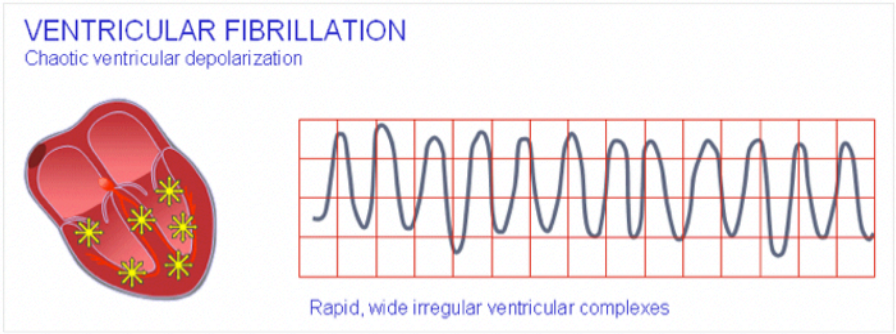
- Ventricular tachycardia.



Ventricular fibrillation When ventricular depolarization occurs chaotically,the situation is called ventricular fibrillation. This is reflected in the ECG,which demonstrates coarse irregular undulations without QRS-complexes. The cause of fibrillation is the establishment of multiple re- entry loopsusually involving diseased heart muscle. In this arrhythmia the contraction of the

ventricularmuscle is also irregular and is ineffective at pumping blood. The lack of blood circulation leads to almost immediate loss of consciousness and deathwithin minutes. The ventricular fibrillation may be stopped with an externaldefibrillator pulse and appropriate medication.

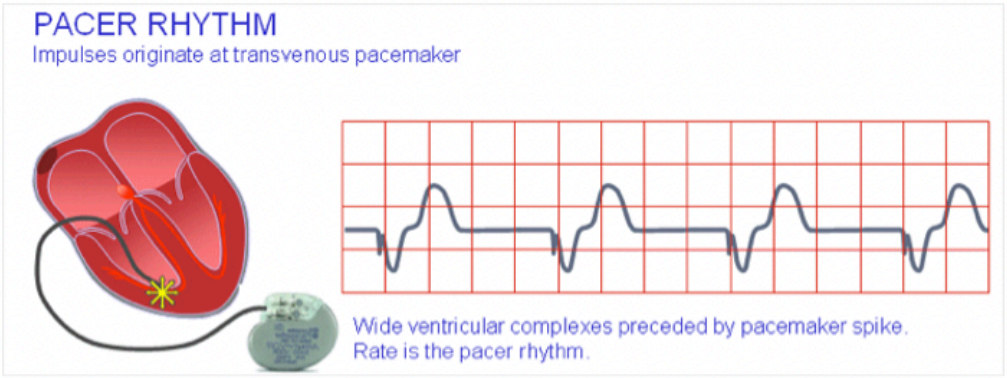
- Ventricular fibrillation.



Pacer rhythm

A ventricular rhythm originating from a cardiac pacemaker is associated with wide QRS-complexes because the pacing electrode is (usually) located in the right ventricle and activation does not involve the conduction system. In pacer rhythm the ventricular contraction is usuallypreceded by a clearly visible pacer impulse spike. The pacer rhythm is usually set to 72/min..

- Pacer rhythm



DISORDERS IN THE ACTIVATION SEQUENCE

Atrioventricular conduction variations

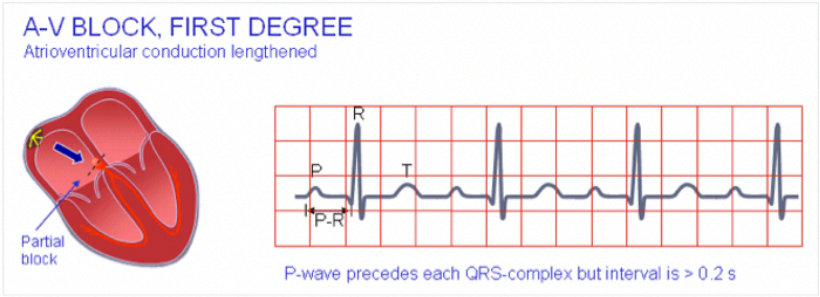
Definition

As discussed earlier, if the

P-waves always precede the QRS-complex with a PR- interval of 0.12-0.2 s,the AV conduction is normal and a sinus rhythm is diagnosed. If the PR-interval is fixed but shorter than normal, either the origin of the impulse is closer to the ventricles (see Section 19.4.2) or the atrioventricular conductionis utilizing an (abnormal) bypass tract leading to pre- excitation of the ventricles. The latter is called the Wolff- Parkinson-White syndrome and is discussed below. The PR-interval may also be variable, such as in awandering atrial pacemaker and multifocal atrial tachycardia.

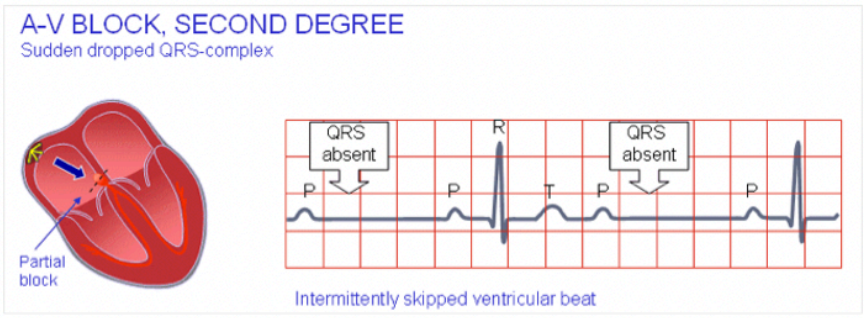
First-degree atrioventricular block When the P-wave always precedes the QRS-complex but the PR-interval is prolonged over 0.2 s, first- degreeatrioventricular block is diagnosed.

- First-degree atrioventricular block.



Second-degree atrioventricular block

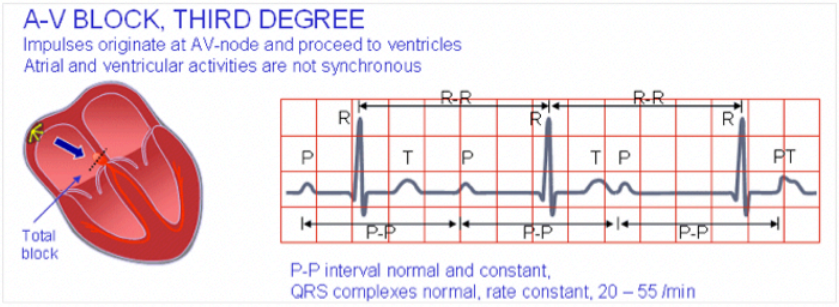
If the PQ-interval is longer than normal and the QRS- complex sometimes does not follow the P-wave, the atrioventricular block is of second-degree. If the PR- interval progressively lengthens, leading finally to the dropout of a QRS- complex,the second degree block is called a Wenkebach phenomenon.



- Second-degree atrioventricular block.

Third-degree atrioventricular block Complete lack of synchronismbetween the P- wave and the QRS-complex is diagnosed as third-degree (ortotal) atrioventricular block. The conduction system defect in third degree AV-block may arise at different locations such as:

- Over the AV-node
- In the bundle of His
- Bilaterally in the upper part of both bundle branches
- Trifascicularly, located still lower, so that it exists in the right bundle-branch and in the two fascicles of the left bundle-branch.



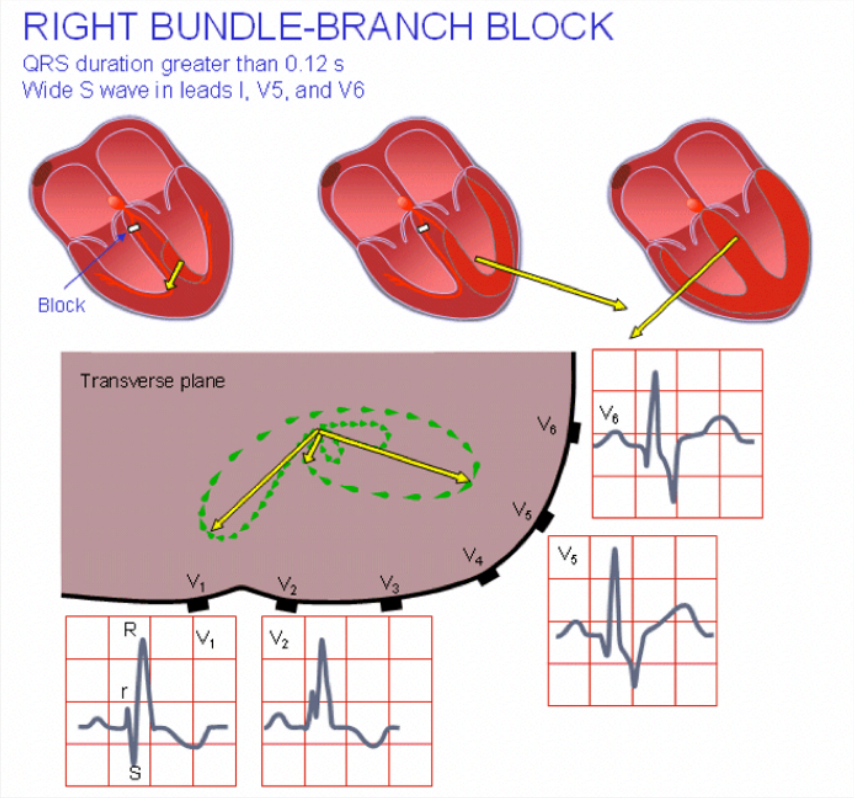
- Third-degree atrioventricular block.

Bundle-branch block

Definition

Bundle-branch block denotes a conduction defect in either of the bundle- branches or in either fascicle of the left bundle-branch. If the two bundle-branches exhibit a block simultaneously, the progress of activation from theatria to the ventricles is completely inhibited; this is regarded as third-degree atrioventricular block (see the previous section). The consequence of left or right

bundle- branch block is that activation of the ventricle must await initiation by the opposite ventricle. After this, activation proceeds entirely on a cell-to-cell basis. The absence of involvement of the conduction system,which initiates early activity of many sites, results in a much slower activation process along normal pathways. The consequence is manifest inbizarre shaped QRS-complexes of abnormally long duration.



Right bundle-branch block

If the right bundle-branch is defective so that the electrical impulse cannot travel through it to the rightventricle, activation reaches the right ventricle by proceeding from the leftventricle. It then travels through the septal and right ventricular musclemass.

This progress is, of course, slower than that through the conduction system and leads to a QRS-complex wider than 0.1 s. Usually the duration criterion for the QRS-complex in right bundle-branch block (RBBB) as well as for the left brundle- branch block (LBBB) is >0.12 s.

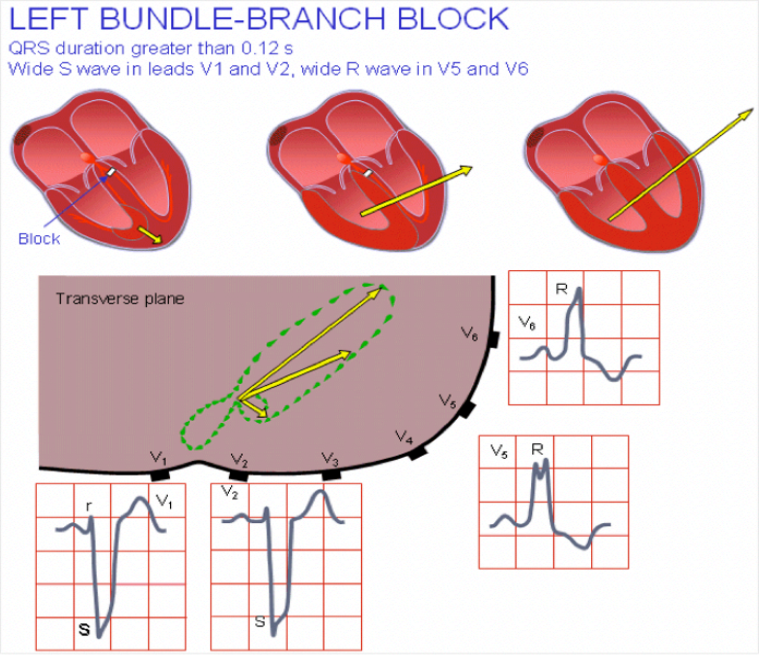
With normal activation the electrical forces of the right ventricle are partiallyconcealed by the larger sources arising from the activation of the leftventricle. In right bundle- branch block (RBBB), activation of the rightventricle is so much delayed, that it can be seen following the activation of the left ventricle. (Activation of the left ventricle takes place normally.)

RBBB causes an abnormal terminal QRS-vector that is directed to the rightventricle (i.e., rightward and anterior). This is seen in the ECG as a broad terminal S- wave in lead I. Another typical manifestation is seen in lead V1 as a double R- wave. This is named an RSR'-complex.

- Right bundle-branch block.

Left bundle-branch block The situation in left bundle-branch block (LBBB) issimilar, but activation proceeds in a direction opposite to RBBB. Again theduration criterion for complete block is 0.12 s or more for the QRS-complex. Because the activation wavefront travels in more or less the normal direction in LBBB, the signals' polarities are generally normal.

However, because of the abnormal sites of initiation of the left ventricularactivation front and the presence of normal right ventricular activation the outcome is complex and the electric heart vector makes a slower and larger loop to the left and is seen as a broad and tall R-wave, usually in leads I,aVL, V5, or V6.

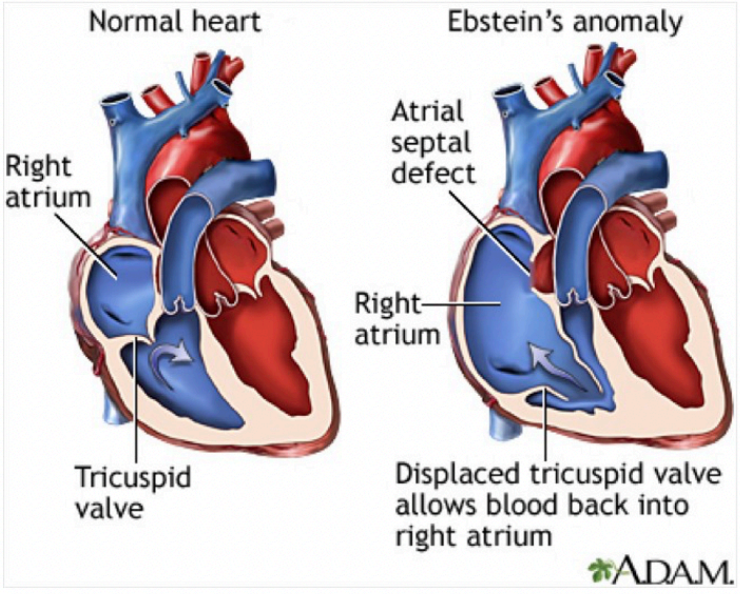


- Left bundle-branch block.

Wolff-Parkinson-White syndrome

One cause for a broad QRS- complex that exceeds over 0.12 s, may be the Wolff- Parkinson-White syndrome (WPW syndrome). In the WPW syndrome the QRS- complex initially exhibits an early upstroke called the delta wave. The interval from the P-wave to the R spike is normal, but the early ventricular excitation forming the delta wave shortens the PQ-time.

The cause of the WPW syndrome is the passage of activation from the atrium directly to the ventricular muscle via an abnormal route, called the bundle of Kent, which bypasses the AV junctions. This activates part of the ventricular muscle before normal activation reaches it via the conductionsystem (after a delay in the AV junction). The process is called pre-excitation, and the resulting ECG depends on the specific location of theaccessory pathway.



INCREASE IN WALL THICKNESS OR SIZE OF ATRIA AND VENTRICLES

Definition

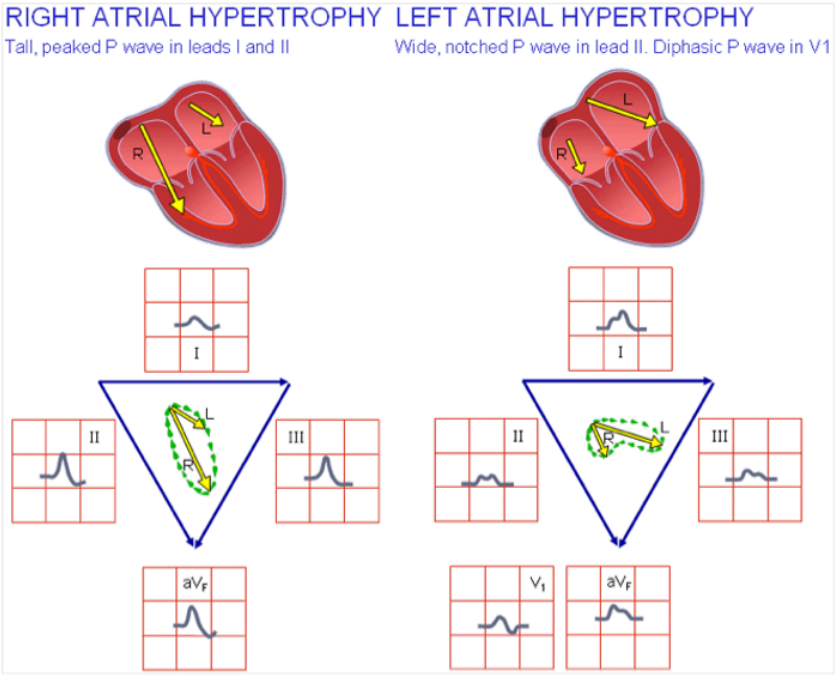
Atrial and ventricular muscles react to physical stress in the same way asskeletal muscles: The muscles enlarge with increased amount ofexercise. The extra tension may arise as a result of increased pressureload or volume load.

Pressure overload is a consequence of increased resistance in the outflowtract of the particular compartment concerned (e.g., aortic stenosis).

Volume overload means that either the outflow valve or the inflow valve of the compartment is incompetent, thus necessitating a larger stroke volumeas compensation for the regurgitant backflow.

The increase in the atrial or ventricular size is called atrial or ventricular enlargement. The increase of the atrial or ventricularwall thickness is called atrial or ventricular hypertrophy.

Very often they both are called hypertrophy, as in this presentation.



Atrial hypertrophy

Right atrial hypertrophy Right atrial hypertrophy is a consequence of right atrial overload. This may be a result of tricuspid valve disease (stenosis orinsufficiency), pulmonary valve disease, or pulmonary hypertension (increased pulmonary blood pressure). The latter is most commonly a consequence of chronic obstructive pulmonary disease or pulmonaryemboli.

In right atrial hypertrophy the electrical force due to the enlargened right atrium is larger. This electrical force is oriented mainly in the direction of lead II but also in leads aVF and III. In all of these leads an unusually large (i.e.,0.25 mV) P-wave is seen.

Left atrial hypertrophy Left atrial hypertrophy is a consequence of left atrial overload. This may be a result of mitral valve disease (stenosis or insufficiency), aortic valve disease, or hypertension in the systemic circulation.

In left atrial hypertrophy the electrical impulse due to the enlargened left atrium is strengthened. This electrical impulse is directed mainly along lead I or opposite to the direction of lead V1.

Because the atrial activation starts from the right atrium, the aforementioned left atrial activation is seen later, and therefore, the P-wave includes two phases. In lead I these phases have the same polarities and in lead V1 the opposite polarities. This typical P-waveform is called the mitral P-wave.

The specific diagnostic criterion for left atrial hypertrophy is the terminalportion of the P-wave in V1, having a duration ≥ 0.04 s and negativeamplitude ≥ 0.1 mV.

- Atrial hypertrophy. (Right and Left)

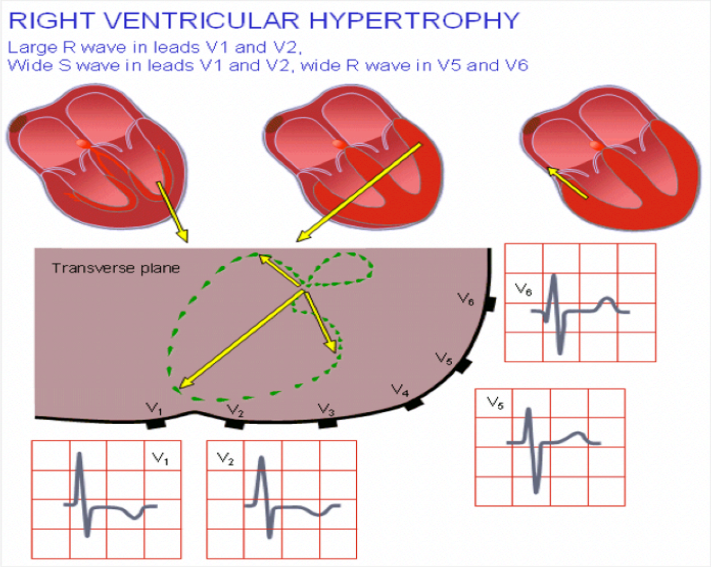
Ventricular hypertrophy

Right ventricular hypertrophy

Right ventricular hypertrophy is a consequence of right ventricular overload. This is caused by pulmonaryvalve stenosis, tricuspid insufficiency, or pulmonary hypertension .Also manycongenital cardiac abnormalities,

such as a ventricular septal defect, maycause right ventricular overload.

Right ventricular hypertrophy increases the ventricular electrical forcesdirected to the right ventricle - that is, to the right and front. This is seenin lead V1 as a tall R-wave of ≥ 0.7 mV.



- Right ventricular hypertrophy.

Left ventricular hypertrophy

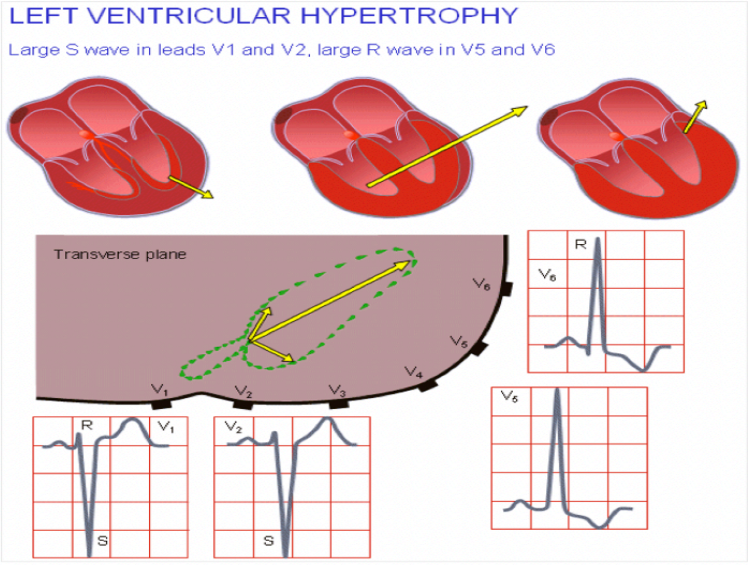
Left ventricular hypertrophy is a consequence of left ventricular overload. Itarises from mitral valve disease, aortic valve disease, or systemic hypertension. Left ventricular hypertrophy may also be a consequence ofobstructive hypertrophic cardiomyopathy, which is a sickness of the cardiacmuscle cells.

Left ventricular hypertrophy increases the ventricular electric forces directed to the left ventricle - that is, to the left and posteriorly.

Evidence of this is seen in lead I as a tall. R-wave and in lead III as a tall S-wave

(≥ 2.5 mV). Also a tall S- wave is seen in precordial leads V1 and V2 and a tall R- wave in leads V5 and V6, (≥3.5 mV).

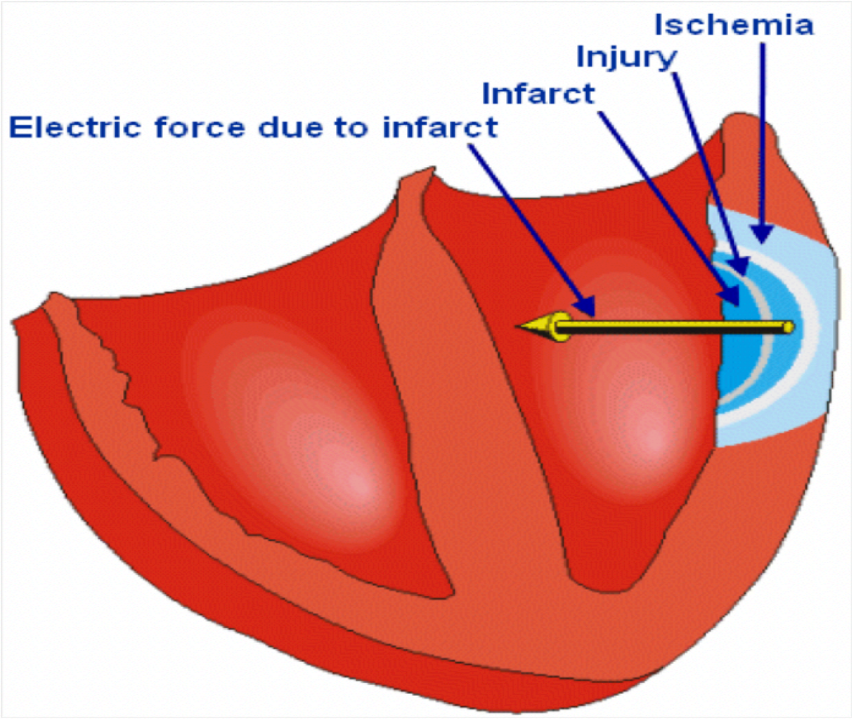
- Left ventricular hypertrophy.



MYOCARDIAL ISCHEMIA AND INFARCTION

If a coronary artery is occluded, the transport of oxygen to the cardiacmuscle is decreased, causing an oxygen debt in the muscle, which is calledischemia. Ischemia causes changes in the resting potential and in the repolarization of the muscle cells, which is seen as changes in the T-wave. Ifthe oxygen transport is terminated in a certain area, the heart muscle dies inthat region. This is called an infarction.

An infarct area is electrically silent since it has lost its excitability. According tothe solid angle theorem , the loss of this outward dipole is equivalent to an electrical force pointing inward. With this principle it is possible to locate the infarction. (Of course, the infarct region also affects the activationsequence and the volume conductor so the outcome is more complicated.)



- Myocardial ischemia and infarction.

Study done on industrial / establishment workers

Investigation done were checking —BP, ECG, Trop-t, 2 D-Echo, Lipid profile,Hb,WBC, Hb1ac and RBS

DIAGNOSIS.	Mala	Female
Hypertension	6534	6360
Hypotension.	847	673
Cardiacinvestig		
ation.		
Hb	6534	6360
RBS	6534	6360
WBC	6534	6360
Lipid profile	6534	6360
Electro cardiog ram	6282	6108
Trop -t.	3216.	2345
2D Echo	3216.	2345

REMEDIES

Regular health check-ups Proper Investigation and treatment is alwaysimportant to save lives Preventive care is important than treatment afterserious ailments

REFERENCES

1. Goldman MJ (1986): Principles of Clinical Electrocardiography, 12th ed.,460 pp. Lange Medical Publications, Los Altos, Cal. Macfarlane PW, LawrieTDV(eds.) (1989):
2. Comprehensive Electrocardiology: Theory and Practice in Health andDisease, 1st ed., Vols. 1, 2, and 3, 1785 pp. Pergamon Press, New York.
3. Netter FH (1971): Heart, Vol. 5, 293 pp. The Ciba Collection of MedicalIllustrations, Ciba Pharmaceutical Company, Summit, N.J.
4. Scheidt S (1983): Basic Electrocardiography: Leads, Axes, Arrhythmias, Vol.2/35, 32 pp. Ciba Pharmaceutical Company, Summit, N.J. Scheidt S (1984): Basic Electrocardiography: Abnormalities of ElectrocardiographicPatterns, Vol. 6/36, 32 pp.