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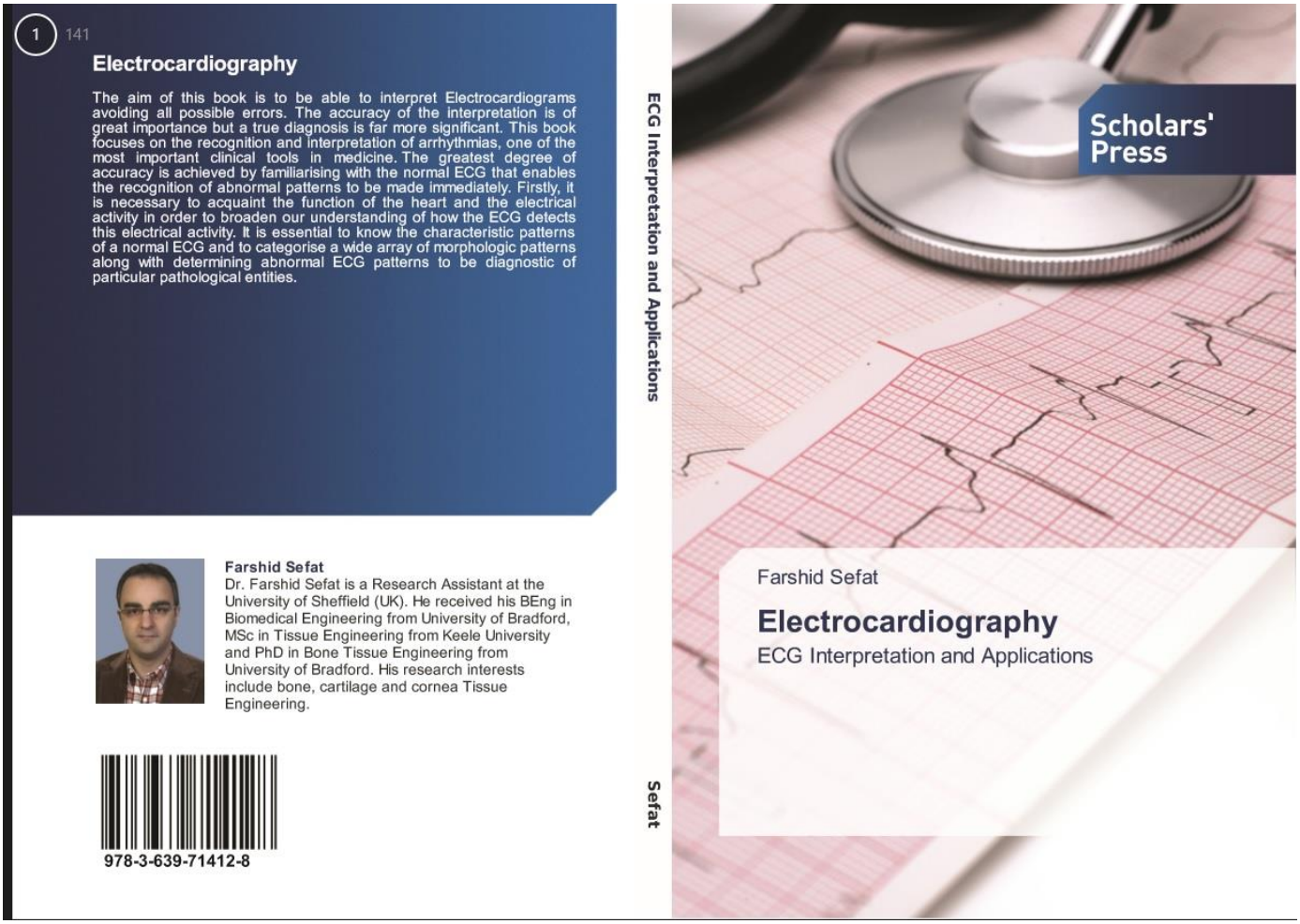

This Book is dedicated to:

My parents

Mr. Hossein Sefat,

Mrs. Maryam Molazem

&

My wife

Dr. Ehsaneh Daghigh Ahmadi

ABSTRACT

The aim of this book is to be able to interpret Electrocardiograms avoiding all possible errors. The accuracy of the interpretation is of great importance but a true diagnosis is far more significant.

This book focuses on the recognition and interpretation of arrhythmias, one of the most important clinical tools in medicine. The greatest degree of accuracy is achieved by familiarising with the normal ECG that enables the recognition of abnormal patterns to be made immediately. Firstly, it is necessary to acquaint the function of the heart and the electrical activity in order to broaden our understanding of how the ECG detects this electrical activity. It is essential to know the characteristic patterns of a normal ECG and to categorise a wide array of morphologic patterns along with determining abnormal ECG patterns to be diagnostic of particular pathological entities.

A series of practical experiments have been carried out on various subjects using the BIOPAC system to record electrical signals of the heart. Subjects were asked to perform various tasks such as lying down, sitting, deep breathing and exercising to detect electrical signals in different conditions and eventually interpret the data. The ethical issue toward each subject is also too important, so it was necessary to let the subject know about any risk factors during experiment. For this purpose, a Volunteer Information Sheet was designed during this work for each subject to read and be aware of all the ethical issues. Also, another Patient Consent Form was designed to make sure that each volunteer fully understands the procedures. Volunteer Questionnaire is necessary to make sure volunteer that there is no problem, which can affect the experimental results.

Finally, ECG results were interpreted using a systematic approach and the precise findings were correlated with the pathophysiology and clinical status of the patient. This book concludes with a thorough investigation into the essential techniques and skills required to accurately interpret an ECG, eliminating as many errors as possible.

ACKNOWLEDGEMENTS

All praise is due to Allah who guided us to this, and we would not have found the way had it not been that Allah guided us (Holy Qur'an, Ch. 7, Ver. 43). Thanks God for giving me a chance to study the wonderful field of Biomedical Engineering.

The greatest credit for my success goes to my father **Mr Hossein Sefat** and my mother **Mrs Maryam Molazem** for their help, encouragement, motivation and love.

I would also like to express my sincere love and care to my wife, **Dr. Ehsaneh Daghigh Ahmadi**, for her patience, encouragement, and moral support.

I pay special thanks to **Dr. Mansour Youseffi** for his support, help and encouragement during this work. Also, I would like to thank **Miss Anne Lesley Butler** for her assistance throughout the project.

I would like to thanks Mr Chris Rule and Mr Prabuhaharan Elangovan for being volunteers in my case study.

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List of abbreviations

AF	Atrial Fibrillation
APB	Atrial Premature Beat
AV	Atrio Ventricular
CAD	Coronary Artery Disease
ECG	Electrocardiogram
LAD	Left Axis Deviation
LGL	Lown-Ganong-Levine
LVH	Left Ventricular Hypertrophy
MAT	Multifocal Atrial Tachycardia
MI	Myocardial Infarction
PAF	Paroxysmal Atrial Fibrillation
RAD	Right Axis Deviation
RVH	Right Ventricular Hypertrophy
SA	Sino Atrial
SVT	Supraventricular Tachycardia
SSS	Sick Sinus Syndrome
VF	Ventricular Fibrillation
VPB	Ventricular Premature Beat
VT	Ventricular Tachycardia
WAP	Wandering Atrial Pacemaker
WPW	Wolff-Parkinson-White
bpm	Beats per minute

Chapter 1

INTRODUCTION

ECG is a heart monitoring technique. It records the electrical signals produced by each heartbeat. The cardiac muscle (myocardium) is unique in its rhythmic beating. This rhythm is analysed with an electrocardiogram. What the ECG measures is the depolarisation (losing their electrical charge) and repolarization of the heart muscle as the cells contract. The ECG line that is produced measures the direction of the electrical activity along with its magnitude. Since the atria are smaller in size than the ventricles, their magnitude on the ECG strip is smaller. Interpreting the ECG is a medical art, and requires the familiarity with the electrical differences of many different breeds of person.

Flow of blood through the four chambers of the human heart in ECG helps doctors to read ECG for correct diagnosis. The electrical signals produced by two nodes in heart, SA nodes (Sino-Atrial node) and AV node (Atrio-ventricular node). SA node is located in the walls of right atrium and this region contract 72 times per minute. SA node is known as natural pacemaker of the heart since its contracts more than the other node. ECG trace shows us the electrical signal generated by the heart. It also shows depolarisation, repolarisation, opening and closing the heart valves.

The ECG is of greatest use in diagnosing cardiac arrhythmias, evaluation of the effectiveness of antiarrhythmic drug therapy, to surgical and critical care monitoring. It is not possible to find out a heart disease in a patient only by using ECG, as it does not provide information about mechanical function of the heart. Also ECG is not usable to measure cardiac output or blood pressure. Therefore, the disease should be decided upon conjunction with other medical tools.

To facilitate the interpretation of rate and rhythm along with variation in the wave, the ECG is usually recorded on the graph paper. It is essential that these variations are correctly interpreted in order to get correct diagnosis. The ECG contains lots of information and these information need to picked up in each reading to achieve a good diagnosis. We need to remember that, the ECG should not be over read.

Reading an ECG is a matter of pattern recognition and deductive logic. When enough experience is not available, adopting a systematic manner so that all aspects of reading the ECG are covered can make an accurate diagnosis.

The aim of this report is to look at different type of abnormalities that can be seen in the ECG, interpret the arrhythmias and compose a systematic approach to aid with the interpretation of ECG's.

The important point is that electrical activity of the heart is only made visible via the ECG. These electrical activities provide powerful indicators on the cardiac function. To get a good treatment, a correct diagnosis is necessary whilst misinterpretation can make serious problems.

Chapter 2

ECG HISTORY

Heart is an important organ in the body and its function is vital. Electrocardiography is the most commonly used diagnostic test in cardiology. Many years scientist tried to find a good method to know more about the heart activities and related diseases. Investigations started from 17th century and many scientists were involved to find a part of ECG's body.

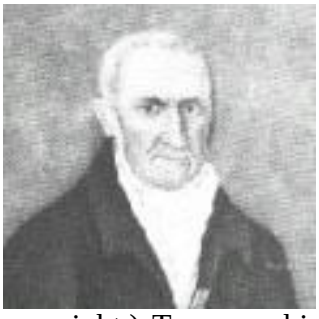
William Gilbert (1600) was one of the oldest scientists that he was involved in part of ECG history. William Gilbert, Physician to Queen Elizabeth I, President of the Royal College of Physicians, and creator of the 'magnetic philosophy' introduces the term 'electrica' for objects (insulators) that hold static electricity.

Jan Swammerdam (1664), a Dutchman, disproves Descartes' mechanistic theory of animal motion by removing the heart of a living frog and showing that it was still able to swim. On removing the brain all movement stopped but then, when the frog was dissected and a severed nerve end stimulated with a scalpel the muscles twitched. This proved that movement of a muscle could occur without any connection to the brain and therefore the transmission of 'animal spirits' was not necessary [16].

Edward Bancroft (1769), an American Scientist, suggests that the 'shock' from the Torpedo Fish is electrical rather than mechanical in nature. The Torpedo fish and other species were widely known to deliver shocks and were often used in this way for therapeutic reasons.

Abildgaard (1775) Shows that hens can be made lifeless with electrical impulses and he could restore a pulse with electrical shocks across the chest. "With a shock to the head, the animal was rendered lifeless, and arose with a second shock to the chest; however, after the experiment was repeated rather often, the hen was completely stunned, walked with some difficulty, and did not eat for a day and night; then later it was very well and even laid an egg [16].

Luigi Galvani (1780) Italian Anatomist notes that a dissected frog's leg twitches when touched with a metal scalpel. He explained a part of his observations as below:
"I had dissected and prepared a frog in the usual way and while I was attending to something else I laid it on a table on which stood an electrical machine at some distance from its conductor and separated from it by a considerable space. Now when one of the persons present touched accidentally and lightly the inner crural nerves of the frog with the point of a scalpel, all the muscles of the legs seemed to contract again and again as if they were affected by powerful cramps" [16]. Luigi showed that electrical generator could contact directly to a muscle cause a muscle contraction. Galvani's name is given to the 'galvanometer' which is an instrument for measuring (and recording) electricity - this is essentially what an ECG is a sensitive galvanometer.



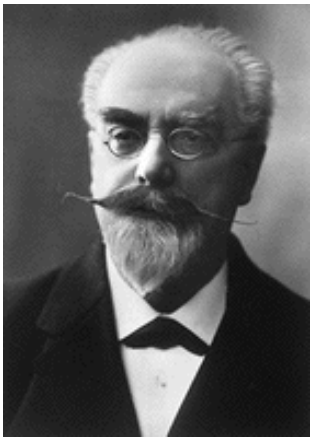
Alessandro Volta (1792), Italian Scientist and inventor, attempts to disprove Galvani's theory of "animal electricity"' by showing that the electrical current is generated by the combination of two dissimilar metals. His assertion was that the electrical current came from the metals and not the animal tissues. (We now know that both Galvani and Volta were right.) To prove his theory he develops the voltaic pile in 1800 [16].

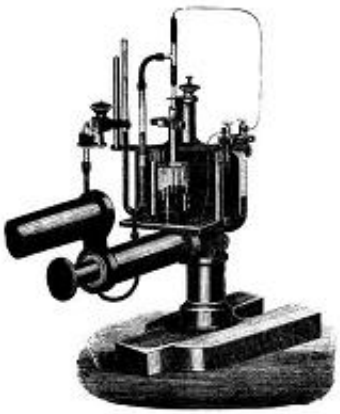
Carlo Matteucci (1842), Professor of Physics at the University of Pisa, shows that an electric current accompanies each heartbeat

Emil Dubois-Reymond (1843) German physiologist describes an "action potential" accompanying each muscular contraction.

Alexander Muirhead (1869-70), an electrical engineer, may have a recorded a human electrocardiogram at St Bartholomew's Hospital, London but this is disputed. If he had he is likely to have used a Thompson Siphon Recorder [16].

Gabriel Lippmann (1872) French physicist invents a capillary electrometer. It is a thin glass tube with a column of mercury beneath sulphuric acid. The mercury meniscus moves with varying electrical potential and is observed through a microscope [17].



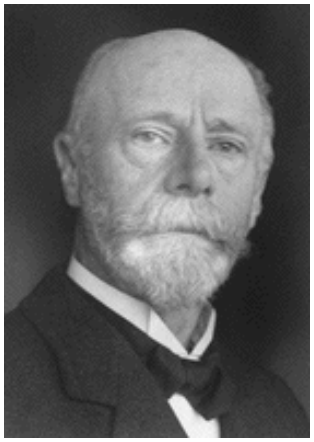


Guillaume Benjamin (1872) describes the resuscitation of a drowned girl with electricity in the third edition of his textbook on the medical uses of electricity. This episode has sometimes been described as the first 'artificial pacemaker'

Electrical smile made by electricity

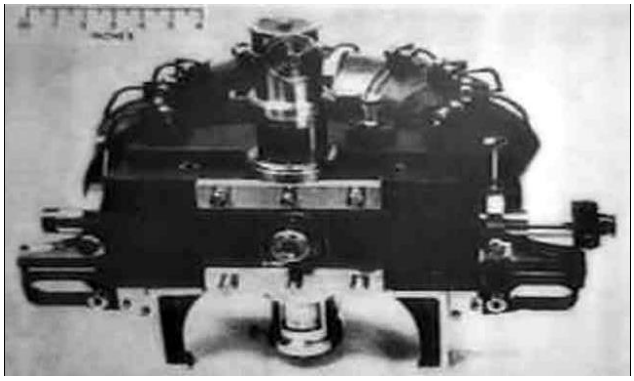
John Burden Sanderson and Frederick page 91878) record the heart electrical current and shows it consists of two phases, which later called QRS and T.

Augustus D. Waller (1887) publishes the first human electrocardiogram.



Willem Einthoven (1895) using an improved electrometer and a correction formula developed independently of Burch distinguishes five deflections, which he names P, Q, R, S and T.

Einthoven (1901) invents a string galvanometer for producing electrocardiograms using a fine quartz string coated in silver based on ideas by Deprez and d'Arsonval. Source:



Source: http://www.brad.ac.uk/acad/mecheng/Ptwigg/index.html

Einthoven (1902) publishes the first electrocardiogram recorded on a string galvanometer.

Einthoven (1906) publishes the first organised presentation of normal and abnormal electrocardiograms recorded with a string galvanometer.

Arthur Cushny (1907), professor of pharmacology at University College London, publishes the first case report of atrial fibrillation.

Einthoven (1912) addresses the Chelsea Clinical Society in London and describes an equilateral triangle formed by his standard leads I, II and III later called 'Einthoven's triangle'.

Willem Einthoven (1924) wins the Nobel Prize for inventing the Electrocardiograph [17].

Source: http://www.brad.ac.uk/acad/mecheng/Ptwigg/index.html

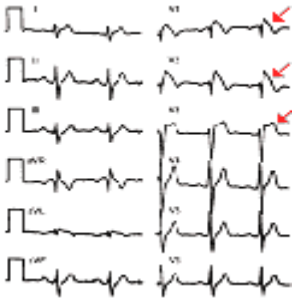
Charles Wolferth and Francis Wood (1932) describe the clinical use of chest leads. By joining the wires from the right arm, left arm and left foot with 5000 Ohm resistors Frank Wilson (1934) defines an 'indifferent electrode' later called the 'Wilson Central Terminal'. Wilson defines the unipolar limb leads VR, VL and VF where 'V' stands for voltage [16].

(1938) American Heart Association and the Cardiac Society of Great Britain define the standard positions, and wiring, of the chest leads V1 - V6.

Emanuel Goldberger (1942) increases the voltage of Wilson's unipolar leads by 50% and creates the augmented limb leads aVR, aVL and aVF. When added to Einthoven's three limb leads and the six chest leads we arrive at the 12-lead electrocardiogram that is used today [16].

Montana physician Norman Jeff Holter (1949) develops a 75-pound backpack that can record the ECG of the wearer and transmit the signal.

Pedro Brugada and Josep brugada (1992) of Barcelona publish a series of 8 cases of sudden death, Right Bundle Branch Block pattern and ST elevation in V1 - V3 in apparently healthy individuals.



Robert Zalenski (1993), Professor of Emergency Medicine, Wayne State University Detroit, and colleagues publish an influential article on the clinical use of the 15-lead ECG, which routinely uses V4R, V8 and V9 in the diagnosis of acute coronary syndromes [16].

Mac 5000, 15-lead ECG
Source: http://www.ecglibrary.com/ecghist.html

Chapter 3

THE HEART AND ITS FUNCTIONS

The heart is a hollow, cone shaped, and muscular pump located within the mediastinum of the thorax and resting upon the diaphragm. The heart is the most significant muscle in the body known as cardiac muscle. Its function is vital as pumping blood around the body in order for survival because without circulation, tissues lack a supply of oxygen and nutrition, and waste material accumulates [1].

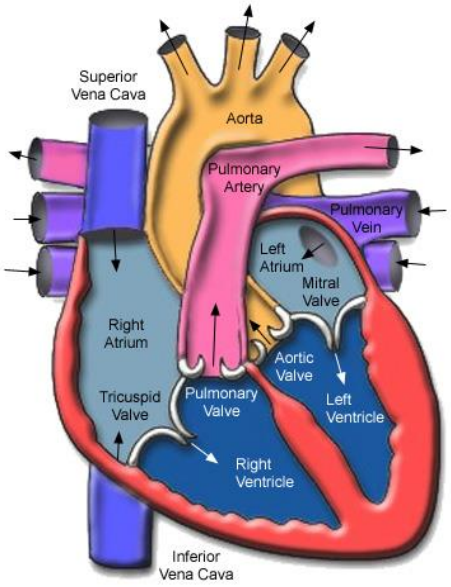


Figure 3.1 cross section of the heart
Source: http://www.tmc.edu/thi/anatomy2.html

Under such condition, cells soon begin to undergo irreversible changes that quickly lead to the death of the organism. Arteries are one type of blood vessels in order to delivery oxygenated blood from the heart to the rest of the body and veins carry deoxygenated

blood back to the heart and finally to the lung to eliminate excess carbon dioxide and gain more oxygen.

3.1 Anatomy of the heart

The heart is about the size of the individual closed fist; weight about 300 grams in adult and is about 12cm long, 9cm wide at the widest point and 6cm thick. It pumps 4300 gallons of blood a day.

3.2 Location of the heart

The heart is located in the chest cavity just posterior to the breastbone (sternum), between the lungs and superior to the diaphragm. Two thirds of heart bulk to the left of the body's midline. A fluid filled sac called the pericardium surrounds the heart. Arteries and veins are responsible to take blood to the body cells and return it to the heart respectively [18].

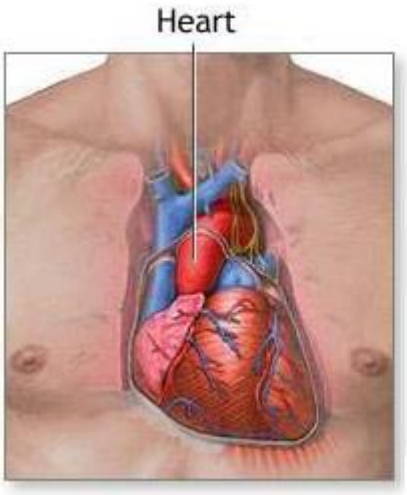


Fig 3.2 Position of the heart

3.3 Chambers

The heart is divided by a partition or septum into two halves [19]. The halves are in turn divided into two chambers. So in fact the internal cavity of the heart is divided into four chambers:

- Right atrium

- Right ventricle
- Left atrium
- Left ventricle

The upper two chambers of the heart are called atria and the lower two chambers are called ventricles. Valves allow blood to flow in one direction between the chambers of the heart.

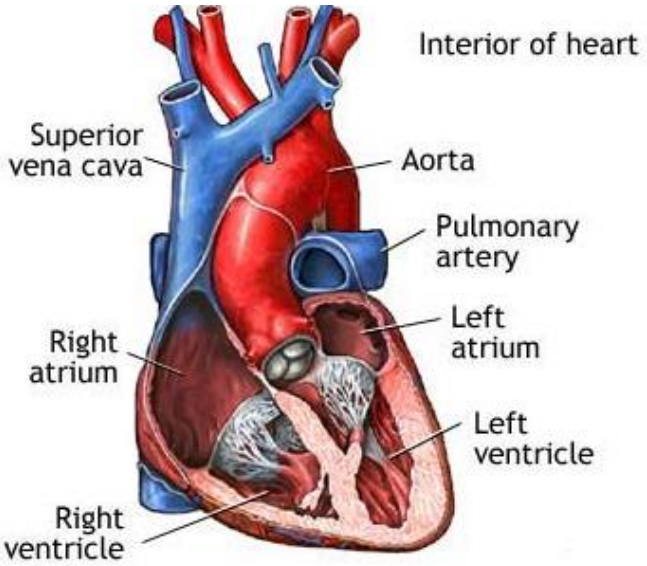


Fig 3.3 Chambers of the heart

The left atrium collects oxygenated blood from the pulmonary circulation, whereas the right atrium collects deoxygenated blood from the circulatory (systematic) system. The left ventricle receives the oxygenated blood from the left atrium and pumps it to the whole body, excluding the lungs. The right ventricle receives deoxygenated blood from the right atrium, and pumps it into the lungs, to become rich in oxygen.

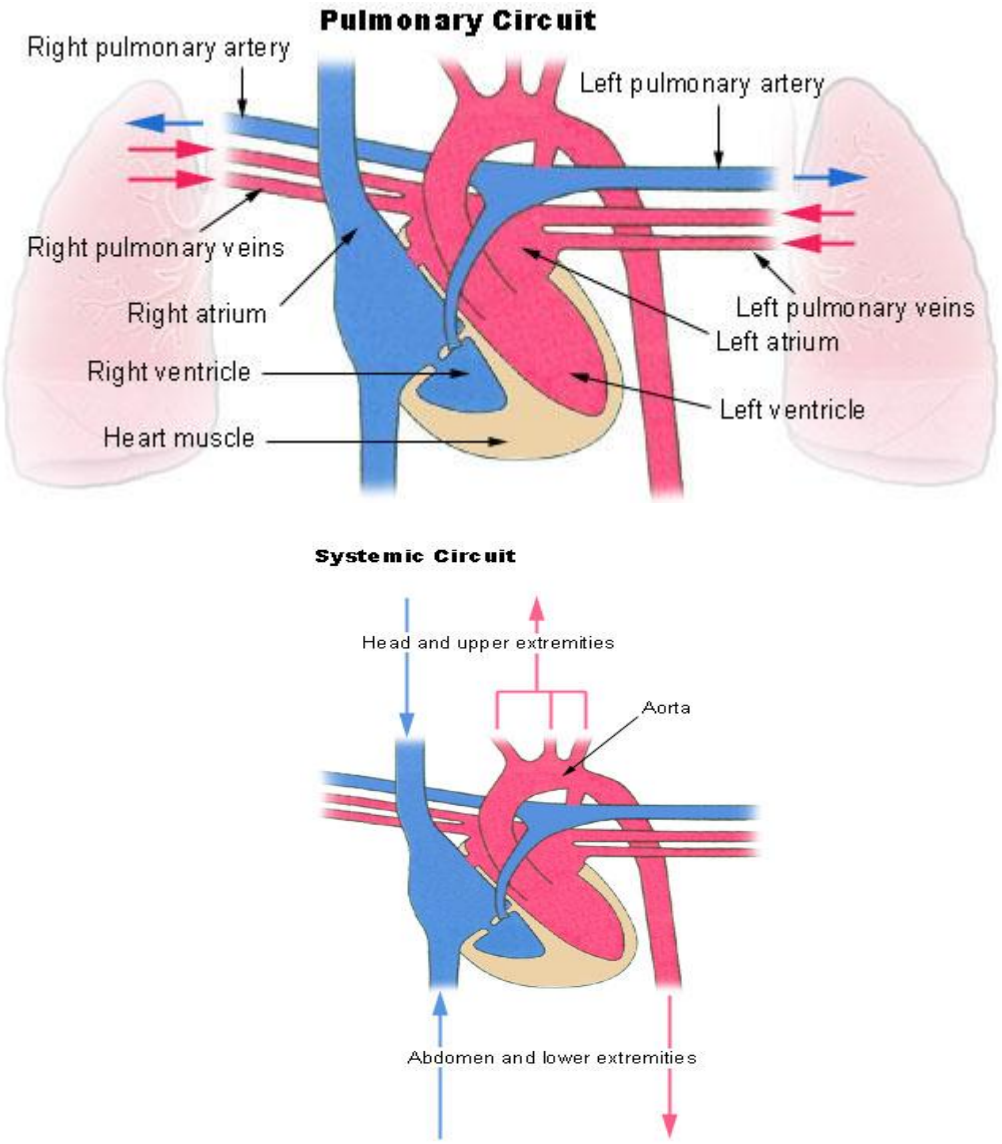


Fig 3.4 systematic and pulmonary circuit

The walls of the chambers thicken in relation with their individual function. This means the atria have thin walls as they only have to expel blood to the ventricles. With the walls of the ventricles being thicker with having to pump the blood further. The thickest wall of all four chambers is the left ventricle as it expels blood to the whole body [5].

3.4 Heart Wall

Pericardium (parietal pericardium) is the protective layer that covers the heart and the proximal ends of the large blood vessels to which it is attached. It is a double layer that also known as pericardial sac. These two layers are:

- Fibrous pericardium (outer layer = rough)
- Serous pericardium (inner layer)

The parietal pericardium is a rough, protective sac composed largely of white fibrous connective tissue and a serous membrane. It is attached to the central portion of the diaphragm, the back of the sternum, the vertebral column and the large blood vessels emerging from the heart. Between the parietal and visceral membrane is a space called pericardial cavity that contains small amount of serous fluid. This fluid reduces the friction between the pericardial membranes as heart move within them and acts as a shock absorber.

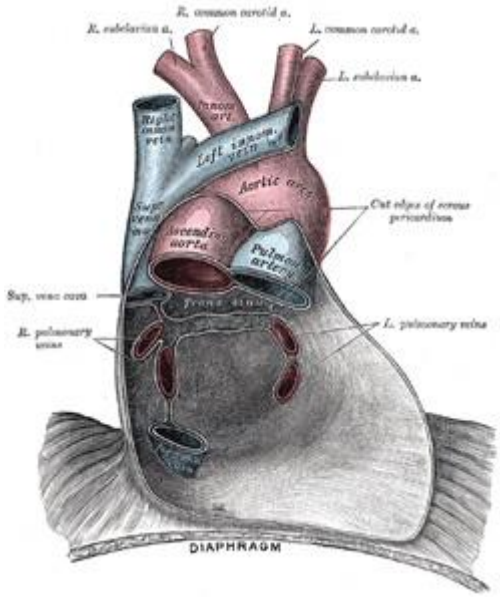


Fig. 3.5 Posterior wall of the pericardial sac, showing the lines of reflection of the serous pericardium on the great vessels.
Source: http://www.bartleby.com/107/137.html

The heart wall is divided into three layers:

- Epicardium (Outer layer also known as Visceral layer)
- Myocardium (middle layer)
- Endocardium (innermost layer)

At the base of the heart the serous pericardium turns back on itself to form the **Epicardium**. It is the outer layer of the wall of the heart. It is composed of connective tissue covered by epithelium. The Epicardium is also known as the visceral

pericardium. Function of the pericardium is to provide an outer protective layer for the heart.

Myocardium is the muscular middle layer of the wall of the heart. It is composed of spontaneously contracting cardiac muscle fibres, which allow the heart to contract. The function of the Myocardium is to stimulate heart contractions to pump blood from the ventricles and relaxes the heart to allow the atria to receive blood. Also provide an outer protective layer for the heart [20].

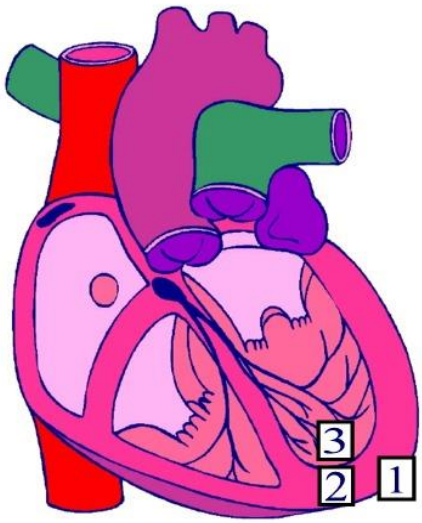


Fig 3.6 Heart wall (1) pericardium
(2) Myocardium (3) Endocardium
Source: http://mtsu32.mtsu.edu:11259/heartlayers.htm

The **Endocardium** is the inner layer of the heart. It consists of epithelial tissue and connective tissue. The function of the Endocardium is to lines the inner cavities of the heart, covers heart valves and is continuous with the inner lining of blood vessels. Purkinje fibres are located in the Endocardium. They participate in the contraction of the heart muscle [20].

3.5 Heart Valves

Pumps need a set of valves to keep the fluid flowing in one direction and the heart is no exception. The heart has two types of valves that keep the blood flowing in the correct direction. The valves between the atria and ventricles are called atrioventricular valves (also called cuspid valves), while those at the bases of the large vessels leaving the ventricles are called semilunar valves [21].

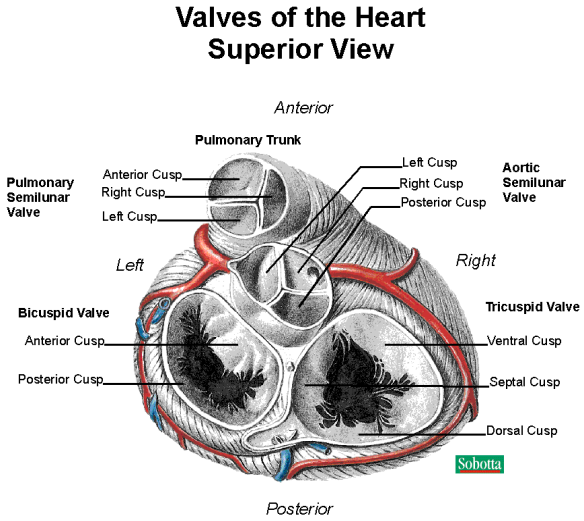


Fig 3.7 valves of the heart

Source: http://biology.about.com/library/organs/heart/blmyocardium.htm

The right atrioventricular valve is the tricuspid valve. The left atrioventricular valve is the bicuspid, or mitral, valve. The valve between the right ventricle and pulmonary trunk is the pulmonary semilunar valve and the valve between the left ventricle and the aorta is the aortic semilunar valve.

When the ventricles contract, atrioventricular valves close to prevent blood from flowing back into the atria. When the ventricles relax, semi- lunar valves close to prevent blood from flowing back into the ventricles.

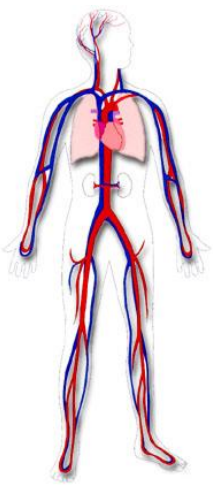
HEART VALVES	FUNCTION
TRICUSPID	Controls the blood between the right atrium and the right ventricle.
PULMONARY	Controls the blood between the right ventricle into the pulmonary artery.
MITRAL	Allows the oxygen rich blood to flow through the left atrium into the left ventricle.
AORTIC	Is the largest artery in the body and enables the oxygen rich blood to flow between the left ventricles and into the aorta.
SEMI-LUNAR	Comprises the aortic and pulmonary valves and enables the flow of blood in one direction within the heart

Table 3.1 Heart valves

3.6 Blood Vessels

The blood vessels are organs of the cardiovascular system that form a closed circuit of tubes carrying blood from the heart to the body cells and back again. These vessels include:

- Arteries
- Arterioles
- Capillaries



- Venules
- Veins

Fig 3.8 blood vessels
Source: http://www.tmc.edu/thi/anatomy1.html

The arteries and arterioles conduct blood away from the ventricles of the heart and lead to the capillaries. The capillaries function to exchange substances between the blood and the body cells, and the venules and veins return blood from the capillaries to the atria.

Arteries are strong, elastic vessels adapted for carrying blood away from the heart under relatively high pressure [22].

The wall of an artery consists of three distinct layers known as:

- **Tunica intima**: innermost layer composed of a layer of Endothelium rich in elastic and collagen fibers that provides a smooth surface that allows blood cells and platelets to flow through without being damaged.
- **Tunica media:** the middle layer makes up the bulk of the arterial wall. This layer contains many elastic fibers as well as smooth muscle cells.
- **Tunica adventitia:** the outer layer is relatively thin and consists chiefly of connective tissue with irregularly arranged elastic and collagen fibers. This layer attaches the artery to the surrounding tissue.

The walls of **veins** are similar to those of arteries in that they are composed of three distinct layers. However the middle layer of the venous wall is poorly developed. Consequently, veins have thinner walls and contain less smooth muscle and less elastic tissue than arteries [23].

Capillaries are the smallest blood vessels. They form the connection between the smallest arterioles and the smallest venules. They consist of endothelium (a single layer of squamous epithelial cells) [24].

Vessel	Type of wall	Function
Artery	Thick, strong wall with three layer Inner layer: endothelial lining Middle layer: smooth muscle and connective tissue Outer layer: connective tissue	Carries blood from the heart to arterioles.
Arteriole	Thinner wall than an artery but still with three layer	Connect an artery to a capillary. Help control the blood flow in to a capillary by vasodilation
Capillary	Single layer of squamous epithelium	Exchange food, waste materials and gases between the blood and tissue.
Venule	Thinner wall, less smooth muscle and elastic tissue than an arteriole	Connects a capillary to a vein
Vein	Thinner wall than artery but with similar layer. The middle layer more poorly.	Carries blood from venules to the heart.

Table 3.2 Blood vessels

3.7 Blood Supply to the Myocardium

The myocardium of the heart wall is a working muscle that needs a continuous supply of oxygen and nutrients to function with efficiency. For this reason, cardiac muscle has

an extensive network of blood vessels to bring oxygen to the contracting cells and to remove waste products [21].

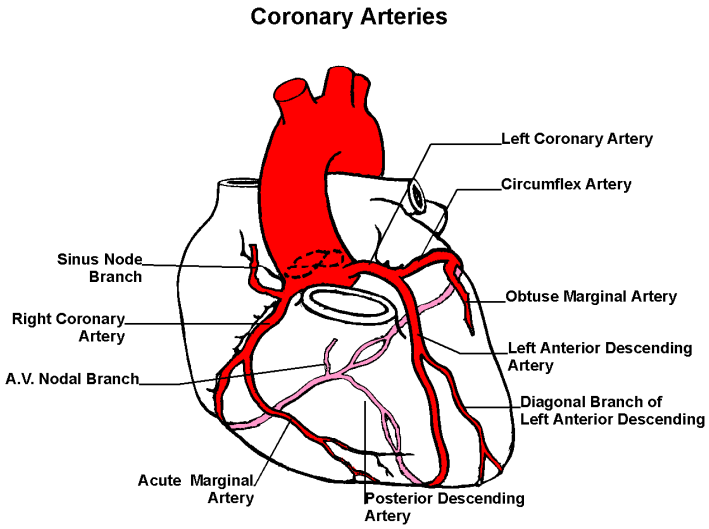


Fig 3.9 Blood Supply to the Myocardium
Source: http://www.accessexcellence.org/AE/AEC/CC/heart_anatomy.html

The right and left coronary arteries, branches of the ascending aorta, supply blood to the walls of the myocardium. After blood passes through the capillaries in the myocardium, it enters a system of cardiac (coronary) veins. Most of the cardiac veins drain into the coronary sinus, which opens into the right atrium [21].

3.8 Pulmonary and systemic circuit:

The blood vessels of the cardiovascular system can be divided into two major pathways:

- Pulmonary circuit
- Systemic circuit

In the pulmonary circuit, blood leaves the heart through the pulmonary arteries, goes to the lungs, and returns to the heart through the pulmonary veins.

In the systemic circuit, blood leaves the heart through the aorta, goes to all the organs of the body through the systemic arteries, and then returns to the heart through the systemic veins. Thus there are two circuits. Arteries always carry blood away from the heart and veins always carry blood toward the heart. Most of the time, arteries carry oxygenated blood and veins carry deoxygenated blood. There are exceptions. The pulmonary arteries leaving the right ventricle for the lungs carry deoxygenated blood and the pulmonary veins carry oxygenated blood [25].

3.9 The cardiac Muscle action Potential:

Change in the membrane potential of a muscle that causes contraction.

Action potentials are produced rapidly in the Sinoatrial (SA) node and spontaneously discharge action potential at an approximate rate of 100-120 per minute.

The action potential made of a rapid depolarisation phase followed by a rapid but partial repolarisation. This continues with a prolonged period of slow repolarisation known as plateau. By completing plateau phase, it terminates with a rapid repolarisation phase during which the membrane potential returns to its resting level.

3.9.1 Rapid depolarisation

Self-excitable means that cell generate their own action potentials such as neurons and small muscle fibres. The membranes of the cells of SA node are very permeable to sodium (Na+) ions and also we know that in resting phase, the resting membrane potential is about 90mV. However when they are brought to threshold by excitation in neighbouring fibres, voltage gated fast sodium ion channels open very fast allowing sodium ion to diffuse in to the cell causing rapid depolarisation.

3.9.2 Plateau

The voltage gated ‘slow’ calcium (Ca^{2+}) channels open in the plasma membrane and the membrane of the sacroplasmic reticulum. The Ca^{2+} from the extracellular fluid and the sacroplasmic reticulum diffuses into the cell and combines with Na^{+}. The combined Na^{+} and Ca^{2+} ions maintain the depolarisation for about 250ms, causing plateau [5].

3.9.3 Rapid Repolarisation

The voltage-gated potassium (K^+) channels opens, allowing K^+ ions to diffuse into the extracellular fluid. At the same time, the Na^+ and Ca^{2+} channels close, resting the membrane potential to -90mV causing repolarisation.

3.9.4 Refractory period

What is refractory means? Refractory in science refer to the period of time when a nerve or muscle cell is unresponsive to stimulation.
Refractory also divided into two stages:

- Absolute refractory period
- Relative refractory period

In absolute refractory period there are no action potentials transmitted as the muscle is drained and requires a moment to recharge. It lasts approximately as long as the muscle action potential. But in the relative refractory period the muscle is not fully charged but will attempt to contract if it receives a signal [5].

3.10 Conduction System of the Heart

Cardiac conduction is the rate at which the heart conducts electrical impulses. Cardiac muscle cells contract spontaneously and are coordinated by nodal tissue, specifically the sinoatrial node. There are other factors that influence heart rate as well. These include endocrine hormones, body temperature and exercise [26].
In fact conduction system of the heart involves the:

1. Sinoatrial (SA) node
2. Atrioventricular (AV) node
3. Atrioventricular bundle (bundle of His)
4. Right and left bundle branches
5. Conduction of the Purkinjie fibres.

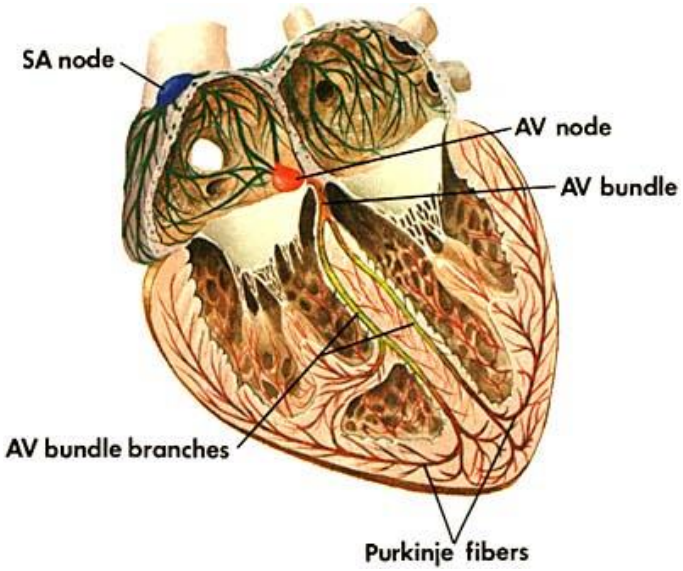


Fig 3.10 conduction system of the heart
Source: http://biology.about.com/library/organs/heart/blcardiacconduction.htm

Cardiac conduction is the rate at which the heart conducts electrical impulses. Cardiac muscle cells contract spontaneously. **Sinoatrial (SA) node** is known as natural pacemaker of the heart, which is located in the upper wall of the right atrium. The SA node is composed of nodal tissue that has characteristics of both muscle and nervous tissue. When the SA node contracts it generates nerve impulses that travel throughout the heart wall causing both atria to contract. The impulse from the SA node generates an electrical impulse of approximately 72 beats per minute in a resting adult [26].

SA node ➡ Contract two atria ➡ stimulate AV node ➡ Blood squeezed in to ventricles ➡ delay of 0.1(s) ➡ AV node connected to Bundle of His ➡ Right & left bundle of His ➡ Purkinjie fibres ➡ Ventricular contraction

Atrioventricular (AV) node is another part of nodal tissue located on the right side of the partiation that divides the atria, near the bottom of the right atrium. Impulses reach the AV node they are delayed for about a tenth of a second. This delay allows the atria to contract and empty their contents first.

Along with the SA node, other structures that conduct impulses through the cardiac muscle causing the atria and ventricle to contract shown in Fig. 3.12.

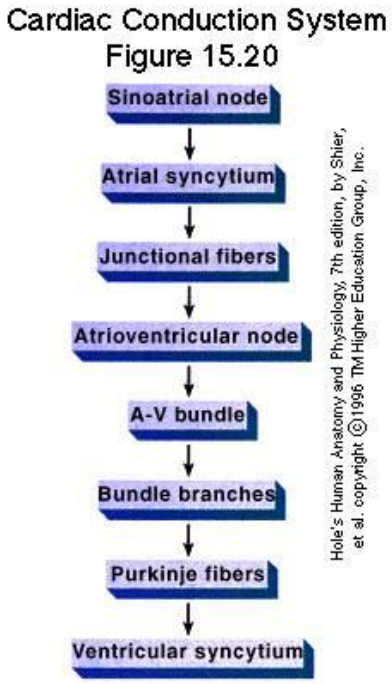


Fig 3.11 Cardiac Conduction system.
Source: http://distance.stcc.edu/AandP/AP/AP2pages/heart/cardiac1.htm

The impulses are then sent down the **atrioventricular bundle**. This bundle of fibres branches off into two bundles and the impulses are carried down the centre of the heart to the left and right ventricles.

At the base of the heart the atrioventricular bundles start to divide further into **Purkinje fibers**. When the impulses reach these fibers they trigger the muscle fibers in the ventricles to contract. The electrical current travels through the two ventricles within 0.06 seconds. The contraction will end up to the top of the ventricles where the blood leaves the ventricles through the pulmonary artery and aorta. The cycle is then repeated continuously [26].

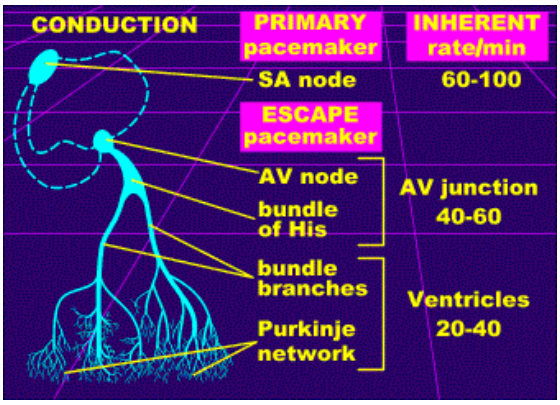


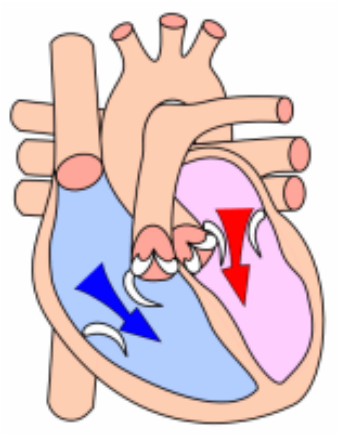
Fig. 3.12Cardiac Conduction

3.11 Cardiac Cycle

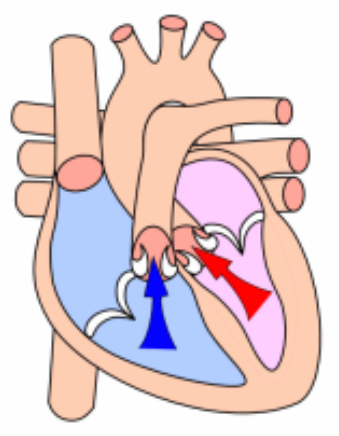
The cardiac cycle refers to the alternating contraction and relaxation of the myocardium in the walls of the heart chambers, coordinated by the conduction system, during one heartbeat. The two stages are:

- Systole (contraction phase of ventricle)
- Diastole (relaxation phase of ventricle)

At a normal heart rate, one cardiac cycle lasts for 0.8 second. In fact cardiac cycle is the sequence of events that occur when the heart beats [27].



Heart Diastole



Heart Systole

Fig 3.13 Cardiac Cycle

During the diastole phase some events occur as below:

- The atria and ventricles are relaxed and the atrioventricular valves are open.

- De-oxygenated blood from the superior and inferior vena cava flows into the right atrium.
- The open atrioventricular valves allow blood to pass through to the ventricles.
- The SA node contracts triggers the atria to contract.
- The right atrium empties its contents into the right ventricle.
- The tricuspid valve prevents the blood from flowing back into the right atrium [28].

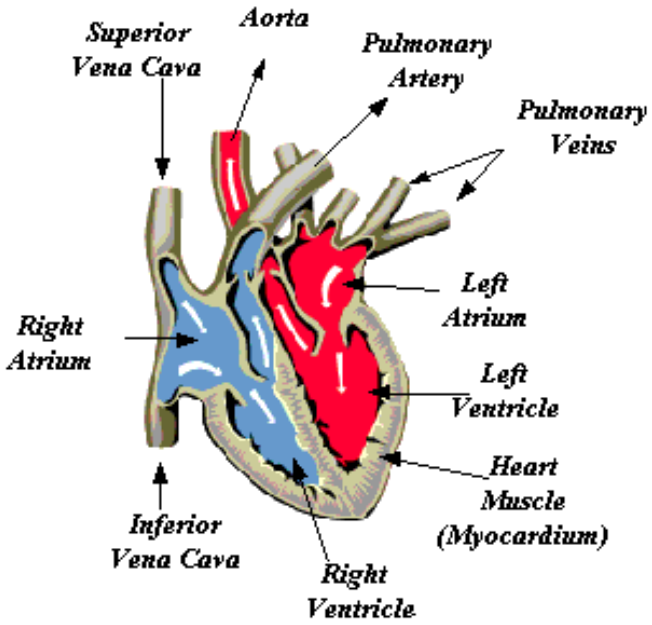


Fig.3.14 Heart Cycle (Systole & Diastole)
http://biology.about.com/library/organs/heart/blcardiaccycle.htm

Also during the Systole phase some events occur as below:

- The right ventricle receives impulses from the Purkinje fibers and contracts.
- The atrioventricular valves close and the semilunar valves open.
- The de-oxygenated blood is pumped into the pulmonary artery.
- The pulmonary valve prevents the blood from flowing back into the right ventricle.
- The pulmonary artery carries the blood to the lungs.

- Blood picks up oxygen and is returned to the left atrium of the heart by the pulmonary veins.

CARDIAC CYCLE	ACTION
Early Diastole	Ventricle relaxes. Semilunar valves close. Atrioventricular valves open. Ventricles fill with blood.
Mid Diastole	Atria and Ventricles are relaxed. Semilunar valves are closed. Atrioventricular valves are open. Ventricles continue to fill with blood.
Late diastole	SA node contracts. Atria contract. Ventricles fill with more blood. Contraction reaches AV node.
Systole	Contraction passes from AV node to Purkinje fibers and ventricular cells. Ventricles contract. Atrioventricular valves open. Semilunar valves open. Blood is pumped from the ventricles to the arteries.

Table 3.3 Cardiac cycles

In the next diastole period:

- The semilunar valves close and the atrioventricular valves open.
- Blood from the pulmonary veins fills the left atrium.
- The SA node contracts again triggers the atria to contract.
- The left atrium empties its contents into the left ventricle.

- The mitral valve prevents the oxygenated blood from flowing back into the left atrium [28].

Also during systole phase:

- The atrioventricular valves close and the semilunar valves open.
- The left ventricle receives impulses from the Purkinje fibers and contracts.
- Oxygenated blood is pumped into the aorta.
- The aortic valve prevents the oxygenated blood from flowing back into the left ventricle [28].

3.12 Sound of the Heart

The sounds associated with the heartbeat are due to vibrations in the tissues and blood caused by closure of the valves. Abnormal heart sounds are called murmurs.

3.13 Heart Rate

The sinoatrial node, acting alone, produces a constant rhythmic heart rate. Regulating factors are reliant on the atrioventricular node to increase or decrease the heart rate to adjust cardiac output to meet the changing needs of the body. Most changes in the heart rate are mediated through the cardiac centre in the medulla oblongata of the brain. The centre has both sympathetic and parasympathetic components that adjust the heart rate to meet the changing needs of the body [21].

Peripheral factors such as emotions, ion concentrations, and body temperature may affect heart rate. These are usually mediated through the cardiac centre.

Chapter 4

Cardiovascular Diseases

Cardiovascular Disease (CVD) includes dysfunctional conditions of the heart, arteries, and veins that supply oxygen to vital life-sustaining areas of the body like the brain, the heart itself, and other vital organs. If oxygen doesn't arrive the tissue or organ will die [25].

In order to diagnosis Heart disease we need to know how the diseases look and how they start. To interpret the Electrocardiography, specialists need to understand different diseases and also how these diseases present themselves in an ECG trace, so by fully understanding diseases ECG (Heart trace) and heart sound specialist feel comfortable to diagnose. This chapter aims to discuss some definitions and diseases.

- **Ischemic Heart Disease:** is the technical term for obstruction of blood flow to the heart. (Excess fat or plaque deposits are narrowing the veins that supply oxygenated blood to the heart).
- **Arteriosclerosis and atherosclerosis**: Excess builds up of fat or plaque are respectively termed arteriosclerosis and atherosclerosis.

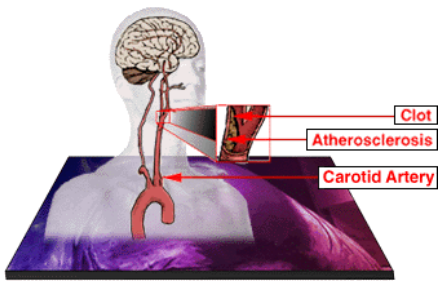


Fig 4.1 atherosclerosis of the cardiac artery
Source:
http://www.healingwithnutrition.com/cdisease/cardiovascular/cardiovascular.html#A1

- **Stroke**: inadequate oxygen flows to the brain.
- **High Blood Pressure (hypertension):** high blood pressure causes excess fat or plaque build up because of the extra effort it takes to circulate blood. The other name of hypertension is "The Silent Killer" because the first warning sign is an angina attack or a deadly heart attack or a stroke. There are many facts causes hypertension such as, disorders (which leave extra fluids, sodium, and toxins in the body), obesity, diabetes, birth control pills, pregnancy, smoking, excess alcohol, stress, and thyroid gland problems [25].

4.1 Irregular heartbeat (Arrhythmia)

The adult heart, beats at a rate of 60-80 beats per minute. Of course during exercise and emotional stress, heart beat faster. Arrhythmia or irregular heartbeat is an abnormality in this rhythm. As mentioned in chapter 3 the heart rhythm controls by groups of cells called Sinoatrial (SA) node.

These cells generate electrical impulses that travel along special muscle cell pathways to the four muscular chambers of the heart. The electrical impulses cause the heart chambers to contract in sequence, forcing the blood to flow in the correct direction [2].

If the heart beats in an irregular rhythm it will not function efficiently. In common the most types of irregular heartbeat are:

- **Premature Ventricular Contraction** (PVCs):

The ventricles contract permanently, before receiving the electrical impulse.

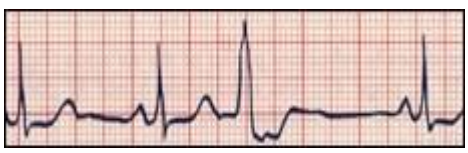


Fig 4.2 PVCs

Source: http://www.txai.org/edu/irregular/premature_beats.htm

- **Premature Atrial Contraction (PACs):**

The atrial contraction permanently, before receiving the electrical impulse.

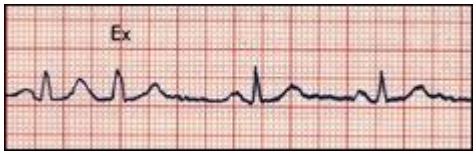


Fig 4.3 PACs
Source:http://www.txai.org/edu/irregular/premature_beats.htm

- **Sick Sinus Syndrome (SSS):**

The Sinoatrial node malfunctions, leading to a slow heart rate, alternating periods of slow and fast heart rate, or the heart may stop for brief periods.

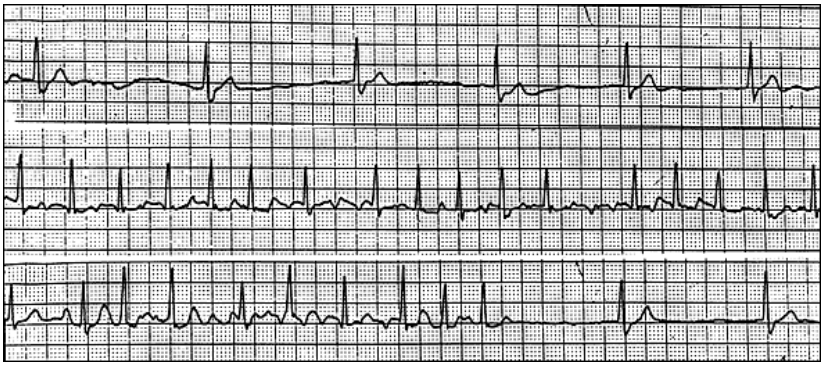


Fig 4.4 Electrocardiogram exhibiting alternating patterns of bradycardia and tachycardia as seen in patients with sick sinus syndrome.
Source: http://www.aafp.org/afp/20030415/1725.html

- **Atrial flutter:**

The atria contract regularly, but too fast (up to 400 bpm). The ventricles are unable to respond, and beat at a much slower rate (100-200 beats).

- **Ventricular flutter:**

Ventricular 'flutter' is a bizarre sine wave like rhythm, and usually degrades into ventricular fibrillation. You won't see it often (or for long) [29].

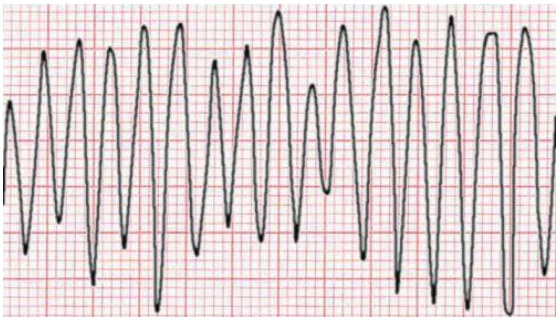


Fig 4.5 Ventricular flutter
Source: http://www.anaesthetist.com/icu/organs/heart/ecg/

- **Atrial fibrillation:**
  The atria beat rapidly (up to 500 bpm) and irregularly, leading to slower, irregular ventricular contractions (around 150 bpm).

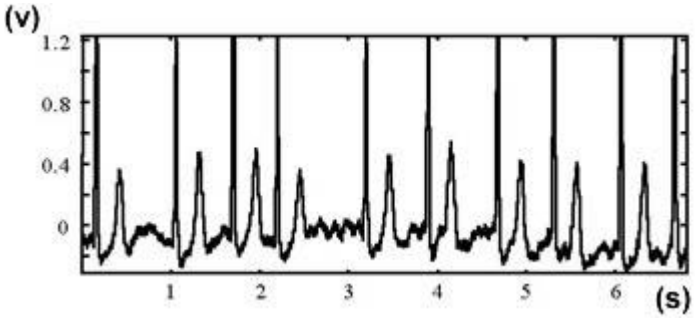
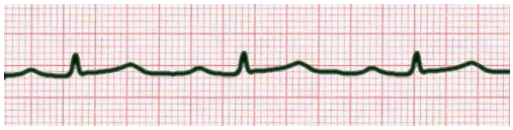
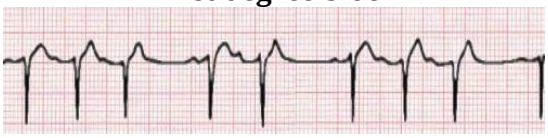


Fig 4.6 Atrial fibrillation
Source: http://www.cardiodigital.com/Research_AF/research_af.htm

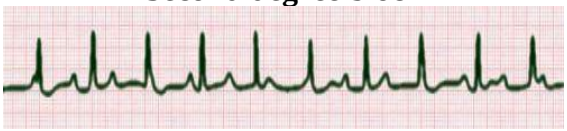
- **Heart block:**
  This is a delay or blockage, to the electrical impulses in the heart muscle, leading to slow and irregular beats. High rates of beating may alternate with low rates.



First degree block



Second degree block



Third degree block

Fig 4.7 Heart block

Source: http://www.anaesthetist.com/icu/organs/heart/ecg/

- **Ventricular fibrillation:**
  Disorganized electrical activity in the heart causes the ventricles to quiver, rather than beating in an effective manner [2].

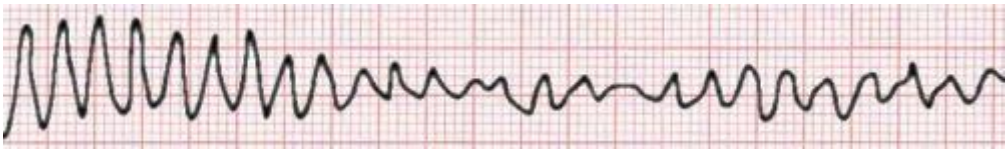


Fig 4.8 Ventricular fibrillation
Source: http://www.anaesthetist.com/icu/organs/heart/ecg/

4.2 What is a Sinus rhythm?

Sinus rhythms are a class of rhythms, which originate at the SA node. Sinus rhythms generally travel through the entire conduction system without inhibition.

- **Sinus Bradycardia** (Slow heart rate):

  If the heart rate is below 60 beats a minute but the rest is the same it is a Sinus Bradycardia. P-R interval is between 0.12- 0.20seconds. QRS duration is between 0.04-0.12 seconds.

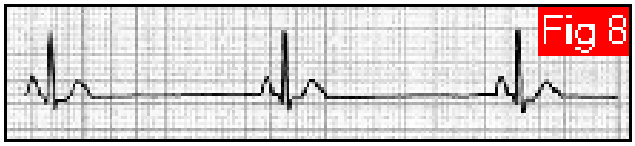


Fig 4.9 Sinus Bradycardia
Source: http://www.nda.ox.ac.uk/wfsa/html/u11/u1105_02.htm

- **Sinus Tachycardia** (Fast heart rate):

  When the heart rate is between 100-150 beats a minute with the same intervals. P-R interval is between 0.12- 0.20seconds. QRS duration is between 0.04-0.12 seconds.

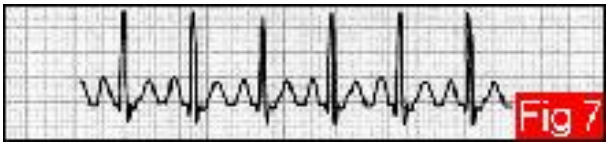


Fig 4.10 Sinus Tachycardia
Source: http://www.nda.ox.ac.uk/wfsa/html/u11/u1105_02.htm

- **Sinus Arrhythmia**

There is normally a slight degree of chaotic variation in heart rate, called sinus Arrhythmia [14]. This is irregular spacing of normal complexes associated with respiration. The R-R interval will change in each beat, as P-R is constant. Scientist believes this is normal finding particularly in young individuals.

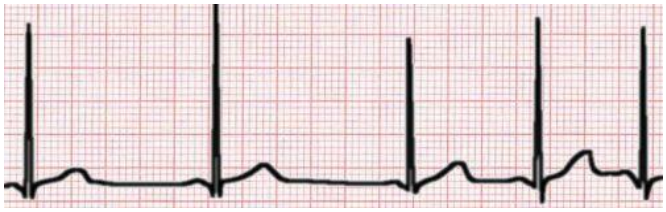


Fig 4.11 Sinus Arrhythmia
Source: http://www.anaesthetist.com/icu/organs/heart/ecg/

	P-R interval	QRS Duration	Rate (beats per minute)
Normal Sinus Rhythm	0.12- 0.20	0.04-0.12	60-100 (Normal)
Sinus Bradycardia	0.12- 0.20	0.04-0.12	Less than 60 (Slow heart rate)
Sinus Tachycardia	0.12- 0.20	0.04-0.12	100-150 (Fast heart Rate)
Sinus Arrhythmia	0.12- 0.20	0.04-0.12	60-100 (regular rhythm with periodic irregularity)

Table 4.1-Sinus rhythm

4.3 Myocarditis

Myocarditis is inflammation of the myocardium (heart muscle). It is often accompanied by pericarditis, inflammation of the pericardium (the membrane surrounding the heart) [2]. Viral infection causes Myocarditis due to the Coxsackie A or B virus. This disease usually accompanies viral infection of the lungs.

Another cause of Myocarditis is Enterovirus. This virus grows up in intestines. Infections with a bacterium, fungus or parasite are some more reason to get Myocarditis. In fact acute rheumatic fever will cause Myocarditis. This disease may be due to sensitivity to certain drugs such as Methyldopa, Penicillin, or Tetracycline.

Myocarditis Symptoms:

- History of preceding viral illness
- Fever
- Chest pain that may resemble a heart attack
- Joint pain/swelling
- Abnormal heart beats
- Fatigue
- Shortness of breath
- Leg edema
- Inability to lie flat

A physical examination may detect a rapid heartbeat (tachycardia) or abnormal heart beats, abnormal heart sounds (murmurs, extra heart sounds), fluid in the lungs and fluid in the skin of the legs. In addition, other signs suggestive of an infection may be present: fever, rashes, red throat, itchy eyes, and swollen joints [30].

Tests used in the diagnosis of Myocarditis are:

- Electrocardiogram (ECG)
- Chest X-Ray
- Ultrasound of the heart (echocardiogram) -- may show weak heart muscle, an enlarged heart, or fluid surrounding the heart.
- White blood cell count
- Red blood cell count
- Blood cultures for infection

- Blood tests for antibodies against the heart muscle and the body itself
- Heart muscle biopsy - rarely performed [30].

ECG is the main method to diagnosis Myocarditis. ECG produces a record of the electrical impulses that immediately precede each contraction of the heart muscle; it is used to help diagnosis disorders of the heart, which produce deviation from the normal electrical pattern. To help confirm the diagnosis, ECG will show up the characteristic electrical abnormalities associated with the generation of the heartbeat.

There is no specific treatment for myocarditis. Bed rest is usually recommended and exercise should be avoided until ECG shows a normal pattern of electrical activity in the heart.
Most of the suffer causes by myocarditis will recover after few weeks (of course it is depend upon how badly the heart muscle is affected). Surgery may be necessary if severe damage has occurred to the pericardium.

4.4 Heart Attack (Myocardiac infarction)

Heart attack also known as coronary thrombosis occurs when the blood supply to the heart muscle is reduced or completely blocked due to obstruction in one or more of the coronary arteries that encircle the heart [4]. It is very common in the UK as a quarter of a million people suffer each year from heart attack and one third of these die. The main symptom is pain located in the centre of the chest, or spreading in to the neck, jaw or shoulders, or down the arms.

4.4.1 What causes a heart attack and how is it diagnosed?

Formation of blood cloth in a narrowed coronary artery is one of the reasons that cause heart attack. Atherosclerosis is the other important cause of heart attack. A mild heart attack may last less than an hour and cause little damage to the muscle. Diagnosis of a heart attack is based on the description of the chest pain as mentioned

earlier. The diagnosis will be confirmed by an electrocardiogram (ECG) test, which measures the electrical activity inside the heart muscle.

4.4.2 Factors contribute to a heart attack

Some factors increase the risk of heart attack as below:

- Age
- Smocking
- High blood cholesterol
- Gender
- High blood pressure
- Diabetes
- Family history
- Obesity

The most important steps to reduce heart attack are to stop smoking and follow a low fat, high fibre diet. Regular exercise and keep your ideal weight can help to prevent heart attack.

4.5 Arteriosclerosis (Atherosclerosis)

It is also known as Artery Hardening. It is a term to use to describe a group of disorders involving the gradual thickening and loss of elasticity of the artery walls. Atherosclerosis causes the blood vessels to become narrowed by a build up of fatty deposits [4].

Various risk factors that make the development of atherosclerosis more likely included:

- A high blood pressure (result of eating to much saturated fat)
- Smocking
- Lack of sufficient physical activity
- Excessive alcohol
- Family history
- Diabetes

The most popular diagnosis of arteriosclerosis is to get a good X-ray. Some medication helps to reduce blood cholesterol to increase blood flow in the blood

vessels. Artery hardening is very dangerous. It is a major factor in heart attacks, strokes, kidney disease and other circulatory disorders.

4.6 Infection of the heart

Infection of the heart, Carditis and Endocarditis, is an additional complication that can occur as a result of a weak immune system, liver problems, heart surgery, or from an autoimmune disorder like rheumatic fever. Endocarditis is quite common in persons with compromised immune systems from HIV or AIDS. If not appropriately handled, permanent heart muscle damage can occur from the infection [31].

Chapter 5

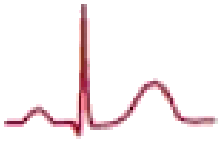
THE ELECTROCARDIOGRAM

The basic of electrocardiography is based on flowing ions between inner cell and outer cell of the cardiac cells. In fact impulse travels along the cardiac muscle fibres and then electric current is generated by the flowing ions. As mentioned in chapter 3 there is a fluids around the heart and the electric current generated by ions flows into the fluid around the heart, and a minute portion actually flows to the surface of the body. An Electrocardiogram is a record of this electrical activity, generated by the heart muscle during a heartbeat. An encyclopaedia definition for an ECG is:

"A test that records the electrical activity of the heart. The ECG is used to measure the rate and regularity of beats as well as the size and position of the chambers, any damage to the heart, and effects of drugs or devices to regulate the heart (pacemaker) [35]."

Source: http://health.yahoo.com/health/encyclopedia/003868/0.html

As mentioned in history of the ECG, the basic of the ECG and electrical heart activity were first introduced in the in the year 1600. The tracing is recorded with an



electrocardiograph and includes the rate and regularities of beats as well as the size and position of the chambers [5].

Diagnosis and predetermination of cardiac problems:

To form a diagnostic network, transmitting ECG's to a central computer and diagnostic centres is necessary. Electrodes are play an important role in surgical recovery and intensive care can monitor these bioelectric functions.
Usually by monitoring a normal ECG physicians are able to predict heart problems before the symptoms become apparent.
When atria and ventricles of the normal heart start to contract, typical peaks on the ECG paper shows physicians the normality of individual. Any difference on an ECG trace shows a disorder. An ECG can be used to determine hundreds of abnormalities that will be discussed later in chapter 8.

5.1 Recording of the ECG

By putting electrodes to different part of the body set up an electrocardiogram. To get a clear ECG, the subject needs to be relaxed. Lying down is the best position to record a good ECG without any interference. However, if the patient is tense then an irregular baseline is observed as a result of electrical interferences.

Attaching electrodes to each limb and to the chest all of which are connected to a central computer enables an ECG to be performed. To record a good ECG without any interference the areas that electrodes need to attach must be clean. Hairs need to be shaved so the electrodes are not affected and has good contact with the skin. Rubber straps, suction cups, or sticky pads help to attach electrodes on the skin to prevent any movement. Special cream or gel also helps to achieve good electrical contact to record the tiny signals generated by the heart.

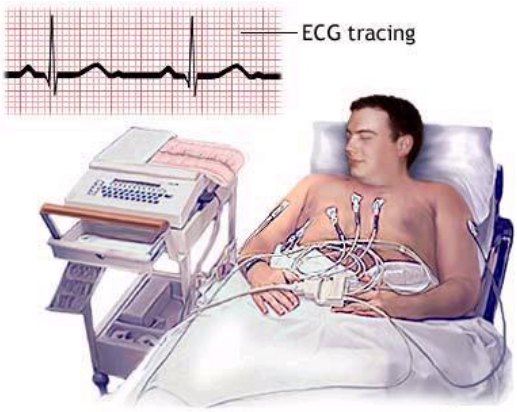
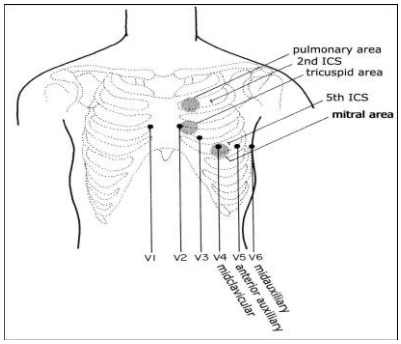


Figure 5.1 Example of ECG
Source: http://www.nlm.nih.gov/medlineplus/ency/imagepages/1135.htm

A number of leads are attached to the body in any standard ECG test. These leads are usually different from 12-15 in diagnostic purpose or 3-5 in ECG monitoring. Minimum movement of the chest and limbs help to get a real ECG and sometimes to reach this aim technicians ask patients to hold their breath for short period of time. A reason for keeping the person still is that during ECG test, any factors that may cause artifact or interference are kept constant.



By attaching electrodes on the surface of the body help physicians to look at the heart from different directions so it is essential that those electrodes connected properly to the skin surface otherwise wrong diagnosis will be made.

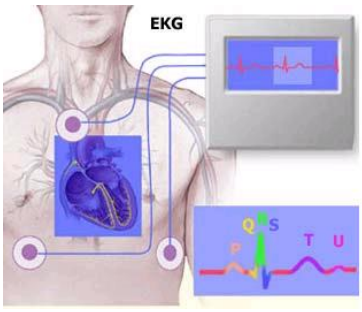


Figure 5.2 Detection of signals from ECG

5.2 Limb Leads (Bipolar)

The bipolar limb leads record the voltage between electrodes placed on the wrists and legs. Bipolar recording is represented by standard limb lead configuration.

Fig 5.3 Bipolar leads
Source: http://www.anaesthetist.com/icu/organs/heart/ecg/

Lead I has the positive electrode on the left arm, and the negative electrode on the right arm. This therefore measures the potential difference between the two arms. In this and the other two limb leads, an electrode on the right leg serves as a reference electrode for recording purposes. In the lead II configuration, the positive electrode is on the left leg and the negative electrode is on the right arm. Thus measures electricity moving down and leftward. Lead III has the positive electrode on the left leg and the negative electrode on the left arm and best measures electrical activity moving down and rightward. These three bipolar limb leads roughly form an equilateral triangle (with the heart at the centre) that is called **Einthoven's triangle** [5].

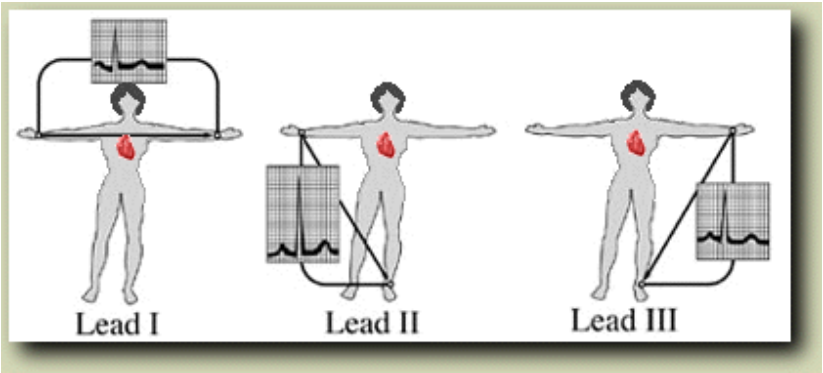


Figure 5.4 ECG Set-up
Source: *http://biomedia.bio.purdue.edu/GenBioLM/GBECG/html/std_limb_leads.html*

It is not make any difference in the recording if the limb leads are attached to the end of the limb or at the origin of the limb because the limb can simply be viewed as a long wire conductor originating from a point on the trunk of the body. If the three limbs of Einthoven's triangle (assumed to be equilateral) are broken apart, then the positive electrode for each Lead is as below:

LEAD Number	Degree relative to the heart
Lead I	Zero degrees
Lead II	+60°
Lead III	120°

Table 5.1 Axial reference systems

This new construction of the electrical axis is called the axial reference system. With this system, a wave of depolarisation travelling at +60° will produce the greatest positive deflection in Lead II. A wave of depolarisation oriented +90° relative to the heart will produce equally positive deflections in both Lead II and III. In this latter case, Lead I will show no net deflection because the wave of depolarisation is heading perpendicular to the 0°, or Lead I axis [5].

5.3 Augmented Limb Leads

There are another three leads called Augmented Unipolar Limb leads. The reason that they called Unipolar is a single positive electrode that is referenced against a combination of the other limb electrodes.

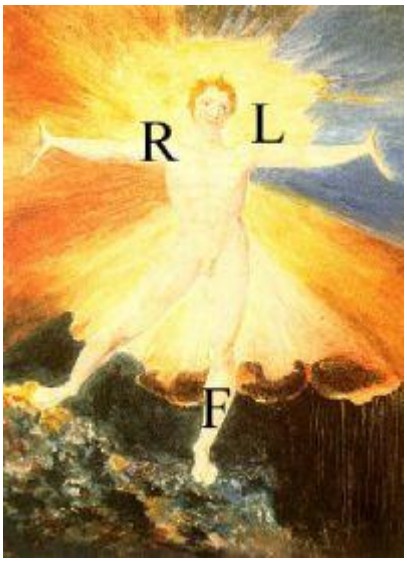


Fig 5.5 Unipolar leads
Source: http://www.anaesthetist.com/icu/organs/heart/ecg/

The positive electrodes for these augmented leads are located on the left arm (aVL), the right arm (aVR), and the left leg (aVf). In practice, these are the same electrodes used for Leads I, II and III.

LEAD Number	Degree relative to the Lead I axis
Lead I (aVL)	–30°
Lead II (aVR)	–150°
Lead III (aVF)	+90°

Table 5.2 Axial reference systems

Three unipolar and three bipolar leads are making six limb leads of the ECG. These leads record electrical activity along a single plane, termed the frontal plane relative to the heart. Using the axial reference system and these six leads, it is rather simple to define the direction of an electrical vector at any given instant in time.

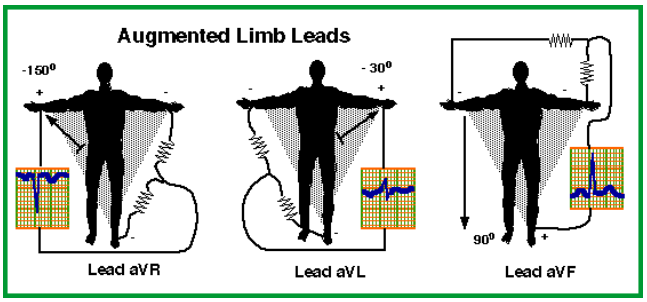


Figure 5.6 Augmented limb leads
Source: ***http://www.coheadquarters.com/PennLibr/MyPhysiology/lect0/figecg12.htm***

If a wave of depolarisation is spreading from right-to-left along the 0° axis, then Lead I will show the greatest positive amplitude. Likewise, if the direction of the electrical vector for depolarisation is directed towards (90°), then aVF will show the greatest positive deflection. If a wave of depolarisation is moving from left-to-right at +150°, then aVL will show the greatest negative deflection.

5.4 Precordial Chest Leads

The V leads, which extend across the Precordium, V1 in the fourth right interspaced, V2 4th left, V4 at the apex (5th interspaced, Midclavicular line), V3 halfway in between V2 and V4, and V5 & V6 in the 5th interspace at the anterior and mid axillary lines respectively [35].

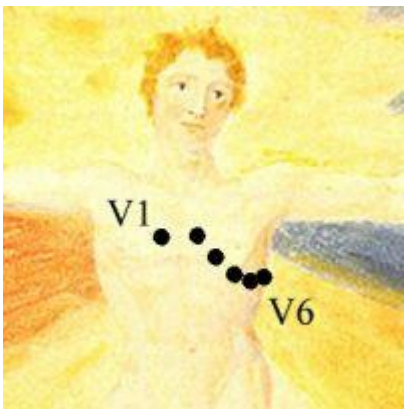


Fig 5.7 Unipolar leads
Source: **http://www.anaesthetist.com/icu/organs/heart/ecg/**

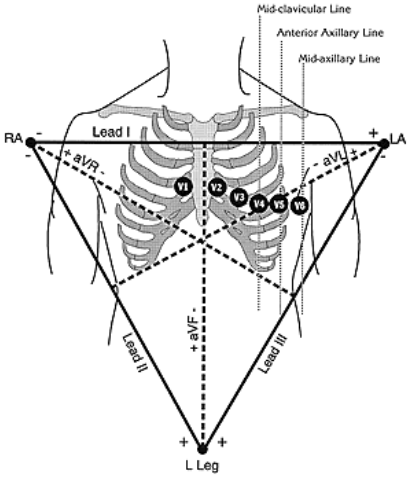


Figure 5.8 Precordial Chest leads

Source: ***http://www.nursingceu.com/NCEU/courses/ecg/ecg_outline/Lesson1/index.htm***

Precordial Chest Leads	POSITION
V1	Fourth intercostals, right strernal border
V2	Fourth intercostals, left strernal border
V3	Equal distance between V2 & V4
V4	Fifth intercostals, left mid clavicular line
V5	Anterior axillary line, same level as V4
V6	Mid-axillary line, horizontal to V5

Table 5.3 Precordial Chest Leads positions

5.5 ECG Paper

ECG paper is traditionally divided into 1mm squares. Vertically, ten blocks usually correspond to 1 mV, and on the horizontal axis, the paper speed is usually 25mm/s, so one block is 0.04s (or 40ms). Note that we also have "big blocks" which are 5mm on their side. Small Square is equal to 0.04 and large square is equal to 0.2.

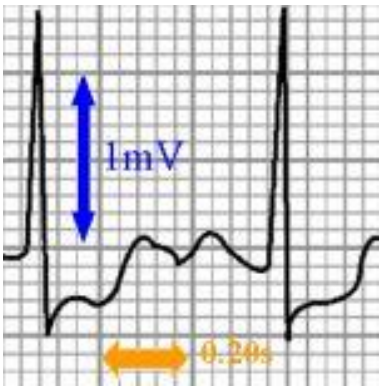


Fig 5.9 ECG paper

Source: http://www.anaesthetist.com/icu/organs/heart/ecg/

5.6 Elements of the ECG

ECG machine detect small voltage changes in the heart, which is in millivolts. The size of the wave depends on the amount of voltage generated by the heart. It is obvious that the greater the voltage make the larger the wave.

ECG consists of a series of waves. These waves are starting from a baseline on the ECG paper called Isoelectric baseline. These are P, Q, R, S and T waves.

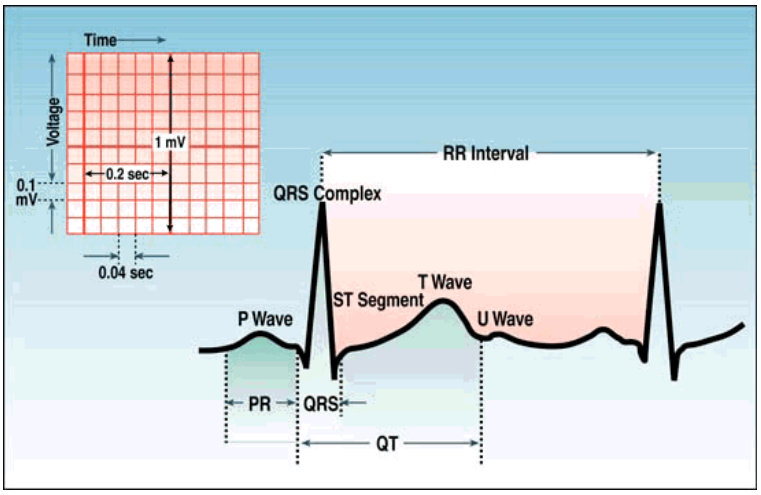


Figure 5.10 Elements of the ECG

Source: ***http://medlib.med.utah.edu/kw/ecg/ecg_outline/Lesson2/index.html***

5.7 The Baseline or Isoelectric line

This is a straight line on the ECG paper where the positive and negative electrical charges are balanced; as a result there is no deflection.

5.8 Waveforms

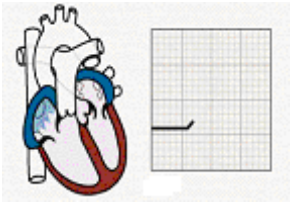
The electrical activity that generates the depolarisation and repolarisation of the atria ventricles is represented by waveforms. If the electrical current is moving towards a lead then a positive deflection is obtained and this is indicated by an upward deflection, above the isoelectric line. If the electrical current is flowing away from the lead then a negative deflection is obtained and is indicated by a downward deflection, below the isoelectric line [5].

WAVE	Represent	Duration
The P wave	Depolarisation of the atria i.e. sequentially depolarising right and left atrial muscles causing contraction of atrial muscles	About 0.1 seconds
The QRS complex	Depolarisation of the left and right ventricles	About 0.08 to 0.12 seconds
The T wave	Repolarisation of the ventricles	About 0.27 seconds
The P-R interval	This is the time taken for an electrical impulse to travel from the atrial nodes (SA Node) to the Purkyne fibres	About 0.12 to 0.20 seconds
The Q-T interval	Ventricular systole	Approximately 0.3 seconds

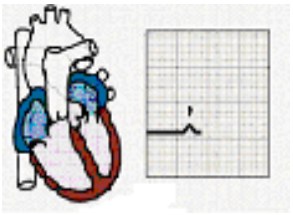
Table 5.4 Different waves of the ECG

5.9 Chronological Activation

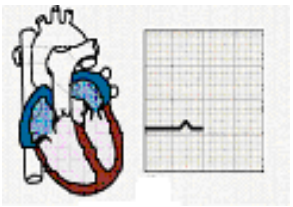
The first part of the P wave is obtained when the sinus impulse activates the right atrium.



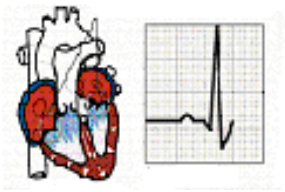
Complete P wave, the left atrium and AV node have been activated.



The His-Purkinje system is activated at the beginning of the ventricular complex.



The activation of the walls of the heart is reflected by the ventricular complex (QRS) with the larger left ventricle dominating.



The ST segment and the T wave indicate the electrical current generated during the repolarisation of the ventricles

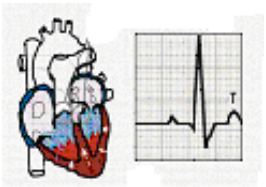


Figure 5.11 Electrical activation of the heart
Source: ***http://www.cardioliving.com/consumer/Heart/Electrocardiogram.shtm***

5.10 Heart Rate

The heart rate is the number of the heartbeat that takes place over one minute.

The easiest way to measure heart rate is by dividing the duration between two identical points of consecutive waveforms (e.g. R-R duration) by 60.

$$HEART\,RATE = \frac{60}{R - R duration}$$

An alternative method can estimate the rate by counting the large squares between two identical points and dividing it by 300

$$\text{Rate} = \frac{Counting.the.L\arg e.squares.between.two.identical.point s}{300}$$

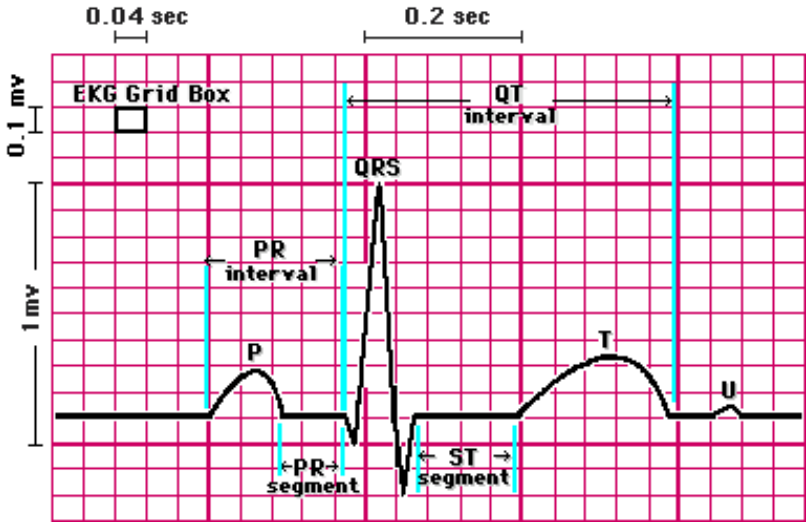


Figure 5.12 Intervals, segments and box sizes
Source: ***http://cal.nbc.upenn.edu/lgcardiac/ecg_tutorial/heartrate.htm***

5.11 Rhythm

"The term rhythm refers to the regularity of a pattern, which in this case is the regularity of the cardiac cycle illustrated along a strip of ECG paper." [8]

To understand the rhythm it is necessary to measure the distance between two complexes then compare the measurement with the next complex (e.g. PP interval

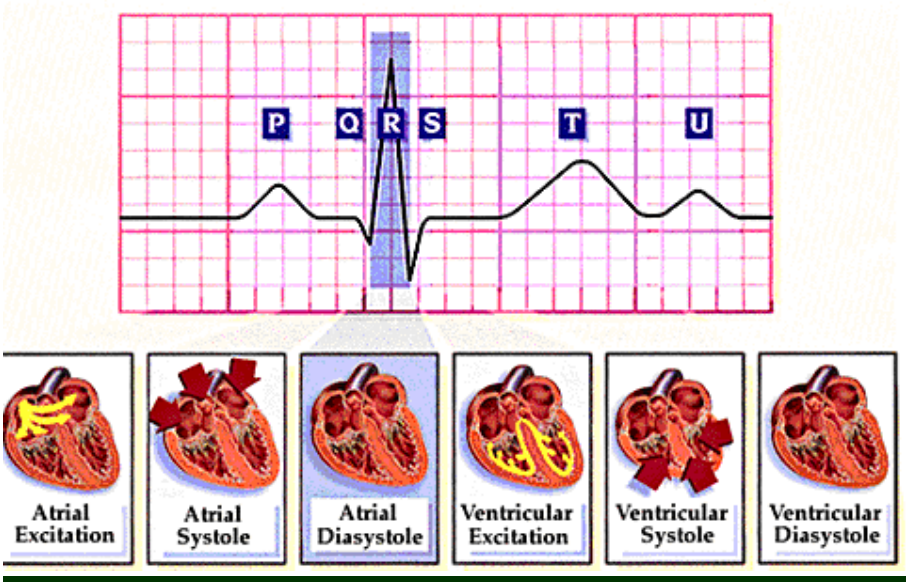
compared with RR interval). Coherent measurements indicate a normal rhythm whereas incoherent measurements indicate an irregular or abnormal rhythm.

Chapter 6

Experimental ECG Procedures, Results & Discussion Using BIOPAC during This Work

The following points are the experimental objective of this practical experiment:

1. To become familiar with the electrocardiograph as a primary tool for evaluating electrical events within the heart.
2. To correlate electrical events as displayed on the ECG with the mechanical events that occur during the cardiac cycle.
3. To observe rate and rhythm changes in the ECG associated with body position and breathing.



Just as the electrical activity of the pacemaker is communicated to the cardiac muscle, "echoes" of the depolarization and re-polarization of the heart are sent through the rest of the body. By placing a pair of very sensitive receivers (electrodes) on other parts of the body, the echoes of the heart's electrical activity can be detected. The record of the electrical signal is called an electrocardiogram (ECG). You can infer the heart's mechanical activity from the ECG [32].

6.1 Components of the ECG

The electrical events of the heart are recorded on the ECG as a pattern of a baseline broken by a P wave, a QRS complex, and a T wave.

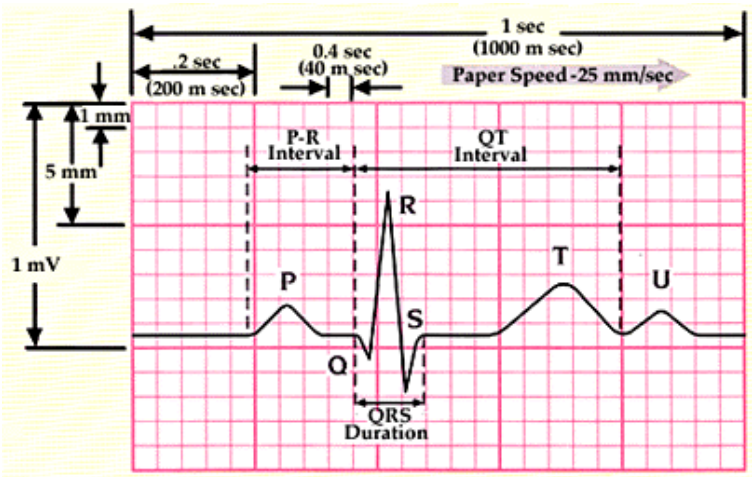


Fig 6.1 component of the ECG

- Isoelectric is the baseline of ECG with a straight line. It is the point of departure for the electrical activity of depolarizations and repolarizations of the cardiac cycles.
- The P wave results from atrial depolarization.
- The QRS complex is a result of ventricular depolarization and indicates the start of ventricular contraction.
- The T wave results from ventricular repolarization and signals the beginning of ventricular relaxation.
- The larger QRS complex marks the electrical signal for atrial repolarization.

6.2 Intervals and Segments

In addition to the wave components of the ECG, there are intervals and segments.

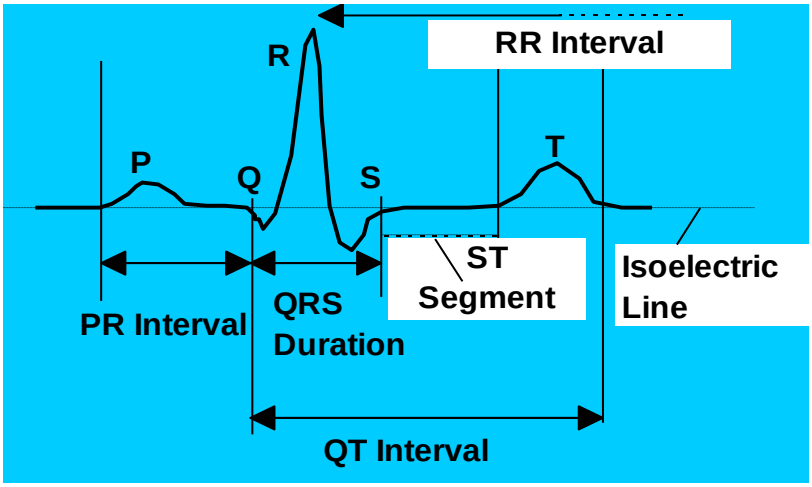


Fig 6.2 component of the ECG

An **interval** is part of the ECG containing at least one wave and a straight line. For example, the PR interval includes the P wave and the connecting line before the QRS complex. The PR interval represents the time it takes for the impulse sent from the SA node to the ventricles. **Segments** only refer to a period of time from the end of one wave to the beginning of the next wave. For example, the PR segment represents the time of AV nodal and transmission to the ventricles. Because the ECG reflects the electrical activity, it is a useful "picture" of heart activity. If there are interruptions of the electrical signal generation or transmission, the ECG will change. These changes can be useful in diagnosing changes within the heart.

6.3 What is an ECG lead used for?

The particular arrangement of two electrodes (one positive, one negative) with respect to a third electrode (the ground) is called a lead. The positions of electrodes for the different leads have been standardized.

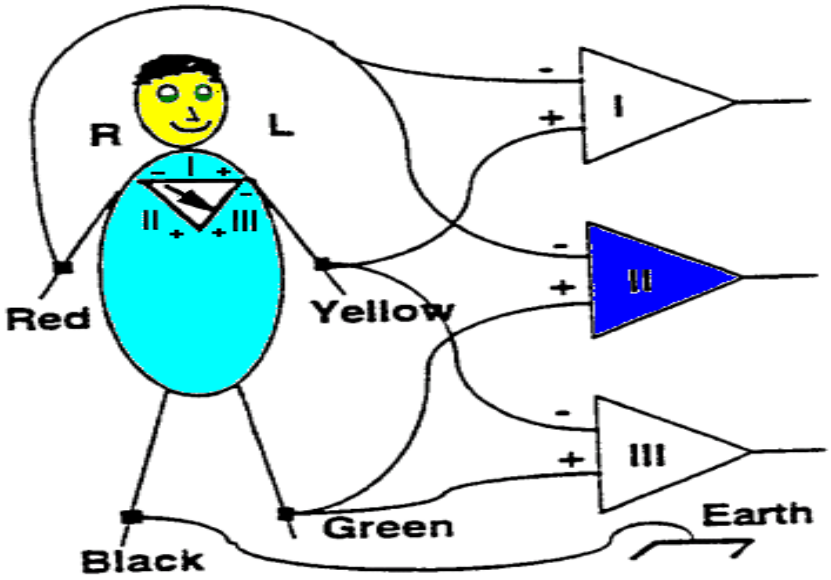
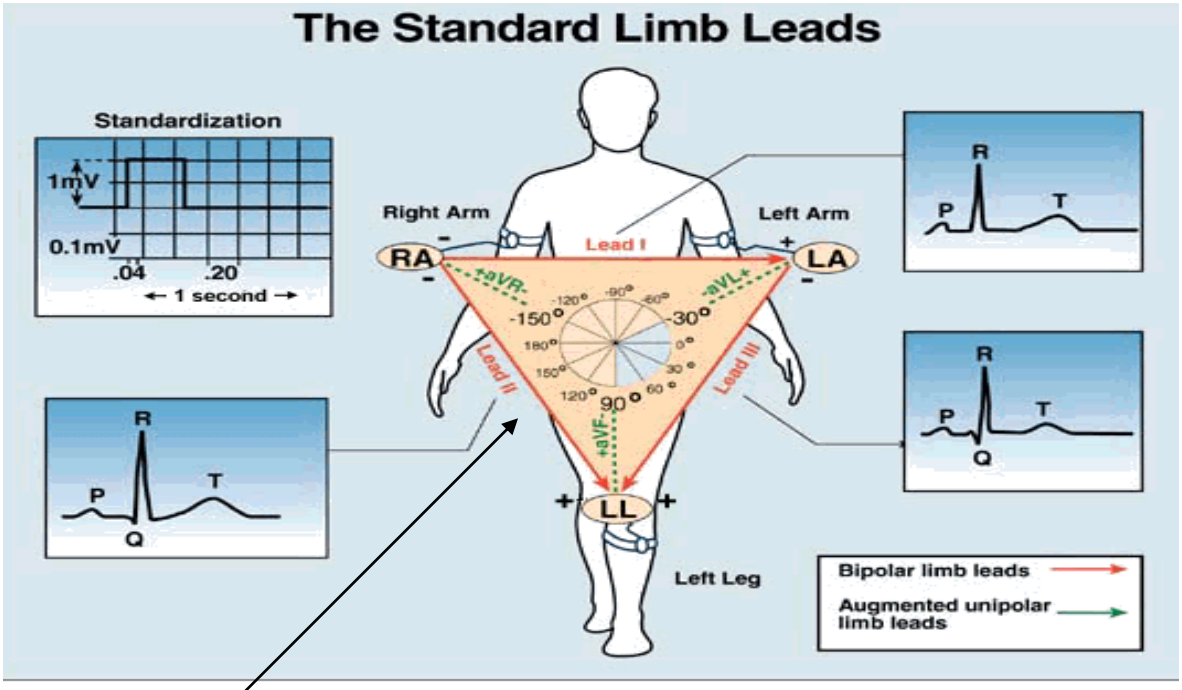


Fig.6.3 attach ECG Electrode to the body

By using Lead **II** this experiment will be carried out which has a positive electrode on the left ankle, a negative electrode on the right wrist, and the ground electrode on the right ankle. In this experiment ECG is being recorded under four conditions. Because ECGs are widely used, basic elements have been standardized to simplify reading ECGs. ECGs have standardized grids of lighter, smaller squares and, superimposed on the first grid, a second grid of darker and larger squares. The smaller grid always has time units of 0.04 seconds on the x-axis and the darker vertical lines are spaced 0.2 seconds apart. The horizontal lines represent amplitude in mV. The lighter horizontal lines are 0.1mV apart and the darker grid lines represent 0.5mV.



Typical Lead II values are given in table below.

Fig 6.4 the standard limb leads

Normal Lead II ECG Values

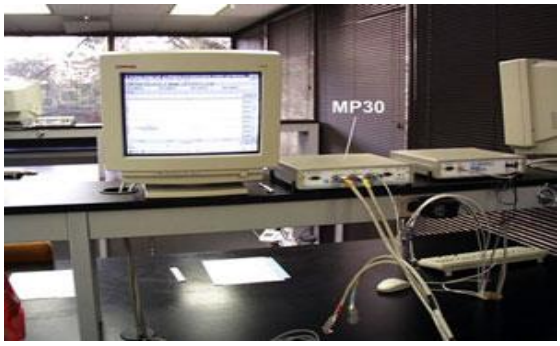
PHASE	DURATION (Second)	AMPLITUDE (millivolt)
P wave	0.06-0.11	< 0.25
P-R interval	0.12-0.20	----------
P-R segment	0.08	----------
QRS complex (R)	< 0.12	0.8-1.2
S-T segment	0.12	----------
Q-T interval	0.36-0.44	----------
T wave	0.16	< 0.5

Table 6.1 Normal Lead II ECG Values

The average resting heart rate for adults is approximately 70 beats/min. Slower heart rates are typically found in individuals who regularly exercise. Athletes are able to pump enough blood to meet the demands of the body with resting heart rates as low as 50 beats/min. Athletes tend to develop larger hearts, especially the muscle in the left ventricle; a condition known as "left ventricular hypertrophy." Because of their larger and more efficient hearts, athletes also exhibit other differences in their ECGs. For instance, low heart rate and hypertrophy exhibited in sedentary individuals can be an indication of failing hearts but these changes are "normal" for well-trained athletes [32].

6.4 MATERIALS

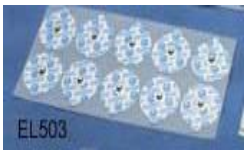
BIOPAC Materials



1. Computer system: PC running Windows95® or Macintosh ®_ minimum 68020
2. BIOPAC acquisition unit (MP30)
3. BIOPAC student *PRO* software (V.3.6.2)
4. Tape (To fix the electrode on the skin with out any movement).

5. BIOPAC electrode gel (GEL1) and abrasive pad (ELPAD) or skin cleanser or alcohol prep

6. BIOPAC electrode lead set (SS2L)



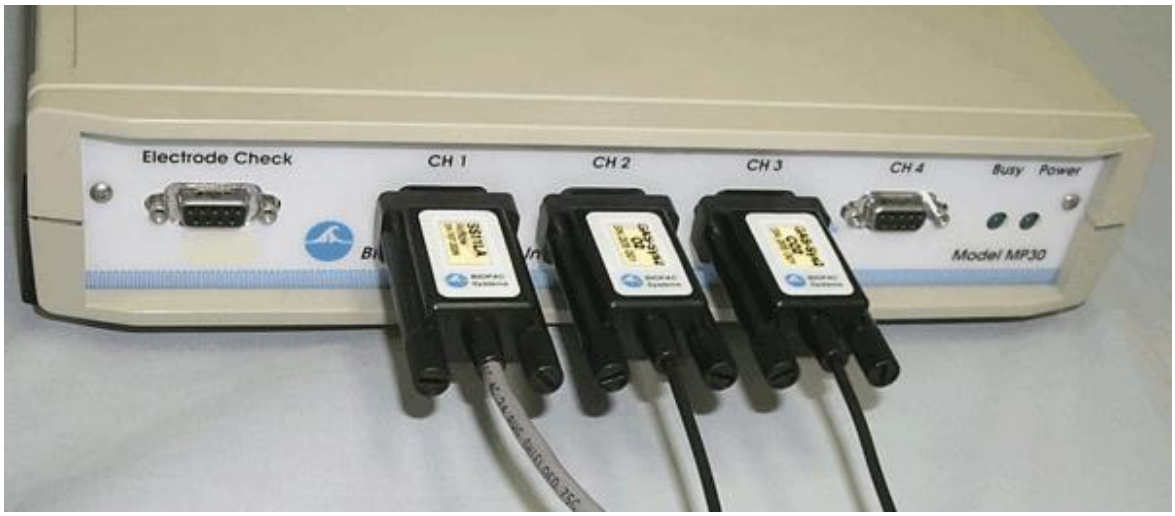
7. BIOPAC disposable vinyl electrodes (EL503), 3 electrodes per subject
8. Step Box (Stepping in place exercise)
9. Cot or lab table and pillow
10. Memory requirements: The Biopac student Lab application needs to have at least 4MB of RAM available for its needs. This is 4MB above and beyond the operating system needs and any other programs that are running.
11. BIOPAC wall transformer (AC100A)
12. BIOPAC serial cable (CBLSERA)

6.5 Detailed Procedure [References 32]

Step1. Three Channels used for this experiment.

- Plug the transducers into the MP30 as follows:

Transducer	MP30 CH NUMBER
Electrode lead set (SS2L)	CH2



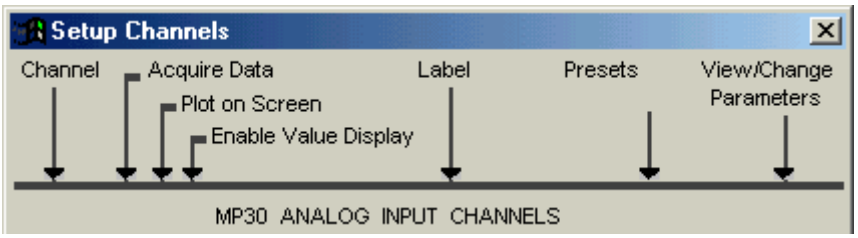
Step 2.

- Turn the computer ON.

Step3.

- Select Student Lab PRO V.3.6.2
- Select MP30 -Setup Channels

Channel 2 – (0.05-150HZ)



ECG

- Set up calculation channels (ECG heart rate) (Filtered ECG)
- Set up parameters

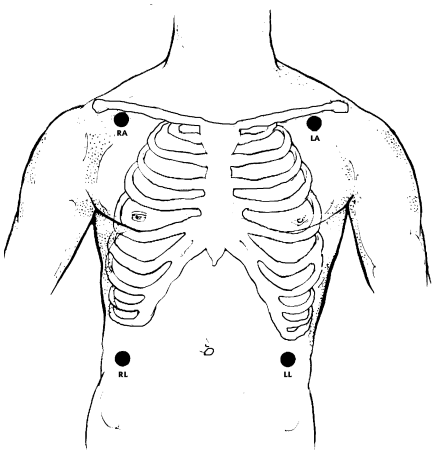
Step 4.

- Electrode Placement, as shown in the diagram, trace the

approximate position for the RA, RL, and LL electrodes on your own chest.

- Attach each wire from the BIOPAC lead set (SS2L) to the electrodes as follows:

WIRE COLOUR	POSITION
WHITE	**RA electrode**
RED	**LL electrode**
BLACK	**RL electrode**



Step 5.

- Check to make sure electrodes securely to the skin.
  If they are being pulled up, you will not get a good ECG signal.

Step 6.

- Go to MP30 and Set-up Acquisition (300 second) 5 minutes.
- Record continuously through the rest and exercise.
- Recorder should insert marker (PC = F9), at beginning of exercise, if subject begins sweat, when subject stop exercising. Keep recording while subject is recovering from exercise.
- Recording will stop automatically after 300 seconds.

Step 7.

- Before start recording the subject must be relaxed, breathing normally.
- The recorder should instruct the subject, follow the point's bellow after recording starts:

1. Lay down for 20 seconds.
2. Sit down relax for another 20 seconds.

3. Breath deep (in and out) by each breath recorder need to insert marker (F9) to show which ECG component is inhale or exhale. This also takes 20 seconds.
4. Exercise for 2 minutes (120 seconds), (By using step box or cycle)
5. Stop exercise after 120 seconds of recording
6. Subject should sit down and relax for the rest of recording

Step 8.

- Press the START button in the *PRO* software.
- Press SUSPEND bottom if you want to stop the recording due to electrode peeled up or too much muscle (EMG) artifact.
- If you are not happy with the graph you can press REDO bottom and start recording again and delete the previous one.
- RESUME bottom let us to carry on recording from the place with suspend the recording.
- Remember you can mark the graph as where as you like just by pressing F9 on the keyboard (it is really useful for analysing inhale and exhale).

Step 9.

- In order to save the file, insert a formatted diskette into drive A. Go to File, select Save AS and then save your file.

Step 10.

- **Data Analysis**

1. Open your saved file. Maximize the screen if required.

2. If needed and if you wish, you may turn the grid on or off through clicking on the icon present to the left of the sigma (show/hide measurements) icon on the top tool bar. Usually clinical grid size is 0.2 horizontal scales and 0.5 vertical scales.

3. Go to the start of the recording by moving the horizontal scroll box to the extreme left site.

4. Click on the I-beam tool located in the lower right hand corner of the display. Move the I-beam cursor to that area of the waveform you wish to begin an interval or amplitude measurement. Click, hold, and drag the pointer over this interval. Doing so will create a highlighted vertical strip on the display. The width of this strip – in seconds – will appear in the 'delta t' box located at the top of the screen. (Click on the Show/Hide Measurements icon if this measurements bar is not present). Notice that if you select an interval between two ECG R-waves, heart rate, in beats per minute (bpm) is also displayed in an additional measurements box. At the end, change the right-most measurement box to 'mean'. This will display the average voltage associated with the highlighted waveform.
5. Once you become comfortable in extracting measurements from the ECG proceed to take the Results necessary where the measurements should be obtained.

    1. Q-T interval (Corresponds to Ventricular Systole)
    2. End of T wave to subsequent R wave (Corresponds to Ventricular Diastole)
    3. BPM and ΔT

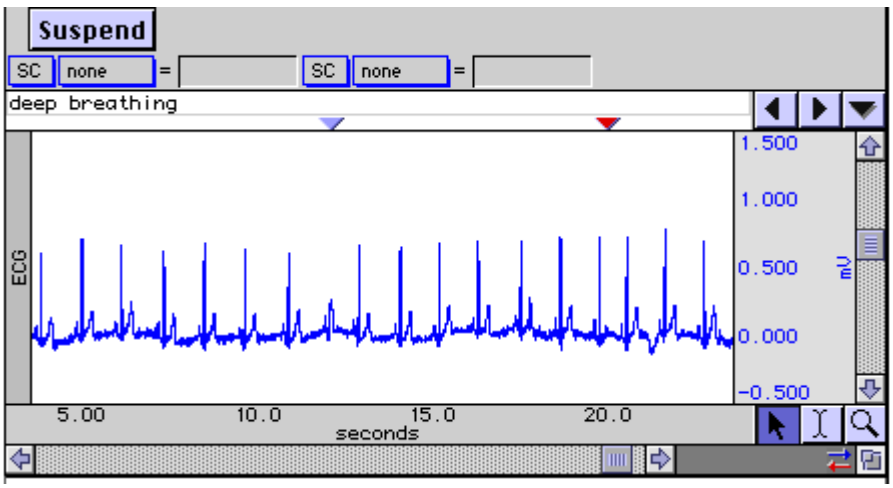
- Take same measurements for before, during and after exercise.

Step 11.

- Print the graphs
- Interpret the results and draw conclusion

End.

Accessories used:



KEY FEATURES:

The display uses classic, clinical grid markings for quick and easy evaluations.

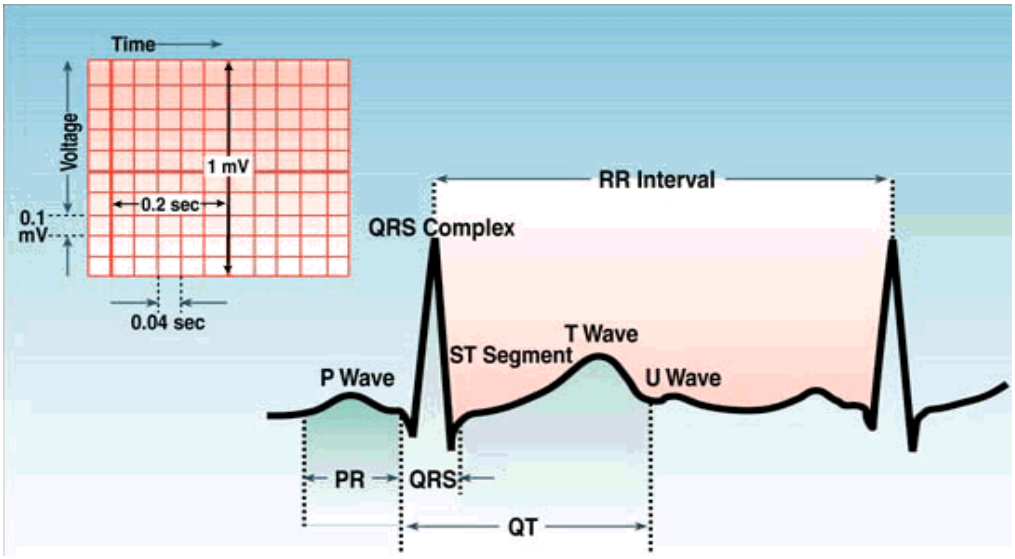
- The user can turn the grid markings on or off.
- The baseline can be adjusted for precise analysis and printing.
- Zoom-in for a closer look at an individual ECG complex.
- The student can perform a detailed analysis of the ECG data.
- See how the ECG complex changes under different conditions.
- Place markers for each of the ECG components (P,Q,R,S &T).
- On-screen commands prompt the students throughout the lesson.
- BIOPAC'S Simple Sensors warn students if they plug the wrong device into the system.

6.6 Method

1. Lead II ECG Record.
2. Event is marked for each new activity.

6.7 Performance Of Various Tasks By The Subject

- Lying down and at a relaxed state.
- After Sitting
- Breathing in and Breathing out.
- Performing a physical exercise to increase the heart rate.
- Recording ECG after exercise.



6.8 Function of ECG Components

Table below show us various waves with different functions.

WAVES	REPRESENT
P wave	Atrial activation
PR interval	The time from onset of atrial activation to onset of ventricular activation.
QRS complex	Ventricular activation
QRS duration	Duration of ventricular activation
ST-T wave	Ventricular repolarization
QT interval	The duration of ventricular activation and recovery
U wave	After depolarizations in the ventricles

Table 6.3 Functions of ECG components

Before starting the experiment it is our responsibility to make sure volunteer(s) read and fill the following forms:

- It is necessary to ask volunteer(s) to read the volunteer information sheet to make sure he/she know everything about experiment.
- It is necessary to ask volunteer(s) to fill the patient consent form to make sure he/she knows all the risk factors.
- Also volunteer(s) need to fill the volunteer questionnaires to make sure he/she has not got any kind of illness.

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6.9 VOLUNTEER INFORMATION SHEET

Study title: (ECG)

ELECTROCARDIOGRAPHY INTERPRETATION AND APPLICATIONS

PLEASE HELP OUR RESEARCH GROUP TO HELP PEOPLE WHO SUFFER FROM HEART DISEASE.
HELP NEW GENERATION
WE ARE LOOKING FOR
VOLUNTEERS
(REGARDLESS OF CONDITION)

Q1. What is the purpose of the study?

- To become familiar with the electrocardiograph as a primary tool for evaluating electrical events within the heart.
- To correlate electrical events as displayed on the ECG with the mechanical events that occurs during the cardiac cycle.
- To observe rate and rhythm changes in the ECG associated with body position and breathing.

Q2. Why have I been chosen?

There is no particular reason.

Q3. Do I have to take part?

No. Taking part in this study is volunteer work

Q4. What will happen to me if I take part?

- This research will last just one day but we need you to repeat the experiment three times.
- We will give you the most suitable time for you to do the test.
- There is no harm or pain in this study.

Q5. What are the possible disadvantages and risks of taking part?

There are no disadvantages for you in this test. We will pay for your travelling cost and any cost related to this research. There is risk involved in this research.

Q6. What is the possible benefit of taking part?

By you taking apart you will help us to find a way to improve our existing knowledge about those people who suffer from heart disease.

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Q7. Which kind of test I will be involved in?

Our team member will attach three electrodes on your body and ask you to relax and follow five different positions to record your heart signals. These positions are: lay down, sitting, deep breathing in and out, exercising with a bike and finally resting after exercise. We will then analyse the data collected from you.

Q8. Will taking part in this study be confidential?

All the information you give to us will stay confidential.

Q9. What will happen to the result of this work?

The outcome of the research will be used for a medical engineering final year project and no name or any identification will be mentioned.

FARSHID SEFAT
Department of Medical Engineering
University of Bradford, Richmond Road,

UNIVERSITY OF BRADFORD

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6.10 Patient Consent Form

FARSHID SEFAT
Department of Medical Engineering
University of Bradford,
Richmond Road
Bradford BD7 1DP

fsefat@bradford.ac.uk

Project:

ELECTROCARDIOGRAPHY INTERPRETATION & APPLICATIONS

Patient Reference number: .....

Personal details

Title Mr ___ Mrs ___ Ms___ Miss___

First name	
Surname	
Date of birth	
Nationality	
Tel number: (including area code)	

	YES	NO
1. I have read and understood the volunteer information sheet.		
2. Details of the project explained to me including aims, methods, etc.		
3. All the risk factors explained to me and I fully understand them.		
4. All risk factors base on local research ethics committee (LREC) regulation explained to me by one member of research team.		

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	YES	NO
5. I had an opportunity to talk to researchers.		
6. I am satisfied with the answers to my questions from researchers.		
7. I had an opportunity to talk to the head of the research group.		
8. I have spoken to all sponsors including University of Bradford and NHS (please specify which one did you meet.) ……		
9. I understand that I am free to withdraw from this study at anytime without giving reason.		
10. I understand that my withdrawal will not affect my future care.		
11. I agree to take part in this research.		

Signature …….. Date ……

Full name (BLOCK CAPITALS) …….

Signature of witness ……. Date ……

Full name (BLOCK CAPITALS) …….

Research Team

- Dr. M.Youseffi: Lecturer and Tutor in Medical Engineering.
- Dr P.Twigg: Lecturer and director of study in Medical Engineering.
- Mr.P.Widdop: Laboratory Technician. (University of Bradford, WG37)

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6.11 Volunteer Questionnaires

Personal details

Title Mr ___ Mrs ___ Ms___ Miss___

First name

Surname

Date of birth

Nationality

Home address (Permanent address)

House name or number

Street name

District

Town

County

Post code

Contact Telephone numbers

Home number

Mobile number

Email address

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The University of Bradford will be advised as to suitable volunteer sympathetic to any medical requirements on the basis of the information provided below.
The University of Bradford will honour its obligations under the Disability Discrimination Act 1995, Health and safety at Research legislation, the research time regulations 1998 and the data protection Act.

- This information is for the use of the University of Bradford Medical Advisor, head of the research and research members. The form will be retained as part of the University of Bradford's **confidential** records. Should your volunteer work not be confirmed this form will be destroyed.
- In accordance with legal requirements the contents will **NOT** be disclosed without your permission to any unauthorised personnel.
- Please fill in this form as accurately as possible. Before completing the form, please read it carefully to the end.
- Please print your answers or thick one of the boxes as appropriate.
- There is space provided at the end of the form for further information. If you answer **YES** to any of the following questions, **YOU MUST USE THIS SPACE TO GIVE DETAILS, INCLUDING DATES**.

	YES	NO
1. At any time has your health seriously interfered with your normal school or work activities?		
2. In the past year have you had any sickness absence lasting more than 2 weeks?		
3. How many periods of absences lasting less two weeks have you suffered in the pat 2 years?		
4. Do you suffer from any long standing illness, injuries, or medical complaints?		
5. Have you had any operations?		
6. Have you suffered any injuries at work, at home, recreational or road traffic accident etc?		

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7. Do you have any allergies?

8. Do you have any problems with the following?
a) Heart eg. Angina high blood pressure, Palpitations, Leaking valves, chest pain, Raynauds Disease etc?

b) Chest eg. Asthma, Bronchitis, TB, etc?

c) Skin eg. Eczema, Dermatitis, Psoriasis, etc?

d) Consciousness eg. Fits, dizziness, blackouts or fainting attacks?

e) Persistent headaches?

f) Stomach or bowel disorders, including indigestion, ulcers and diarrhoea etc?

g) Hernias or ruptures?

h) Kidneys / bladder?

i) Liver or pancreas eg. Jaundice, Hepatitis?

9. Have you ever suffered from?
a) Arthritis? (Is so which joints?)

b) Injuries to bones, joints, hands or fingers?

c) Sports injuries?

d) Strain injuries to back or neck at home or at work?

e) Tennis elbow and Golfer's elbow?

f) Vibration white finger?

g) Carpal tunnel syndrome?

h) Lumbago Fibrositis, Sciatica, or Prolapsed disc, etc?

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	YES	NO
i) Neuritis or trapped nerve?		
j) Shoulder problems eg. Frozen shoulder?		
10. Have you suffered from Repetitive strain injury or Tenosynovitis?		
11. Do you suffer from any hearing loss? If yes which ear? ----------		
a) Have you ever had a discharging ear?		
b) Have you had a perforated eardrum?		
c) Have you had any other ear disease?		
12. What sports have you played in the past or present?		
13. List your main leisure activities?		
14. Have you ever suffered from any: a) alcohol related illness?		
b) Psychiatric or mental disorder?		
c) Form of drug dependency?		
d) Depression or nervous problem?		
15. Have you ever been exposed to any health hazard at work eg. Dusts, fumes, chemicals?		
16. Do you wear glasses/contact lenses for reading or distance vision?		

17. When did you last have your eye tested? - (Date :……)

18. Do you operate a VDU?
(if so ,show how many hours per day: ……)

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19. Have you suffered any problems with using a VDU?

20. Have you been involved in heavy lifting?

21. Are you aware of any reason why you should avoid lifting?

22. Do you smoke? (If so, how many a day:……)

23. Are you on a hospital waiting list for treatment?

24. Are you taking any medication at present?
    a. From your doctor?

    b. Self prescribed?

    c. For recreational purpose?

25. Are you undergoing any form of medical treatment including physiotherapy, Chiropracty, Osteopathy, Acupuncture?(in last 2 years)

26. Have you ever suffered fro many type of food poisoning, including: Enteritis, Eiahorrea or vomiting lasting more than 48 hours including:
a) Salmonella/Typhoid?

b) Dysentery?

c) Campylobacter/ Cryptosporidia infection?

27. Have you ever lived or spent significant time abroad?
If so, have you suffered from any tropical diseases?

28. Are there any medical condition you wish the University of Bradford Medical advisor to be aware of in confidence or which may requires special consideration?

29. In an average week, how much of each of the following do you drink?
a) Beer …… b) Wine ……...
c) Fizzy Drinks (eg. Coke) ……

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30. If you have ever had vaccinations for the following, give approximate dates:

a) Tetanus: ……..
b) TB: …….
c) Polio: ……..

31. What is your:
a) Height: ……..
b) Weight: …….
c) Shoe Size: …….

If you want to provide further information in confidence, please write to the University of Bradford medical advisor at the following address:

Dr. George Day
Chesham Building
Richmond Road
Bradford
BD7 1BR

If you answered YES to any of the questions, please explain in the space below:

UNIVERSITY OF BRADFORD

MAKING KNOWLEDGE WORK

I declare that to the best of my knowledge I am not suffering from any illness or disability, which would affect me carrying out the research as a volunteer.

Having read the information on the front page, I have answered these questions to the best of my knowledge.

Volunteer Signature: …….

Please print your name here: ……...

Date: ……

FARSHID SEFAT
Department of Medical Engineering
University of Bradford, Richmond Road,

6.12 Data and Calculations

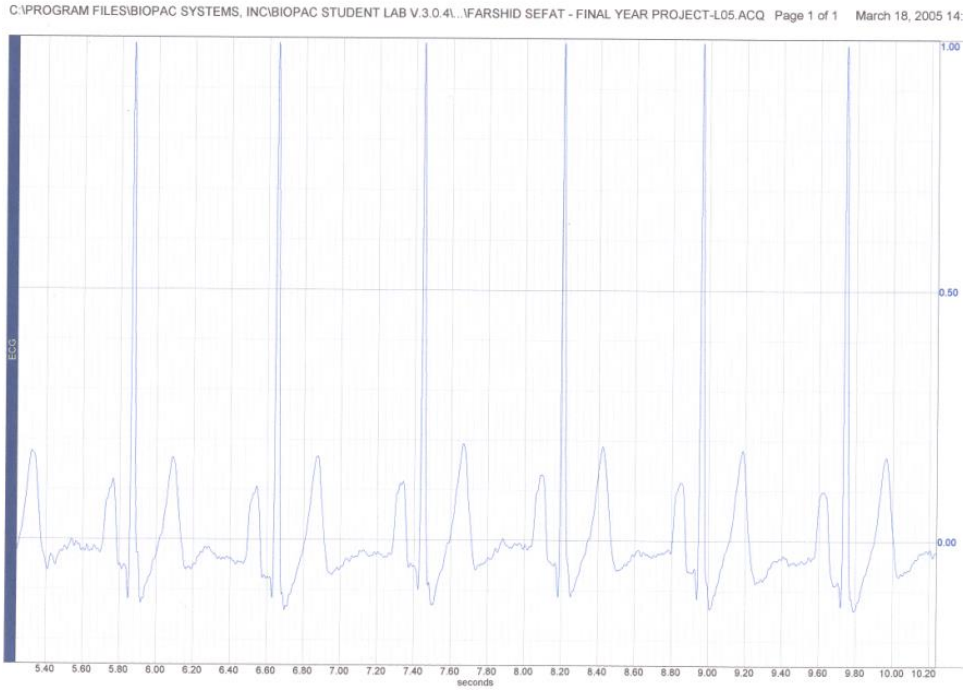
Subject Profile

Name:	**CHRIS RULE**	**Height:**	**172 Cm**
Age/ Gender:	23/Male	Weight:	63 Kg

6.12.1 Supine, Resting, Regular Breathing

Table 6.4

Measurements	From Channel	Cardiac cycle			Mean	Range
		1	2	3		
∆ T	CH 2	0.80	0.78	0.76	0.78	0.76-0.8
BPM	CH 2	76	75	78	76	75-78



Graph 6.1 Supine, Resting, Regular Breathing

Table 6.5

ECG Component	Duration Δ T (CH2)				Amplitude (mV) Δ (CH2)			
	Cycle 1	Cycle 2	Cycle 3	Mean	Cycle 1	Cycle 2	Cycle 3	Mean
P Wave	0.10	0.08	0.09	0.10	0.15	0.17	0.15	0.16
PR interval	0.12	0.14	0.12	0.13	----------	----------	----------	----------
PR Segment	0.04	0.04	0.04	0.04	----------	----------	----------	----------
QRS Complex	0.16	0.12	0.18	0.15	1.09	1.11	1.12	1.11
QT Interval	0.32	0.34	0.32	0.33	----------	----------	----------	----------
ST Segment	0.08	0.08	0.08	0.08	----------	----------	----------	----------
T Wave	0.16	0.16	0.16	0.16	0.21	0.22	0.25	0.23

** Corresponds to ventricular systole*** Corresponds to ventricular diastole

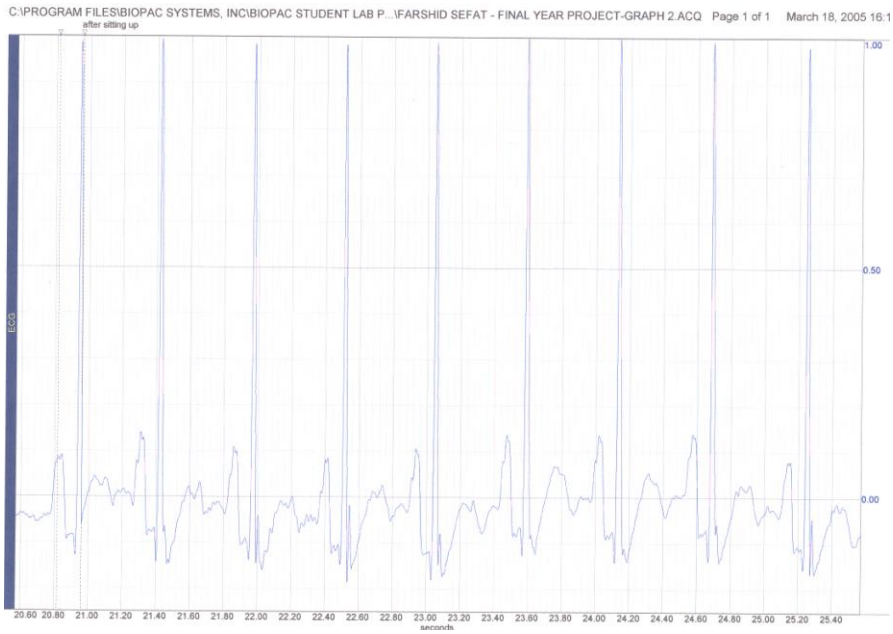
Table 6.6

	CH 2 Delta T			
Ventricular Readings	Cycle 1	Cycle 2	Cycle 3	Mean
QT Interval (Corresponds to Ventricular Systole)	0.32	0.34	0.32	0.33
End of T wave to subsequent R wave (Corresponds to Ventricular Diastole)	0.50	0.49	0.47	0.49

6.12.2 Sitting

Table 6.7

Heart Rate	Channel No.	Cycle 1	Cycle 2	Cycle 3	Mean
Δ T	CH 2	0.55	0.56	0.52	0.54
BPM	CH 2	89	91	93	91



Graph 6.2 Sitting

6.12.3 Supine, deep Breathing

Table 6.8

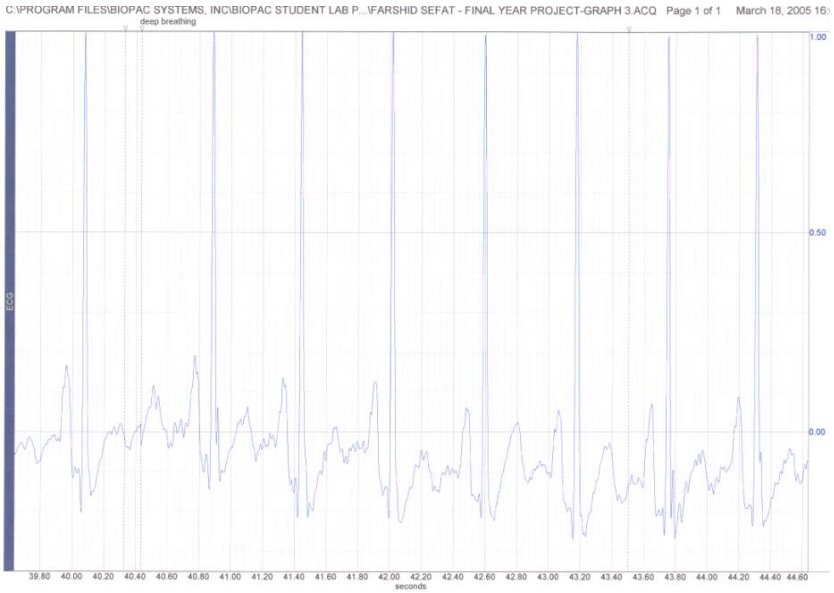
Inspiration

Heart Rate	Channel No	Cycle 1	Cycle 2	Cycle 3	Mean
Δ T	CH 2	0.56	0.58	0.52	0.55
BPM	CH 2	106	102	103	104

Table 6.9

Expiration

Heart Rate	Channel No	Cycle1 1	Cycle 2	Cycle 3	Mean
Δ T	CH 2	0.60	0.62	0.60	0.61
BPM	CH 2	101	98	96	98

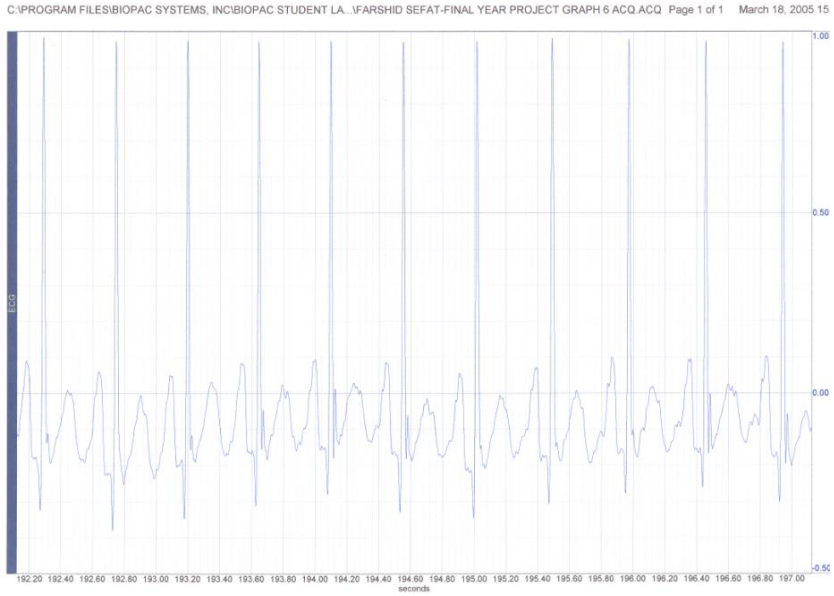


Graph 6.3 Supine, deep Breathing

6.12.4 After Exercise

Table 6.10

	Channel 2 Delta T			
Ventricular Readings	**Cycle 1**	**Cycle 2**	**Cycle 3**	**Mean**
QT Interval (Corresponds to Ventricular Systole)	**0.26**	**0.28**	**0.28**	**0.27**
End of T wave to subsequent R wave (Corresponds to Ventricular Diastole)	**0.22**	**0.21**	**0.23**	**0.22**



Graph 6.4 after exercises

		Start recording				End of recording			
Heart Rate	**CH No.**	**Cycle 1**	**Cycle 2**	**Cycle 3**	**Mean**	**Cycle 1**	**Cycle 2**	**Cycle 3**	**Mean**
ΔT	CH 2	**0.42**	**0.46**	**0.44**	**0.44**	**0.48**	**0.48**	**0.52**	**0.49**
BPM	CH 2	**136**	**129**	**133**	**132**	**129**	**136**	**133**	**132**

Table 6.11

6.13 Data Summary and Questions to be answered

E. Heart Rate (BPM) Table 6.12

Condition	Mean	Range
Supine, regular breathing	**76**	**75-78**
Supine, deep breathing, inhalation	**104**	**102-106**
Supine, deep breathing, exhalation	**98**	**96-101**
Sitting, regular breathing	**91**	**89-93**
After exercise- start of recording	**132**	**129-136**
After exercise- end of recording	**132**	**129-136**

Explain the changes in heart rate between conditions. Describe the physiological mechanisms causing these changes.

The heart rate of a human varies from 150 beats per minute in young children, to about 60 in the aged. However, doing exercise this will alter the rate substantially. As this

shown in our results, in sitting (regular breathing) the heart rate is 91 BPM, after exercise it increases to 132 BPM. This is because during exercise the supply of Oxygen to the muscles will decrease, which increases, respiration and the cardiac output rises as a result of that to supply working tissues with increased amount of oxygen and nutrients, this causes heart rate to rise, as the body has to work harder. When the exercise is complete the heart rate will gradually decrease back to the resting pulse rate. As shown in the results obtained after exercise, at the start and the End of recording, the heart rate was the same 132 BPM, this is because it will take sometime for the heart rate to decrease back to the resting state condition. Moving from supine regular breathing to sitting regular breathing, the sympathetic output increases which increases the heart rate. The heart rate is faster during inhalation than exhalation. This is because for inhalation to occur the lungs must expand, which increases long volume. The first step in expanding the lung is contraction of the diaphragm, the main muscle of inhalation, which increases the size of the thoracic cavity, which increases the heart rate. These followed by decrease in heart rate by exhalation. The reason for that is that in expiration no muscular contractions are involved. Exhalation begins when the diaphragm relaxes, which decreases the volume of the thoracic cavity.

6.14 Duration ΔT

Rhythm Table

Table 6.13 Rhythm

Measurement	Mean	Range
Supine, deep breathing, inhalation	0.55	0.52-0.58
Supine, deep breathing, exhalation	0.61	0.60-0.62

Are there differences in the cardiac cycle with the respiratory cycle?

The duration (ΔT) for the deep breathing is less than regular breathing (0.78s), the reason for that is that the heart rate increases during deep breathing. During deep, forceful inhalations (0.55s) accessory muscles of inhalation also participate in increasing the size of the thoracic cavity, which increases the heart rate. Consequently there is difference in the cardiac cycle and the respiratory cycle.

Measurement	Mean	Range
Supine, regular breathing Ventricular systole	**0.33**	**0.32-0.34**
Supine, regular breathing Ventricular diastole	**0.49**	**0.47-0.50**
After Exercise Ventricular systole	**0.27**	**0.26-0.28**
After Exercise Ventricular diastole	**0.22**	**0.21-0.23**

Table 6.14

What changes occurred in the duration of systole and diastole between resting and exercise?

Duration of systole in regular breathing (0.33s) is higher than duration of systole post exercise (0.27s). The duration of diastole as also decreased from regular breathing (0.49s) to post exercise (0.22s), this shows that the person heart rate must have been faster in post exercise compared to regular breathing.

6.15 Data Review

Number	Data Review	Answer
1	Is there always one P wave for every QRS complex?	**Yes**
2	Describe the P and T wave shapes:	**********
3	Do the wave durations and amplitudes for all subjects fall within the normal ranges?	**Yes**
4	Does the ST-segment mainly measure between-0.1mV and 0.1mV?	**Yes**
5	Is there baseline “drift” in the recording?	**Yes**
6	Is there baseline “noise” in the recording?	**Yes**

Table 6.15

6.16 Describe the P and T wave shapes:

P Wave

Electrical impulses originating in the SA node trigger atrial depolarization. The normal P wave is no more than 0.1 second in duration and 2.5mm high. The direction of electrical activity is from SA to AV node. The P wave is a representation of the time it takes for atrial depolarisation. It is viewed normally as small and curved with a positive deflection

T Wave

Ventricular repolarization, which follows ventricular depolarisation, is represented by the T wave. Its shape is rounded and taller and wider than the P wave**.** It is also more sensitive to physiologic and hormonal changes in shape but usually presents as a

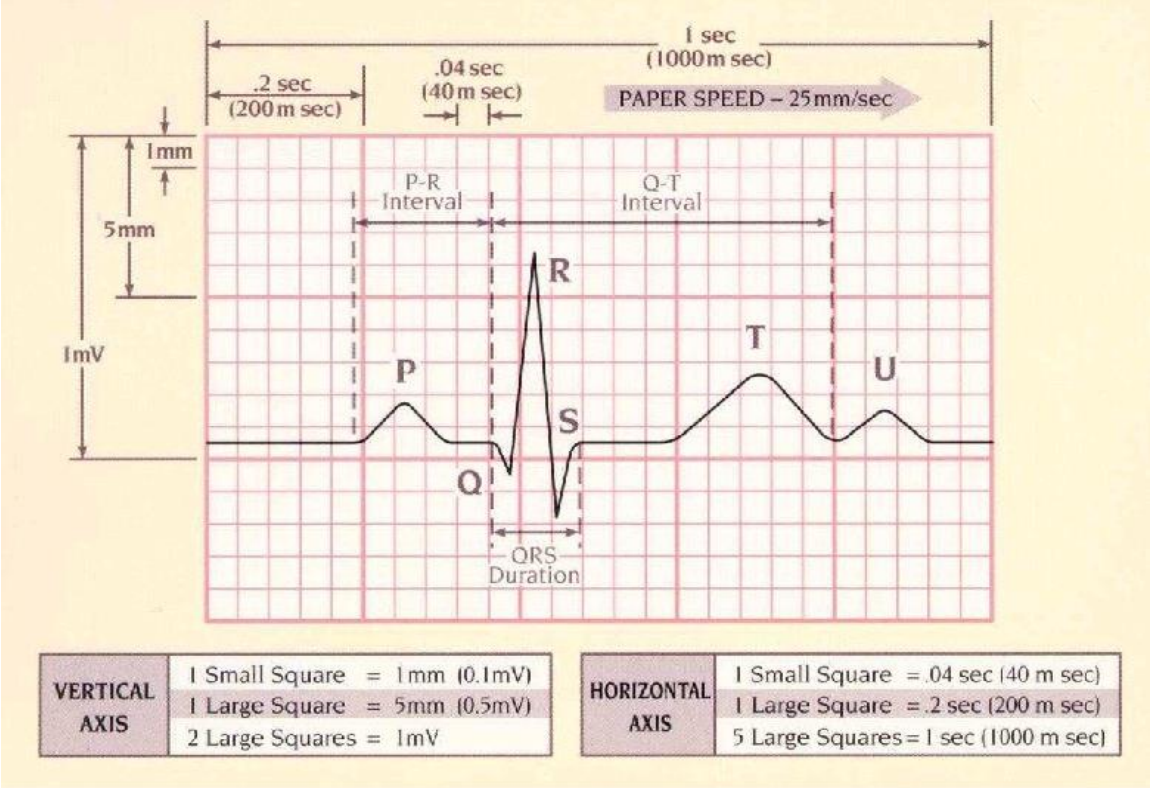
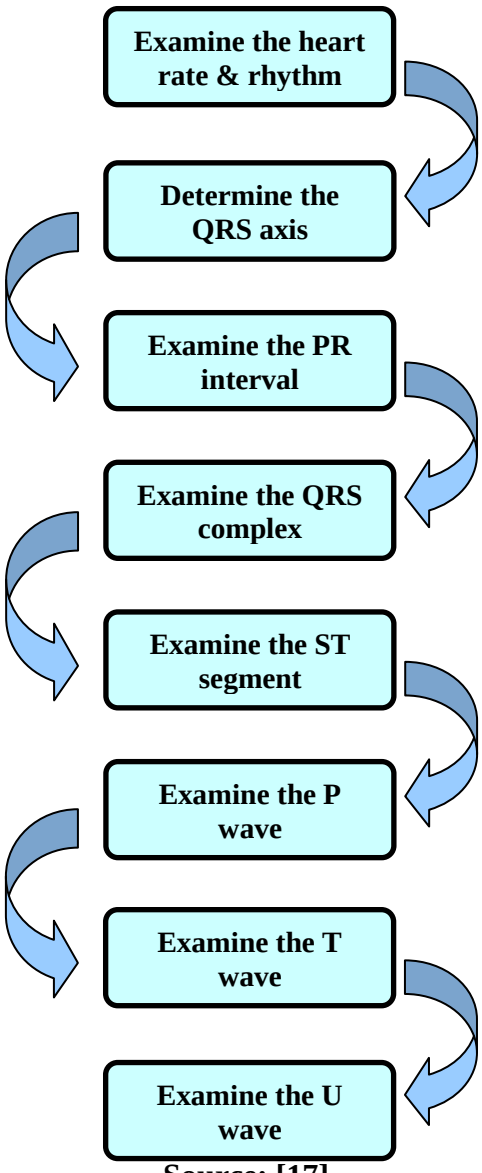


Figure 6.5 ECG graph

Chapter 7

ECG INTERPRETAITON OF RESULTS OBTAINED IN THIS WORK

It is always questionable a question that what does happen if a patient come to the hospital without any previous history and complain about a pain in his/her chest. Cardiologists always follow the procedure below:



Source: [17]

Heart rate is an important element in the ECG interpretation and need to be calculated. As mentioned in chapter 6 the two formulas to calculate heart rate are:

$$\text{Rate} = \frac{60}{R-R\ duration}$$

(OR)

$$\text{Rate} = \frac{Counting.the.Large.squares.between.two.identical.points}{300}$$

But these days life become easier and by using high technology electrocardiography machine in hospitals, heart rate can be achieved. Remember the normal heart rate is between 60-100 beat per minute. If there is an abnormal heart rate, then the following arrhythmias can be diagnosis:

- **Bradycardia**- if heart rate is under 60 bpm, it affects the pressure receptors in the atria and a reduction in this pressure causes the heart to slow down.
- **Narrow complex Tachycardia**, (occur over short period of time).
    1. **Sinus Tachycardia**- when heart rate is over 100 bpm.
    2. **Superventricular Tachycardia (SVT)** - heart rate varies in different ages and usually is faster in children and even faster in infants. If heart rate is over 220 bpm in infants, over 180 in children and over 160 in adults then the cardiac tissue above the ventricle initiates fast beating. Electrical signals in the heart create a short circuit, which causes heart to beat faster.
- **Wide complex Tachycardia**- (occur over long period of time)

Ventricular Tachycardia

The arrhythmias associated with a low rhythm are Sick sinus syndrome (SSS) and

Heart Blocks. Also the arrhythmias associated with a fast rhythm are Atrial fibrillation (AF), Ventricular Tachycardia, Ventricular fibrillation and sudden cardiac arrest. Now we discuss each one in brief.

- **Sick sinus syndrome (SSS)** As mentioned in chapter 4, SSS is a type of Bradycardia. The sinoatrial node malfunctions, leading to a slow heart rate. ECG Shows heart rate falls to 40-50 bpm or less.

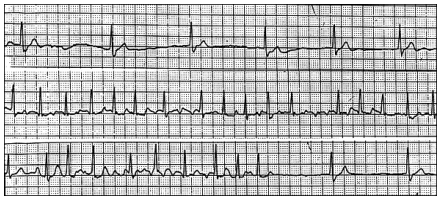


Fig 7.1 Electrocardiogram shows sick sinus syndrome.
Source: http://www.aafp.org/afp/20030415/1725.html

Heart rhythm is irregular, there are pauses between signals from the SA node and escape rhythms may also be obtained.

- **Atrial fibrillation (AF),** the atria beat rapidly (up to 500 bpm) and irregularly, leading to slower, irregular ventricular contractions (around 150 bpm). In AF multiple wavelets of electrical impulses are detected and there is no normal sinus rhythm observed. AF can occur either recurrently or periodically. Paroxysmal Atrial Fibrillation is the name given to periodic type of AF.

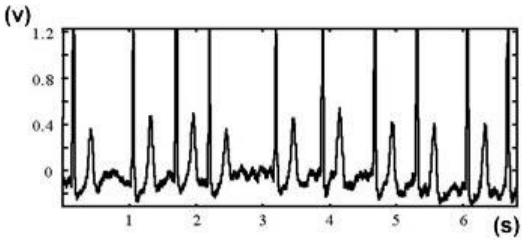


Fig 7.2 Atrial fibrillation
Source: http://www.cardiodigital.com/Research_AF/research_af.htm

- **Ventricular Tachycardia,** (discussed in disease chapter)
- **Ventricular fibrillation**, disorganized electrical activity in the heart causes the ventricles to quiver, rather than beating in an effective manner [2].

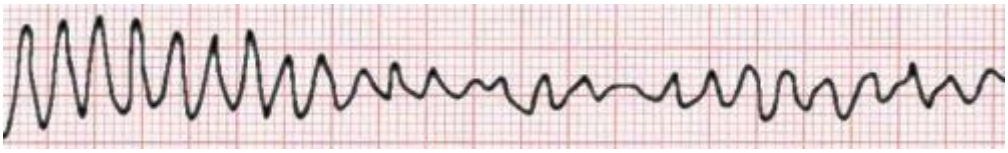


Fig 7.3 Ventricular fibrillation
Source: http://www.anaesthetist.com/icu/organs/heart/ecg/

- **Sudden cardiac arrest** is a sudden and unexpected loss of heart function that results in death.

7.1 Axis in ECG

The peculiar system we use in electrocardiography is non-Cartesian, and rather arbitrary! [36]

ECG can be interpreted by measure the direction of vectors in degrees, and zero is indeed facing `East', but +90^{o} is south. You can work out that ± 180^{o} is 'West', and that minus 90^{o} is 'North'. Axis in any ECG is same as ECG depolarisation and we should not misunderstood it with frontal plane QRS axis as lots of people do.

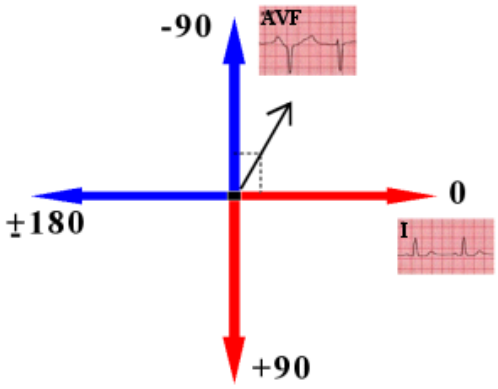


Fig 7.4 ECG Axis
Source: http://www.anaesthetist.com/icu/organs/heart/ecg/

Axis position	Related diseases
Where the axis is up and to the left (North west axis) eg. -135^{o}	Congenital heart disease, dextrocardia, and sometimes in severe chronic obstructive airway disease
Marked right axis deviation (e.g. +150^{o})	May signify significant `right-sided' heart disease.
If the T wave axis is more than about 45 to 60^{o} different from the QRS axis,	This is abnormal
The P-wave axis (The normal axis is about +40 to +60^{o})	Moving right with chronic obstructive disease OR The axis may move left with congenital heart disease,
Northern' shift in axis	An ectopic atrial focus low down in the atrium (`coronary sinus rhythm')

Table 7.1 ECG axis and related diseases

7.2 Determination of the QRS axis

The QRS axes are considered normal between -30˚ and +100˚. Left Axis Deviation (LAD) and Right Axis Deviation (RAD) are two QRS axis deviations. Any variation outside the above range can lead to LAD and RAD. If the leftward axis quadrant is

more negative than –30 degrees it known as left axis deviation (LAD) and if rightward axis quadrant is more positive than 100 degrees it known as right axis deviation (RAD). In LAD overall positive observed in lead 3 and negative QRS is in leads 2 and 3. In RAD overall positive QRS observed in lead1.

7.3 Causes of Right Axis Deviation (RAD) & Left Axis Deviation (LAD)

Causes for RAD are:

- Right ventricular hypertrophy (RVH)
- Right bundle branch block (RBBB)
- WPW syndrome
- Ventricular Ectopy

And causes for LAD are:

- Left ventricular hypertrophy (LVH)
- Left bundle branch block (LBBB)
- Left anterior Hemiblock
- WPW syndrome
- Ventricular Ectopy

Now we discuss each cause in brief:

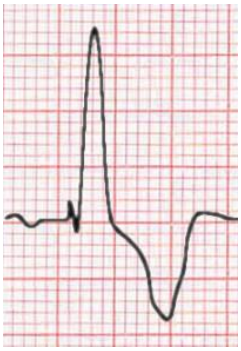
7.3.1 Right Axis Deviation (RAD) causes:

- **Right ventricular hypertrophy (RVH)**

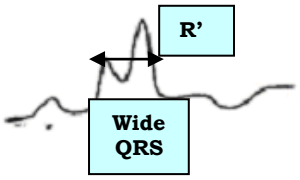
  There are some characteristics, which can be observed with RVH such as right atrial enlargement, pulmonary hypertension or very type of right ventricular outflow obstruction. It is important to know what causes RVH. There are few causes such as increased filling of the right ventricle, augmented resistance to right ventricular evacuation and incompetent emptying.

- **Right bundle branch block (RBBB)**

  When there is a block in the right bundle branch the impulse slow down resulting in the electrical impulse to find an alternative route. It is obvious on ECG to diagnosis RBBB by observing wide QRS



complex with a distinct pattern of a second R wave (R')



RBBB

Figure 7.5 A RBBB pattern
Source: http://heartdisease.about.com/library/weekly/aa020101b.htm

- **WPW syndrome**

This syndrome will affect the heart rhythm. An additional pathway created to conduct electrical impulse from atria to the ventricles. Hence, a wide QRS is observed.

- **Ventricular Ectopy** - irregular heartbeats caused by disturbance to the hearts electrical system [5].

7.3.2 Left Axis Deviation (LAD) causes

- **Left ventricular hypertrophy (LVH)**

LVH is weakening and function inefficiently due to prolonged strain to the heart.

- **Left bundle branch block (LBBB)**

LBBB is similar to RBBB except that the left bundle branch is damaged or diseased.

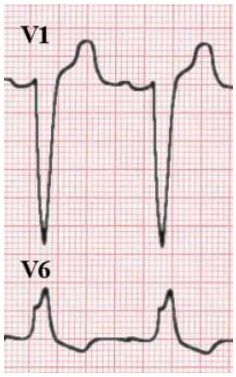


Figure 7.6 A LBBB pattern

LBBB

Source: *http://heartdisease.about.com/library/weekly/aa020101b.htm*

- **Left anterior Hemiblock**

When the QRS axis is to the left by more than 30 degrees it is known as left anterior Hemiblock. In fact it is an abnormality of the electrical conduction system.

7.4 Examining the P-R interval

P-R interval in a normal heart is between 0.12-0.2 seconds. A prolonged interval can indicate an AV nodal block, Hyperkalemia and occasionally Hyperthyroidism. The possible AV nodal blocks that can be diagnosed from the ECG are:

1. First degree AV block where the impulses are moving very slow through the AV node.
2. Type I Second Degree AV Node Block is characterised on the ECG as the progressive lengthening of the PR interval until a beat is dropped.
3. Type I1 Second Degree AV Node Block is characterised on the ECG as a series of non-conducting P waves.
4. Third degrees AV block where no impulses are reaching the AV node therefore the ventricles use their own pacemaker that operates at a slower rate [5].

A shortened P-R interval can diagnose following:

- An AV nodal rhythm
- Low atrial nodal rhythm
- WPW syndrome
- Lown-Ganong-Levine (LGL)
- Hypertension.

7.5 Examining the QRS complex

Table below represent normal durations of the QRS complex.

LEAD	DURATION OF THE QRS COMPLEX
Leads (1, 11, 111)	**0.05- 0.10 (Sec)**
Leads (V1-V6)	**0.06- 0.12 (Sec)**

Table7.2 Duration of the QRS complex

There are some variations in this duration that can be observed are shown in the following table:

QRS COMPLEX	ABNORMALITIES
Narrow QRS complex of less than 0.06 seconds	Supraventricular Tachycardia

Wide QRS complex of over 0.12 seconds	Hyperkalemia, BBB, WPW syndrome, ventricular or supraventricular Tachycardia.
When too much fluid collects in the pericardium increasing the pressure in the heart	Pericardial effusion
When excess mucus inside the tissues causes them to swell up.	Myxedema
High QRS amplitude	Left ventricular hypertrophy
Irregular QRS complex rate with uniform shape	Atrial flutter

Table 7.3 Variations in QRS complex

Also low QRS amplitude and diffuse coronary artery disease can be diagnoses.

7.6 Examining the S-T segment

Normally ST segment is 0.04 second (1mm) from the J-point (end of QRS). ST segment is a good reference to indicate some abnormalities such as Ischemia or infarction. Following table show us different abnormalities that can be observed in different ECG leads.

ST-SEGMENT & RELATED LEADS	ABNORMALITIES
V1, V2	ST segment depression
ST segment accompanied with a large R wave	Posterior wall infarction.
V1, V2	Anterior wall infarct
V3, V4	Lateral wall infarct
Leads 1, V5, V6 and aVL	Inferior wall infarct
ST segment elevation	Indicate early stages of a Myocardial Infarction (MI), also known as a heart attack

Table 7.4 Examining the S-T segment

7.7 Examining the P wave

In a normal ECG, the P wave is generally can be observed as below:

LEADS	P-WAVE SHAPE
Leads 1, 11, aVF, V4, V5, V6	Upright
aVR	Inverted
Leads 111, aVL, V1, V2, and V3	Variable

Table 7.5 P wave and its shapes

In an abnormal ECG the variations observed in P waves are:

P-WAVE SITUATION	ABNORMALITIES
Inverted in leads other than aVR	Ectopic atrial focus or AV nodal rhythm.
High amplitude	Atrial dilatation or atrial hypertrophy due to increased depolarisation force within the atrium.
Biphasic, wide or notched P waves (look at the Figure 7.7)	A left atrial enlargement as a result of a delay in the electrical activation of the enlarged left atrium
Pointed or peaked P wave (Look at the Figure 7.8)	Right atrial enlargement
Absences of the P wave	Hyperkalemia, AV nodal rhythm or SA node block
Saw tooth configuration in leads 11, 111 and aVF	Atrial flutter that is a rhythm disturbance of the atria resulting in a rapid but regular heartbeat

Table 7.6 P-wave and its related abnormalities

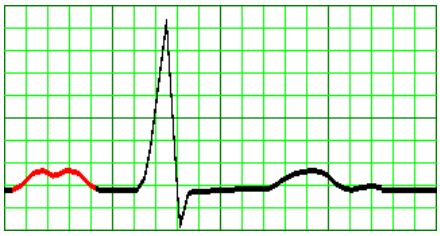


Figure 7.7 Biphasic, wide or notched P waves
Source ***http://www.madsci.com/manu/ekg_hypr.htm***

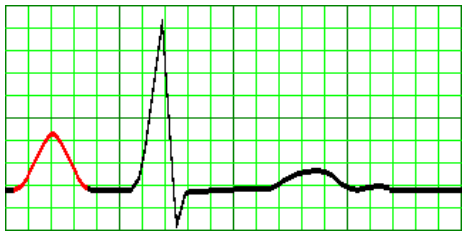


Figure 7.8 Pointed or peaked P wave
Source:http://www.madsci.com/manu/ekg_hypr.htm

Figure 7.9 SA node block reflecting a missed beat
Source: ***http://endeavor.med.nyu.edu/student-org/erclub/ekgexpl4.html***

SA node block is caused by the SA node failing to transmit an electrical impulse and an AV nodal rhythm that is a conduction block that causes the electrical impulses travelling between the atria and the ventricles in the AV node to be delayed [5].

7.8 Examining the T wave

A normal T wave is observed as following figure:

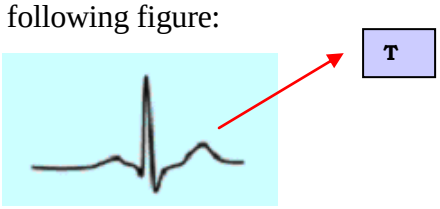


Figure 7.10 Normal T wave

Source: ***http://bmj.com/cgi/content/full/324/7344/1023***

In a normal ECG, the T wave is generally can be observed as below:

ECG LEADS	T-WAVE SHAPE
Leads 1, 11, V3, V4, V5, V6	Smooth, upright
aVR and V1	Inverted
aVL and aVF	High amplitude
In all other leads.	Variable

Table 7.7 T wave shapes

Also the common T wave variations found are:

T-WAVE SITUATION	ABNORMALITIES
Tall T waves	Hyperkalemia, Myocardial infarction, Myocardial Ischemia or left bundle branch block.
Notched T waves	Pericarditis, (The tissues of the pericardium are inflamed).
Pointed T waves	MI (which is caused by the closure of the coronary artery that supplies the heart muscle with blood)
Small flattened or inverted T waves	Myocardial-Ischemia, hyperventilation, anxiety, LVH, drug use (e.g. digoxin) or Pericarditis

Table 7.8 T-wave and its related abnormalities

Figures 7.11 and 7.12, below, show various kind of T wave shape.

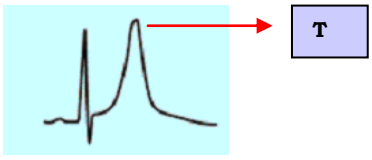


Figure 7.11 Tall T waves

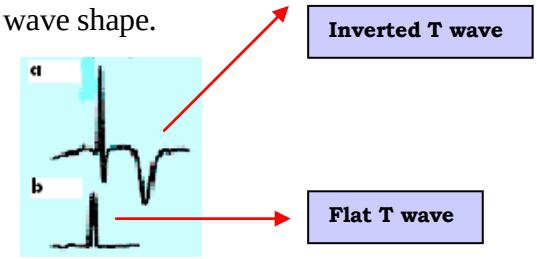


Figure 7.12 (a) Inverted or (b) flattened T wave

Source: ***http://bmj.com/cgi/content/full/324/7344/1023***

7.9 Examining the U wave

Lead V3 is the best place to look at U wave with the same polarity as the T wave. Abnormal U waves are shown in the table below:

U-WAVE SITUATION	ABNORMALITIES
Prominent	Hypokalemia
Inverted	Myocardial Ischemia, a shortage of blood flow and oxygen to the heart muscle, or a Myocardial strain pattern
Increased amplitude	Hypercalcemia, Epinephrine or Thyrotoxicosis.

Table 7.9 U-wave and its related abnormalities

7.10 Overall Analysis of the ECG

The first wave appear on the ECG paper is P wave and it caused by atrial depolarisation. 3mm height and duration of 0.1 seconds are the normal characteristics of the P wave. A broad notched P wave corresponds to the late depolarisation vector caused by the left heart failure and is called the left atrial enlargement. Cardiologist can diagnose right atrial enlargement by observing a tall peaked P wave on ECG trace, which is due to an early depolarisation vector in pulmonary hypertension. Absence of the P wave represents failure of atrial depolarisation, which occurs in sinus arrest.

P-R interval is the time taken from onset of atrial depolarisation (P wave) to the onset of ventricular contractions. It has duration of 0.12-0.20 seconds. First-degree heart block can be diagnoses by observing a prolonged P-R interval when the conduction through the AV node is delayed. A short P-R interval of duration <0.12 seconds occurs when the AV node is bypassed and results in early ventricular activation known as Wolff-Parkinson-White (WPW) or Lown–Banong-Levine syndrome.

The QRS complex represents the depolarisation of the left and right ventricles. QRS complex should not exceed 0.12 seconds and should precede a P wave. Slow ventricular depolarisation caused by either excitation Hypokalaemia or bundle branch block can be recognised by observing a prolonged QRS complex. Exaggerated QRS deflections indicate ventricular hypertrophy. Failure of a QRS complex indicates pericardial effusion or emphysema.

T wave represents repolarisation of the ventricles. The duration of T wave is about 0.27 seconds. Myocardial infarction that is dangerous matter in cardiology and patients with myocardial infarction need special care to prevent further problem. A tall T-wave occurs in acute myocardial infarction. A prolonged T wave inversion occurs as a non-specific response to viral infection, hypothermia and hyperventilation.

The Q-T interval represents ventricular systole and is approximately 0.3 seconds in duration. It is known as very rate-sensitive, so therefore shortens with increasing heart rate. Ventricular arrhythmias, Hypokalaemia, rheumatic fever or drugs can diagnose by observing prolonged Q-T intervals. Shortening of the Q-T interval is caused by Hyperkalaemia digitalis therapy.

The U wave is an upward deflection followed by the T-wave. U wave abnormalities occur in Ischaemic disease. By looking at the next page table we are able to see summary of waves and related abnormalities associated with each one, which can be diagnoses by ECG.

WAVE	DURAITON	OBSERVATION
P **WAVE**	About 0.1 Seconds	Caused by: Atrial depolarisation Broad notched P wave: late depolarisation vector caused by the left heart failure (left Atrial enlargement). Tall peaked P wave: an early depolarisation vector in pulmonary hypertension (right Atrial enlargement). Absence of the P wave: Failure of Atrial depolarisation

P-R INTERVAL	0.12-0.20 Seconds	Prolonged P-R interval: first-degree heart block Short P-R interval of duration <0.12 seconds: Wolff-Parkinson-White (WPW) or Lown–Banong-Levine syndrome.
QRS COMPLEX	0.8-0.12 Seconds	Prolonged QRS complexes: excitation Hypokalaemia or bundle branch block Exaggerated QRS deflections: ventricular hypertrophy Failure of a QRS complex: pericardial effusion or emphysema.
T Wave	About 0.27 Seconds	Prolonged T wave inversion: occurs as a non-specific response to viral infection, hypothermia and hyperventilation. Tall T-wave: acute myocardial infarction.
Q-T INTERVAL	0.35-0.45 Seconds	Prolonged Q-T interval: ventricular arrhythmias, Hypokalaemia, rheumatic fever or drugs.
U WAVE	Very small	U wave abnormalities: occur in Ischaemic disease.

Table 7.10 ECG Waves and related diseases

Chapter 8

INTERPRETATION OF ARRHYTHMIAS AND RELATED CALCULATIONS CARRIED OUT IN THIS WORK

Arrhythmia is another name for irregular heartbeat, which is an abnormality in this rhythm. As mentioned earlier the rhythm is controlled by a group of cells called the sinoatrial node. The question is what normal cardiac rhythm is? Usually the sinus rhythm is the normal cardiac rhythm where the heart rate is regular, between 60-100 beats per minute, the P wave is upright in lead 11 and inverted in lead aVR and a QRS complex follows every P wave. If the heart beats in an irregular rhythm it will not function efficiently.

The most common types of arrhythmia are known as:

- Bradycardia
- Tachycardia
- Heart blocks

8.1 Bradycardia

As just mentioned the heart rate is regular, between 60-100 beats per minute, but when the sinus rhythm is below 60 bpm it known as Bradycardia. The ECG pattern repeats itself at intervals of greater duration resulting in lengthy QT intervals.

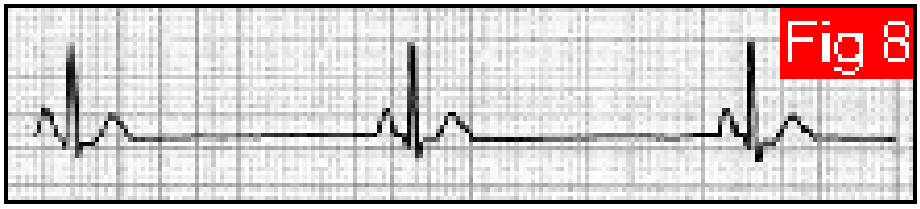


Fig 8.1 Sinus Bradycardia

Source: http://www.nda.ox.ac.uk/wfsa/html/u11/u1105_02.htm

Some people, particularly those young individuals who doing a heavy sport, a rate less than 60 bpm is normal. Usually the main causes are Sleep, Athletic heart, Meningitis, Mental depression, Increased vagal tone or decreased sympathetic tone.

There is no treatment available unless Bradycardia is chronic and associated with low cardiac output, in which case a pacemaker will be required.

8.2 Ventricular Tachycardia

When the SA node start beating faster about 100 bpm is known as Ventricular Tachycardia (VT). P-R interval is between 0.12- 0.20seconds. QRS duration is between 0.04-0.12 seconds.

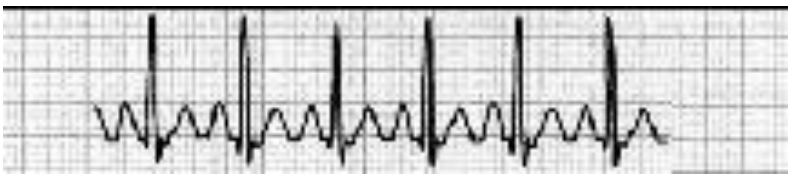


Fig 8.2 Sinus Tachycardia

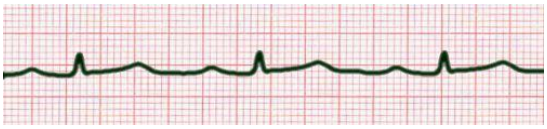
P waves may not be distinguishable in Ventricular Tachycardia. Also P-R interval is not measurable. QRS complex duration is greater than 0.12 sec and T wave may not be separated from the QRS complex.

Usually congestive heart failure, Cardiogenic shock, alcohol, nicotine, caffeine are the most important cause of VT.

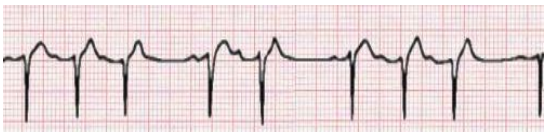
8.3 Heart Blocks

This is a delay or blockage, to the electrical impulses in the heart muscle, leading to slow and irregular beats. In this case there is no signal will pass from upper chambers to the lower chambers of the heart. Cardiologist classified heart blocks to the three main classes and these classes are to do with severity of impairment. These three types are:

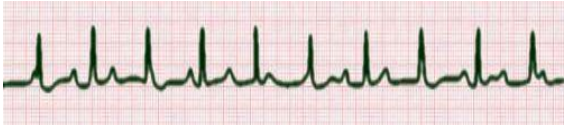
- First-degree heart block
- Second-degree heart block
- Third-degree heart block



First-degree heart block



Second-degree block



Third-degree block

Fig 8.3 Heart block

8.3.1 First-degree heart block

When the electrical impulses takes longer to travel through the AV node it known as first-degree heart block. The P-R interval which is the time taken for the impulse to travel from upper chamber to the lower chambers is longer than usual. The P-R interval is greater than 0.20 sec. Rates may range from Bradycardia to Tachycardia.

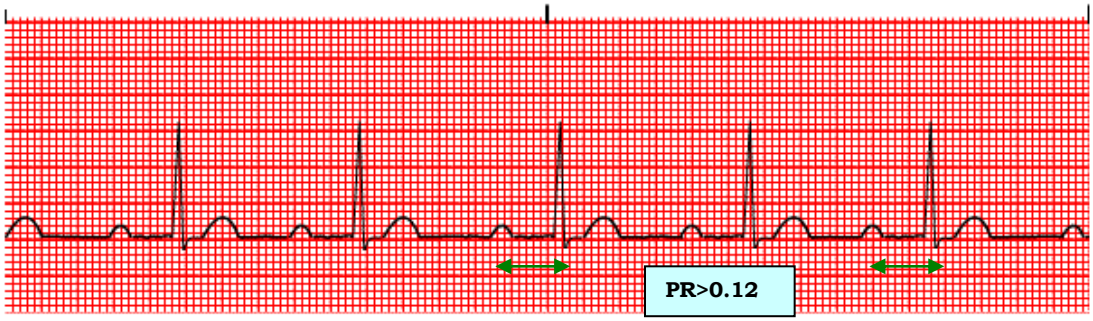


Figure 8.4 ECG tracing of a first degree heart block

Source: ***http://www.rnceus.com/ekg/ekqfdav.html***

Usually person with the first-degree heart block has no problem in normal life and there is no treatment. Some drugs can cause a first-degree heart block. These drugs are Beta-Blockers, Calcium channel blockers and Digitalis glycosides. Basically they increase refractory time of the AV node and finally reducing AV conduction.

8.3.2 Second-degree heart block

When some of the electrical impulses from the atrium don't reach the lower chambers and there is no QRS complex generating, this situation is known as second degree heart blocks. This type of heart blocks consists of two types which known as:

- Mobitz I
- Mobitz II

Mobitz I AV block is distinguished by a repeating cycle of increasing PR intervals. As the interval get longer, a P wave is either not conducted. In fact there is no ORS complex or the P wave is simply dropped. The cycle then repeated again.

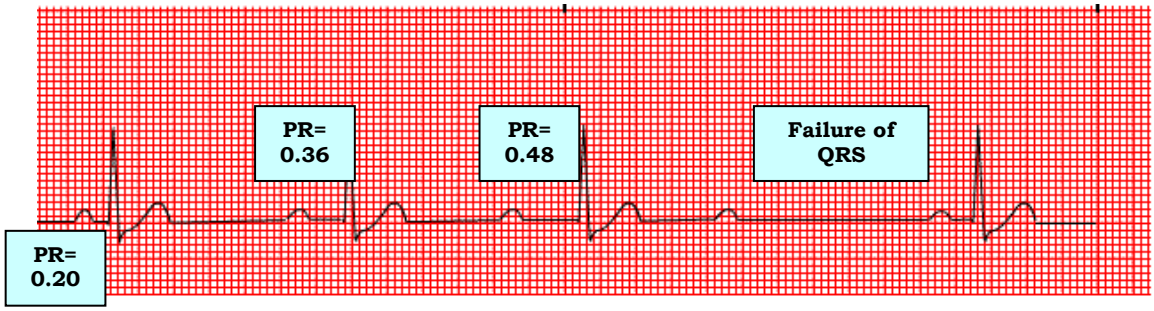


Figure 8.5 ECG tracing of a Mobitz 1heart blocks

Source: ***http://www.rnceus.com/ekg/ekgsecond1.html***

Mobtiz type I sometimes take place when the individual is sleep or in disease. This type occurs due to abnormal conduction through the AV node itself.

Mobitz II can be recognised by a consistent PR interval and frequently non-conductive P waves. QRS complex may appear widened depending on the location of the block. Wide QRS complex indicate that the ventricles are depolarising from an action potential in the ventricular tissue, rather than from or above the AV junction [3].

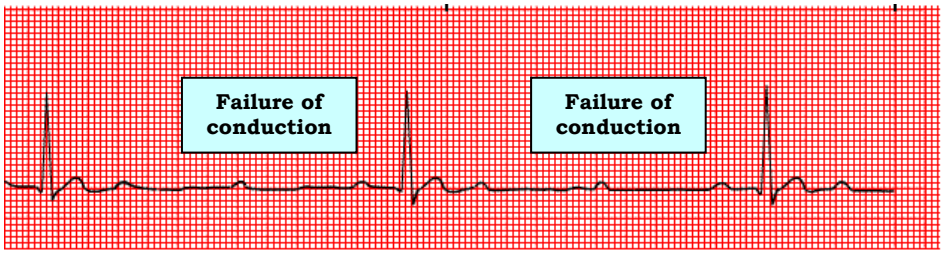


Figure 8.6 ECG tracing of a Mobitz 11 heart block

Source: ***http://www.rnceus.com/ekg/ekgsecond2.html***

In fact Mobitz type II AV block is a result of abnormal conduction below the AV node, in the bundle of His. Cardiologists believe that if type II develops it can end up to the third degree heart block.

8.3.3 Second-degree heart block: 2:1 or 3:1 conduction block

There may be alternate conducted and none conducted atrial beats giving rise to 2:1 conduction or one conducted atrial beat followed by two non-conducted beats giving 3:1 conduction.

8.3.4 Third degree heart block

This type of heart block is the most dangerous one. There is no conduction through the AV node at all.

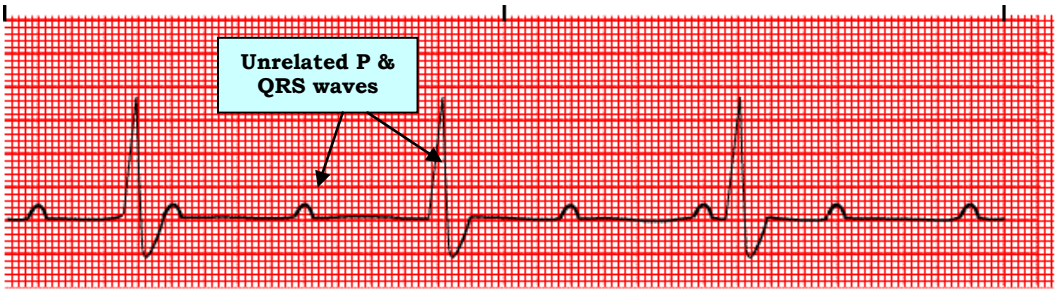


Figure 8.7 ECG tracing of a third degree heart block

Source: ***http://www.rnceus.com/ekg/ekgthird.html***

Since the different part of the heart can develop their own heart beat pattern, the atria beat at their intrinsic rate (60-80 BPM) and the ventricles, which are completely isolated from the atria beat at their slower rate of 20 –40 BPM. Some characteristics features are:

- The QRS complex will often be wide (depending on the origin of the ventricular action potential, they may remain narrow)
- The P-P interval and R-R interval will each be regular and consistent.
- The P-P interval will be faster than R-R (no relation between the two).
- Ventricular contraction will not always be preceded by an atrial contraction.

Treatment for the high degree heart blocks can be one of the following:

- Pharmacological
- Invasive
- Autonomic drugs (to inhibit vagal stimulation)
- Artificial pacemakers

We discussed various types of arrhythmias in the disease chapter but it is necessary to interpret arrhythmias by considering all the waves.

The arrhythmias will be divided in to two major types. The first type of Arrhythmia occurs in the atria, which is known as a Superventricular Arrhythmia. The second type occurs in the ventricles, which is known as a Ventricular Arrhythmia. Each type of arrhythmia has various disorders that will briefly described in this chapter.

1. **Supraventricular Arrhythmias** (occur in the atria).

- Atrial Premature Beats (APBs)
- Wandering atrial pacemaker (WAP)
- Multifocal Atrial Tachycardia (MAT)
- Supraventricular Tachycardia (SVT)
- Atrial Fibrillation (AF)
- Atrial Flutter
- Junctional Escape
- Junctional Tachycardia

2. **Ventricular Arrhythmias** (occur in the ventricles).

- Ventricular premature beats (VPB)
- Ventricular Tachycardia (VT)
- Ventricular Fibrillation (VF)

8.4 Supraventricular Arrhythmias

8.4.1 Atrial Premature Beats (APBs)

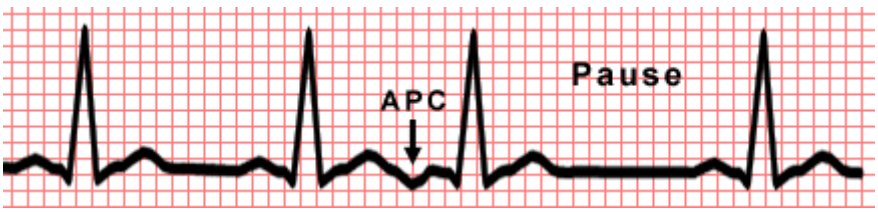


Figure 8.8 Atrial premature beats

Source: ***http://home.earthlink.net/~avdoc/infocntr/htrhythm/hrapbs.htm***

Above ECG showing that heart rate and rhythm are not normal. P wave display with a different shape and does not always precede the QRS complex, this is observed by a buried P wave in the preceding T wave. This happens due to abnormal electrical signals that arise in the atria and is known as a premature beat. A pause can be observed and it last for about1-2 seconds before it fires again. In the above ECG, there is a wait of 1.7 seconds, which is again consistent with an APB.

8.4.2 Wandering Atrial Pacemaker (WAP)

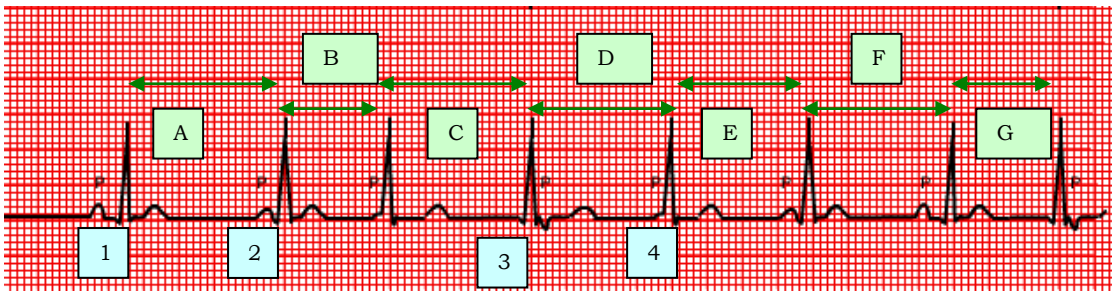


Figure 8.9 Wandering Atrial Pacemaker (WAP)

Source: ***http://www.rnceus.com/ekg/ekgwap.html***

Heart rate in points A to G in the above ECG are irregular and can be calculated by using following formula:

$$HEART\,RATE = \frac{60}{R - Rduration}$$

Point A $\Rightarrow$ Rate = $\frac{60}{(22 \times 0.04)}$ =68bpm Point B $\Rightarrow$ Rate = $\frac{60}{(15 \times 0.04)}$ =100bpm

Point C $\Rightarrow$ Rate = $\frac{60}{(20 \times 0.04)}$ =75bpm Point D $\Rightarrow$ Rate = $\frac{60}{(20 \times 0.04)}$ =75bpm

Point E $\Rightarrow$ Rate = $\frac{60}{(19 \times 0.04)}$ =79bpm Point F $\Rightarrow$ Rate = $\frac{60}{(20 \times 0.04)}$ =75bpm

Point G $\Rightarrow$ Rate = $\frac{60}{(15 \times 0.04)}$ =100bpm

It is obvious to see from above ECG that the rhythm is regular with a QRS complex subsequent to each P wave. The P-R intervals differ and QRS complex is normal in

duration. The P waves in the above ECG have different shapes with four different morphologies (1, 2, 3, 4).

Hence, this suggests a wandering Atrial pacemaker as each pacemaker gives rise to its own distinctive P wave morphology and the irregular rhythm (usually <100) obtained is a result of the multiple foci within the atria firing instead of sinus node alone [5].

8.4.3 Multifocal Atrial Tachycardia (MAT)

The medical encyclopaedia defines MAT as:

"A rapid heart rate caused by inappropriate electrical impulses arriving at the ventricles (the lower chambers of the heart) from multiple locations within the atria (the upper chambers of the heart)" [37].

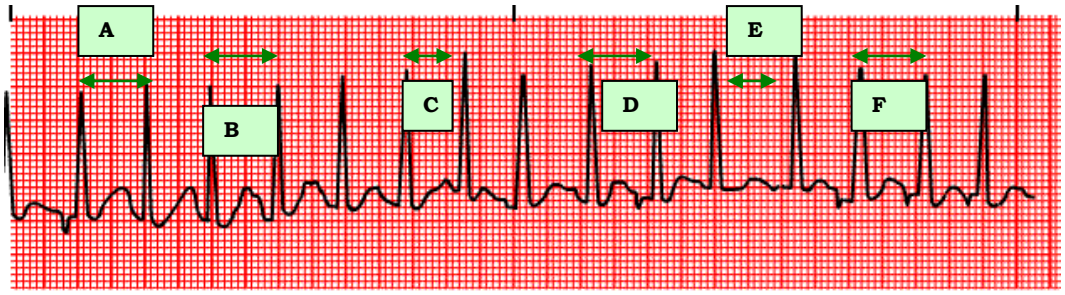


Figure 8.10 Multifocal atrial tachycardia

Source: *http://www.rnceus.com/ekg/ekgmat.html*

Above ECG represent irregular heart rate, taking random intervals gives:

Point A ⇒ Rate =150 bpm Point B ⇒ Rate =150 bpm

Point C ⇒ Rate =167 bpm Point D ⇒ Rate =167 bpm

Point E ⇒ Rate =125 bpm Point F ⇒ Rate =167 bpm

As mentioned in chapter 7, a fast heart rate is known as Tachycardia, which causes heart, to beat at more than 100 beats per minute. QRS complex is normal but P-R intervals differ. The P wave morphology differs, as there is multiple atrial pacemaker foci's firing, giving rise to different P wave morphologies.

8.4.4 Supraventricular Tachycardia (SVT)

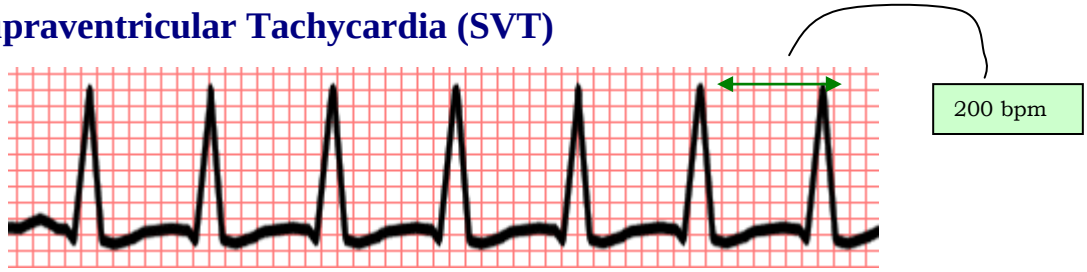


Figure 8.11 Supraventricular Tachycardia at 200 bpm

Source: ***http://home.earthlink.net/~avdoc/infocntr/htrhythm/hrapbs.htm***

As you can see above ECG has a regular heart rate at 200 bpm. The heart beat is fast and represent SVT which is also known as Atrial Tachycardia with more than 160 bpm..

8.4.5 Atrial Fibrillation (AF)

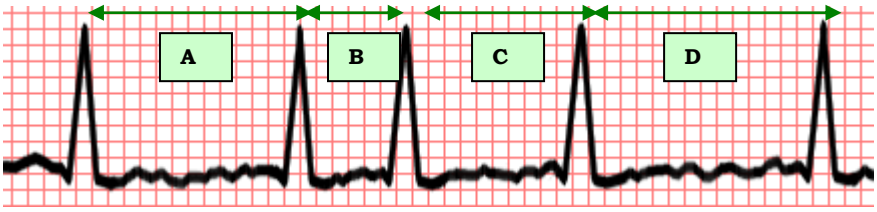


Figure 8.12 Atrial Fibrillation

Source: ***http://home.earthlink.net/~avdoc/infocntr/htrhythm/hrapbs.htm***

The heart rate is irregular at the following intervals:

Point A $\Rightarrow$ Rate = $\frac{60}{(13.5\times 0.04)}$ =111bpm Point B $\Rightarrow$ Rate = $\frac{60}{(6.5\times 0.04)}$ =231bpm

Point C $\Rightarrow$ Rate = $\frac{60}{(11\times 0.04)}$ =136bpm Point D $\Rightarrow$ Rate = $\frac{60}{(14.5\times 0.04)}$ =103bpm

Above ECG represent ventricular rate with rapid and irregular rhythm.

WAVE	OBSERVATION
P	Unclear
QRS complex	Wide
P-R interval	None

Table 8.1 PQRS observation

There is no association between the P waves and the QRS complexes.

8.4.6 Atrial Flutter

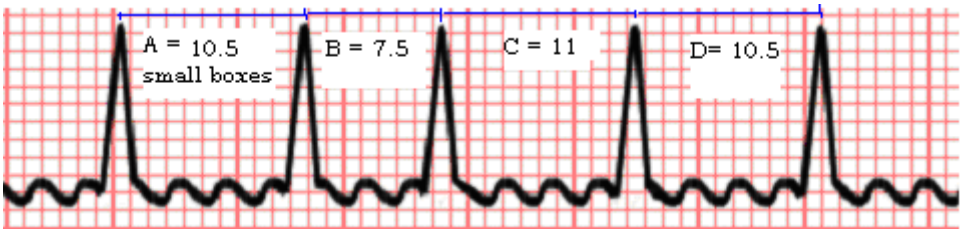


Figure 8.13 Atrial Flutter

Source: ***http://home.earthlink.net/~avdoc/infocntr/htrhythm/hraflut.htm***

From the ECG, the rate can be determined at each as:

$$\text{Point A} \Rightarrow \text{Rate} = \frac{60}{(10.5 \times 0.04)} = 143\text{bpm} \qquad \text{Point B} \Rightarrow \text{Rate} = \frac{60}{(7.5 \times 0.04)} = 200\text{bpm}$$

$$\text{Point C} \Rightarrow \text{Rate} = \frac{60}{(11 \times 0.04)} = 136\text{bpm} \qquad \text{Point D} \Rightarrow \text{Rate} = \frac{60}{(10.5 \times 0.04)} = 143\text{bpm}$$

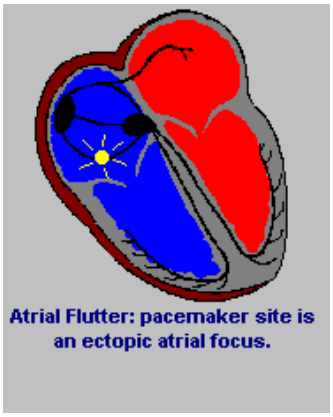


Figure 8.14 Cardiac illustration of Atrial Flutter
Source: ***http://www.cssolutions.biz/ecg2s.html***

Figure 8.13 represent rapid heart rate, as the pacemaker site is an ectopic atrial focus, the calculations also represent this irregularity. There are some points can be observed from the ECG:

- The QRS complexes are uniform in shape but irregular in rate
- There is no P-R interval
- P waves are not associated with the QRS complex
- Saw tooth figured P waves are observed

8.4.7 Junctional Escape

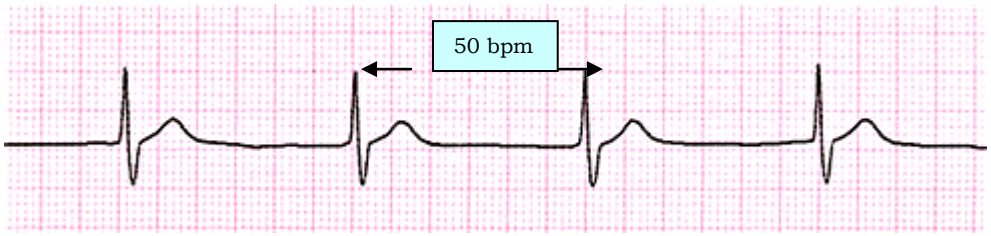


Figure 8.15 Junctional Escape rhythms

Source: ***http://medlib.med.utah.edu/kw/ecg/mml/ecg_junctional.html***

This ECG shows us that the pacemaker in the junctional tissue beats at a rate of 50 bpm and no sinus rhythm is obtained. The P waves are absent; P-R interval and QRS duration are < 0.12 seconds.

8.4.8 Junctional Tachycardia

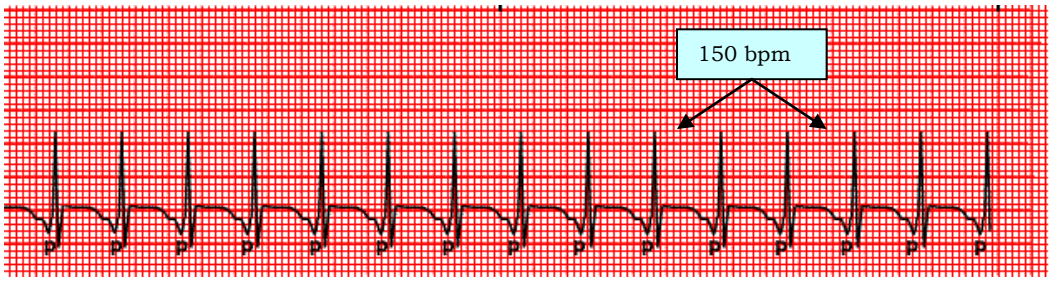
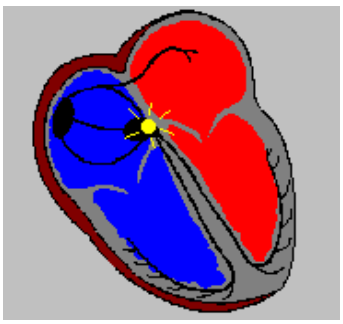


Figure 8.16 Junctional Tachycardia

Source: ***http://www.rnceus.com/ekg/ekgjt.html***

The heart rate is regular at 150 bpm. As you can see form the following diagram Junctional Tachycardia has a rapid rate, the sinus node doesn't fire and the pacemaker in the junctional tissue initiates the impulses, as seen in the diagram.



WAVE	OBSERVATION
P	Inverted
QRS complex	Normal
Rhythm	Regular

Table 8.2 PQRS observation

Figure 8.17 Cardiac illustration of Junctional Tachycardia

Source: ***http://www.cssolutions.biz/ecg2s.html***

8.5 Ventricular Arrhythmias

8.5.1 Ventricular premature beats (VPB)

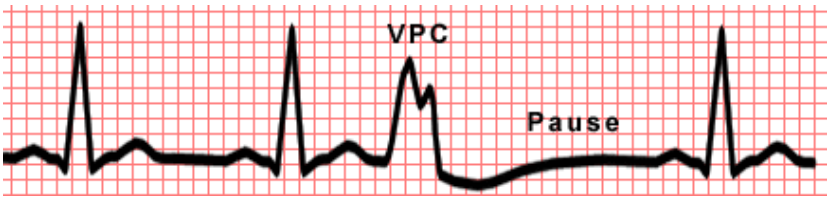


Figure 8.18 Ventricular premature beat

Source: ***http://home.earthlink.net/~avdoc/infocntr/htrhythm/hrvpbs.htm***

Ventricular premature beats (VPB) are also known as premature ventricular contractions (PVC). Heart rate and rhythm in the above ECG are irregular. Some QRS complexes are not preceded with a P wave and then followed with a pause. The reason is the beats arise from the ventricles and interrupt the heart rhythm, giving an irregular pulse and is then followed with a compensatory pause, as the next atrial beat is not conducted.

Bigeminy is the name given to VPB when every other beat is VPB and if every third beat is VPB then it is called Trigemeny.

8.5.2 Ventricular Tachycardia (VT)

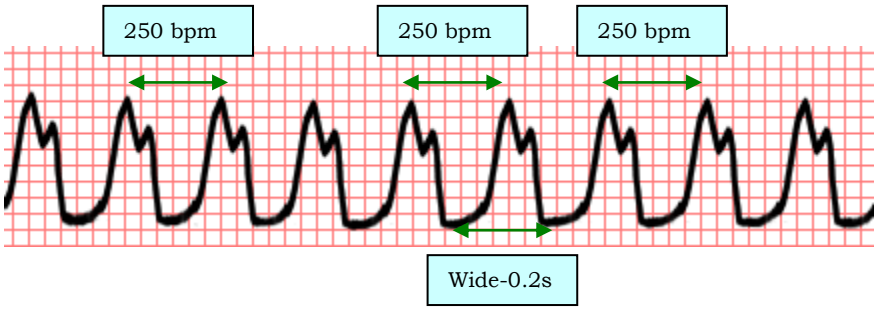


Figure 8.19 Ventricular Tachycardia

Source: ***http://home.earthlink.net/~avdoc/infocntr/htrhythm/hrvtach.htm***

A rapid and regular heart rate of 250 bpm is observed, the QRS complex is >0.12 seconds and is not correlated with the P wave.

Hence VT is diagnosed and the disturbance in the rhythm causes the ventricles to produce electrical impulses that take over the heart beat, resulting in a rapid heartbeat

When VT occurs for more than 30 seconds, it is known as sustained VT and if it exceeds 30 seconds, then it is known as unsustained VT.

8.5.3 Ventricular Fibrillation (VF)

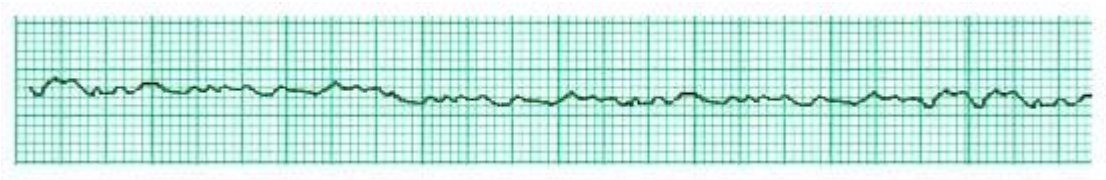


Figure 8.20 Ventricular Fibrillation

Source: ***http://sprojects.mmi.mcgill.ca/cardiophysio/venticularfibrillation.htm***

It can be clearly observed that the hearts electrical activity is disorganised causing the heart to beat in rapid, unsynchronised manner. An undulating Isoelectric line with no identifiable QRS complexes is obtained. Therefore VF is diagnosed from this interpretation.

Chapter 9

DISCUSSION

ECG is an important instrument in the medical world for diagnoses of various diseases but it is useful only if the interpretations made are accurate. It is some times not possible for a specialist to make a full accurate ECG interpretation and this is not being used to its full potential and this is why surgeons and cardiologist do not always wish to interpret the ECG as a full proof test.

9.1 Skills required for interpreting an ECG

The person wishing to work with ECG and to interpret has to know different types of ECG including normal and abnormal ECG patterns. The physician needs to have enough knowledge about various arrhythmias, heart diseases and how ECG will change in each situation. As mentioned in chapter 4, using cardiac drugs can treat some of the heart diseases so it is useful for a physician to know these drugs and the effect of each drug regarding depolarisation, repolarisation and its relevant ECG patterns.

Interpreting the ECG pattern cannot be obtained unless knowing each wave in normal and abnormal situations. In fact, ECG interpretations need lots of practice to become familiar with the meaning of each wave.

9.2 Maintaining competence in ECG interpretation

To maintain competence in ECG interpretation physicians need to make themselves update with new information in the medical world. By attending certain courses physicians can gain knowledge of the new data technology. Interpreting ECG regularly make physicians more confident in this field. Quality assurance programs enable for reviewing a sample of ECG, which is then examined by a qualified physician. By receiving the feedback physician can increase his/her accuracy to interpret.

9.3 Correct use of the ECG as a diagnostic instrument

Development of a clinical discrepancy based on the ECG allows the accurate use of the ECG as a diagnostic tool. The individuals, who want to interpret the ECG, need to be familiar with diseases associated with the heart. Wishes a diagnosis with a degree of discrepancy should be created for all abnormal ECG's and should include the disease that is responsible for the abnormal ECG along with the possible causes.

Hence, the interpretation of the ECG requires a dynamic method of thinking with the objective being the establishment of a clinical differential diagnosis [5].

9.4 Influencing factors

One of the most important issues in ECG that needs to be considered is the different factor that influences the ECG. These factors are initiated from the individuals who come to the clinic or hospitals either healthy or non-healthy. These factors are:

- Height
- Weight
- Age
- Sex
- Heart size
- Heart position
- Heart orientation

It is obvious that each two individual are different and we need to consider the above issues for each one. Individuals are different and so is their ECG. Two healthy persons may have completely different ECG's, not representative of one normal and one abnormal ECG. A person with a heart disease may be have an ECG representing no arrhythmias due to the above influencing factors Technicians would recognise that ECG for adults are different to infants and this different interpretation will be obtained and the definition of Bradycardia and Tachycardia will differ.

9.5 Recommended systematic approach to interpreting ECG

Different types of arrhythmias were discussed in chapter 8 but it is important to have systematic approach. This will exclude the data from being overlooked and enable a more reliable diagnosis. This approach is as follow:

A. Measure the rate

- Normal rate: 60-100 bpm
- <60 is Bradycardia
- >100 is Tachycardia

B. Evaluate the rhythm

- A QRS after each P is normal
  1. If not then a second or third degree AV block is diagnosed for rate <100
  2. If rate >100 then it is atrial or nodal tachycardia or atrial flutter
- A P wave before each QRS is normal if not then:
  1. Single slow beat: escape beat
  2. Slow rhythm: escape rhythm
  3. Single fast beat: premature beat (PAC if different P, PVC if QRS >0.12)
- Tachycardias if:
  1. Wide (>0.12) and rate >120 then VT
  2. Narrow and regular: no P, then AV nodal tachycardia: with preceding P then atrial Tachycardia.
  3. No P waves and unrefined baseline, then atrial fibrillation
  4. Three different P wave morphologies, then MAT

C. Measure the P-R interval, QRS interval and the axis

- P-R interval of 0.12-0.20 is normal
  1. >0.12 is 1st degree AV block
  2. <0.12 is accelerated AV conduction (if delta wave, then WPW)
- QRS complex of 0.10 is normal
  1. >0.12 is BBB, SVT, VT, WPW or Hyperkalemia
  2. <0.08 is SVT
- Axis is normal if leads 1 and 11 are positive (-30 to 90)
  1. If lead 1 is negative then RAD (90 to 150)

2. If lead 11 is negative then LAD (-30 to -90)
3. If leads 1 and 11 are negative, then extreme left axis (150 to 270)

D. Evaluate P wave

- Inverted in leads other than aVF, then ectopic atrial
- High amplitude then atrial dilation or atrial hypertrophy

E. Evaluate ST segment

- Inverted T waves can suggest Ischemia
- ST depression indicates Ischemia
- ST elevation indicates injury, Pericarditis or MI

F. Evaluate U wave

- Prominent U wave indicates Hypokalemia
- Inverted indicates MI
- High amplitude indicates Hypercalcemia, Epinephrine or Thyrotoxicosis

The above systematic approach is summarized in the following table so as we can refer to it without having any problem and understand it fully.

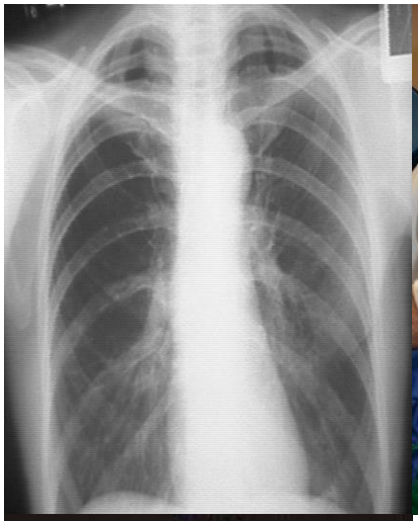
WAVES, INTERVALS & SEGMENTS	ABNORMALITIES
Normal rate: 60-100 bpm	• <60 →→→ Bradycardia • >100 →→→ Tachycardia
QRS after each P is normal	• Rate <100 →→→ second or third degree AV block • Rate >100 →→→ atrial or nodal tachycardia or atrial flutter
P wave before each QRS is normal	• Single slow beat →→→ escape beat • Slow rhythm →→→ escape rhythm •Single fast beat (premature beat) if different P→ PAC, •if QRS>0.12→PVC
	• Wide (>0.12) and rate >120 →→→ VT

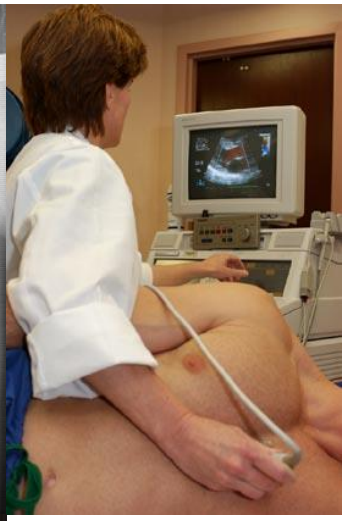
Fast heart beat	• Narrow and regular: no P →→→AV nodal tachycardia • With preceding P →→→ atrial Tachycardia. • No P waves and unrefined baseline→→ atrial fibrillation • Three different P wave morphologies→→ MAT
P-R interval of 0.12-0.20 is normal	• >0.12 →→→1st degree AV block • <0.12 →→→accelerated AV conduction (if delta wave →→→WPW)
QRS complex of 0.10 is normal	• >0.12 →→→ BBB, SVT, VT, WPW or Hyperkalemia • <0.08 →→→SVT
Axis is normal if leads 1 and 11 are positive (-30 to 90)	• If lead 1 is negative →→→ RAD (90 to 150) • If lead 11 is negative →→→ LAD (-30 to -90) • If leads 1 and 11 are negative→→→ extreme left axis (150 to 270 degree)
Evaluate P wave	• Inverted in leads other than aVF →→→ ectopic atrial • High amplitude →→→ atrial dilation or atrial hypertrophy
Evaluate ST segment	• Inverted T waves→→→ Ischemia • ST depression→→→ Ischemia • ST elevation →→→ injury, Pericarditis or MI
Evaluate U wave	• Prominent U wave →→→ Hypokalemia • Inverted →→ → MI • High amplitude →→→ Hypercalcemia, Epinephrine or Thyrotoxicosis

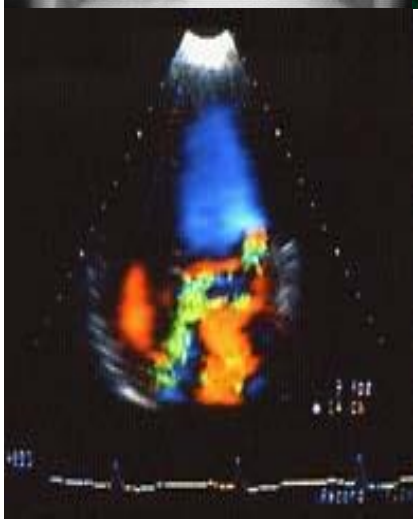
Table 9.1 Newly designed Table for the systematic approach for the ECG Evaluation

9.6 Failures to correlate the data

In order to get an accurate diagnosis it is essential to see a good correlation between ECG of the patient in the past and the current ECG. Result of different tools such as X-ray and Echocardiography can also confirm the correct diagnosis.







Failure to correlate data obtained from X-ray, Echocardiogram and ECG can have severe consequence for the patient. It is important to remember that the same condition may not be diagnosed by different methods used. It is essential to review the ECG to determine that the arrhythmias have not been overlooked.

CONCLUSION

The ECG has been used for many years and the role of the ECG can be redefined in order to increase its values and decrease its limitations. The simplicity of recording and the reasonably low cost of the routine ECG, indicates a potential value as a cost effective screening test.

It was found in the report that despite the great uses of the ECG, it is not always used to its full potential. A better and more reliable diagnosis can be obtained if regular examinations were made. This would allow the physician to make comparisons with all the ECG's obtained from routine examinations to see if any significant changes have occurred that can have important implications for clinical management decisions. It is also essential that a systematic approach is adopted in order to avoid missing subtle abnormalities that may have clinical importance. The approach that I recommended and used was very simple and enabled an easy to follow interpretation of the ECG. On completion of this report, I feel that I have acquired great knowledge on the use and interpretation of the ECG along with the numerous arrhythmias that can be detected using the ECG. I can also conclude that the level of reliability of the interpretation will improve with experience, maintaining competence and using the ECG correctly.

There have been great advances in technology; the recent development in ECG interpretation entails the use of a computerised program. This has provided good results but is still not able to outperform highly skilled physicians. Current work in this field is to construct a 3D version of the heart using visible human images from which the conducting fibres and heartbeat can be detected, both normal and abnormal.

REFERENCES

1. Human Anatomy by John W. Hole, Jr / Karen A. Koos, 1991
2. Irregular heartbeat, M.Youseffi's handout (Lecture Notes), University of Bradford.
3. Electrocardiography lecture notes, M.Youseffi (August 2002). University of Bradford.
4. Heart disease Lecture notes M.Youseffi (2002)
5. Electrocardiography interpretation and Applications by M.Youseffi (August 2003), University of Bradford.
6. BIOPAC student Lab V3.0 (Lesson 5), Medical Engineering lab sheet, University of Bradford.
7. Human biodynamic Coursework, Farshid Sefat 2004
8. Conover, M.B., Pocket Guide Series Electrocardiography, 4th edition, USA, 1998
9. ECG Lab Report, Farshid Sefat, 2003
10. 150 ECG problems by Hampton, John R, 2003
11. Cardiology by Swanton, R. H. (Robert Howard) 1989, second edition.
12. The ECG in practice by Hampton, John R1992, second edition.
13. The ECG made easy by Hampton, John R1992, 4th edition.
14. Electrocardiography, a programmed text for self-tuition in the principles of electrocardiography and the interpretation of electrocardiograms.By Owen, S. G. (Samuel Griffith), 1966.
15. Heart Disease Earl N.Silber, Louis N.Katz 1975.
16. http://www.ecglibrary.com/ecghist.html
17. http://www.brad.ac.uk/acad/mecheng/Ptwigg/index.html
18. http://biology.about.com/library/weekly/aa062801a.htm
19. http://biology.about.com/library/weekly/aa062801a.htm
20. http://biology.about.com/library/organs/heart/blmyocardium.htm
21. http://training.seer.cancer.gov/module_anatomy/unit7_2_cardvasc_heart1_structure.html
22. http://webschoolsolutions.com/patts/systems/heart.htm

23. http://www.physioweb.org/blood_vessels.html
24. http://www.maexamhelp.com/id103.htm
25. http://www.accessexcellence.org/AE/AEC/CC/heart_anatomy.html
26. http://biology.about.com/od/physiology/a/aa052104a.htm
27. http://training.seer.cancer.gov/module_anatomy/unit7_2_cardvasc_heart2_physiology.html
28. http://biology.about.com/library/organs/heart/blcardiaccycle.htm
29. http://www.anaesthetist.com/icu/organs/heart/ecg/
30. http://www.nlm.nih.gov/medlineplus/ency/article/000149.htm
31. http://www.healingwithnutrition.com/cdisease/cardiovascular/cardiovascular.html
32. http://www.biopac.com/
33. http://biology.about.com/library/weekly/aa062801a.htm
34. http://biology.about.com/library/organs/heart/blmyocardium.htm
35. http://health.yahoo.com/health/encyclopedia/003868/0.html
36. http://www.anaesthetist.com/icu/organs/heart/ecg/
37. http://www.nlm.nih.gov/medlineplus/ency/article/000186.htm
38. http://www.rjmatthewsmd.com/Definitions/pop/104b.htm
39. http://www.patrol.org/resource/davntink/heart.htm
40. http://my.webmd.com/encyclopedia/article/1675.56774
41. http://my.webmd.com/encyclopedia/article/1675.56433
42. http://www.ebme.co.uk/arts/basiccard/basiccard2.htm
43. http://www.tmc.edu/thi/valves.html
44. http://health.yahoo.com/health/encyclopedia/003868/0.html
45. http://www.nlm.nih.gov/medlineplus/ency/imagepages/1135.htm
46. http://www.heartcenteronline.com/myheartdr/common/artprn_rev.cfm?filename=&ARTID=483
47. http://biomedia.bio.purdue.edu/GenBioLM/GBECG/html/std_limb_leads.html
48. http://www.coheadquarters.com/PennLibr/MyPhysiology/lect0/figecg12.htm
49. http://www.nursingceu.com/NCEU/courses/ecg/ecg_outline/Lesson1/index.htm
50. http://medlib.med.utah.edu/kw/ecg/ecg_outline/Lesson2/index.html
51. http://www.cardioliving.com/consumer/Heart/Electrocardiogram.shtm.

52. http://cal.nbc.upenn.edu/lgcardiac/ecg_tutorial/heartrate.htm
53. http://www.rnceus.com/ekg/ekgfdav.html
54. http://www.rnceus.com/ekg/ekgsecond1.html
55. http://www.rnceus.com/ekg/ekgsecond2.html
56. http://www.rnceus.com/ekg/ekgthird.html
57. http://heartdisease.about.com/library/weekly/aa020101b.htm
58. http://heartdisease.about.com/library/weekly/aa020101b.htm
59. http://www.madsci.com/manu/ekg_hypr.htm
60. http://www.madsci.com/manu/ekg_hypr.htm
61. http://endeavor.med.nyu.edu/student-org/erclub/ekgexpl4.html
62. http://bmj.com/cgi/content/full/324/7344/1023
63. http://home.earthlink.net/~avdoc/infocntr/htrhythm/hrapbs.htm
64. http://www.rnceus.com/ekg/ekgwap.html
65. http://www.nlm.nih.gov/medlineplus/ency/article/000186.htm
66. http://www.rnceus.com/ekg/ekgmat.html
67. http://home.earthlink.net/~avdoc/infocntr/htrhythm/hrapbs.htm
68. http://home.earthlink.net/~avdoc/infocntr/htrhythm/hrapbs.htm
69. http://home.earthlink.net/~avdoc/infocntr/htrhythm/hraflut.htm
70. http://www.cssolutions.biz/ecg2s.html
71. http://medlib.med.utah.edu/kw/ecg/mml/ecg_junctional.html
72. http://www.rnceus.com/ekg/ekgjt.html
73. http://www.cssolutions.biz/ecg2s.html
74. http://home.earthlink.net/~avdoc/infocntr/htrhythm/hrvpbs.htm
75. http://home.earthlink.net/~avdoc/infocntr/htrhythm/hrvtach.htm
76. http://sprojects.mmi.mcgill.ca/cardiophysio/venticularfibrillation.htm
77. http://www.fpnotebook.com/CV61.htm
78. http://www.nda.ox.ac.uk/wfsa/html/u03/u03_011.htm