Whanganui District Health Board

FUNDAMENTAL CARDIAC WORKBOOK

SELF DIRECTED LEARNING PACK
(12 HOURS)

NAME:_____

WARD/PRACTICE AREA:_____

MAILING ADDRESS:

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INTRODUCTION

Welcome to the Fundamental Cardiac Workbook. This book covers basic aspects of cardiology which, as a registered nurse, you are expected to have a certain degree of knowledge in. Completion of this workbook and associated set of tests will enable you to progress toward completion of the Advanced Cardiac Study Day should you wish to do so.

To pass the Fundamental Cardiac Workbook and be awarded the 12 hours of professional development towards your portfolio, you must complete the workbook in full and achieve a 100% pass in the tests. You may however resubmit any questions you fail in if you wish.

MODULE AIMS

The aim of the modules is to achieve the following:

Learning outcomes and objectives

1. To have an understanding of the anatomy and physiology of the heart.
2. To have an understanding of the pathophysiology, disease process within the coronary arteries and areas of the myocardium supplied by the different coronary arteries.
3. Understand the normal electrical conduction processes of the heart.
4. To be able to interpret basic cardiac arrhythmias.
5. To understand assessment and treatment of chest pain.
6. To understand the principles of acute coronary syndrome and angina.
7. To understand the basic pharmacological principles of cardiac drugs.
8. Have an understanding of the administration of intravenous amiodarone.
9. Have an understanding of cardiac telemetry.

Module Assessment

- Read the Fundamental Cardiac Workbook
- Complete the self directed learning and all of the questions
- Complete the smokefree ABC online learning and present a copy of the certificate to a nurse educator
- Give the completed workbook to a nurse educator in your area for marking
- A certificate of completion will be sent to you once you have completed the module and answered all of the related questions correctly

CONTENTS

MODULE ONE

ANATOMY AND PHYSIOLOGY OF THE HEART

The heart is a muscular organ which weighs approximately 300 grams and is located within the mediastinum between the lungs. It is made up of four chambers, two atria and two ventricles, which beat independently.

ATRIA

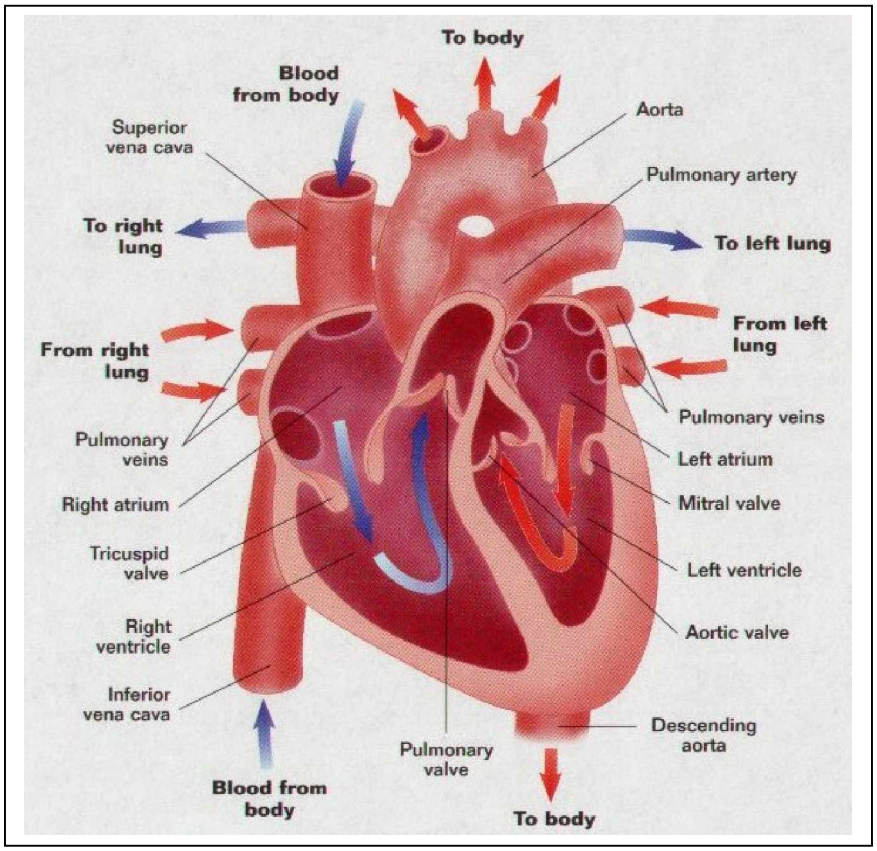
The two upper chambers are called atria. The left and right atria are separated by the intra atrial septum, and should beat at the same time. The right atria, upon contraction, sends blood to the right ventricle and the left atria sends blood to the left ventricle.

VENTRICLES

The two lower chambers are called ventricles. These are higher pressure chambers than the atria and are separated by thick intraventricular septum. Blood is ejected from the right ventricle and travels to the lungs to be oxygenated and the blood from the left ventricle is transported throughout the body via the aorta.

The blood enters the right atrium via the Superior Vena Cava from the top of the body or the Inferior Vena Cava if returning from the lower half of the body.

See following page for Structure of the Heart Diagram



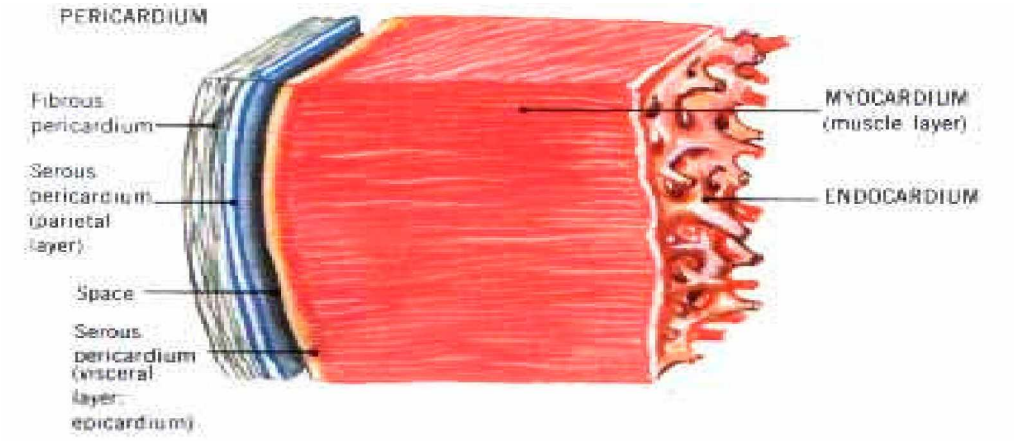
THE LAYERS OF THE HEART

ENDOCARDIUM

This is the inner layer of the heart. It is made up of endothelial tissue and covers the valves and chordae tendinae. The endocardium is continuous with the lining of the great vessels of the heart.

MYOCARDIUM

The myocardium is the middle layer of the heart. It is made up of striated fibre cell, separated by intercalated disk so that electrical impulses can move easily from cell to cell. This is referred to as the syncytial nature of cardiac muscle.



http://www.heartfoundationjm.org/v/Deco/ServicesAboutHumanHeartHeartWall.jpg

EPICARDIUM

This is the outer layer and is continuous with the visceral pericardium.

VALVES

Forward blood flow through the heart is controlled by valves. There are four valves situated in the heart, two atrioventricular and two semilunar valves, all of which open and close passively, in response to where the pressure upon them is generated. For example the valves close when backward pressure from the blood is exerted on them. Conversely they open when they sense a forward pressure gradient.

The valves are connected to the heart by chordae tendinae which further connect to muscular extensions of the myocardium called papillary muscles.

ATRIOVENTRICULAR VALVES

1. Tricuspid Valve
   Three leaflets
   Lies between the right atrium and the right ventricle

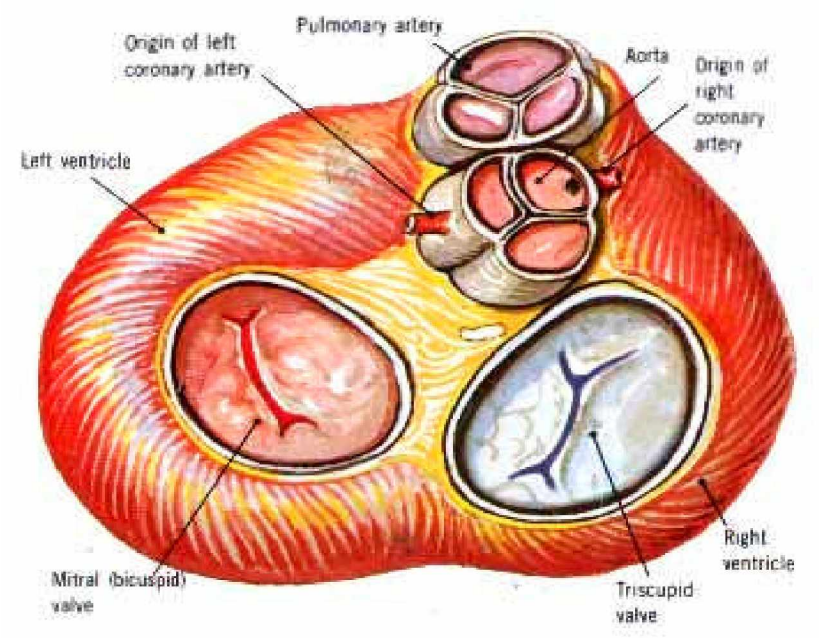
2. Mitral Valve
   Two leaflets
   Lies between the left atrium and left ventricle

3. Semilunar Valves

PULMONIC VALVE
   Two leaflets
   Lies between the right ventricle and the pulmonary artery

AORTIC VALVE
   Two leaflets
   Lies between the left ventricle and the aorta

See below for Valvular Diagrams



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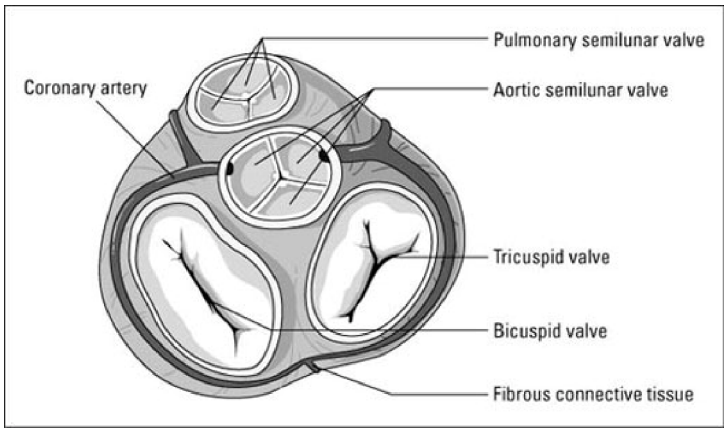

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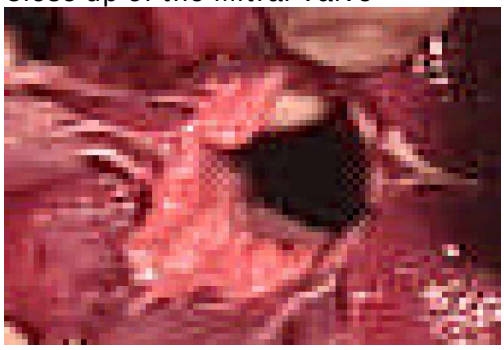
Close up of the Mitral Valve



This close-up of the two leaflet of the Mitral Valve shows the chordae tendinae nicely. The chordae are connected to the myocardium of the left ventricle by large muscular bundles called papillary muscle. This chordae/papillary muscle structure helps to maintain valve geometry during ventricular contraction

to reduce regurgitation back through the valve. Blood flows out towards the viewer in this picture.

CORONARY BLOOD SUPPLY

Due to its high demand for oxygen the heart has its own blood supply. The right and left coronary arteries originate behind the cusps of the aorta.

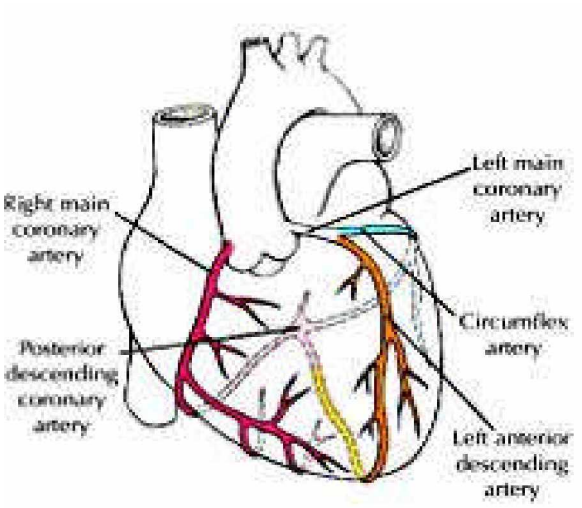
The right coronary artery (RCA) supplies
- the right side of the heart
- the posterior septum
- the Sino-atrial node in 60% of people
- the atrio-ventricular node in 90% of people

The left coronary artery further divides into the left anterior descending (LAD) and the circumflex branch. The LAD travels down the anterior portion of the left ventricle and the circumflex wraps around the side of the left ventricle and down the posterior portion of the left heart.

The LAD supplies
- the left ventricle
- the anterior septum
- the anterior papillary muscles

The circumflex branch supplies
- the lateral and posterior portion of the left heart
- the Sino-atrial node of 40% of people
- the atrio-ventricular node in 10% of people

http://www.cardiologist.uk.com/images/heart.jpg



CARDIAC VEINS

There are three main cardiac veins which form a single drainage system which empties the deoxygenated blood into the right atrium at the coronary sinus (just above the tricuspid valve).

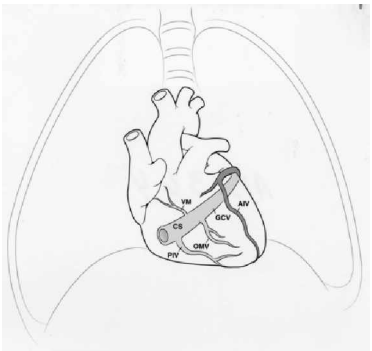


Figure A

http://www.ajronline.org/cgi/content-nw/full/177/6/1447/FIG1

Fig. 1A. — Major epicardial coronary veins. Drawing in frontal projection shows that anterior interventricular (AIV) and obtuse marginal (OMV) veins drain into great cardiac vein (GCV). Oblique vein of Marshall (VM) drains into coronary sinus (CS) at level of venous valve of Vieussens, marking point of transition of coronary sinus and great cardiac vein in mid atrioventricular groove. Posterior interventricular vein (PIV) joins coronary sinus near ostium to right atrium.

CONDUCTIVE SYSTEM OF THE HEART

Before the heart can contract it must be stimulated. The stimulus must be delivered quickly and efficiently to all areas of the myocardium. In order to achieve these two functions the heart relies first on self excitation and secondly on rapid conduction via specialised conductive pathways.

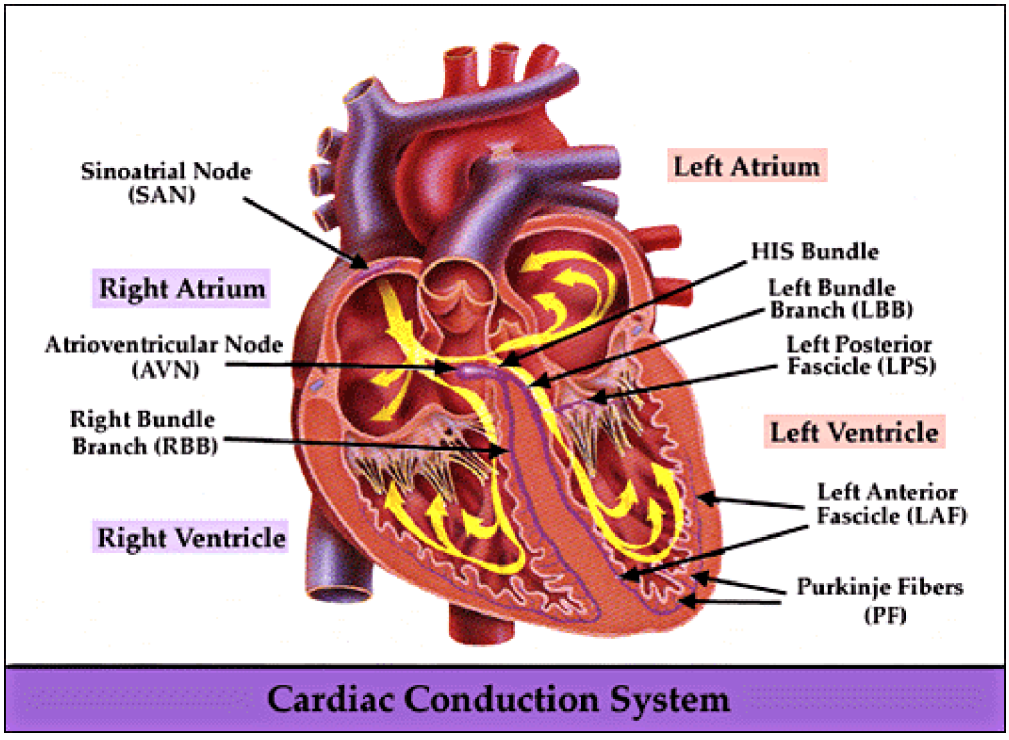
Automatically and at regular intervals an electrical impulse arises in the Sino-atrial (SA) Node. The impulse travels through pathways in the right and left atrium called internodal pathways until it reaches the Atrio-ventricular (AV) node which is located between the right atrium and the right ventricle.

The impulse is delayed in the AV node momentarily to allow time for the atria to contract.

The impulse then travels though the bundle of HIS and then down the right and left bundle branches.

The impulse then spreads down through the myocardium to the purkinje fibres. Ventricular contraction then occurs.

For the impulse to move quickly though the heart muscle it must travel down specialised conductive pathways. If there is a blockage in an area of the conductive system then the impulse will travel through the heart much more slowly.



HTTP://MEDSTAT.MED.UTAH.EDU/KW/ECG/MML/ECG_CCS.GIF

DEPOLARISATION

The initial spread of the impulse through a muscle is known as depolarisation. This electrical impulse

moves from cell to cell causing muscle contraction. The signal causes sodium to rush into the cell and potassium to move out. In turn this results in cellular contraction or systole.

REPOLARISATION

This is when the sodium/potassium pump on the cell membrane realigns the electrolytes. This causes the cell to relax and return to its resting state, otherwise known as diastole. On the ECG both depolarisation (systole) and repolarisation (diastole) are distinguishable.

PACEMAKERS OF THE HEART

The primary pacemaker of the heart is the sinoatrial (SA) node. It discharges impulses at a resting rate of 60-100 times per minute, but is capable of producing up to a person's maximal heart rate, which is roughly 220 minus the person's age.

Should the SA node fail to fire the Bundle of His fibres, situated in the atrio-ventricular nodal region can discharge electrical activity at a rate of 40-60 times per minute.

Failing this the Purkinje fibres deep in the bundle branches take over the pacing of the heart, but are only capable of eliciting a heart rate of 15-40 beats per minute.

Thus the SA node is the pacemaker responsible for normal heart rate control, hence the term normal sinus rhythm.

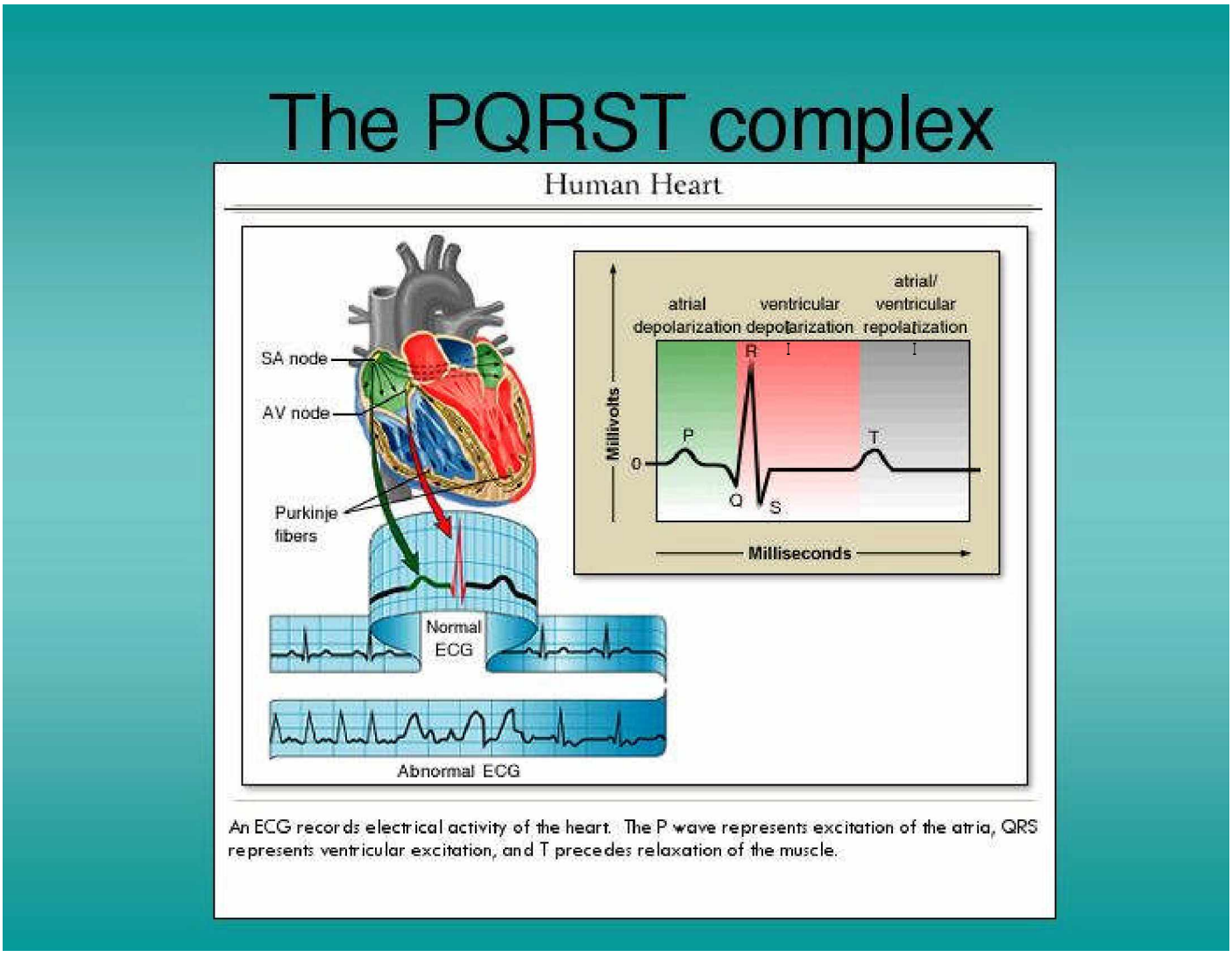
NOTES

MODULE TWO

THE ELECTRICAL EVENTS OF THE CARDIAC CYCLE

Each beat of the heart is represented on the ECG by three deflections:

P Wave
QRS Complex
T Wave



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REMEMBER
Each of these deflections represents either depolarisation or repolarisation of the atria and ventricles. Note that even though the atria do engage in a relaxation period (repolarisation), electrically the impulse this generates is too small to have been evidenced on the actual ECG

THE P WAVE

This represents atrial depolarisation and begins as soon as the impulse leaves the sinus node and initiates atrial contraction.

The P wave is gently rounded in shape and is usually about 2-3mm in height.

QRS COMPLEX

The QRS complex represents ventricular depolarisation and may have various components, depending on which lead of the ECG is recorded.

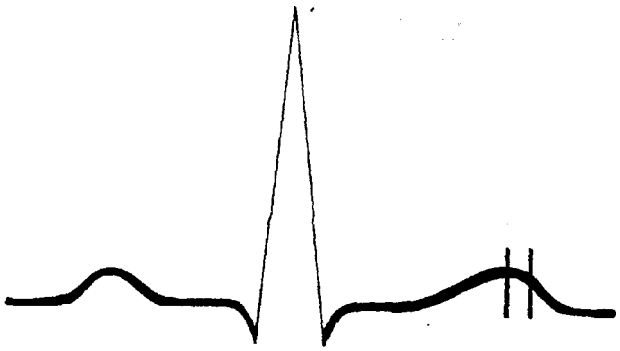
q wave – the initial negative deflection preceding an r wave
r wave – the first positive deflection
s wave – the negative deflection following the r wave. The amplitude or height of the QRS complex should be greater than 5mm and less than 20mm. In order to measure amplitude both regular and positive deflection should be added together.

Duration and width represents the time the impulse takes to pass over the ventricles. This should not exceed 0.12 seconds.

THE T WAVE

The T wave results from the repolarisation or diastole of the ventricles. The T wave should be rounded and symmetrical.

Towards the end of the T wave is an area called the vulnerable period. A stimulus generated at this time, either intrinsically or extrinsically, may result in ventricular fibrillation; an abnormal rhythm which left untreated is fatal.



Approximate location of the vulnerable period.

(Conover, 1996)

INTERVALS

PR INTERVAL

The distance from beginning of the p wave to the beginning of the QRS complex is referred to as the PR interval. It represents the length of time it takes for the impulse to travel from the atria to the ventricles.

The normal PR interval is between 0.12 and 0.20 seconds (3 - 5 little boxes).

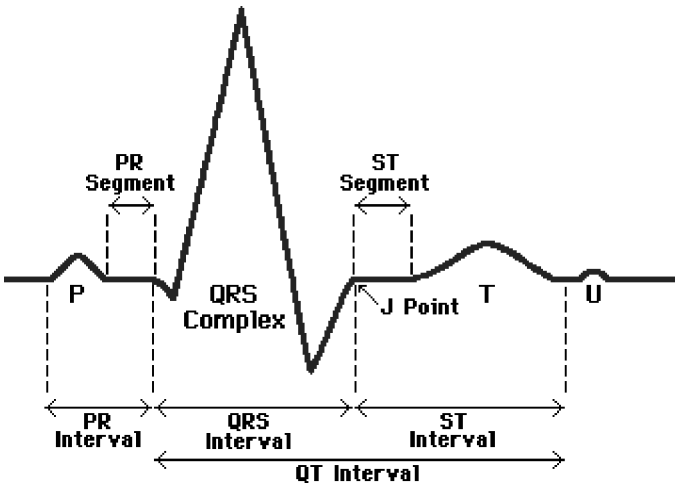
QRS INTERVAL

As stated on the previous page this should not exceed 0.12 seconds (3 little boxes)

ST SEGMENT

The ST segment is the interval that occurs between the end of the QRS complex and the beginning of the t wave. It represents the end of depolarisation and the beginning of repolarisation.

It should be isoelectric, which means flat on the baseline. Elevation or depression of the ST segment may indicate an abnormality of the myocardial tissue due to injury or ischaemia.



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Systematic Analysis of Rhythm Strip

1. Determine Ventricular Rate.
   60-100 bpm = normal
   < 60 = bradycardia
   > 100 = tachycardia

2. Assess rhythm/regularity.
   regular or irregular?

3. Identify and examine P Waves.
   Is there an upright, rounded P wave before each QRS?

4. Assess intervals.
   PR (0.12 - 0.20)
   QRS (0.04 – 0.10)
   QT (less than ½ the R-R interval = normal)

5. Evaluate overall appearance of Rhythm.
   ST segment – evaluation or depression/
   T wave – upright, normal height?
   any extra or unusual beats?

6. Interpret the rhythm and evaluate clinical significance.

MODULE THREE

RATE CALCULATION

There are several methods for calculating heart rate. However the most reliable method, even in the setting of an irregular rhythm is to calculate the number of cycles in six seconds and multiply that number by ten.

1. Choose a place to start and count 30 large squares (= 6 seconds).

2. Count the number of R waves in that period.

3. Multiply that number by 10 to give you the rate for one minute.

ECG PAPER

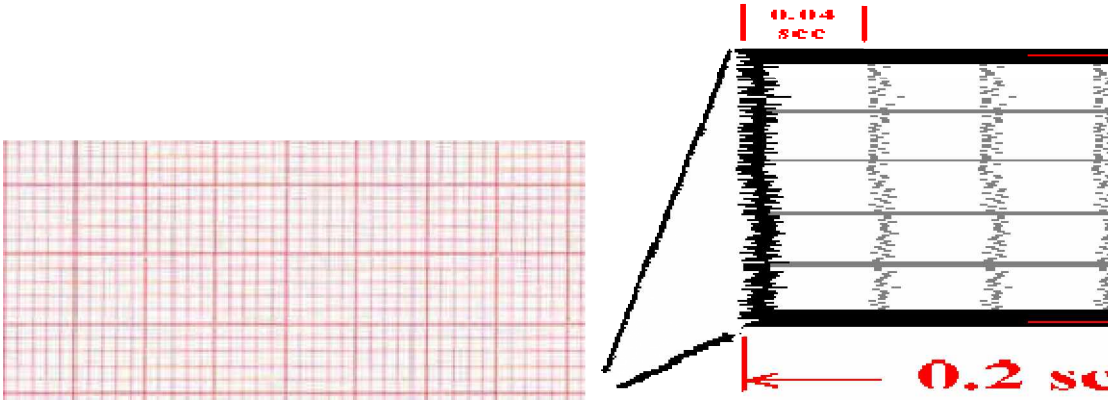
HORIZONTAL PLANE

Time is measured on the horizontal plane. Each small square on the ECG paper is equal to 1mm in length and represents 0.04 seconds. The large squares are 5mm in length and equal 0.20 seconds in time.

VERTICAL PLANE

Amplitude or voltage is measured on the vertical plane. One mil volt iis equal to 10 mm or two large squares.

Important - These measures are accurate only for standardised ECG paper speed which is 25 mm/second. 15 Large boxes are equivalent to a 3 second time strip



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DETECTING NORMAL SINUS RHYTHM

Rate	60-100bpm
Rhythm	Regular
Components	P wave, followed by QRS complex followed by T wave, with normal intervals.

LEAD PLACEMENT AND RECORDING THE 12 LEAD ECG

Depolarisation of the cardiac cells causes contraction of the heart muscle. These electrical changes set up an electrical field which can be recorded from electrodes which are placed on the body. The 12 lead ECG view these impulses from twelve different angles.

The twelve recording leads are separated into two groups - the limbs leads and the chest leads.

THE LIMB LEADS

These are further divided into:
- Standard Leads
- Augmented Leads

STANDARD LEADS

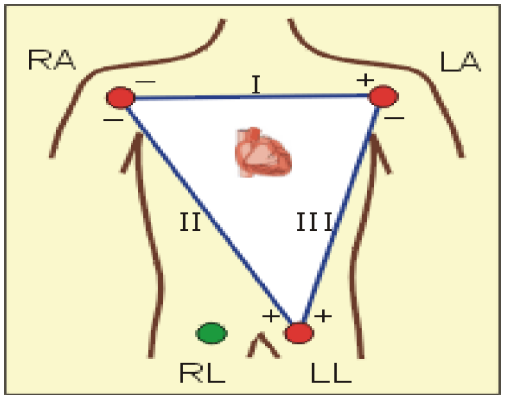
These leads are bipolar because they pick up electrical messages at two places spontaneously, as they have two electrodes, one negative and one positive. The limb leads are made up of the following:

Lead I	right arm negative to left arm positive
Lead II	right arm negative to left leg positive
Lead III	left arm positive to left leg positive

The axes of the three bipolar leads forms Einthoven's triangle.

Axis of the leads

Right Arm



Left Arm

Left Leg

The axis of a lead an imaginary line drawn between the two electrodes of the bipolar lead or between the positive electrode and a reference point of the unipolar lead. Einthoven's triangle is formed by the axes of the three bipolar limb leads, 1, 11, and 111. The diagram shows the placement of the positive and negative electrodes for each lead.
www.cvphysiology.com/ Arrhythmias/A013a

AUGMENTED LEADS

As these leads pick up impulses at only one point they are know as unipolar. There are three unipolar leads as follows:

AVR	Right arm
AVL	Left arm
AVF	Left foot

In reference to unipolar leads the V stands for unipolar and the A stand for augmented, meaning that the ECG machine increases the size of these low voltage leads.

The lead attached to the right leg is an earth lead only.

CHEST LEADS

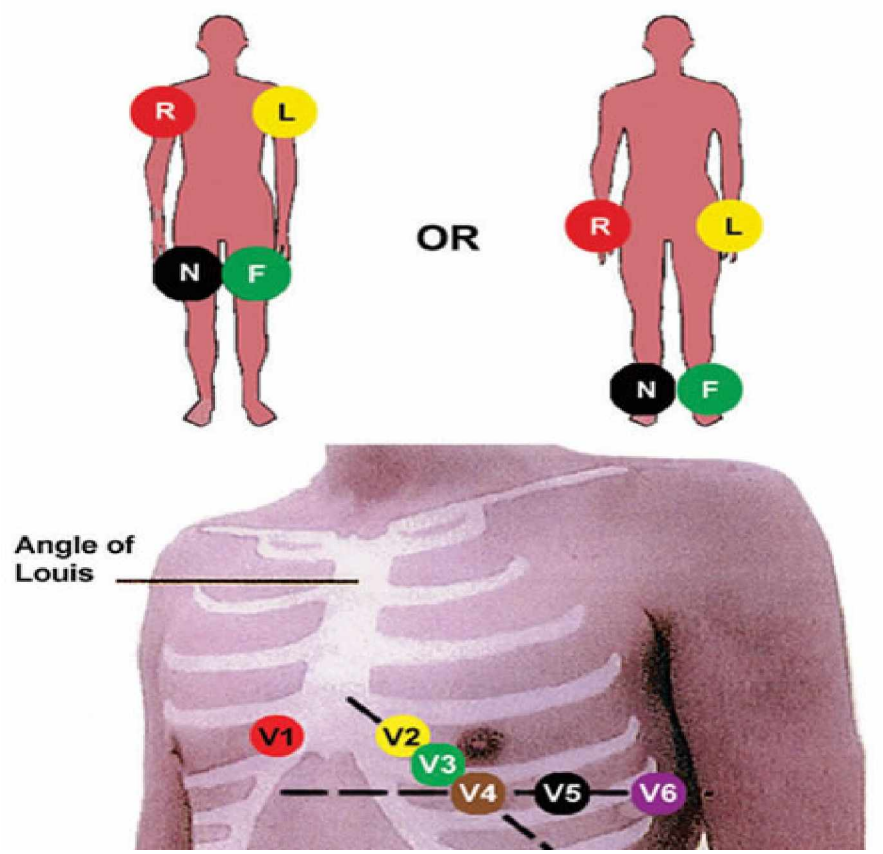
There are six chest leads, V1-V6. Again V represents unipolar, hence the impulse that is recorded is transmitted to that lead only.

NOTES

DID YOU KNOW
The chest leads can also be called precordial leads

PLACEMENT OF CHEST LEADS

V1	4th intercostal space, right sternal border
V2	4th intercostal space, left sternal border
V3	Directly between V2 and V4
V4	5th intercostal space, midclavicular line
V5	On the same plane as V4, anterior axillary line
V6	On the same plane as V4, mid axillary line



Electrode sites for the chest leads.

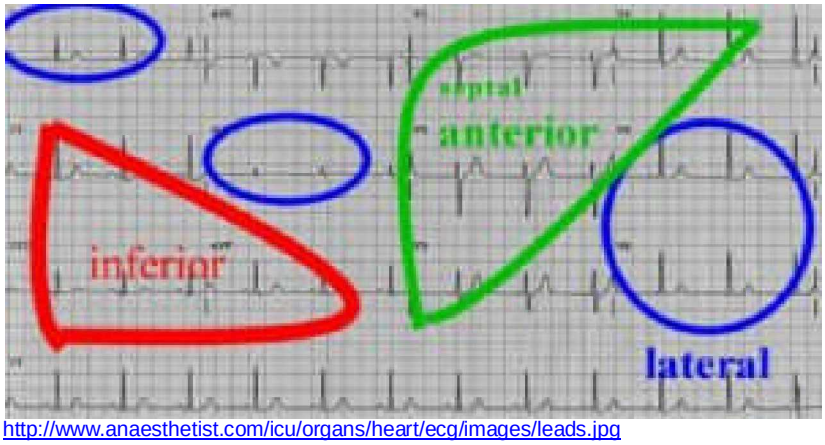
The precordial leads are on the left from V1 on the right sternal border to V6 at the left midaxillary line. (Conover, 1996)

GROUPING OF LEADS

LEADS ARE GROUPED TOGETHER AND REFLECT A PARTICULAR WALL OF THE HEART

Anterior leads	V3, V4
Anteroseptal leads	V1, V2, V3
Lateral leads	V5, V6, AVL, Lead I

Inferior leads	II, III, AVF
Posterior leads	V1, V2 (indirect)



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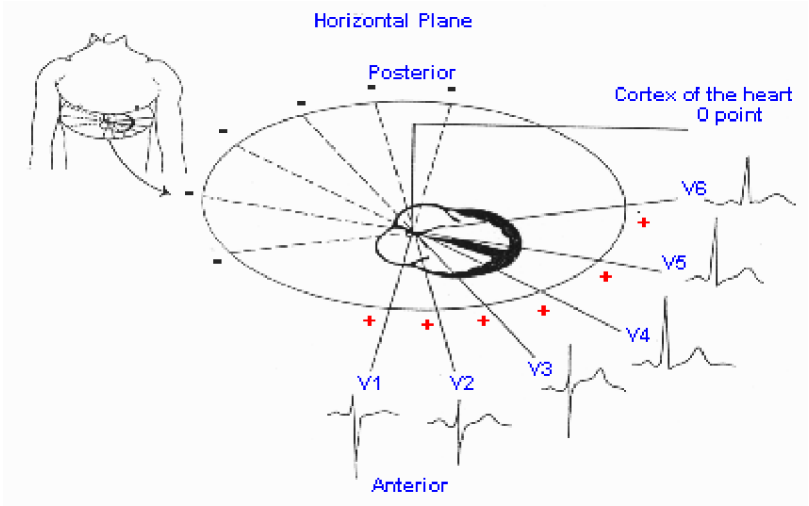

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RECORDING AN ECG

1. Ensure correct placement of chest leads. If a serial ECG is required it is a good idea to mark the spots where the electrodes have been placed to maintain consistency of graphs.

2. Limb leads may be placed on the wrist and angle, or the arm, shoulder or thigh.

3. Avoid placing leads over hairy, sweaty or bony prominences.

4. Use the form of contact medium, such as gel pads or liquid gel.

5. Remove anything which may cause interference and hence reduce the quality of the ECG. These include removing any metal objects from the person's body such as a watch, as well as turning off anything electrically powered in close proximity of the person, e.g. IV pumps.

6. Ask the patient to breathe quietly and hold their breath while you record the ECG. Also ensure that the patient is warm and comfortable to avoid tremors and fidgeting.

7. Label the ECG appropriately; including any symptoms the patient may have, such as chest pain.

NOTES

MODULE FOUR

THE NORMAL 12 LEAD ECG

The normal electrocardiogram is composed of a P wave, a QRS complex and a t wave. The P wave represents ventricular depolarisation. The T wave reflects the phase of rapid repolarisation of the ventricles.

If electrical currents flow towards an electrode a positive or upright deflection will be recorded on the ECG paper. Conversely if the current flows away from the electrode then the resultant deflection on the ECG will be negative or downward.

In the standard 12 lead ECG the individual recordings for each lead may seem different but this is only because they are recorded from different positions on the body, from different angles.

All these different recording angles are employed to arrive at a true picture of what is really happening with the heart.

Atrial depolarisation speeds from the SA node in the top right hand corner of the right atrium through both atria and down to the AV node. It travels in leftward and inferior direction.

The current passes through the AV node, bundle of HIS and both ventricular walls at the same time, but the impulse traverses the right ventricular wall first because it is so much thinner than the left.

The septum is depolarised from left to right then both ventricles are depolarised. As the left ventricle has a much thicker muscle mass most of the electrical forces are picked up there.

Therefore most electrical impulses travel in a downward leftward direction.

Lead I
- positive p wave
- none or small q wave (due to septal depolarisation)
- positive r wave
- positive t wave

Lead II
- positive p wave
- positive r wave
- may have an s wave

Lead III
- positive p wave (can be flat or biphasic or negative)
- positive r wave
- S wave deeper than the r wave (due to left sided depolarisation)

AVL
- same as lead I as they record from the same position

AVF
- similar to lead III as it is in a similar position

AVR
- negative p wave
- negative QRS complex (s) wave
- negative t wave

V1

negative p wave (may be positive)
small R wave due to both septal and right ventricular forces. When right ventricular activation is still beginning the dominant leftward force of the left ventricle produces a deep s wave.

V2 positive P wave
slightly bigger R wave

V3 more R wave progression, usually biphasic

V4 mainly positive with a smaller s wave

V5 totally positive as a r wave progression continues

V6 totally positive (no s wave)

RULES FOR ECG INTERPRETATION

CHECKS

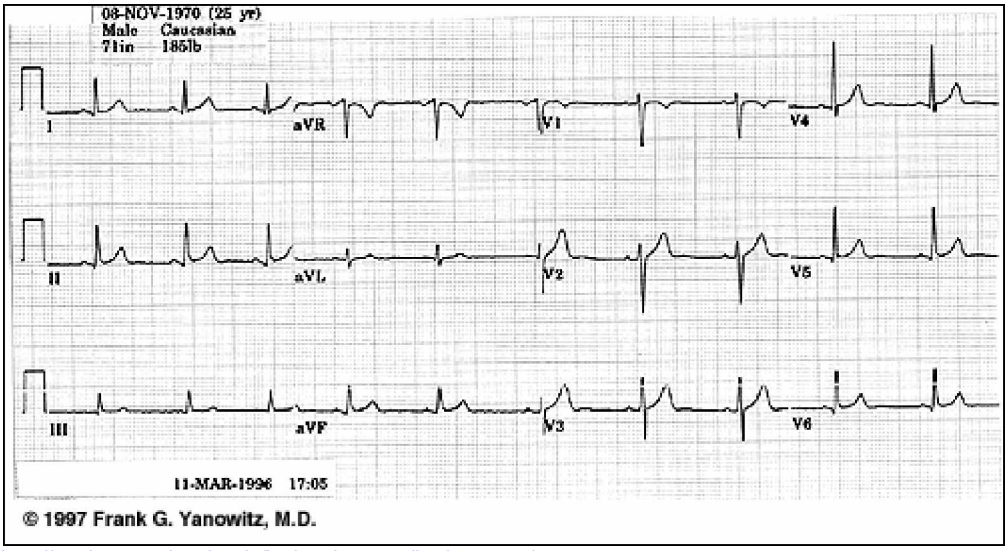
1. Look at AVR. It should be negative
2. Calibration mark should be 2 large boxes high (= 10 mm or 1 mV)
3. Speed of paper should be 25 mm per second

INTERPRETATION

1. Diagnose the rhythm strip (lead II)
2. Check r wave progression across anterior leads
3. Look at leads in groups
4. Look at ST segments, t wave and check for the presence of pathological Q waves.
5. Make your final diagnosis.

NB. If a rhythm on interpretation is not deemed to be sinus rhythm or is recognised as life threatening please call a doctor for further interpretation.

EXAMPLES OF NORMAL ELECTROCARDIOGRAPHS



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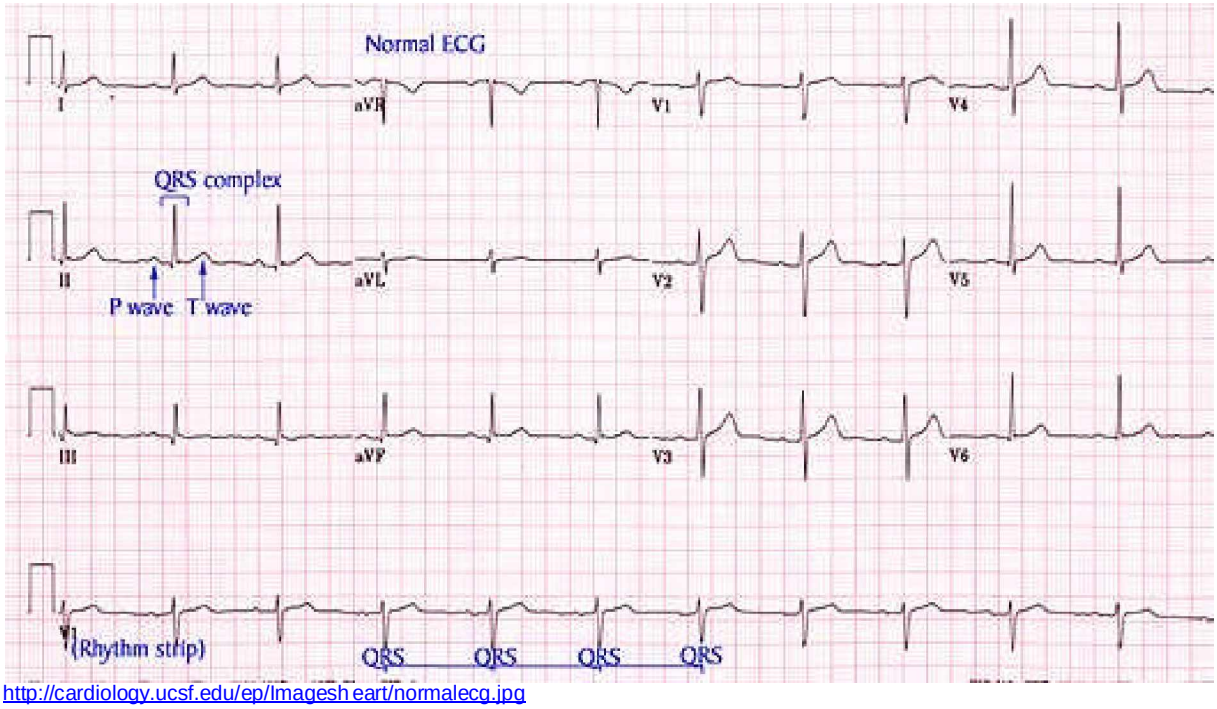

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MODULE FIVE

ANGINA

Angina is a reversible condition in which the myocardial oxygen demand temporarily exceeds the oxygen supply.

The major symptom of angina is chest pain, but it is often associated with shortness of breath, sweating, nausea.

Angina results from the narrowing of the coronary arteries. This may be permanent due to structural abnormality such as atherosclerosis or transient due to spasm of the artery wall. As arterial flow is decreased the myocardial tissue's need for oxygen and nutrients continues. The same work of pumping blood must be accomplished with less available energy and oxygen.

The tissue that depends on the blood supply becomes ischaemic as it functions within less oxygenated blood.

ECG CHANGES

ST Segment depression usually greater than 0.5 mm (1/2 a little box) in 2 or more leads
T wave flattening or inversion

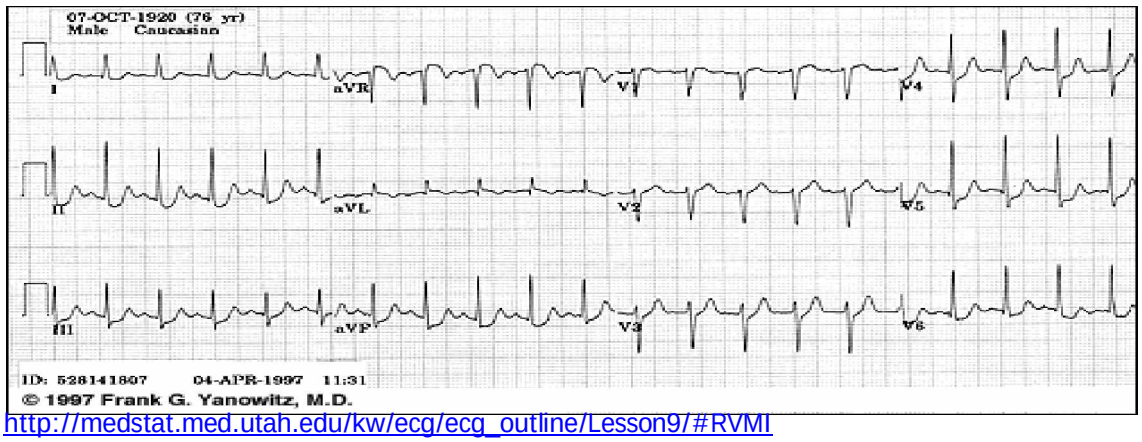
The ST segment depression can be widespread over the ECG leads indicating three vessel involvement or it can be specific to one area of the heart. Widespread ST segment elevation suggests spasm of the coronary arteries and not myocardial infarct.

SYMPTOMS

pain due to lactic acid formation (anaerobic metabolism)
associated breathlessness
diaphoresis
dizziness

Most people with chronic angina feel the pain when they exercise, also emotions which constrict the arteries and increase heart rate (sympathetic control).

AN EXAMPLE OF AN ECG SHOWING ST DEPRESSION WITH UNSTABLE ANGINA



http://medstat.med.utah.edu/kw/ecg/ecg_outline/Lesson9/#RVMI

ACUTE CORONARY SYNDROME

ACS encompasses any presentation with chest pain.

It is classified into three categories: High risk, Intermediate risk and Low risk.
High risk would encompass the ST elevation MI or prolonged chest pain,
Intermediate would encompass what we know as unstable angina where the pain has resoved but they have other risk factors,
Low risk would be pain but few risk factors.

Follow http://www.heartfoundation.com.au/downloads/NHF_ACS_chart0506.pdf to see the algorithm from the Heart Foundation regarding treatment and risk factors of each category.

NB: You may see Non STEMI and STEMI now written as Non STEACS and STEACS – which is ST elevation Acute Coronary Syndrome.

MODULE SIX

ACUTE MYOCARDIAL INFARCTION

A life threatening condition characterised by the formation of localised necrotic areas within the myocardium. Myocardial infarction usually follows the sudden cessation of blood flow and oxygen flow to the heart muscle.

PATHOPHYSIOLOGY

Complete or nearly complete occlusion of a coronary vessel by severe chronic atherosclerotic plaque with a superimposed thrombus formation.

SYMPTOMS

chest pain, which may radiate to arms, jaw, shoulder or back
diaphoresis
dizziness
shortness of breath
nausea and vomiting
feeling of impending doom

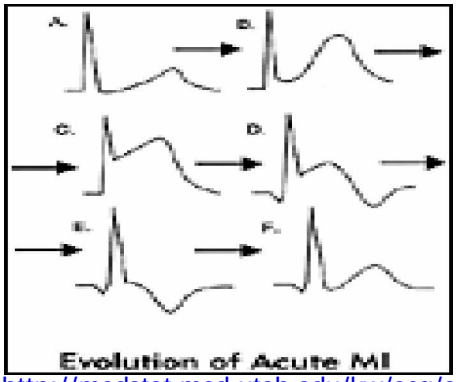
ST SEGMENT INFARCTION (STEMI) ECG CRITERIA

Initially <u>persistent</u> ST segment elevation of
- o≥2 mm (2 little boxes) in 2 or more chest leads or
- o≥1 mm in 2 or more limb leads

followed by t wave inversion
Q waves will develop and are permanent

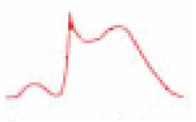
During a myocardial infarction the initial ECG change is that of ST segment elevation in the leads reflecting the involved surface of the myocardium.

The evolution of ST segment changes in acute myocardial infarction (An explanation follows)

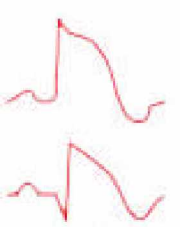


http://medstat.med.utah.edu/kw/ecg/ecg_outline/Lesson9/#RVMI

Normal appearances in a lead, which by the QRS morphology clearly lies over the L Ventricle



Within hours of the clinical onset of infarction there is ST segment deviation. At this stage no QRS changes or T wave changes have occurred. Although such a pattern is frequently spoken of, loosely, as following " acute infarction", no definitive evidence of infarction is shown. There is evidence of myocardial damage. There is an unstable situation. In the vast majority of cases evolutionary changes of infarction follow. Occasionally the record returns to normal.



Within days the R wave voltage has fallen and abnormal Q waves have appeared. These changes are sufficient to prove the occurrence of infarction. In addition T wave inversion has appeared. The ST elevation becomes less pronounced.

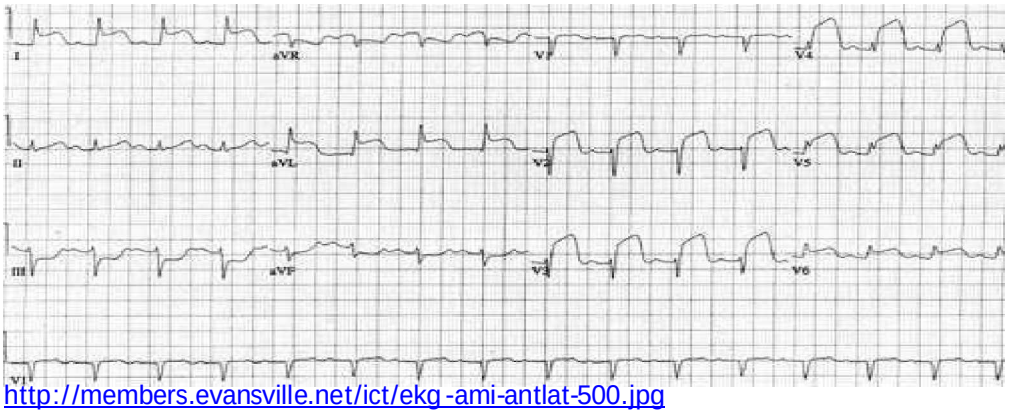
Within one or more weeks the S-T segment changes revert completely to normal. The R wave voltage remains reduced and the abnormal Q waves persist. Deep symmetrical T wave inversion may develop at this stage. In some patients this pattern remains permanently, in others it progresses to the appearance in the next figure.

Months after the clinical infarction the T waves may gradually return to normal. The abnormal Q waves and reduced R wave voltage persist.

ANTERIOR WALL INFARCTION

An infarction of the anterior wall of the heart results from the occlusion of the left anterior descending coronary artery or one of its branches.

The following ECG is an antero-lateral myocardial infarction. Notice the ST elevation in the limb leads as well as in most of the chest leads



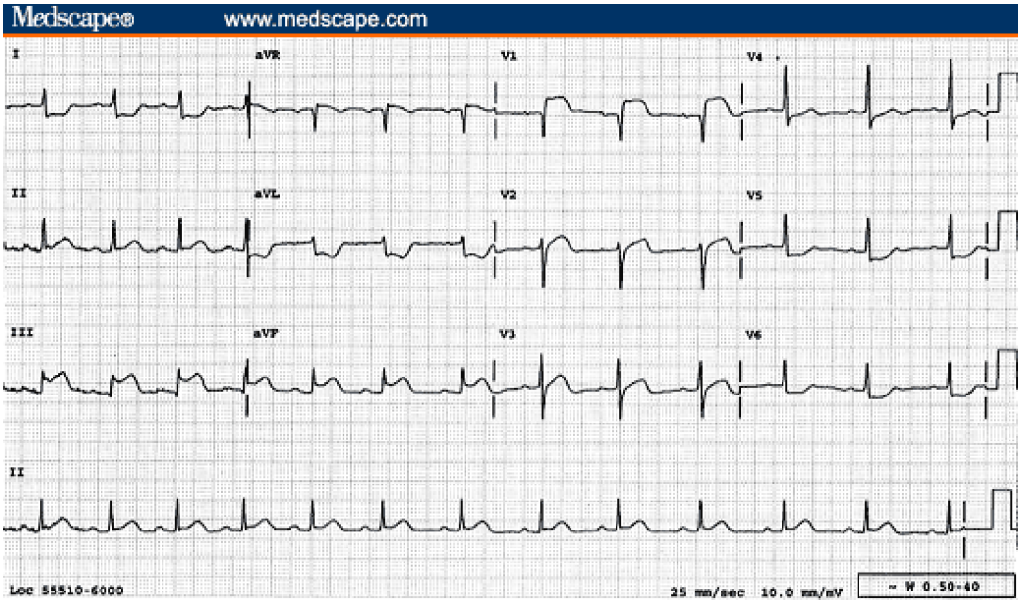
http://members.evansville.net/ict/ekg-ami-antlat-500.jpg

INFERIOR WALL INFARCTION

In order for the inferior wall to become infarcted the circumflex or the right coronary artery must be occluded.

The following ECG is an example of an inferior myocardial infarction.

Some of you may have noted the ST elevation in V1. This is due to myocardial infarction of the R ventricle. Remember the R ventricle is also fed by the circumflex and right coronary artery.



RIGHT VENTRICULAR (RV) INFARCTION

Right ventricular infarcts are reflected in lead V4R. Hence in order to determine this it is first necessary to do another ECG with right sided leads.

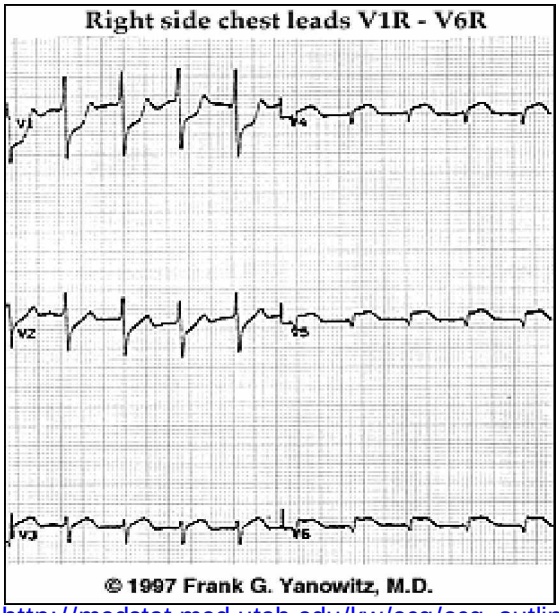
ECG USING RIGHT SIDED LEADS

V1 (becomes V2R) and V2 (essentially becomes V1R) are placed in the usual position.
V3R directly between V2R (where V1 would normally be) and V4R

V4R = mid clavicular line, 5th intercostals space on the right side of the chest
V5R = on the same plane as V4R, anterior axillary line, on the right side of the chest
V6R = on the same plane as V4R, mid axillary line, on the right side of the chest

RV infarcts happen in the setting of inferior wall infarction, when the occlusion is in the right coronary artery. As RV infarcts can carry a poorer prognosis than an inferior infarction alone, it is important, in the setting of an inferior AMI to perform right sided leads in order to determine if a RV infarct is also present to avoid possible mismanagement.

ST elevation, $\geq$2mm, in right chest leads, especially V4R (see below)



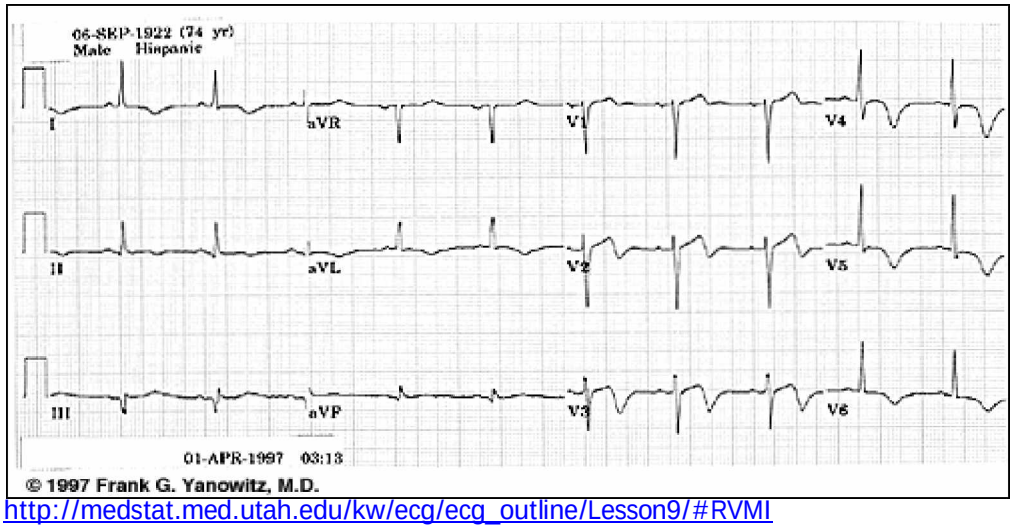
http://medstat.med.utah.edu/kw/ecg/ecg_outline/Lesson9/#RVMI

NON ST ELEVATION AMI (NON-STEMI)

The diagnosis of a non-STEMI infarct can only truly be made when there is a characteristic rise in the levels of serum troponin and creatine-kinase-MB, in association with transient non specific findings on their ECG. These changes may include ST depression or T wave abnormalities without the evolution of Q waves, or both.

Whilst non-STEMI's result in smaller infarct size, people do have a higher incidence of post infarction angina and rate of reoccurrence than those who have STEMI's.

> Although it is tempting to localise the non-Q MI by the particular leads showing ST-T changes, this is probably only valid for the ST segment elevation pattern
>
> Evolving ST-T changes may include any of the following patterns: Convex downward ST segment depression only (common). Or Convex upwards or straight ST segment elevation only (uncommon) or symmetrical T wave inversion only (common) or combinations of above changes



http://medstat.med.utah.edu/kw/ecg/ecg_outline/Lesson9/#RVMI

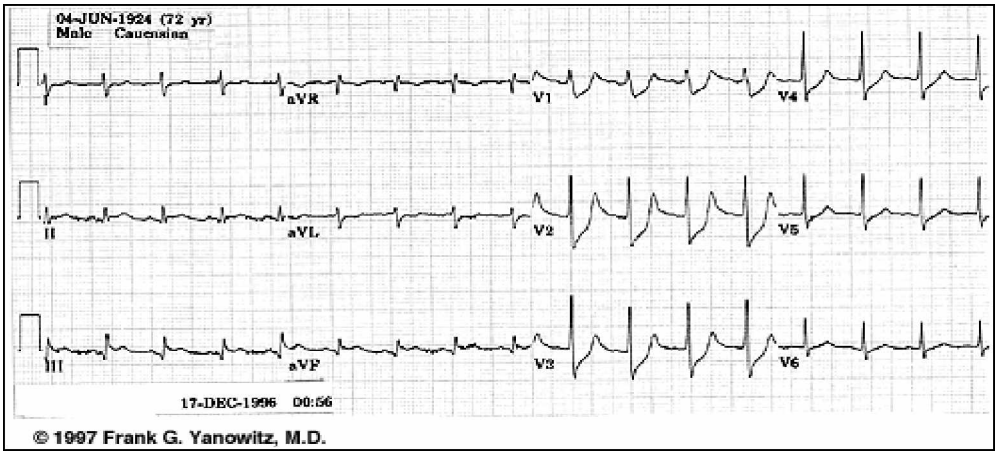
POSTERIOR WALL INFARCTION

There are no leads directly over the true posterior wall. The changes are reflected in the leads on the opposite wall. Therefore these changes are seen back to front.

Instead of an abnormal Q wave, there is a tall, broad initial R wave. Also instead of ST segment elevation resulting from an injury current travelling away from the electrode, there is ST segment depression resulting from injury current travelling toward the electrode. The t wave is upright rather than inverted.

As with right ventricular infarcts, posterior infarcts often accompany inferior wall infarctions. In the acute stage ST depression is usually evident in V3 and V4, giving way to an R wave in V1 as the infarct progresses.

This ECG is an example of an Acute inferoposterior MI (note tall R waves V1-3, marked ST depression V1-3, ST elevation in II, III, aVF)



http://medstat.med.utah.edu/kw/ecg/ecg_outline/Lesson9/#RVMI

ECG changes are seen in anterior precordial leads V1-3, but are the mirror image of an anteroseptal MI:

- Increased R wave amplitude and duration (i.e., a "pathologic R wave" is a mirror image of a pathologic Q)
- R/S ratio in V1 or V2 >1 (i.e., prominent anterior forces)
- Hyperacute ST-T wave changes: i.e., ST depression and large, inverted T waves in V1-3
- Late normalisation of ST-T with symmetrical upright T waves in V1-3

THE MIRROR TEST IN READING POSTERIOR INFARCTION ECG

APPLICATION OF THE MIRROR TEST

The anterior precordial leads (V1, V2, V3) provide a mirror image view of the posterior wall of the left ventricle.

Thus, the tall R waves and ST depression, that is seen in V1, V2, and V3 look like Q waves and coved ST elevation when the Mirror Test is performed.

If this was an actual paper ECG tracing, you would flip the page over, rotate it 180° and hold it up to the light

Thus, the purpose of the Mirror Test is to facilitate recognition of ECG changes that might represent acute posterior MI.

Reciprocal ECG Changes

ST segment depression occurring in the leads opposite or distal to the affected wall or region is referred to as reciprocal ECG changes.

For example in the setting of an acute anterior infarct ST depression may be seen in the inferior leads.

Generally, ST depression mirrors the degree of ST elevation occurring. Whilst the exact mechanism of reciprocal changes is not understood there are several thoughts. Firstly it may be a sign of ischaemia on the opposite wall; or it simply may be an electrographic phenomena. It is however universally accepted that these types of infarcts carry a worse prognosis.

WHANGANUI DHB ACUTE CORONARY SYNDROME PATHWAY

Whanganui District Heath Board has a dedicated pathway which related exclusively to angina, unstable angina, acute non stemi and ST elevation MI. All patients admitted with such conditions should be placed on the pathway. The pathway is developed to enable standardisation and continuity of care through multiple disciplinary input.

Nurses working within the health board who look after patients admitted with an acute cardiac event are expected to complete the core care plans generated for each day and enter all notes in the corresponding section for each day post infarct. The pathway is expected to be completed for the length of the patient's stay or for up to five days.

Please study Acute Coronary Syndrome pathway found in the reading section.

NOTES

MODULE SEVEN

RHYTHMS ASSOCIATED WITH CARDIAC ARREST

Cardiac arrest is defined as the cessation of cardiac function resulting in the loss of effective cardiac output which, if left untreated, will cause brain tissue damage and ultimately death.

VENTRICULAR TACHYCARDIA (VT)

This life threatening dysrhythmia indicates the presence of significant underlying cardiac disease and occurs most commonly in the setting of acute myocardial infarction and coronary artery disease. It is also preventable in people with cardiomyopathy or mitral valve prolapse.

ECG RECOGNITION

Four or more consecutive premature ventricular contractions
Essentially regular
Rate 110-250 beats per minute
No associated p wave, as the complexes originate in the ventricles
Complexes are wide and bizarre in appearance

CLINICAL IMPLICATIONS

People with VT may be conscious, initially, but left untreated will often be rendered unconscious, and rapidly progress to ventricular fibrillation and ultimately death.

TREATMENT

CONSCIOUS VT

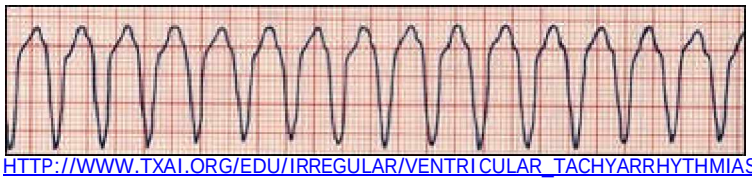
Lignocaine 1-1.5mg/kg IVI stat, potentially followed by a lignocaine infusion &/Or Amiodarone, usually as an infusion of up to 15 mg/kg for 24 hours

UNCONSCIOUS VT

Direct Current Reversion (DCR):

- Monophasic defibrillation of 1 x 360 Joules & 360 J for all subsequent shocks
- Biphasic defibrillation of 1x 200 Joules & 200 J for all subsequent shocks
- Stacked shocks are only delivered if it is a witnessed, monitored arrest

When reverted, an infusion of lignocaine or amiodarone is usually commenced.



HTTP://WWW.TXAI.ORG/EDU/IRREGULAR/VENTRICULAR_TACHYARRHYTHMIAS.HTM

VENTRICULAR FIBRILLATION

Ventricular fibrillation is defined as chaotic depolarisation of individual myocardial muscle fibres which do not produce an effective ventricular contraction, therefore the heart does not pump and no pulses are felt.

ECG RECOGNITION

Irregular, chaotic undulations
No clear-cut ventricular complexes

CLINICAL IMPLICATIONS

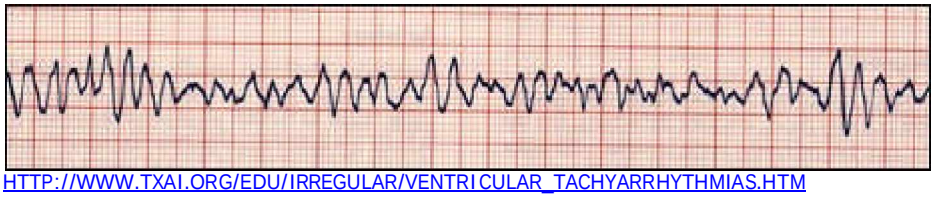
Rapid loss of consciousness, with no detectable pulse or blood pressure is inevitable with VF. Left untreated all people with VF will die

TREATMENT

Direct Current Reversion (DCR):

- Monophasic defibrillation of 1 x 360 Joules & 360 J for all subsequent shocks
- Biphasic defibrillation of 1 x 200 Joules & 200 J for all subsequent shocks
- Stacked shocks are only delivered if it is a witnessed, monitored arrest

1mg of IVI adrenaline may be required to increase the electrical activity within the heart and hence make the heart more responsive to defibrillation.



HTTP://WWW.TXAI.ORG/EDU/IRREGULAR/VENTRICULAR_TACHYARRHYTHMIAS.HTM

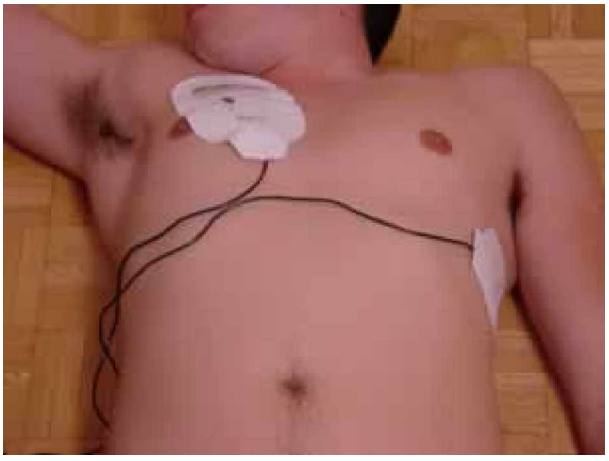
DEFIBRILLATION

Transthoracic cardioversion delivers electrical energy to the heart by means of metal paddles placed on the intact chest or placed directly on the heart when the chest is opened, such as during cardiac surgery.

This procedure depolarises the excitable myocardium, thereby interrupting re-entrant circuits and discharging automatic pacemaker foci to establish electrical homogeneity.

Cardioversion restores sinus rhythm if the sinus node is the first pacemaker to fire after the electrical shock.

Defibrillator Pad Position



ASYSTOLE

Asystole is recognised by an absence of any electrical activity at all in the myocardium.

CLINICAL IMPLICATIONS

A heart in a systole has no electrical impulse being initiated and hence is incapable of any mechanical activity. At this point the person is clinically dead.

ECG RECOGNITION

An essentially flat line on the ECG signal
There may be odd undulations but no discernible complexes

TREATMENT

Cardiopulmonary Resuscitation (CPR)
Adrenaline 1mg IVI stat, repeat every 3 minutes.

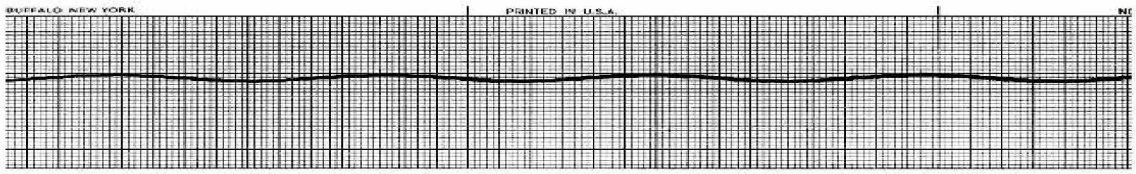


Figure 27-19 Asystole. (Always check two different leads to confirm rhythm.)

It is the recommendation of the editors of this package, to ensure that information you are using is current, to access the standards for practice regarding advanced life support. Please refer to the following website for the New Zealand Resuscitation Guidelines for Basic Life Support and Advanced Life Support for the most current updates.

http://www.nzrc.org.nz
Go to policy statements and guidelines.

NOTES

MODULE EIGHT

ATRIAL DYSRHYTHMIA

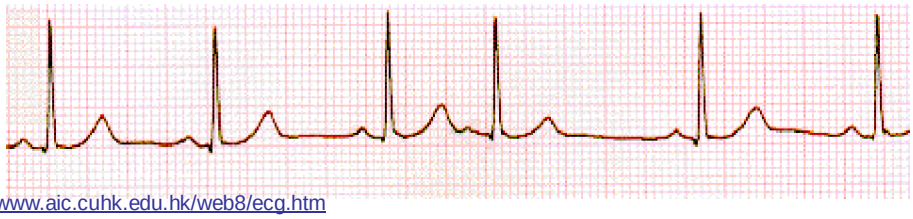
PREMATURE ATRIAL CONTRACTIONS (PAC)

When the atrial impulse originates at a point other than the sinus node a premature atrial contraction will be the result. The ectopic focus may be anywhere in the atrial. The resultant deflection is called a p prime wave.

One the ECG an abnormal p wave (p prime) is seen, followed by a normal QRS and t wave. The complex is premature and therefore is closer to the preceding sinus beat than expected. The sinus node then resets itself and there is a compensatory pause before the next normal sinus beat.

CLINICAL IMPLICATIONS

Normal: emotion, caffeine and other stimulants, alcohol, tobacco
Abnormal: warning sign of congestive heart failure (due to AMI) from atrial stretch

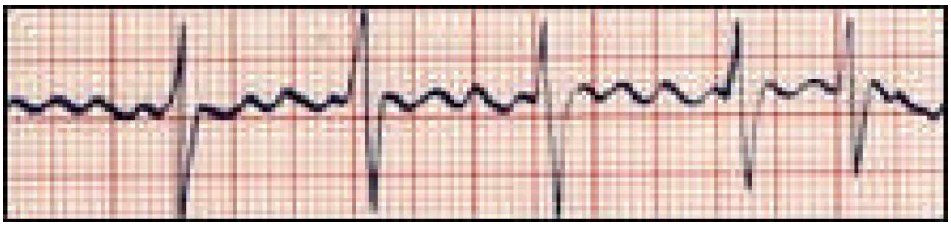


http://www.aic.cuhk.edu.hk/web8/ecg.htm

ATRIAL FLUTTER

Atrial flutter is caused by an ectopic focus from the atria, firing at a very fast rate, usually 300 times per minute. The AV node generally refuses to transmit all the impulses and only allows the occasional message through to the ventricles, in order to control the heart rate.

http://www.txai.org/edu/irregular/bradycardias.htm



CLINICAL IMPLICATIONS

Atrial flutter may be seen in association with any of the following conditions:

- Heart failure
- Valvular disease
- Pulmonary embolus
- Digitalis toxicity
- Ischaemic heart disease

ECG RECOGNITION

Flutter waves have a saw tooth configuration. QRS is normal and may be regular or irregular depending on the degree of the AV block.

Flutter rates are usually divisions of 300. For example:

- 1:1 = rate of 300 beats per minute
- 2:1 = rate of 150 beats per minute
- 3:1 = rate of 100 beats per minute
- 4:1 = rate of 75 beats per minute

ATRIAL FIBRILLATION (AF)

When many cells in the atria fire impulses haphazardly the result is disorganisation of the electrical stimuli, hence the atria do not beat effectively, but rather quiver or fibrillate.

CLINICAL IMPLICATIONS

- Congestive Cardiac Failure
- COPD
- Hyperthyroidism
- Hypertension
- Idiopathic

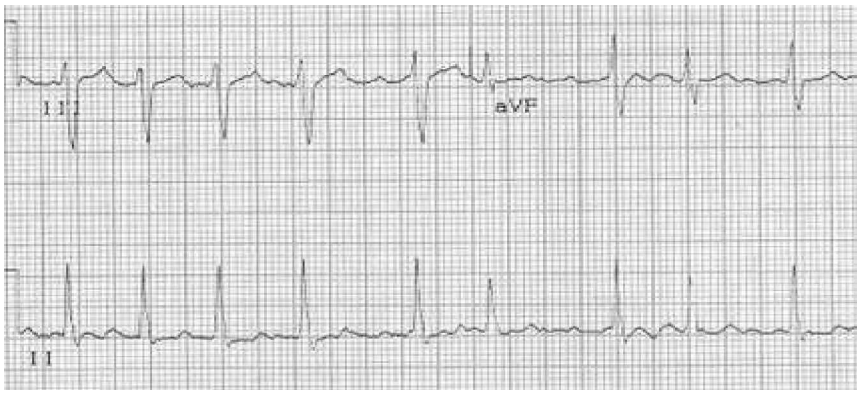
DANGER OF THROMBUS FORMATION

As the atria do not contract properly they do not fully empty. For this reason there is a greater risk of clots forming due to stasis of the blood, which may be then circulated through the body.

ECG RECOGNITION

- No p waves
- Fibrillatory line between QRS complexes
- "Irregularly irregular" ventricle response

http://www.aic.cuhk.edu.hk/web8/ecg.htm



- P waves may be abnormal, upright, inverted or very difficult to see
- QRS normal, but may be distorted if aberrant conduction is present
- rate 140-220 beats per minute

PREMATURE VENTRICULAR COMPLEXES

CLINICAL IMPLICATIONS

PVC's are present in people with normal heart. The problem with PVC's arises when too many of them become present, as they do not represent a true effective beat.

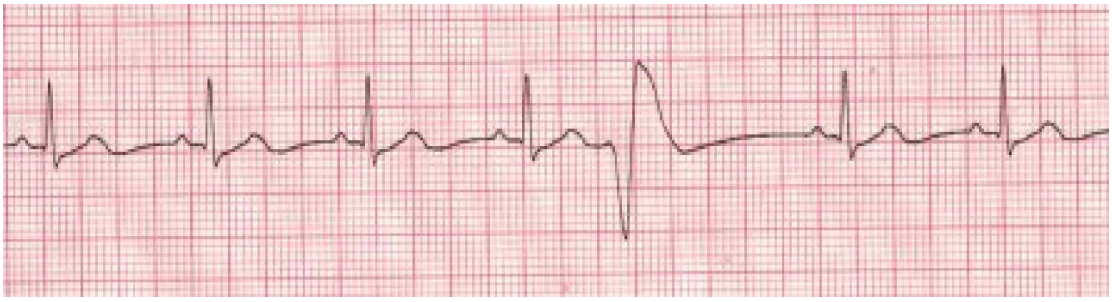
VENTRICULAR BIGEMINY

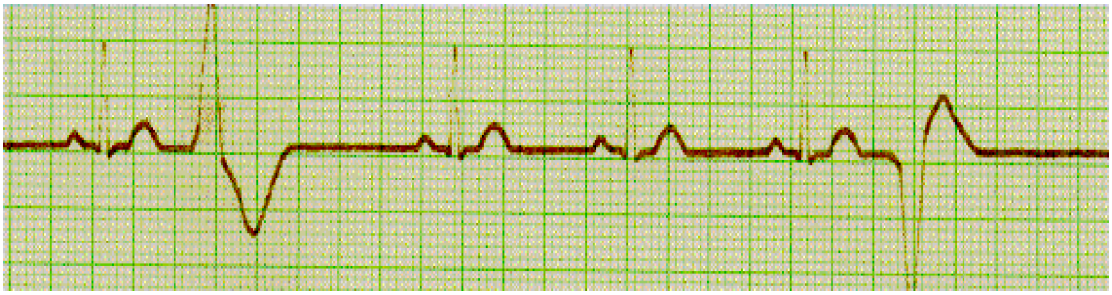
A bigeminal rhythm that consists of pairs i.e. one sinus beat, one PVC, one sinus beat, one PVC, etc

ECG RECOGNITION

- Broad QRS complex (longer than 0.12 seconds)
- Premature
- T wave in opposite direction to the QRS complex
- No related P wave
- Compensatory pause before the next complex

See below





METHOD OF RHYTHM ANALYSIS & ECG INTERPRETATION

This "method" is recommended when reading all 12-lead ECGs. Like the physical examination, it is desirable to follow a standardised sequence of steps in order to avoid missing subtle abnormalities in the ECG tracing, some of which may have clinical importance. The six major sections in the "method" should be considered in the following order:

1. Measurements
2. Rhythm Analysis
3. Conduction Analysis
4. Waveform Description
5. ECG interpretation
6. Comparison with previous ECG (if any)

1. MEASUREMENTS (USUALLY MADE IN LEAD II)

- Heart rate (state atrial and ventricular, if different)
- PR interval (from beginning of P to beginning of QRS) QRS duration (width of most representative QRS)
- QT interval (from beginning of QRS to end of T)
- QRS axis in frontal plane (advanced ECG module only)

2. RHYTHM ANALYSIS

State basic rhythm (e.g. "normal sinus rhythm", "atrial fibrillation", etc). Identify additional rhythm events if present (e.g. "PVC's", "PAC's", etc). Consider all rhythm events from atria, AV junction, and ventricles

3. CONDUCTION ANALYSIS

"Normal" conduction implies normal Sino-atrial (SA), atrio-ventricular (AV), and intraventricular (IV) conduction.

The following conduction abnormalities are to be identified if present:

Sino Atrial (SA) Blocks
Atrio ventricular (AV) Blocks
1^{st}, 2^{nd} (Type I and Type II) and 3^{rd} degree
Intraventricular Blocks (Advanced ECG module only)
RBBB, LBBB, Fascicular Blocks, Non specific intraventricular conduction defects, Wolff Parkinson White Syndrome (WPW)

4. WAVEFORM DESCRIPTION

Carefully analyse the 12-lead ECG for abnormalities in each of the waveforms in the order in which they appear: P-waves, QRS complexes, ST segments, T waves, and Don't forget the U waves.

P Waves: are they too wide, too tall, look funny (i.e., are they ectopic), etc.? QRS complexes: look for pathologic Q waves, abnormal voltage, etc
ST segments: look for abnormal ST elevation and/or depression. T Waves: look
For abnormally inverted T waves.

U Waves: look for prominent or inverted U waves.

5. ECG INTERPRETATION

This is the conclusion of the above analyses. Interpret the ECG as "Normal", or "Abnormal". Occasionally the term "borderline" is used if unsure about the significance of certain findings.

List all abnormalities.

Examples of "abnormal" statements are: Inferior MI, probably acute
Old anteroseptal MI
Left ventricular hypertrophy (LVH)

6. COMPARISON WITH PREVIOUS ECG

If there is a previous ECG in the patient's file, the current ECG should be compared with it to see if any significant changes have occurred. These changes may have important implications for clinical management decisions.

NOTES