Sudan University of Science and Technology

College of Graduate Studies

Characterization of Myocardial Infarction and Therapeutic Response using Echocardiography

توصيف إحتشاء عضلة القلب ومدي الاستجابة العلاجية بواسطة التصوير بالصدي الصوتي

A thesis submitted for fulfillment of requirements of PhD degree in Medical Diagnostic Ultrasound

Bahaaedin Abdelwahid Elkhader Ali

Supervisor;

Associate Prof. Dr. Alsafi Ahmed Abdalla

Co- Supervisor;

Associate. Prof. Dr. Mohammed Ahmed Ali

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(الَّذِينَ آمَنُوا وَتَطْمَئِنُّ قُلُوبُهُمْ بِذِكْرِ اللَّهِ أَلَا بِذِكْرِ اللَّهِ تَطْمَئِنُّ الْقُلُوبُ)

{الرعد :28}

صدق الله العظيم

Dedication

I dedicate this research to My:

loving mother,

family,

friends,

wife,

children(Huthiefa,Asmaa,Aasif)

&

to the soul of my father.

Bahaaedin

Acknowledgment

First of all thanks to ALLAH for helping me to achieve this work.

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Contents

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List of Abbreviations

Abbreviation	Phrase
2 D	Two-dimensional
ACE-I	Angiotensin Converting Enzyme Inhibitors
ACS	Acute coronary syndrome
AMI	Acute myocardial infarction
AV	Atrioventricular
BMI	Body Mass Index
BSA	Body surface area
CAD	Coronary artery disease
CFM	Color-flow mapping
COPD	Chronic obstructive pulmonary disease
CT	Computed tomography
CVD	Cardiovascular disease
CW	Continuous-wave
DM	Diabetes Mellitus
EAP	Early acute pericarditis
ECG	Electrocardiogram
EDTA	Ethylenediaminetetraacetic acid
HDL	High-density lipoprotein
IHD	Ischemic Heart Disease
IVC	Inferior vena cava
IVS	Interventricular septum
LA	Left atrium
LDL	Low-density lipoprotein
LV	Left ventricle
LVEF	Left ventricular ejection fraction
LVOT	Left Ventricular Outflow Tract
MA	Metropolitan area
MHz	Megahertz
MI	Myocardial Infarction
M-mode	Motion mode
MRI	Magnetic resonance imaging
MV	Mitral Valve
PA	Pulmonary artery
PCI	Percutaneous coronary intervention.

PE	Pericardial effusion
PLA	Parasternal long –axis
PMI	Point of maximal impulse
PW	Posterior Wall
PW	Pulsed-wave
RA	Rural area
RA	Right atrium
RV	Right ventricle
SA	Sinoatrial
SHI	Second harmonic
SPSS	Standard Statistical Package for the Social Sciences
TTC	Triphenyltetrazolium chloride
TV	Tricuspid Valve
UA	Nonmetropolitan urban area
VTI	Velocity-Time Integral

ABSTRACT

The term "myocardial infarction" focuses on the myocardium (the heart muscle) and the changes that occur in it due to the sudden deprivation of circulating blood. The main change is necrosis (death) of myocardial tissue.

The word "infarction" comes from the Latin "infarcire" meaning "to plug up or cram." It refers to the clogging of the artery.

Myocardial infarction (MI) is a major health problem, with relatively high rates of morbidity and mortality. Among epidemiologists, there has been increasing interest in the characteristics of communities that influence health.

The aim of this study was to measure the predisposing factors of MI versus some parameters and to test the response of MI to treatment.

This study was conducted in the period of July 2012 to June 2014 in the ultrasound department of the Sudan Heart Center in Khartoum-Sudan, 250 participants (67.2% males and 32.8% females) and their ages range from 22 to 86 years; mean age of 41 ± 1.2 years were included in this study. Echocardiography studies were performed using MyLab 50 XVision-Esaote echocardiography machine equipped with 2.5 MHz phased array probe. Statistical Package for the Social Sciences (SPSS) was used to analyze the results.

The mean and standard deviation (SD) of left ventricle ejection fraction before prescribed drugs was (43.10±4.2). This study had demonstrated a significant increase in left ventricle ejection fraction occurred after treatment with drugs ($p < 0.001$). The value after used treatment ranged from 25 to 75 with mean and standard deviation 56.8±8.7 %.

The study confirm that echocardiography is a valuable tool for evaluation of patients with myocardial infarction.
This study conclud that; age, gender, body mass index, place of residence and patient history show significant association with myocardial infarction risk worldwide ($p < 0.001$).

الخلاصة

مصطلح "احتشاء عضلة القلب" يركز على عضلة القلب والتغيرات التي تحدث في ذلك بسبب الحرمان المفاجئ من الامداد الدموي. التغيير الرئيسي هو نخر (موت) لنسيج عضلة القلب .

كلمة "احتشاء" يأتي من "infarcire" اللاتينية التي تعني "أغلق أو حشر" ويشير إلى انسداد الشريان.

احتشاء عضلة القلب مشكلة صحية كبيرة، مع معدلات عالية نسبيا من معدلات الاعتلال والوفيات. كان هناك اهتمام متزايد بين علماء الأوبئة في خصائص المجتمعات التي تؤثر على الصحة.

الهدف من هذه الدراسة هو قياس العوامل المهيئة لاحتشاء عضلة القلب مقابل بعض المعايير واختبار استجابة احتشاء عضلة القلب للعلاج.

تمت هذه الدراسة في الفترة من يوليو 2012 وحتى يونيو 2014 بقسم الموجات فوق الصوتية لمركز السودان للقلب في الخرطوم-السودان، حيث أجريت علي 250 مشاركا (67.2٪ ذكور و 32.8٪ للإناث)، وتراوحت أعمارهم بين 22-86 سنة؛ بمتوسط عمر 41 ± 1.2 سنة. أجريت دراسات تخطيط صدى القلب باستخدام آلة تخطيط صدى القلب (MyLab 50 XVision-ESAOTE) ذات محول الطاقة 2.5 ميغاهيرتز. لتحليل النتائج تم استخدام برنامج الحزمة الإحصائية للعلوم الاجتماعية (SPSS).

المتوسط و الانحراف المعياري للجزء المقذوف للبطين الأيسرقبل استخدام الادوية كان (41.1±3.9%).وأظهرت هذه الدراسة حدوث زيادة كبيرة في الجزء المقذوف للبطين الأيسر بعد العلاج بالعقاقير (P <0.001).حيث تراوحت بعد استخدام العلاج بين 25-75 بمتوسط وانحراف معياري 56.8 ± 8.7.

أكدت الدراسة أن تخطيط صدى القلب هو أداة قيمة لتقييم المرضى الذين يعانون من احتشاء عضلة القلب.

وخلصت هذه الدراسة إلى أن. العمر والجنس ومؤشر كتلة الجسم، ومكان الإقامة و التاريخ المرضي أظهرارتباط كبير مع خطر احتشاء عضلة القلب في جميع أنحاء العالم (P <0.001).

CHAPTER ONE

INTRODUCTION

1.1 Background

The heart is a muscular organ that pumps blood throughout the various parts of the body. Anatomically, the heart lies within the mediastinum and rests on the diaphragm. Cardiac tissue differs from other muscle tissues of the body in its construction and is termed myocardium. The left side of the heart is responsible for the extensive systemic circulation; thus the left muscle wall is about three times as thick as the right side (Bontrager,2001).

The heart itself is divided into four chambers: the right and left atria and the right and left ventricles. Each chamber functions either to receive and/or pump blood. The blood circulation is a closed system, by which unoxygenated blood enters the right atrium from all parts of the body, is reoxygenated in the lungs, and returned to the body by the left ventricle (Bontrager,2001).

Myocardial infarction is the major cause of disability and death from coronary artery disease. In various industrialised countries it accounts for 10-25% of all deaths. In approximately 50% of patients the condition is fatal, and many of the remainder suffer from impaired cardiac function (Anderson, 1991).

Myocardial infarcts affect mainly the ventricular myocardium. Over 90% of infarcts are regional, i.e. involve part of the myocardium lying within the region supplied by a major coronary artery (Anderson, 1991).

Echocardiography has evolved into a highly specialized field of ultrasound .It originally began with M-mode techniques and developed into two-,three-,and even four –dimensional imaging combined with Doppler and color flow capabilities. Innovative technical advances, such as transesophageal examinations and contrast agents, added yet further diagnostic capabilities. Echocardiology serves as an ideal noninvasive method to examine cardiac anatomy in the normal as well as abnormal states .The combination of anatomical and functional information provided by echocardiography makes it the diagnostic method of choice in a variety of clinical situations (Krebs et.al, 2004).

The heart is an extremely complex organ, and echocardiography provides a variety of techniques that can be applied to obtain comprehensive information about a very dynamic organ. When performing an echocardiographic examination, it is important to consider not only the two-dimensional imaging information but also the Doppler and color flow findings. These techniques are performed as an integral part of an echocardiographic examination and should be used to complement one another (Krebs et.al, 2004).

1.2 Problem of the study:

The increase percentage of sudden death among population due to myocardial infarction. Therer are shortness of myocardial infarction studies facilities and absene of domestic reference up to author knowledge.

Therefore assessment of heart using echocardiography can help in predicting of myocardual infarction.

1.3 Objectives:

1.3.1 *General objectives*

General objectives of this study was to characterize of myocardial infarction and therapeutic response using echocardiography in order to explore the predisposing factors of MI and effect of treatment in cardiac parameters.

1.3.2 *Specific objectives:*

-To predict the occurrence of MI in young.

-To correlate between the age and MI incidence.

-To determine the incidence of MI in gender.

-To test the response of MI to treatment.

1.4 Significance of the study:

This study will have a significant importance in the myocardial infarction patients because it is the first studyin this field. It's results may contribute in health promotion program in Sudan.

This study will improve the quality of the patients' lifeand get them back to living by avoiding the complication of disease.

Also to provide accurrate information not available with standard imaging methods by using echocardiography.

1.5 Overviews of the study:

This study will fall into five chapters with chapter one is an introduction, problem of the study, objectives and overview. Chapter two include literature review while chapter three include material used and the method of data collection and analysis. Chapter four presents the result of the study in a line graphs and table and finally chapter five which include the discussion, conclusion and recommendations.

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction:

The heart is a hollow muscular organ that is somewhat pyramid shaped and lies within the pericardium in the mediastinum. It is connected at its base to the great blood vessels but otherwise lies free within the pericardium (Snell,2003).

2.2 Location and Size of the heart:

The heart is located in the thoracic cavity between the lungs. This area is called the mediastinum. The base of the cone-shaped heart is uppermost, behind the sternum, and the great vessels enter or leave here. The apex (tip) of the heart points downward and is just above the diaphragm to the left of the midline. This is why we may think of the heart as being on the left side, because the strongest beat can be heard or felt here (Scanlon & Sanders,2007).

For all its might, the heart is relatively small, roughly the same size (but not the same shape) as your closed fist. It is about 12 cm (5 in.) long, 9 cm (3.5 in.) wide at its broadest point, and 6 cm (2.5 in.) thick, with an average mass of 250 g (8 oz) in adult females and 300 g (10 oz) in adult males (Tortora& Derrickson,2008).

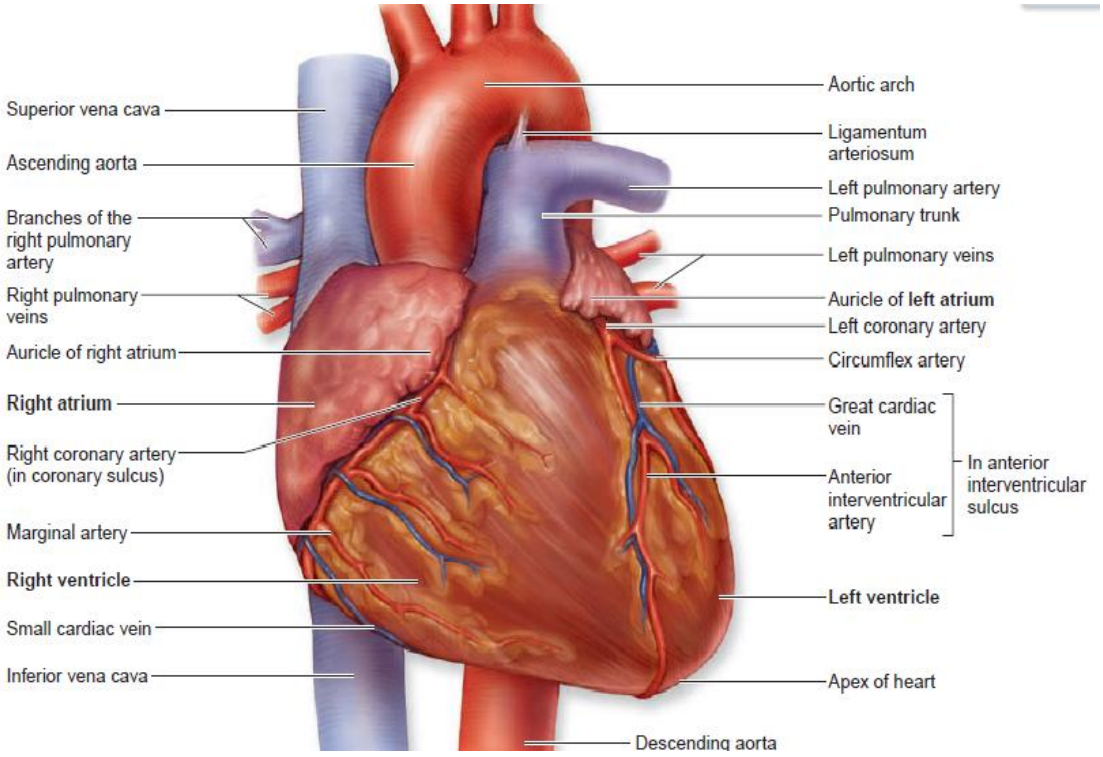

Figure. 2.1 *External Anatomy and Features of the Heart. An illustration shows the heart chambers and associated vessels and the apex of the heart in an anterior view.*

2.3 Surface of the heart:

The heart has three surfaces: sternocostal (anterior), diaphragmatic (inferior), and a base (posterior). It also has an apex, which is directed downward, forward, and to the left (Snell,2003).

The sternocostal surface is formed mainly by the right atrium and the right ventricle, which are separated from each other by the vertical atrioventricular groove. The right border is formed by the right atrium; the left border, by the left ventricle and part of the left auricle. The right ventricle is separated from the left ventricle by the anterior interventricular groove (Snell,2003).

The diaphragmatic surface of the heart is formed mainly by the right and left ventricles separated by the posterior interventricular groove. The inferior surface of

the right atrium, into which the inferior vena cava opens, also forms part of this surface (Snell,2003).

The base of the heart, or the posterior surface, is formed mainly by the left atrium, into which open the four pulmonary veins . The base of the heart lies opposite the apex (Snell,2003).

The apex of the heart, formed by the left ventricle, is directed downward, forward, and to the left . It lies at the level of the fifth left intercostal space, 3.5 in. (9 cm) from the midline. In the region of the apex, the apex beat can usually be seen and palpated in the living patient (Snell,2003).

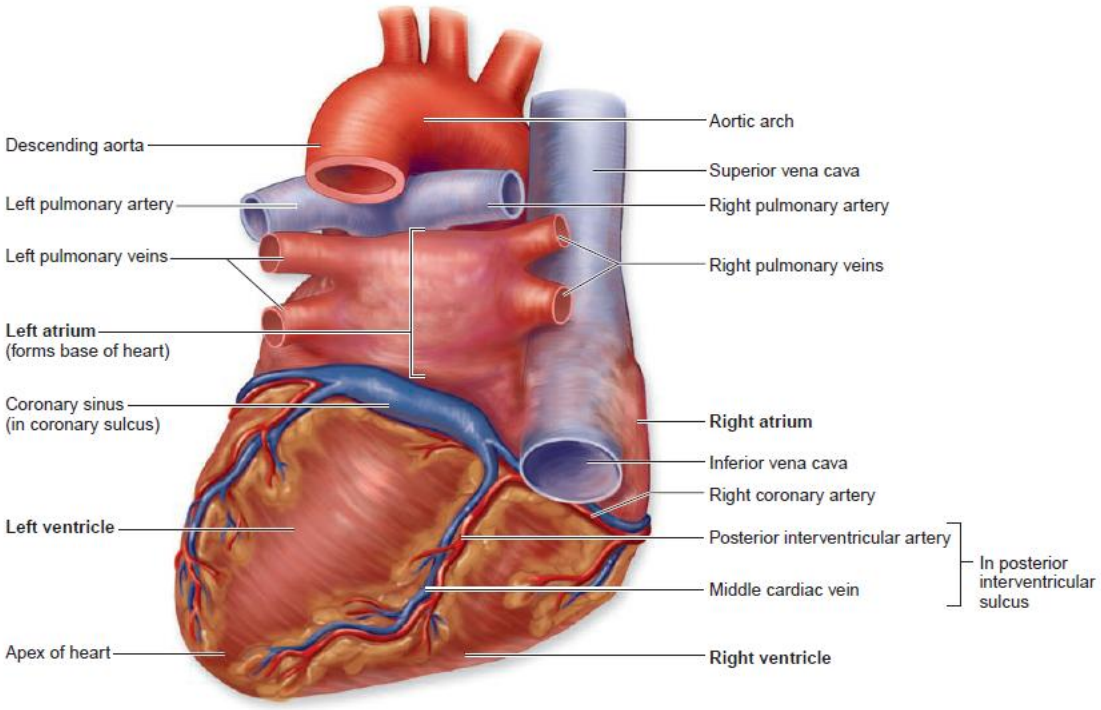

Figure. 2.2 *External Anatomy and Features of the Heart. An illustration and a shows the heart chambers and associated vessels and the base of the heart in a posterior view.*

2.4 Border of the heart:

The ***superior border*** of the heart is formed by the superior margins of the artia and is mainly hidden by the ascending aorta and the pulmonary trunk. The border extend from the upper part of the left second intercostal space (1-2 cm from the margin of the sternum) to the lower part of the same space on the right, close to the margin of the sternum . A line joining these point also marks the line of the pulmonary arteries which lie along this border of the heart (Romanes, 2008).

The ***right border*** of the heart extend from the right end of the superior border to a point on the right sixth costal cartilage 1-2 cm from the margin of the sternum. This convex border formed by the right atrium (Romanes, 2008).

The ***inferior border*** extend from the lower extremity of the right border to the apex of the heart . This lies in the fifth intercostal space immediately medial to a vertical line dropped through the midpoint of the clavicle (midioclavicular line). This border , formed mainly by the right ventricle, lies at the a lower level in the erect than in recumbent posture and in inspiration than in expiration . It is normally slightly concave , but any condition leading to the hypertrophy of the right ventricle, e. g. increased pulmonary arterial pressure, makes it convex, giving the heart a globular shape (Romanes, 2008).

The ***left border*** marked by a convex line joining the left ends of the superior and inferior border. It is formed by the left ventricle except for small part formed by the left auricle superiorly (Romanes, 2008).

The ***coronary sulcus*** lies on a line joining the sternal ends of the third left and sixth right costal cartilage (Romanes, 2008).

The ***great orifices*** of the heart and the valve which guard them lie on a line parallel and slightly inferior to the coronary sulcus.They will be seen when the heart is dissected. The pulmonary orifice lies posterior to the sternal end of the third costal cartilage. The aortic orifice is posterior to the left margin of the sternum at the level of the third intercostal space. The left atrioventricular (mitral) orifice is posterior to the left half of the sternum at the level of fourth costal cartilage. The right atrioventricular (tricuspid) orifice is posterior to the middle of the sternum at the level of fourth intercostal space (Romanes, 2008).

2.5 Heart chamber:

2.5.1 Right atrium

The right atrium receives the superior vena cava in its upper and posterior part, the inferior vena cava and coronary sinus in its lower part, and the anterior cardiac vein (draining much of the front of the heart) anteriorly.

Running more or less vertically downwards between the vena cava is a distinct muscular ridge, the *crista terminalis* (indicated on the outer surface of the atrium by a shallow groove—the *sulcus terminalis*) (Harold ,2006).

The openings of the inferior vena cava and the coronary sinus are guarded by rudimentary valves; that of the inferior vena cava being continuous with the *annulus ovalis* around the shallow depression on the atrial septum, the *fossa ovalis*, which marks the site of the fetal *foramen ovale* (Harold ,2006).

2.5.2 Right ventricle

This chamber projects to the left of the right atrium. The atrioventricular groove between the two is vertical over the front of the heart and anteroposterior on the inferior surface. It lodges the right coronary artery and is usually filled with fat.

The right ventricle narrows as it passes upwards towards the commencement of the pulmonary trunk.

The interior of the cavity, whose walls are much thicker than those of the atrium, is thrown into a series of muscular ridges and bundles, the trabeculae carneae. One of these ridges has broken free and lies in the cavity attached by its two ends to the interventricular septum and the anterior papillary muscle. This is the septomarginal trabecula (formerly the moderator band; it contains part of the right branch of the conducting bundle. Other bundles or bridges of muscle break free from the ventricular wall to form the papillary muscles which are attached to the cusps of the tricuspid valve (McMinn,2009).

The tricuspid valve guards the right atrioventricular orifice. It has three cusps and admits the tips of three fingers (the mitral valve has two cusps and admits two fingers). The three cusps, called anterior, posterior and septal, are attached by their bases to the fibrous atrioventricu1ar ring and are arranged to lie against the three walls of the ventricle - anterior, inferior and septal. The edges and ventricular surfaces of the cusps receive the attachments of the chordae tendineae, inelastic cords which diverge from the papillary muscles and prevent the cusps from being everted when the ventricle contracts. Usually the large anterior papillary muscle is connected by chordae to the anterior and posterior cusps, a smaller posterior papillary muscle is attached to the posterior and septal cusps, and several small papillary muscles join the septal and anterior cusps (McMinn,2009).

The cavity of the right ventricle is flattened by the forward bulge of the intervenoicular septum. Thus the anterior wall and septum are of equal area,

while the inferior wall (floor) is much narrower. The posterior cusp of the tricuspid valve is correspondingly smaller than the other two (McMinn,2009).

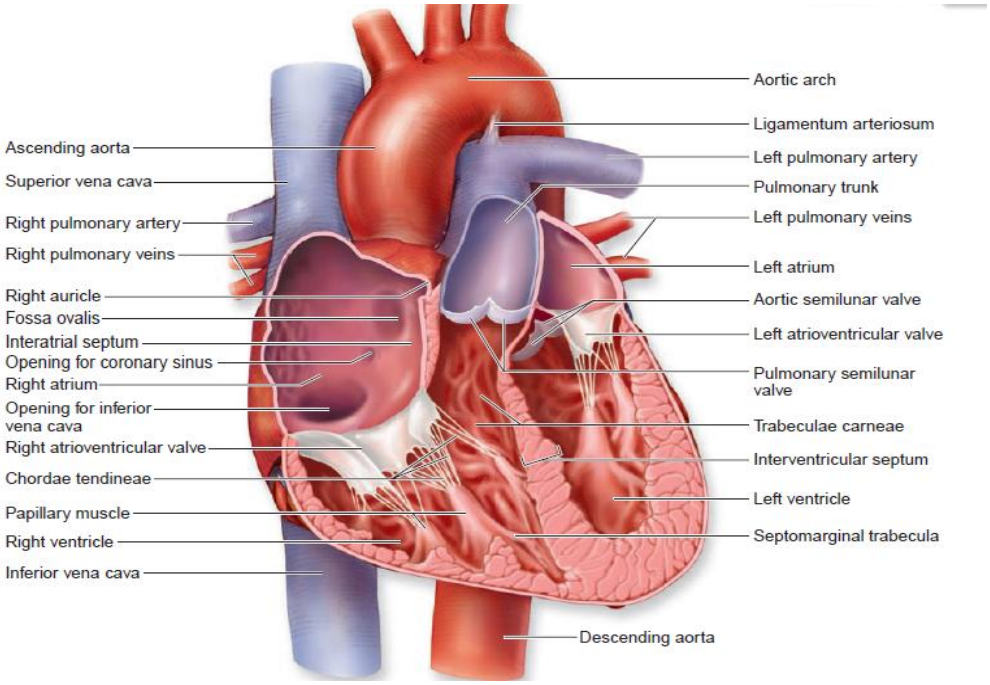

Figure. 2.3 *Internal Anatomy of the Heart. An illustration reveals the internal structure of the heart, including the valves and the musculature of the heart wall.*

2.5.3 Left Atrium

Once gas exchange occurs in the lungs, the oxygenated blood travels through the pulmonary veins to the left atrium .The smooth posterior wall of the left atrium contains openings for approximately four pulmonary veins. Sometimes two of these vessels fuse prior to reaching the left atrium, thus decreasing the number of openings through the atrial wall. Like the right atrium, the left atrium also has pectinate muscles along its anterior wall as well as an auricle (McKinley& O'Loughlin,2008).

Separating the left atrium from the left ventricle is the **left atrioventricular opening**. This opening is covered by the **left atrioventricular (AV) valve** (also

called the bicuspid valve, since it has two triangular cusps). This valve is also sometimes called the mitral valve, because the two triangular cusps resemble a miter (the headpiece worn by a bishop). Oxygenated blood flows from the left atrium, through the left atrioventricular opening when the valve is open, into the left ventricle. The left AV valve is forced closed when the left ventricle begins to contract, preventing blood backflow into the left atrium (McKinley& O'Loughlin,2008).

2.5.4 Left Ventricle

The left ventricle is the thickest chamber of the heart, averaging 10–15 mm (0.4–0.6 in.) and forms the apex of the Like the right ventricle, the left ventricle contains trabeculae carneae and has chordae tendineae that anchor the cusps of the bicuspid valve to papillary muscles (Tortora& Derrickson,2008).

The walls of this cavity are three times as thick as those of the right ventricle . The interventricular septum bulges into the cavity of the right ventricle, so that in cross section the left ventricle is circular, the right crescentic. Trabeculae carneae are well developed. There are two papillary muscles, anterior and posterior, the anterior being the: larger. Both are connected by chordae tendineae to each valve cusp. The posterior cusp receives the chordae on both its margin and its ventricular surface, but since blood is squirted across both surfaces of the anterior cusp (down through the mitral orifice and up to the aortic) the chordae are attached to it only along its margins. The upper and right end of the septal wall is smooth; between the smooth part and the anterior cusp of the mitral valve is the aortic vestibule, which leads up to the aortic orifice (McMinn,2009).

The left intraventricular blood pressure is six times higher than that inside the right ventricle (Snell,2003).

The interventricular septum lies vertically from side to side across the body: the cavity of the right ventricle lies in front of it and that of the left ventricle behind it. It is marked on the surface of the heart by the interventricular branches of right and left coronary arteries. Its muscle wall, equal in thickness to that of the left ventricle, bulges forward into the cavity of the right ventricle. At its attachment to the fibrous skeleton (conjoined atrioventricular rings) it is thinner and more fibrous. This is the membranous part of the septum, and the aortic vestibule lies between it and the anterior cusp of the mitral valve (McMinn,2009).

The aortic orifice is guarded by the aortic valve, at the: entrance to the ascending aorta. It lies at a lower level than the pulmonary orifice, rather to its right side , and is more obliquely placed. Its three semilunar cusps are named right, left and posterior (in contrast to the anterior, right and left cusps of the pulmonary valve (McMinn,2009).

During fetal life, a temporary blood vessel, called the ductus arteriosus, shunts blood from the pulmonary trunk into the aorta. Hence, only a small amount of blood enters the nonfunctioning fetal lungs . The ductus arteriosus normally closes shortly after birth, leaving a remnant known as the ligamentum arteriosum , which connects the arch of the aorta and pulmonary trunk (Tortora& Derrickson,2008).

2.6 Surface markings of valves

As far as the valves are concerned, they all lie behind the sternum, making a line with each other that is nearly vertical . The bases of tricuspid and mitral valves, attached to the atrioventricular ring, are indicated by vertical lines over the lower part of the sternum. The tricuspid valve lies behind the midline of the lower sternum, the mitral valve, overlapping it, lies higher and somewhat to the

left. The aortic and pulmonary orifices lie behind the left border of the sternum at the third costal cartilage; the pulmonary is the higher of the two (McMinn,2009).

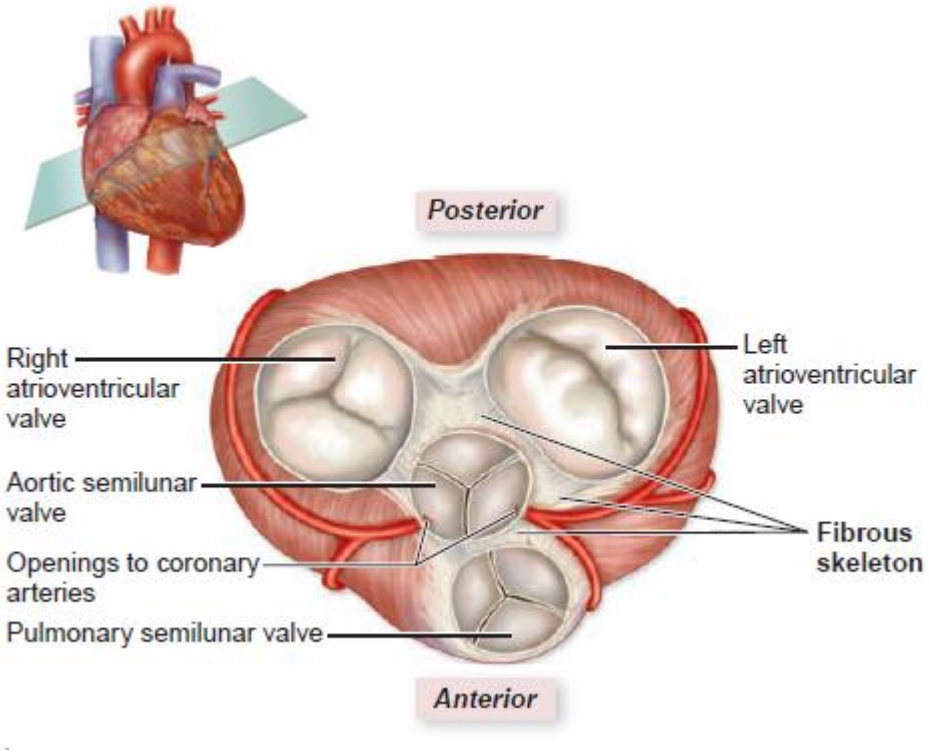

Figure. 2.4 *Heart valves in superior view. The atria have been removed. The fibrous skeleton of the heart is also shown.*

2.7 Heart Wall Structure

The heart wall consists of three distinctive layers: an external epicardium, a middle myocardium, and an internal endocardium.

The epicardium is the outermost heart layer and is also known as the visceral layer of the serous pericardium. The epicardium is composed of a serous membrane and areolar connective tissue. As we age, more fat is deposited in the epicardium, and so this layer becomes thicker and more fatty (McKinley& O'Loughlin,2008).

The myocardium is the middle layer of the heart wall and is composed of cardiac muscle tissue. The myocardium is the thickest of the three heart wall layers. It lies deep to the epicardium and superficial to the endocardium. The myocardial layer is where myocardial infarctions (heart attacks) occur. The arrangement of cardiac muscle in the heart wall permits the compression necessary to pump large volumes of blood out of the heart (McKinley& O'Loughlin,2008).

The internal surface of the heart and the external surfaces of the heart valves are covered by endocardium . The endocardium is composed of a simple squamous epithelium, called an endothelium, and a layer of areolar connective tissue (McKinley& O'Loughlin,2008).

2.8 The development of the heart

Development of the heart commences in the third week, when the embryo becomes too large to receive its nutrients through diffusion alone. At this time, the embryo needs its own blood supply, heart, and blood vessels for transporting oxygen and nutrients through its growing body. The steps involved in heart development are complex, because the heart must begin working before its development is complete (McKinley& O'Loughlin,2008).

By day 19 (middle of week 3), two heart tubes (or endocardial tubes) form from mesoderm in the embryo. By day 21, these paired tubes fuse, forming a single primitive heart tube .This tube develops the following named expansions that ultimately give rise to postnatal heart structures (McKinley& O'Loughlin,2008). The primitive heart is a single tube which soon shows grooves demarcating the sinus venosus, atrium, ventricle and bulbus cordis from behind forwards. As this

tube enlarges it kinks so that its caudal end, receiving venous blood, comes to lie behind its cephalic end with its emerging arteries (Harold ,2006).

The sinus venosus later absorbs into the atrium and the bulbus becomes incorporated into the ventricle so that, in the fully developed heart, the atria and great veins come to lie posterior to the ventricles and the roots of the great arteries (Harold ,2006).

The boundary tissue between the primitive single atrial cavity and single ventricle grows out as a dorsal and a ventral endocardial cushion which meet in the midline, thus dividing the common atrio-ventricular orifice into a right (tricuspid) and left (mitral) orifice.

The division of the primitive atrium into two is a complicated process but an important one in the understanding of congenital septal defects. Apartition, the septum primum, grows downwards from the posterior and superior walls of the primitive common atrium to fuse with the endocardial cushions. Before fusion is complete, however, a hole appears in the upper part of this septum which is termed the foramen secundum in the septum primum (Harold ,2006).

A second membrane, the septum secundum, then develops to the right of the primum but this is never complete; it has a free lower edge which does, however, extend low enough for this new septum to overlap the foramen secundum in the septum primum and hence to close it (Harold ,2006).

The two overlapping defects in the septa form the valve-like foramen ovale which shunts blood from the right to left heart in the fetus . After birth, this foramen usually becomes completely fused leaving only the fossa ovalis on the septal wall of the right atrium as its memorial. In about 10% of adult subjects, however, a probe can still be insinuated through an anatomically patent, although functionally sealed foramen (Harold ,2006).

The primitive sinus venosus absorbs into the right atrium so that the venae cavae draining into the sinus come to open separately into this atrium. The smooth-walled part of the adult atrium represents the contribution of the sinus venosus, the pectinate part represents the portion derived from the primitive atrium (Harold ,2006).

Rather similarly, the adult left atrium has a double origin. The original single pulmonary venous trunk entering the left atrium becomes absorbed into it, and donates the smooth-walled part of this chamber with the pulmonary veins entering as four separate openings; the trabeculated part of the definitive left atrium is the remains of the original atrial wall (Harold ,2006).

2.9 BLOOD SUPPLY

2.9.1 The Arterial Supply of the Heart:

The arterial supply of the heart is provided by the right and left coronary arteries, which arise from the ascending aorta immediately above the aortic valve . The coronary arteries and their major branches are distributed over the surface of the heart, lying within subepicardial connective tissue (Snell,2003).

The right coronary artery arises from the anterior aortic sinus of the ascending aorta and runs forward between the pulmonary trunk and the right auricle . It descends almost vertically in the right atrioventricular groove, and at the inferior border of the heart it continues posteriorly along the atrioventricular groove to anastomose with the left coronary artery in the posterior interventricular groove. The following branches from the right coronary artery supply the right atrium and

right ventricle and parts of the left atrium and left ventricle and the atrioventricular septum (Snell,2003).

Branches

1. The right conus artery supplies the anterior surface of the pulmonary conus (infundibulum of the right ventricle) and the upper part of the anterior wall of the right ventricle (Snell,2003).

2. The anterior ventricular branches are two or three in number and supply the anterior surface of the right ventricle. The marginal branch is the largest and runs along the lower margin of the costal surface to reach the apex (Snell,2003).

3. The posterior ventricular branches are usually two in number and supply the diaphragmatic surface of the right ventricle (Snell,2003).

4. The posterior interventricular (descending) artery .It supplies branches to the posterior part of the ventricular septum but not to the apical part, which receives its supply from the anterior interventricular branch of the left coronary artery. A large septal branch supplies the atrioventricular node.
In 10% of individuals, the posterior interventricular artery is replaced by a branch from the left coronary artery (Snell,2003).
5. The atrial branches supply the anterior and lateral surfaces of the right atrium. One branch supplies the posterior surface of both the right and left atria. The artery of the sinuatrial node supplies the node and the right and left atria; in 35% of individuals it arises from the left coronary artery (Snell,2003).

The left coronary artery, which is usually larger than the right coronary artery, supplies the major part of the heart, including the greater part of the left atrium, left ventricle, and ventricular septum. It arises from the left posterior aortic sinus of the ascending aorta and passes forward between the pulmonary trunk and the

left auricle. It then enters the atrioventricular groove and divides into an anterior interventricular branch and a circumflex branch (Snell,2003).

Branches

1. The anterior interventricular (descending) branch. Its branch supplies the right and left ventricles with numerous branches that also supply the anterior part of the ventricular septum. One of these ventricular branches (left diagonal artery) may arise directly from the trunk of the left coronary artery. A small left conus artery supplies the pulmonary conus (Snell,2003).

2. The circumflex artery is the same size as the anteriorinterventricular artery . It winds around the left margin of the heart in the atrioventricular groove. A left marginal artery is a large branch that supplies the left margin of the left ventricle down to the apex. Anterior ventricular and posterior ventricular branches supply the left ventricle. Atrial branches supply the left atrium (Snell,2003).

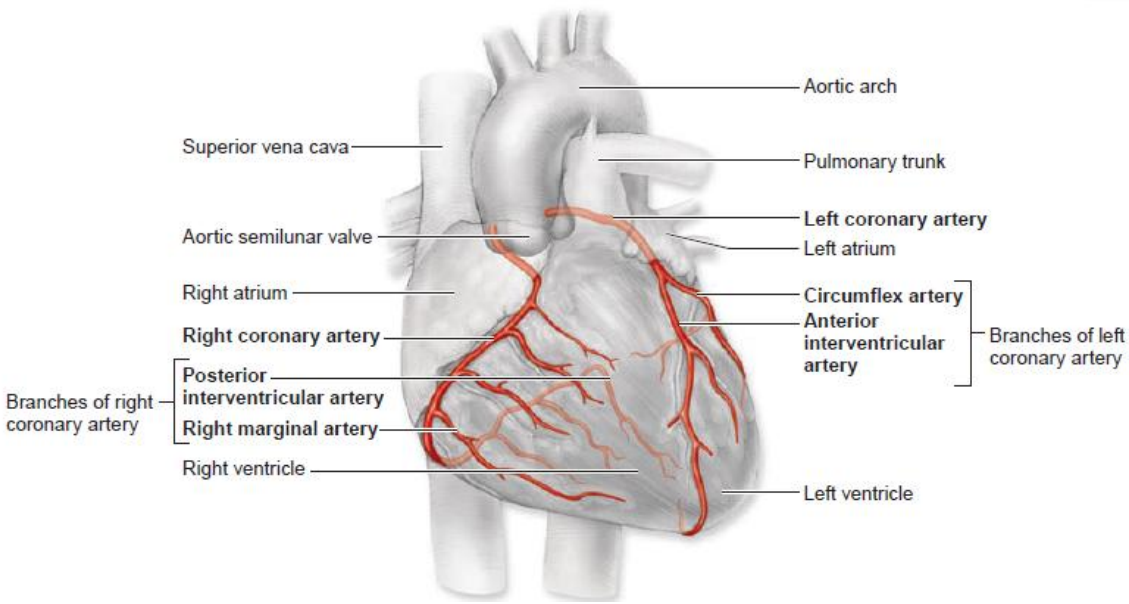

Figure. 2.5 *Coronary Circulation. Anterior view of (a) coronary arteries.*

2.9.2 Venous Drainage:

The bulk of the venous drainage of the heart is achieved by veins which accompany the coronary arteries and which open into the right atrium. The rest of the blood drains by means of small veins (venae cordis minimae) directly into the cardiac cavity (Harold ,2006).

The coronary sinus lies in the posterior atrioventricular groove and opens into the right atrium just to the left of the mouth of the inferior vena cava.

It receives:

1-the great cardiac vein in the anterior interventricular groove;

2-the middle cardiac vein the inferior interventricular groove;

3-the small cardiac vein — accompanying the marginal artery along the lower border of the heart;

4-the oblique vein— descends obliquely on the posterior aspect of the left atrium.

The anterior cardiac veins (up to three or four in number) cross the anterior atrioventricular groove, drain much of the anterior surface of the heart and open directly into the right atrium (Harold ,2006).

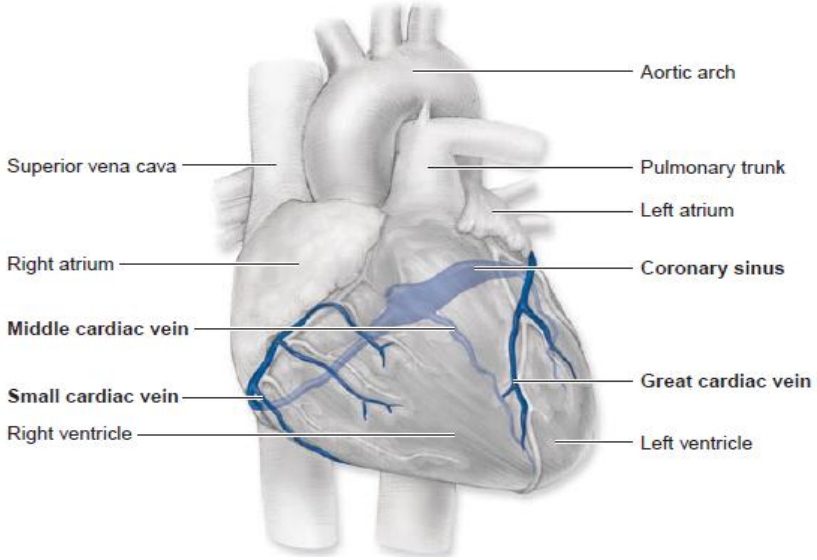

Figure. 2.6 *Coronary Circulation. Anterior view of (a) coronary veins.*

2.10 LYMPH DRAINAGE:

The lymphatics of the heart drain back along the coronary arteries, emerge from the fibrous pericardium along with the aorta and pulmonary trunk, and empty into the tracheobronchial lymph nodes and mediastinal lymph trunks (McMinn,2009).

2.11 NERVES SUPPLY

The nerve supply of the heart is derived from the vagus (cardio-inhibitor) and the cervical and upper 5 thoracic sympathetic ganglia (cardioaccelerator) by way of superficial and deep cardiac plexuses (Harold ,2006).

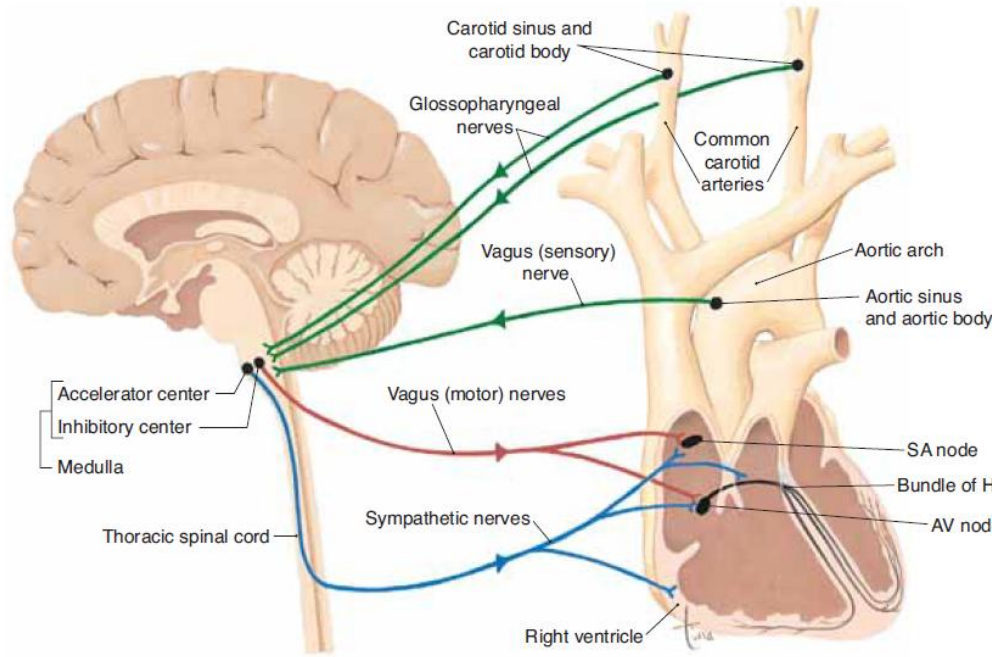

Figure. 2.7 *Nervous regulation of the heart. The brain and spinal cord are shown on the left. The heart and major blood vessels are shown on the right.*

2.12 PHYSIOLOGY OF THE HEART

The parts of the heart normally beat in orderly sequence: Contraction of the atria (atrial systole) is followed by contraction of the ventricles (ventricular systole), and during diastole all four chambers are relaxed. The heartbeat originates in a specialized cardiac conduction system and spreads via this system to all parts of the myocardium. The structures that make up the conduction system are the sinoatrial node (SA node), the internodal atrial pathways, the atrioventricular node (AV node), the bundle of His and its branches, and the Purkinje system. The various parts of the conduction system and, under abnormal conditions, parts of the myocardium are capable of spontaneous discharge. However, the SA node normally discharges most rapidly, depolarization spreading from it to the other regions before they discharge spontaneously. The SA node is therefore the normal cardiac pacemaker, its rate of discharge determining the rate at which the heart beats. Impulses generated in the SA node pass through the atrial pathways to the AV node, through this node to the bundle of His, and through the branches of the bundle of His via the Purkinje system to the ventricular muscle (Ganong,2005).

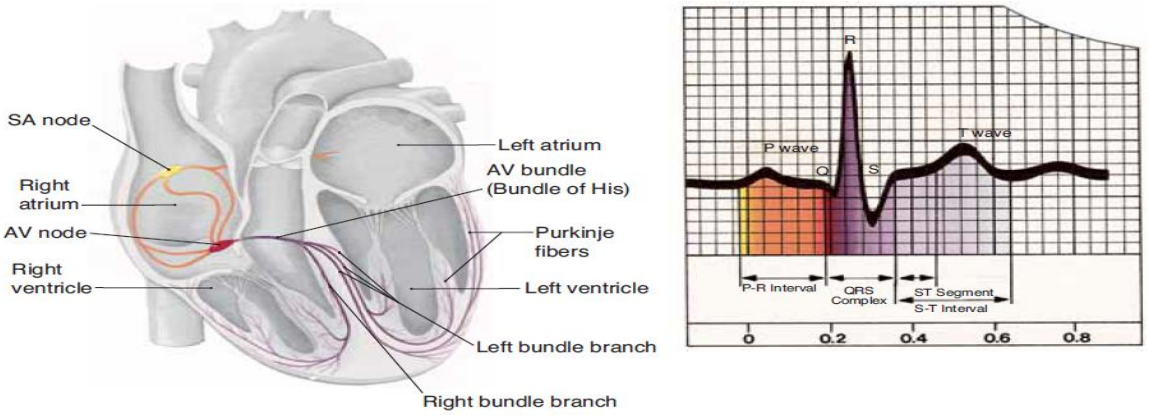

Figure. 2.8 *Conduction pathway of the heart. Anterior view of the interior of the heart. The electrocardiogram tracing is of one normal heartbeat.*

2.13 ORIGIN & SPREAD OF CARDIAC EXCITATION

2.13.1 Anatomic Considerations

In the human heart, the SA node is located at the junction of the superior vena cava with the right atrium. The AV node is located in the right posterior portion of the interatrial septum . There are three bundles of atrial fibers that contain Purkinje type fibers and connect the SA node to the AV node: the anterior internodal tract of Bachman, the middle internodal tract of Wenckebach, and the posterior internodal tract of Thorel. Conduction also occurs through atrial myocytes, but it is more rapid in these bundles. The AV node is normally the only conducting pathway between the atria and ventricles. It is continuous with the bundle of His, which gives off a left bundle branch at the top of the interventricular septum and continues as the right bundle branch. The left bundle branch divides into an anterior fascicle and a posterior fascicle. The branches and fascicles run subendocardially down either side of the septum and come into contact with the Purkinje system, whose fibers spread to all parts of the ventricular myocardium (Ganong,2005).

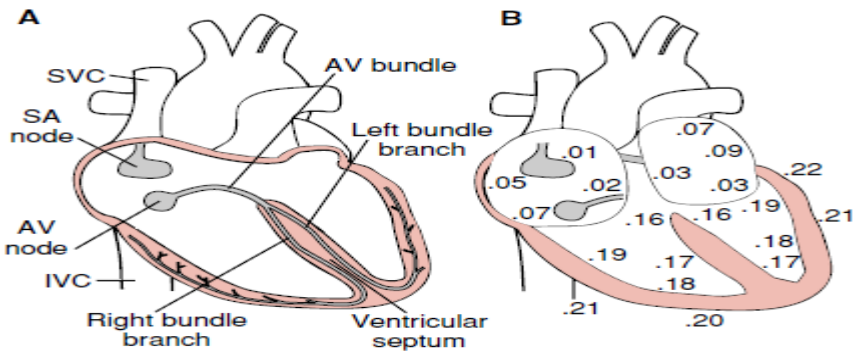

Figure. 2.9 *The timing of excitation of various areas of the heart (in fractions of a second).*

The conduction system is composed for the most part of modified cardiac muscle that has fewer striations and indistinct boundaries. The SA node and, to a lesser extent, the AV node, also contain small round cells with few organelles, which are connected by gap junctions. These are probably the actual pacemaker cells, and therefore they are called P cells. The atrial muscle fibers are separated from those of the ventricles by a fibrous tissue ring, and normally the only conducting tissue between the atria and ventricles is the bundle of His (Ganong,2005).
The heart actually is composed of two syncytiums: the atrial syncytium that constitutes the walls of the two atria, and the ventricular syncytium that constitutes the walls of the two ventricles. Normally, potentials are not conducted from the atrial syncytium into the ventricular syncytium directly through this fibrous tissue. Instead, they are conducted only by way of a specialized conductive system called the A-V bundle (Guyton & Hall,2006).

2.13.2 Action Potentials in Cardiac Muscle

The action potential recorded in a ventricular muscle Fiber, averages about 105 millivolts, which means that the intracellular potential rises from a very negative value, about -85 millivolts, between beats to a slightly positive value, about +20 millivolts, during each beat. After the initial spike, the membrane remains depolarized for about 0.2 second, exhibiting a plateau as shown in the figure, followed at the end of the plateau by abrupt repolarization. The presence of this plateau in the action potential causes ventricular contraction to last as much as 15 times as long in cardiac muscle as in skeletal muscle (Guyton & Hall,2006).

2.13.3 The Cardiac Cycle

The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called the cardiac cycle. Each cycle is initiated by spontaneous generation of an action potential in the sinus node. This node is

located in the superior lateral wall of the right atrium near the opening of the superior vena cava, and the action potential travels from here rapidly through both atria and then through the A-V bundle into the ventricles. Because of this special arrangement of the conducting system from the atria into the ventricles, there is a delay of more than 0.1 second during passage of the cardiac impulse from the atria into the ventricles. This allows the atria to contract ahead of ventricular contraction, thereby pumping blood into the ventricles before the strong ventricular contraction begins. Thus, the atria act as primer pumps for the ventricles, and the ventricles in turn provide the major source of power for moving blood through the body's vascular system (Guyton & Hall,2006).

2.14 MECHANICAL EVENTS OF THE CARDIAC CYCLE

2.14.1 Diastole and Systole

The cardiac cycle consists of a period of relaxation called diastole, during which the heart fills with blood, followed by a period of contraction called systole. The top three curves show the pressure changes in the aorta, left ventricle, and left atrium, respectively. The fourth curve depicts the changes in left ventricular volume, the fifth the electrocardiogram, and the sixth a phonocardiogram, which is a recording of the sounds produced by the heart—mainly by the heart valves—as it pumps (Guyton & Hall,2006).

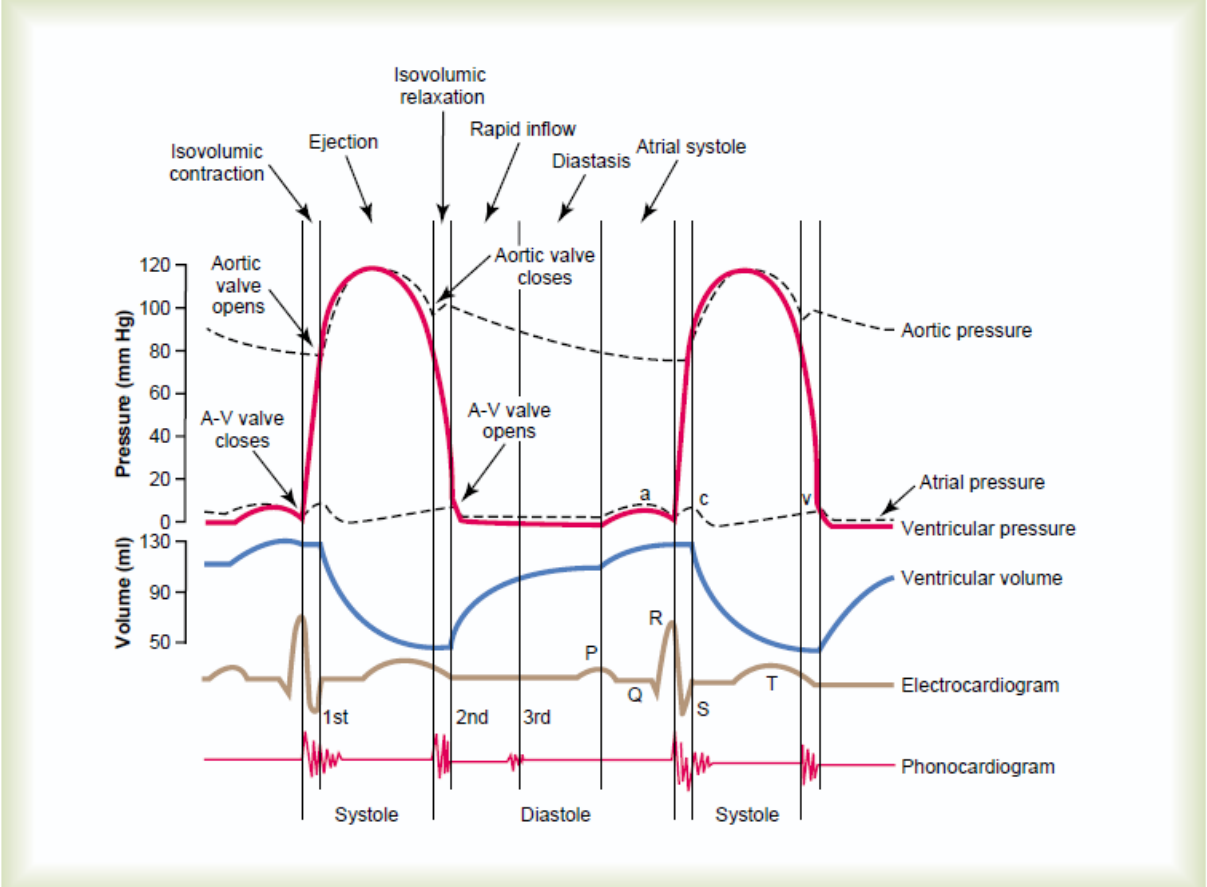

Figure. 2.10 *Events of the cardiac cycle for left ventricular function, showing changes in left atrial pressure, left ventricular pressure, aortic pressure, ventricular volume, the electrocardiogram, and the phonocardiogram.*

2.14.2 Events in Late Diastole

Late in diastole, the mitral and tricuspid valves between the atria and ventricles are open and the aortic and pulmonary valves are closed. Blood flows into the heart throughout diastole, filling the atria and ventricles. The rate of filling declines as the ventricles become distended, and—especially when the heart rate is low—the cusps of the atrioventricular (AV) valves drift toward the closed position . The pressure in the ventricles remains low (Ganong,2005).

Atrial Systole and Diastole. The P wave of the electrocardiogram (ECG) reflects atrial depolarization, which initiates atrial systole. Contraction of the atria "tops off" ventricular filling with a final, small volume of blood from the atria, producing the a wave. Under resting conditions, atrial systole is not essential for ventricular filling and, in its absence, ventricular filling is only slightly reduced. However, when increased cardiac output is required, as during exercise, the absence of atrial systole can limit ventricular filling and stroke volume. This happens in patients with atrial fibrillation, whose atria do not contract synchronously. The P wave is followed by an electrically quiet period, during which atrioventricular (AV) node transmission occurs (the PR segment). During this electrical pause, the mechanical events of atrial systole and ventricular filling are concluded before excitation and contraction of the ventricles begin. Atrial diastole follows atrial systole and occurs during ventricular systole. As the left atrium relaxes, blood enters the atrium from the pulmonary veins. Simultaneously, blood enters the right atrium from the superior and inferior vena cava. The gradual rise in left atrial pressure during atrial diastole produces the v wave and reflects its filling. The small pressure oscillation early in atrial diastole, called the c wave, is caused by bulging of the mitral valve and movements of the heart associated with ventricular contraction (Rhoades & Tanner,2003).

2.14.3 Ventricular Systole

At the start of ventricular systole, the mitral and tricuspid (AV) valves close. Ventricular muscle initially shortens relatively little, but intraventricular pressure rises sharply as the myocardium presses on the blood in the ventricle . This period of isovolumetric (isovolumic, isometric) ventricular contraction lasts about 0.05 s, until the pressures in the left and right ventricles exceed the pressures in the aorta (80 mm Hg; 10.6 kPa) and pulmonary artery (10 mm Hg) and the aortic and pulmonary valves open. During isovolumetric contraction, the AV valves bulge

into the atria, causing a small but sharp rise in atrial pressure (Ganong,2005). When the aortic and pulmonary valves open, the phase of ventricular ejection begins. Ejection is rapid at first, slowing down as systole progresses. The intraventricular pressure rises to a maximum and then declines somewhat before ventricular systole ends. Peak left ventricular pressure is about 120 mm Hg, and peak right ventricular pressure is 25 mm Hg or less. Late in systole, the aortic pressure actually exceeds the ventricular, but for a short period momentum keeps the blood moving forward. The AV valves are pulled down by the contractions of the ventricular muscle, and atrial pressure drops. The amount of blood ejected by each ventricle per stroke at rest is 70-90 mL. The end-diastolic ventricular volume is about 130 mL. Thus, about 50 mL of blood remains in each ventricle at the end of systole (end-systolic ventricular volume), and the ejection fraction, the percent of the end-diastolic ventricular volume that is ejected with each stroke, is about 65%. The ejection fraction is a valuable index of ventricular function. It can be measured by injecting radionuclide-labeled red blood cells, imaging the cardiac blood pool at the end of diastole and the end of systole (equilibrium radionuclide angiocardiography), and then calculating the ejection fraction(Ganong, 2005).

2.14.4 Early Diastole

Once the ventricular muscle is fully contracted, the already falling ventricular pressures drop more rapidly. This is the period of protodiastole. It lasts about 0.04 s. It ends when the momentum of the ejected blood is overcome and the aortic and pulmonary valves close, setting up transient vibrations in the blood and blood vessel walls. After the valves are closed, pressure continues to drop rapidly during the period of isovolumetric ventricular relaxation. Isovolumetric relaxation ends when the ventricular pressure falls below the atrial pressure and the AV valves open, permitting the ventricles to fill. Filling is rapid at first, then slows as

the next cardiac contraction approaches. Atrial pressure continues to rise after the end of ventricular systole until the AV valves open, then drops and slowly rises again until the next atrial systole. (Ganong,2005).

2.15 THE ELECTROCARDIOGRAM

The electrocardiogram (ECG) is a continuous record of cardiac electrical activity obtained by placing sensing electrodes on the surface of the body and recording the voltage differences generated by the heart. The equipment amplifies these voltages and causes a pen to deflect proportionally on a paper moving under it. This gives a plot of voltage as a function of time (Rhoades & Tanner,2003).

2.16 Function of the Atria as Primer Pumps

Blood normally flows continually from the great veins into the atria; about 80 per cent of the blood flows directly through the atria into the ventricles even before the atria contract. Then, atrial contraction usually causes an additional 20 per cent filling of the ventricles. Therefore, the atria simply function as primer pumps that increase the ventricular pumping effectiveness as much as 20 per cent. However, the heart can continue to operate under most conditions even without this extra 20 per cent effectiveness because it normally has the capability of pumping 300 to 400 per cent more blood than is required by the resting body. Therefore, when the atria fail to function, the difference is unlikely to be noticed unless a person exercises; then acute signs of heart failure occasionally develop, especially shortness of breath (Guyton & Hall,2006).

The a wave is caused by atrial contraction. Ordinarily, the right atrial pressure increases 4 to 6 mm Hg during atrial contraction, and the left atrial pressure increases about 7 to 8 mm Hg. The c wave occurs when the ventricles begin to

contract; it is caused partly by slight backflow of blood into the atria at the onset of ventricular contraction but mainly by bulging of the A-V valves backward toward the atria because of increasing pressure in the ventricles (Guyton & Hall,2006).

The v wave occurs toward the end of ventricular contraction; it results from slow flow of blood into the atria from the veins while the A-V valves are closed during ventricular contraction. Then, when ventricular contraction is over, the A-V valves open, allowing this stored atrial blood to flow rapidly into the ventricles and causing the v wave to disappear (Guyton &Hall,2006).

2.17 Function of the Ventricles as Pumps

Filling of the Ventricles. During ventricular systole, large amounts of blood accumulate in the right and left atria because of the closed A-V valves. Therefore, as soon as systole is over and the ventricular pressures fall again to their low diastolic values, the moderately increased pressures that have developed in the atria during ventricular systole immediately push the A-V valves open and allow blood to flow rapidly into the ventricles, as shown by the rise of the left ventricular volume curve.This is called the period of rapid filling of the ventricles (Guyton & Hall,2006).

Period of Ejection. When the left ventricular pressure rises slightly above 80 mm Hg (and the right ventricular pressure slightly above 8 mm Hg), the ventricular pressures push the semilunar valves open. Immediately, blood begins to pour out of the ventricles, with about 70 per cent of the blood emptying occurring during the first third of the period of ejection and the remaining 30 per cent emptying during the next two thirds. Therefore, the first third is called the period of rapid

ejection, and the last two thirds, the period of slow ejection (Guyton & Hall,2006).

Period of Isovolumic (Isometric) Relaxation. At the end of systole, ventricular relaxation begins suddenly, allowing both the right and left intraventricular pressures to decrease rapidly. The elevated pressures in the distended large arteries that have just been filled with blood from the contracted ventricles immediately push blood back toward the ventricles, which snaps the aortic and pulmonary valves closed. For another 0.03 to 0.06 second, the ventricular muscle continues to relax, even though the ventricular volume does not change, giving rise to the period of isovolumic or isometric relaxation. During this period, the intraventricular pressures decrease rapidly back to their low diastolic levels. Then the A-V valves open to begin a new cycle of ventricular pumping. (Guyton & Hall,2006)

2.18 End-Diastolic Volume, End-Systolic Volume, and Stroke Volume Output

Output. During diastole, normal filling of the ventricles increases the volume of each ventricle to about 110 to 120 milliliters. This volume is called the end-diastolic volume. Then, as the ventricles empty during systole, the volume decreases about 70 milliliters, which is called the stroke volume output. The remaining volume in each ventricle, about 40 to 50 milliliters, is called the end-systolic volume. The fraction of the end-diastolic volume that is ejected is called the ejection fraction—usually equal to about 60 per cent. When the heart contracts strongly, the end-systolic volume can be decreased to as little as 10 to 20 milliliters. Conversely, when large amounts of blood flow into the ventricles during diastole, the ventricular end diastolic volumes can become as great as 150 to 180 milliliters in the healthy heart. By both increasing the end-diastolic volume

and decreasing the end-systolic volume, the stroke volume output can be increased to more than double normal Guyton & Hall,2006).

2.19 Function of the Valves

Atrioventricular Valves. The *A-V* valves (the tricuspid and mitral valves) prevent backflow of blood from the ventricles to the atria during systole, and the semilunar valves (the aortic and pulmonary artery valves) prevent backflow from the aorta and pulmonary arteries into the ventricles during diastole. These valves, for the left ventricle, close and open passively. That is, they close when a backward pressure gradient pushes blood backward, and they open when a forward pressure gradient forces blood in the forward direction. For anatomical reasons, the thin, filmy A-V valves require almost no backflow to cause closure, whereas the much heavier semilunar valves require rather rapid backflow for a few milliseconds (Guyton & Hall,2006).

Function of the Papillary Muscles. Papillary muscles that attach to the vanes of the A-V valves by the chordae tendineae. The papillary muscles contract when the ventricular walls contract, but contrary to what might be expected, they do not help the valves to close. Instead, they pull the vanes of the valves inward toward the ventricles to prevent their bulging too far backward toward the atria during ventricular contraction. If a chorda tendinea becomes ruptured or if one of the papillary muscles becomes paralyzed, the valve bulges far backward during ventricular contraction, sometimes so far that it leaks severely and results in severe or even lethal cardiac incapacity (Guyton & Hall,2006).

Aortic and Pulmonary Artery Valves. The aortic and pulmonary artery semilunar valves function quite differently from the A-V valves. First, the high pressures in the arteries at the end of systole cause the semilunar valves to snap to

the closed position, in contrast to the much softer closure of the A-V valves. Second, because of smaller openings, the velocity of blood ejection through the aortic and pulmonary valves is far greater than that through the much larger A-V valves. Also, because of the rapid closure and rapid ejection, the edges of the aortic and pulmonary valves are subjected to much greater mechanical abrasion than are the A-V valves. Finally, the A-V valves are supported by the chordae tendineae, which is not true for the semilunar valves. It is obvious from the anatomy of the aortic and pulmonary valves that they must be constructed with an especially strong yet very pliable fibrous tissue base to withstand the extra physical stresses (Guyton & Hall,2006).

2.20 CARDIAC OUTPUT

Ejection fraction is normally more than 55%. It is dependent on heart rate, preload, afterload, and contractility and provides a nonspecific index of ventricular function. Still, it has proved to be valuable in predicting the severity of heart disease in individual patients (Rhoades & Tanner,2003).

2.20.1 Regulation of Heart Pumping

When a person is at rest, the heart pumps only 4 to 6 liters of blood each minute. During severe exercise, the heart may be required to pump four to seven times this amount. The basic means by which the volume pumped by the heart is regulated are (1) intrinsic cardiac regulation of pumping in response to changes in volume of blood flowing into the heart and (2) control of heart rate and strength of heart pumping by the autonomic nervous system (Guyton & Hall,2006).

2.20.2 Ventricular Function Curves

One of the best ways to express the functional ability of the ventricles to pump blood is by ventricular function curves. Note that as the atrial pressure for each

side of the heart increases, the stroke work output for that side increases until it reaches the limit of the ventricle's pumping ability. Another type of ventricular function curve called the ventricular volume output curve. The two curves of this figure represent function of the two ventricles of the human heart based on data extrapolated from lower animals. As the right and left atrial pressures increase, the respective ventricular volume outputs per minute also increase. Thus, ventricular function curves are another way of expressing the Frank-Starling mechanism of the heart. That is, as the ventricles fill in response to higher atrial pressures, each ventricular volume and strength of cardiac muscle contraction increase, causing the heart to pump increased quantities of blood into the arteries (Guyton & Hall,2006).

2.21 Control of the Heart by the Sympathetic and Parasympathetic Nerves

The pumping effectiveness of the heart also is controlled by the sympathetic and parasympathetic (vagus) nerves, which abundantly supply the heart. For given levels of input atrial pressure, the amount of blood pumped each minute (cardiac output) often can be increased more than 100 per cent by sympathetic stimulation. By contrast, the output can be decreased to as low as zero or almost zero by vagal (parasympathetic) stimulation (Guyton & Hall,2006).

Parasympathetic (Vagal) Stimulation of the Heart. Strong stimulation of the parasympathetic nerve fibers in the vagus nerves to the heart can stop the heartbeat for a few seconds, but then the heart usually "escapes" and beats at a rate of 20 to 40 beats per minute as long as the parasympathetic stimulation continues. In addition, strong vagal stimulation can decrease the strength of heart muscle contraction by 20 to 30 per cent. The vagal fibers are distributed mainly to the atria and not much to the ventricles, where the power contraction of the heart occurs. This explains the effect of vagal stimulation mainly to decrease heart rate

rather than to decrease greatly the strength of heart contraction. Nevertheless, the great decrease in heart rate combined with a slight decrease in heart contraction strength can decrease ventricular pumping 50 per cent or more (Guyton & Hall,2006).

2.22 Increasing the Arterial Pressure Load (up to a Limit) Does Not Decrease the Cardiac Output

Increasing the arterial pressure in the aorta does not decrease the cardiac output until the mean arterial pressure rises above about 160 mm Hg. In other words, during normal function of the heart at normal systolic arterial pressures (80 to 140 mm Hg), the cardiac output is determined almost entirely by the ease of blood flow through the body's tissues, which in turn controls venous return of blood to the heart (Guyton & Hall,2006).

2.23 PATHOLOGY OF THE HEART

Cardiovascular disease (CVD) is the leading cause of death in men and women in the United States. Because of economic advances, social structures, and demographics, it is predicted that CVD will become the leading cause of death worldwide by 2020, surpassing infectious diseases. It is estimated that the direct and indirect costs of CVD in the United States alone were $448.5 billion for 2008. To reduce this increase in morbidity, mortality, and cost, strategies such as population-based public health measures, preventative programs for high-risk subgroups, and the allocation of resources for treatments for CVD can be useful (porth & Mattfin,2009).

Although many diseases can involve the heart and blood vessels, cardiovascular dysfunction results from one or more of five principal mechanisms (Kumar, et al,2005).

• *Failure of the pump*. In the most common circumstance, the cardiac muscle contracts weakly or inadequately, and the chambers cannot empty properly. In some conditions, however, the muscle cannot relax sufficiently to permit ventricular filling (Kumar, et al,2005).

• *An obstruction to flow*, owing to a lesion preventing valve opening or otherwise causing increased ventricular chamber pressure (e.g., aortic valvular stenosis, systemic hypertension, or aortic coarctation). The increased pressure overworks the chamber that pumps against the obstruction (Kumar, et al,2005).

• *Regurgitant flow* causes some of the output from each contraction to flow backward, adding a volume workload to each of the chambers, which must pump the extra blood (e.g., left ventricle in aortic regurgitation; left atrium and left ventricle in mitral regurgitation) (Kumar, et al,2005).

• *Disorders of cardiac conduction*. Heart block or arrhythmias owing to uncoordinated generation of impulses (e.g., atrial or ventricular fibrillation) lead to nonuniform and inefficient contractions of the muscular walls.

• *Disruption of the continuity of the circulatory system* that permits blood to escape (e.g., gunshot wound through the thoracic aorta) (Kumar, et al,2005).

2.24 CORONARY ARTERY DISEASE

The term *coronary artery disease* (CAD) describes heart disease caused by impaired coronary blood flow. In most cases, CAD is caused by atherosclerosis, which affects not only the coronary arteries but arteries in other areas of the body. Diseases of the coronary arteries can cause myocardial ischemia and angina, myocardial infarction or heart attack, cardiac arrhythmias, conduction defects, heart failure, and sudden death. Heart attack is the single largest killer of men and women in the United States, Canada, and other industrialized countries. Each year, more than 1.6 million Americans have new or recurrent myocardial infarctions; one third of those die within the first 24 hours, and many of those who survive suffer significant morbidity. In spite of these numbers, the overall death rate from CAD has declined over the past several decades (porth & Mattfin,2009).

Major risk factors for CAD include cigarette smoking, elevated blood pressure, elevated serum total and low-density lipoprotein (LDL) cholesterol, low serum high-density lipoprotein (HDL) cholesterol, diabetes, advancing age, abdominal obesity, and physical inactivity. Individuals with diabetes and the metabolic syndrome are at particularly increased risk for development of cardiovascular disease (porth & Mattfin,2009).

CAD is a disorder of impaired coronary blood flow, usually caused by atherosclerosis. Myocardial ischemia occurs when there is a disparity between myocardial oxygen supply and demand and can present as chronic ischemic heart disease or ACS. Diagnostic methods for CAD include ECG, exercise stress testing, nuclear imaging studies, CT, MRI, and angiographic studies in the cardiac catheterization laboratory (porth & Mattfin,2009).

The chronic ischemic heart diseases include chronic stable angina, silent myocardial ischemia, and variant (vasospastic) angina. Chronic stable angina is associated with a fixed atherosclerotic obstruction and pain that is precipitated by increased work demands on the heart and relieved by rest. Variant angina can result from spasms of the coronary arteries or other dysfunctions. Silent myocardial ischemia occurs without symptoms (porth & Mattfin,2009).

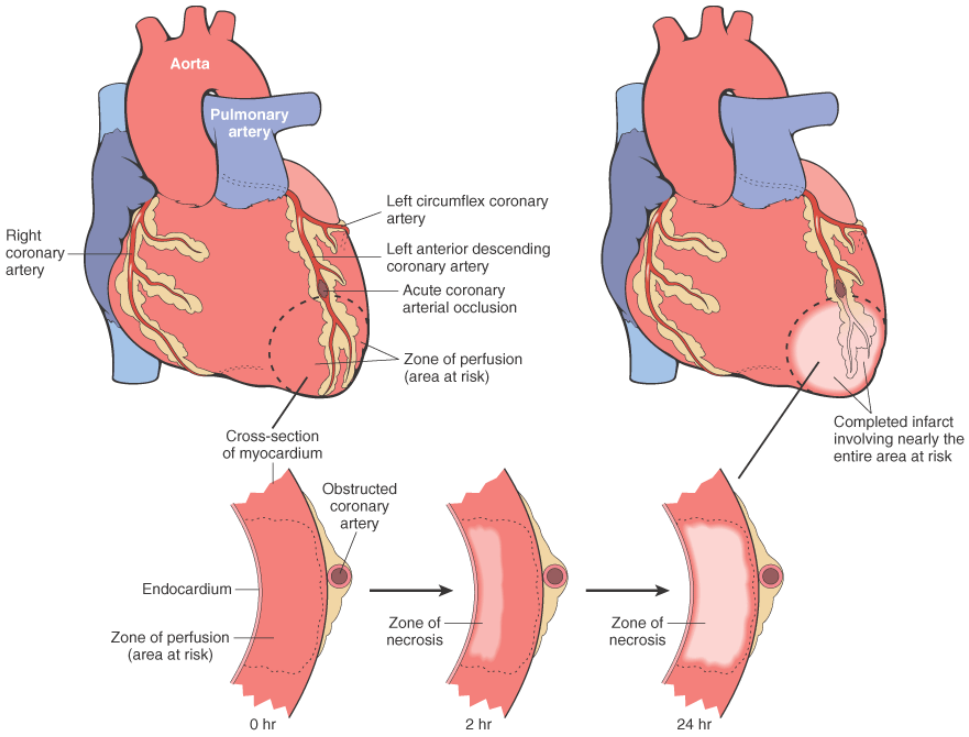

Figure. 2.11 *Schematic representation of the progression of myocardial necrosis after coronary artery occlusion. Necrosis begins in a small zone of the myocardium beneath the endocardial surface in the center of the ischemic zone.*

2.25 MYOCARDIAL INFARCTION (MI)

MI, also known as "heart attack," is the death of cardiac muscle resulting from ischemia. It is by far the most important form of IHD and alone is the leading cause of death in the United States and industrialized nations. About 1.5 million individuals in the United States suffer an acute MI annually and approximately one third of them die. At least 250,000 people a year die of a heart attack before they reach the hospital (Kumar, et al,2005).

2.25.1 Transmural versus Subendocardial Infarction

Incidence and Risk Factors.

Suffice it to say that MI may occur at virtually any age, but the frequency rises progressively with increasing age and when predispositions to atherosclerosis are present, such as hypertension, cigarette smoking, diabetes mellitus, genetic hypercholesterolemia, and other causes of hyperlipoproteinemia. Nearly 10% of myocardial infarcts occur in people under age 40, and 45% occur in people under age 65. Blacks and whites are equally affected. Throughout life, men are at significantly greater risk of MI than women; the differential progressively declines with advancing age. Except for those having some predisposing atherogenic condition, women are remarkably protected against MI during the reproductive years. Nevertheless, the decrease of estrogen following menopause can permit rapid development of coronary artery disease (CAD), and IHD is the overwhelming cause of death in elderly women. Moreover, recent epidemiologic evidence suggests that postmenopausal hormone replacement therapy does not protect women against MI (Kumar, et al,2005).

Pathogenesis.

We now consider the basis for and subsequent consequences of myocardial ischemia, particularly as they relate to the typical transmural myocardial infarct. Coronary Arterial Occlusion. transmural acute MI results from a dynamic interaction among several or all of the following-coronary atherosclerosis, acute atheromatous plaque change (such as rupture), superimposed platelet activation, thrombosis, and vasospasm—resulting in an occlusive intracoronary thrombus overlying a disrupted plaque. In addition, either increased myocardial demand (as with hypertrophy or tachycardia) or hemodynamic compromise (as with a drop in blood pressure) can worsen the situation. Recall also that collateral circulation may provide perfusion to ischemic zones from a relatively unobstructed branch of the coronary tree, bypassing the point of obstruction and protecting against the effects of an acute coronary occlusion (Kumar, et al,2005).

In the typical case of MI, the following sequence of events can be proposed:

• The initial event is a sudden change in the morphology of an atheromatous plaque, that is, disruption—manifest as intraplaque hemorrhage, erosion or ulceration, or rupture or fissuring (Kumar, et al,2005).

• Exposed to subendothelial collagen and necrotic plaque contents, platelets undergo adhesion, aggregation, activation, and release of potent aggregators including thromboxane A2 , serotonin, and platelet factors 3 and 4 (Kumar, et al,2005).

• Vasospasm is stimulated by platelet aggregation and the release of mediators (Kumar, et al,2005).

• Other mediators activate the extrinsic pathway of coagulation, adding to the bulk of the thrombus (Kumar, et al,2005).

• Frequently within minutes, the thrombus evolves to completely occlude the lumen of the coronary vessel (Kumar, et al,2005).

The evidence for this sequence is compelling and derives from (1) autopsy studies of patients dying with acute MI, (2) angiographic studies demonstrating a high frequency of thrombotic occlusion early after MI, (3) the high success rate of therapeutic thrombolysis and primary angioplasty, and (4) the demonstration of residual disrupted atherosclerotic lesions by angiography after thrombolysis. Although coronary angiography performed within 4 hours of the onset of apparent MI shows a thrombosed coronary artery in almost 90% of cases, the observation of occlusion is seen in only about 60% when angiography is delayed until 12 to 24 hours after onset. Thus with the passage of time, at least some occlusions appear to clear spontaneously owing to lysis of the thrombus or relaxation of spasm or both (Kumar, et al,2005).

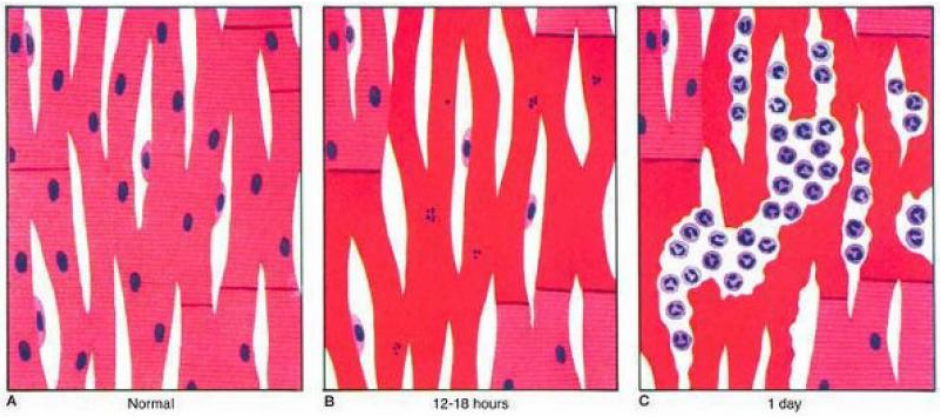

Figure. 2.12 *Development of a myocardial infarct. A. Normal myocardium. B. After about 12 to 18 hours, the infarcted myocardium shows eosinophilia (red staining) in sections of the heart stained with hematoxylin and eosin. C. About 24 hours after the onset of infarction, polymorphonuclear neutrophils infiltrate necrotic myocytes at the periphery of the infarct.*

In approximately 10% of cases, transmural acute MI is not associated with atherosclerotic plaque thrombosis stimulated by disruption. In such situations, other mechanisms may be involved:

• Vasospasm: isolated, intense, and relatively prolonged, with or without coronary atherosclerosis, perhaps in association with platelet aggregation (sometimes related to cocaine abuse) (Kumar, et al,2005).

• Emboli: from the left atrium in association with atrial fibrillation, a left-sided mural thrombus or vegetative endocarditis; or paradoxical emboli from the right side of the heart or the peripheral veins which cross to the systemic circulation, through a patent foramen ovale, causing coronary occlusion (Kumar, et al,2005).

• Unexplained: cases without detectable coronary atherosclerosis and thrombosis may be caused by diseases of small intramural coronary vessels such as vasculitis, hematologic abnormalities such as hemoglobinopathies, amyloid deposition in vascular walls, or other unusual disorders, such as vascular dissection and inadequate protection during cardiac surgery (Kumar, et al,2005).

2.25.2 Myocardial Response.

The consequence of coronary arterial obstruction is the loss of critical blood supply to the myocardium, which induces profound functional, biochemical, and morphologic consequences. Occlusion of a major coronary artery results in ischemia and, potentially, cell death throughout leading cause of mortality in IHD patients, can be caused by massive cell injury with mechanical failure but is most often due to ventricular fibrillation caused by myocardial irritability induced by ischemia or infarction. Interestingly, studies of resuscitated survivors of "sudden death" show that the majority do not develop acute MI; in such cases, myocardial irritability induced by ischemia presumably led directly to the serious arrhythmia (Kumar, et al,2005).

Irreversible injury of ischemic myocytes occurs first in the subendocardial zone. With more extended ischemia, a wavefront of cell death moves through the myocardium to involve progressively more of the transmural thickness of the ischemic zone. The precise location, size, and specific morphologic features of an acute myocardial infarct depend on: (Kumar, et al,2005).

• The location, severity, and rate of development of coronary atherosclerotic obstructions

• The size of the vascular bed perfused by the obstructed vessels

• The duration of the occlusion

• The metabolic/oxygen needs of the myocardium at risk

• The extent of collateral blood vessels

The presence, site, and severity of coronary arterial spasm

• Other factors, such as alterations in blood pressure, heart rate, and cardiac rhythm (Kumar, et al,2005).

The necrosis is largely complete within 6 hours in experimental models and humans, involving nearly all of the ischemic myocardial bed at risk supplied by the occluded coronary artery (Kumar, et al,2005).

Progression of necrosis, however, may follow a more protracted course in some patients (possibly over 6 to 12 hours or longer) in whom the coronary arterial collateral system, stimulated by chronic ischemia, is better developed and thereby more effective (Kumar, et al,2005).

Morphology

Nearly all transmural infarcts involve at least a portion of the left ventricle (including the ventricular septum). About 15% to 30% of those that affect the posterior free wall and posterior portion of the septum transmurally extend into the adjacent right ventricular wall. Isolated infarction of the right ventricle,

however, occurs in only 1% to 3% of cases. Associated infarction of atrial tissue accompanies a large posterior left ventricular infarct in some cases. Transmural infarcts usually encompass nearly the entire perfusion zone of the occluded coronary artery (Kumar, et al,2005).

Almost always there is a narrow rim (approximately 0.1 mm) of preserved subendocardial myocardium sustained by diffusion of oxygen and nutrients from the lumen (Kumar, et al,2005).

The frequencies of critical narrowing (and thrombosis) of each of the three main arterial trunks and the corresponding sites of myocardial lesions resulting in infarction (in the typical right dominant heart) are as follows:

• Left anterior descending coronary artery (40% to 50%): infarct involves anterior wall of left ventricle near apex; anterior portion of ventricular septum; apex circumferentially (Kumar, et al,2005).

• Right coronary artery (30% to 40%): infarct involves inferior/posterior wall of left ventricle; posterior portion of ventricular septum; inferior/posterior right ventricular free wall in some cases

• Left circumflex coronary artery (15% to 20%): infarct involves lateral wall of left ventricle except at apex (Kumar, et al,2005).

solution of triphenyltetrazolium chloride (TTC). This histochemical stain imparts a brick-red color to intact, noninfarcted myocardium where the dehydrogenase enzymes are preserved. Because dehydrogenases are depleted in the area of ischemic necrosis (they leak out through the damaged cell membranes), an infarcted area is revealed as an unstained pale zone (while old scarred infarcts appear white and glistening). Subsequently, by 12 to 24 hours, an infarct can be identified in routinely fixed gross slices owing to a red-blue hue caused by stagnated, trapped blood. Progressively thereafter, the infarct becomes a more sharply defined, yellow-tan, somewhat softened area that by 10 days to 2 weeks is

rimmed by a hyperemic zone of highly vascularized granulation tissue. Over the succeeding weeks, the injured region evolves to a fibrous scar (Kumar, et al,2005).

The histopathologic changes also have a fairly predictable sequence. Using light microscopic examination of routinely stained tissue sections, the typical changes of coagulative necrosis become detectable variably in the first 4 to 12 hours. "Wavy fibers" may be present at the periphery of the infarct; these changes probably result from the forceful systolic tugs by the viable fibers immediately adjacent to the noncontractile dead fibers, thereby stretching and buckling them. An additional but sublethal ischemic change may be seen in the margins of infarcts: so-called vacuolar degeneration or myocytolysis, involving large vacuolar spaces within cells, probably containing water. This potentially reversible alteration is particularly frequent in the thin zone of viable subendocardial cells. Subendocardial valvular insufficiency, only a relatively few mechanisms produce acquired valvular stenosis (Kumar, et al,2005).

2.26 SONOGRAPHY OF THE HEART

The evaluation of cardiac structures by echocardiography has many important parameters that must be fully understood and used in daily practice. Previously M-mode (time-motion mode) echocardiography was used, and it was regarded as an essential diagnostic tool for the practice of cardiology. The reason for its widespread use was its noninvasive, reproducible, and accurate assessment of cardiac structures in the evaluation of cardiac disease. The M-mode technique is limited, however, in that it provides only a one-dimensional or "icepick" view of the heart. The advent of two-dimensional echocardiography has allowed cardiac structures to be visualized in a real time fashion. Thus, the echocardiographer can now assess intracardiac lesions, observe contractility, and estimate valvular function. The combination two-dimensional and M-mode studies provides an extremely accurate means of evaluating wall thickness, valvular orifice an chamber size, and contractility of the left ventricle (Sandra,2011).

To perform a diagnostic echocardiogram examination the sonographer must be aware of anatomic and pathophysiologic parameters of the heart and understand the physical principles of sonography. These parameters are discussed relative to M-mode and two-dimensional techniques. The standard M-mode examination is presented first, followed by evaluation of the heart by combined two-dimensional and M-mode techniques (Sandra,2011).

2.26.1 Patient preparation

A basic EKG is attached to the patient assist with the timing of the cardiac cycle. leads are attached to the right chest left chest and left hip region avoiding hair if possible (Tempkin,1999).

2.26.2 Patient position

- Left lateral decubitus for most views with the left arm extended above the head and the right arm at the patients side (Tempkin,1999).
- Sub xiphoid: left lateral decubitus or supine bend the knees to relax the stomach muscles if needed (Tempkin,1999).
- Supra sterna: the patient is supine with the neck extended A pillow can also be placed under the shoulder allowing the head to drop back hyper extending the neck even further (Tempkin,1999).

2.26.3 Transducers

Several types of transducers are available for echocardiographic techniques. Ideally one should use as high a frequency as possible to improve the resolution of returning echoes. However, the higher the frequency, the less the penetration; therefore compromises have to be made to obtain the best possible image (Sandra,2011).

Many echocardiographers working with adults use a 3.5-MHz transducer with a medium focus. The larger patient may require a 2.25-MHz transducer, whereas a barrel-chested, emphysematous patient needs a 1.6-MHz transducer. The pediatric patient generally requires a 5.0- or a 7-5-MHz transducer for improved resolution and near-field definition (Sandra,2011).

Although many transducers are internally focused to improve resolution by shaping the beam and reducing distortion, most cardiac transducers are of medium focus to concentrate the maximum resolution in the area of the mitral valve (Sandra,2011).

The smaller crystal or diameter of the transducer allows better skin contact between the rib interspaces and also gives more freedom to “sweep the beam.” Thus the transducer remains in one interspace, but the beam angle is swept

obliquely from the right shoulder to the left hip to record cardiac structures (Sandra,2011).

2.26.4 Breathing techniques

For the majority of patients normal respiration. When ribs or lungs interfere having the patients either hold their breath or expel all their air and not breathe may improve the image .you may also need to slide an inter space to follow the movement of the heart. Experiment to find the best possible picture (Tempkin,1999).

2.26.5 Transducers orientation

NOTE: Hold the transducer like a pencil ,keeping two fingers on the patient at all times. This contact helps to prevent unintentional sliding and allows the sonographer to know how much pressure he or she is applying.

NOTE: To simplify the discussion of transducer orientation, imagine a clock on the patient's chest. The indicator on the transducer , which is some type of mark or indentation, will be directed anywhere from one to twelve o'clock. (To check indictor orientation side. There should be movement on the left side of the sector. Adjust L/R invert if necessary) (Tempkin,1999).

NOTE: Most movements are very slight once the proper interspace is found (Tempkin,1999).

NOTE: Remember the heart sits on an angle between the right shoulder and left hip (Tempkin,1999).

2.27 Heart survey

2.27.1 2 D EXAMINATION

- The purpose of the 2 D examination is to:

  (a) Identify the chambers and walls valves of the heart, and evaluated their size, thickness, and motion (Tempkin,1999).

  (b) Assess the anatomical relationships of structures to rule out congenital defects (Tempkin,1999).

  Document the presence of any pathology including tumors or fluid surrounding the heart, or thrombi within (Tempkin,1999).

2.27.2 Parasternal views

1.Begin with parasternal long axis by placing the transducer to the left side of the sternum in the second to third intercostals space with the indicator on the transducer directed towards 10 o'clock. Evaluate the sizes of the LA, LV, aortic root, and the RV. Assess for thickness and motion of the AV, MV, IVS, and posterior wall of the LV (Tempkin,1999).

2.Maintaining the same interspace and 10 o'clock orientation, angle the transducer inferior and medial towards the belly button. This produces the right ventricular inflow view and visualizes the more anterior structure of the heart: the RA, TV, and RV. A remnant of the Eustachian valve (a normal variant) may also be observed in the RA (Tempkin,1999).

3.To obtain the right ventricular out flow view, the transducer is now angled superior and lateral towards the left shoulder. The indicator is still directed towards10 o'clock. This will open the pulmonary artery and allow for assessment of the pulmonic valve (Tempkin,1999).

4.Rotate the transducer 90 degrees clock wise towards 1 o'clock maintaining the same inter space as above and keeping the transducer close to the sternum. The

parasternal short axis views are observed here, beginning with aortic valve level. Tilt the transducer towards the right shoulder .start by visualizing the area above the aortic valve for the presence of pathology, then slowly sweep towards the level of the aortic valve. The AV should be in the center of the screen with the LA, RV, RA, and PA surrounding it. Evaluate for the presence of three aortic cusps and note the thickness and motion of all the valve (Tempkin,1999).

5.Continue to slowly sweep laterally through the left ventricular out flow tract region towards the mitral valve. Only the angle of the transducer has changed and the ultra sound beam is now pointing almost directly anterior to posterior. Both leaflets of the mitral valve should be observed as well as its biphasic motion (Tempkin,1999).

6.Slowly angle the transducer further lateral, towards the left hip. The cross section of the LV appears round with the papillary muscles indenting the inner surface, giving the cavity a mushroom –like appearance. Assess LV function for focal or global abnormalities. Continue to sweep laterally, beyond the papillary muscles as deep in to the ventricles as possible allowing for further assessment of LV function (Tempkin,1999).

NOTE: Occasionally, you need to slide an inter space to obtain the different level of short axis , though angling is usually sufficient (Tempkin,1999).

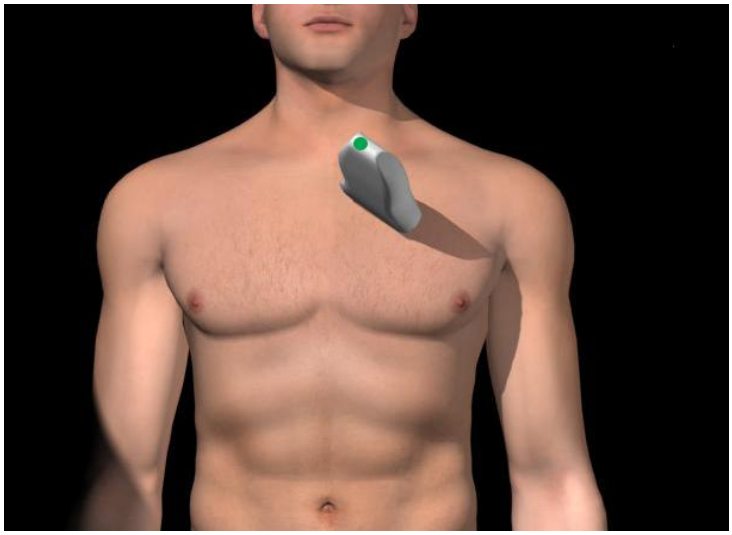

Figure. 2.13 *parasternal long axis view.*

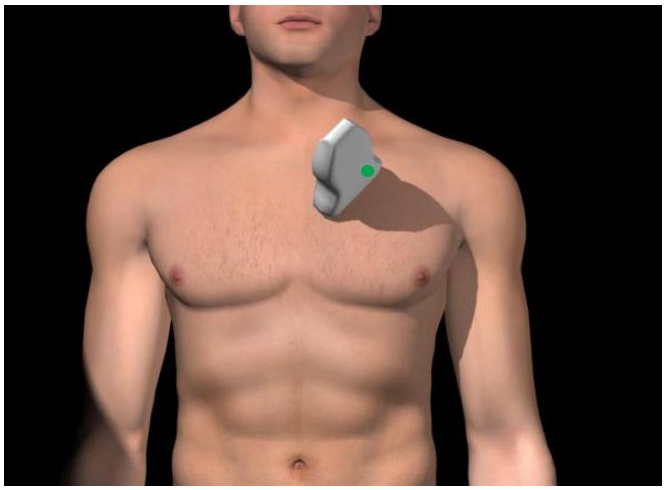

Figure. 2.14 *parasternal short axis views.*

2.27.3 Apical Views

1.Place the transducer on the left flank, lateral to the left breast and point up wards, in the direction of the right shoulder . the indicator is oriented to 3 o'clock . the heart is transected from apex to base and the for chambers, MV, TV, interventricular septum, and lateral walls in the apical region are now

visualized. Each structure is evaluated in respect to its size, thickness, and motion. This is known as the apical four chamber view (Tempkin,1999).

NOTE: If unable to find the proper apical inter space, locate the PMI(point of maximal impulse) by placing two fingers on the left side of the chest and feeling for the heart beat. The transducer is placed at this position. This is the apex of the heart (Tempkin,1999).

NOTE: It is important to visualize the endocardium of the LV in order to assess function. Evaluate the walls to see if they are thickening and there are any focal or global ischemic abnormalities. When estimating motion it is easiest to segmentalize the ventricle, looking first at the proximal, mid, then distal walls, and then to check overall function (Tempkin,1999).

2. For the apical five – chamber view, angle the transducer slightly superior to open the LVOT and the aortic valve. The MV and TV become obscured. Evaluate for the presence of any obstruction in the outflow tract region.

3. Rotate the transducer counterclockwise towards 12 o'clock while still pointing towards the right shoulder. The MV,LA, and LV are visualized and thus called the apical two –chamber view. The inferior, anterior, and apical walls of the LV can now be assessed (Tempkin,1999).

4. Rotate the transducer further counterclockwise towards 11 o'clock opening up the apical long axis view. The structures seen in parasternal long axis are visualized again in this view, but due to the different orientation, the apical region of the heart is now observed (Tempkin,1999).

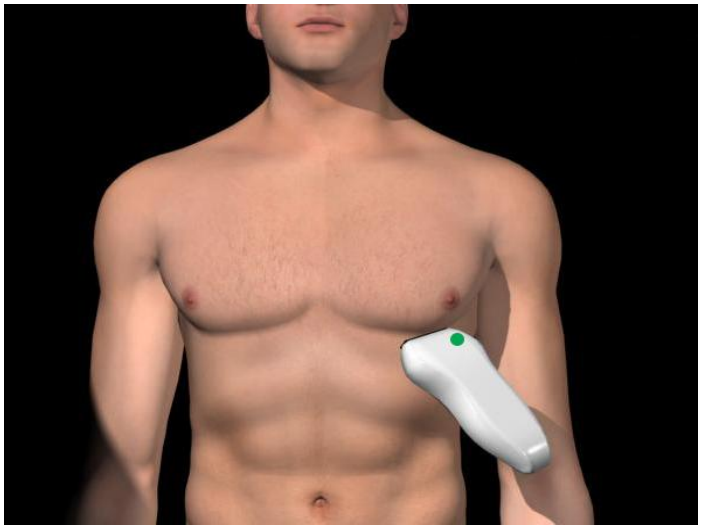

Figure. 2.15 *Apical Views.*

2.27.4 Subxiphoid views

1.To obtain the subxiphoid long axis, place the transducer below the xiphoid process and slightly to the right of midline away from the stomach on a softer portion of the abdomen. Using the liver as a window, point the transducer toward the left shoulder . Hold the hand above the transducer, rather than like a pencil. This enable the transducer to be angled under the ribs and prevents the hand from interfering with the scan. The indicator is pointed towards 3 o'clock. The four chambers of the heart are visualized and assessed for relative sizes. If the chambers appear foreshortened, the transducer should be rotated the transducer should be rotated accordingly. The area around the heart should also be evaluated for the presence of pericardial fluid, tumors, and masses. The interatrial septum is also best evaluated in this view (Tempkin,1999).

2. For the subxiphoid short axis views, rotate the transducer 90 degrees counterclockwise towards 12 o'clock. Sweeping the transducer from the direction of the left shoulder to direction of the right shoulder procedures the same three levels as the parasternal short axis views (papillary muscles, mitral valve, and aortic valves) but with the heart on a slightly different tilt. In addition, the hepatic

veins and IVC can be seen to enter the RA by pointing more rightward, beyond the aortic valve level. Make sure the IVC is clear with no thrombi (Tempkin,1999).

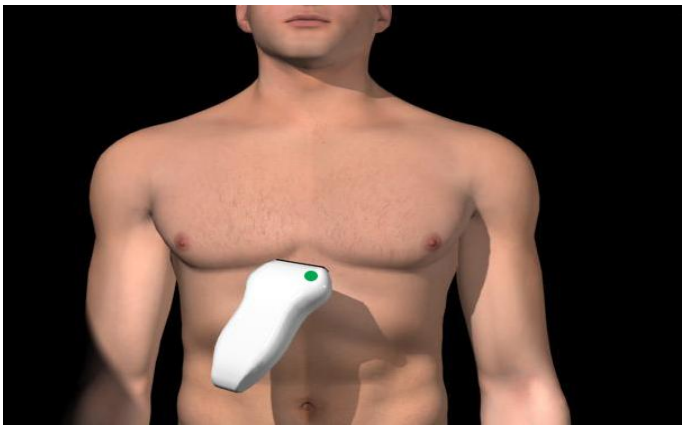

Figure. 2.16 *Subxiphoid short axis views.*

2.27.5 Suprasternal view

1.With the patient in the supine position, neck extended place the transducer at the sternoclavicular groove and angle inferior towards the heart. This will visualize the aortic arch and its branches , along with a cross section of the right pulmonary artery. The transducer is oriented towards 12o'clock.

2.For a short axis of the aorta, rotate the transducer 90 degrees clockwise to 3 o'clock. A longitudinal section of the right pulmonary artery may also be seen anterior to the LA (Tempkin,1999).

NOTE: this view should be used when questions involving the aorta arise, such as in dissection or Marfans syndrome (Tempkin,1999).

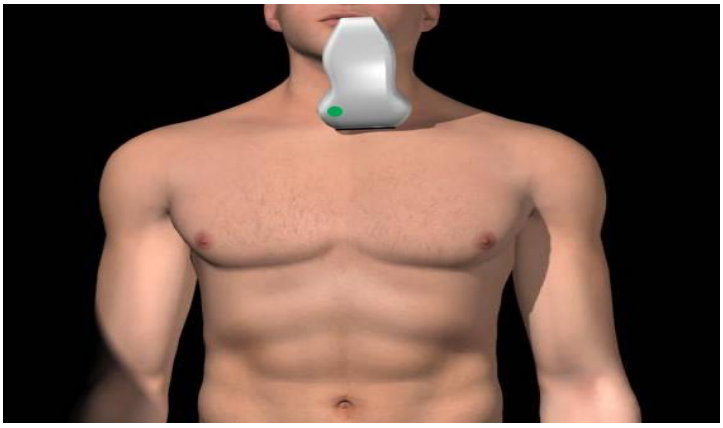

Figure. 2.17 *Suprasternal view.*

2.28 SONOGRAPHIC APPEARACE

- The pericardium is most reflective structure and appears almost white.
- The papillary muscles and myocardium are medium gray and homogeneous in ehotexture (Tempkin,1999).
- The valves are slightly more echogenic than the walls when perpendicular to the ultra sound beam (Tempkin,1999).
- The area within the chambers and great vessels as well as any other fluid space is anechoic (Tempkin,1999).

2.29 DISPLAY OF NORMAL HEART PATTERNS

The patient is generally examined in the supine or left lateral semidecubitus position. The cardiac window is usually found between the third to fifth intercostals spaces, slightly to the left of the sternal border. The cardiac window may be considered that area on the anterior chest where the heart is just beneath the skin surface, free of lung interference . With initial high-gain settings we have found it more advantageous to cover a larger area along the sternal border in the search for typical echocardiographic patterns in an effort to determine which intercostals space is the best window. When the transducer is placed along the left sternal border, the examiner should run the transducer up and down (between

the third to fifth intercostals spaces) the chest wall to define the pericardial echo with the strongest or loudest echo reflection. After the pericardium is defined, one can search for the mitral and aortic valve patterns and determine which interspace is best for demonstrating the continuity of the cardiac structures. The cardiac sonographer must keep in mind that different body shapes require variations in transverse heart, and thus a slight lateral movement from the sternal border may be needed to record cardiac structures. A thin patient may have a long and slender heart, requiring a lower, more medial transducer position. Barrel –chested patients may present with echogaraphic difficulties because of the lung absorption interference. It may be necessary to turn these patients completely on their left sides or even prone to eliminate this lung interference. Sometimes the upright or slightly bent-forward position is useful in forcing the heart closer to the anterior chest wall (Sandra,2011).

The following techniques guidelines for the average patient. In the initial echocardiographic study, moving the transducer freely along the left sterna border until all the cardiac structures are easily identified is a better practice than restricting it to one interspace. This saves time and gives the examiner a better understanding of cardiac relationships. If there is difficulty examining the patient in the supine position, a semidecubitus position should be used. Sometimes, if the heart is actually very medial, the best study is performed with the patient completely on the left side. If too much lung interference clouds the study, the patient should exhale for as long as possible. This usually gives the examiner enough time to record pieces of a valid study (Sandra,2011) .

The gain or power settings are usually increases for the initial search period and then decreases to obtain a clear tracing. The highest gain is used in the area of the left ventricle and mitral valve, with the more anterior structures, such as the aorta, tricuspid, and pulmonary valve, requiring less gain (Sandra,2011) .

2.29.1 Normal mitral valve

Echographically, the mitral valve is one of the easiest cardiac structures to recognize. The transducer should be directed perpendicular to the patient's chest wall, slightly away from the left sternal border, in approximately the fourth intercostals space. With proper gain settings, the M-mode tracings are often the most sensitive recorder of initial mitral valve motion. The cardiac sonographer may recognize the initial echo of the right ventricular wall, the echo-free cavity of the right ventricular cavity, the anterior and posterior walls of the interventricular cavity, the anterior and posterior walls of the interventricular septum, and, finally, the mitral valve apparatus as shown in the left atrial or left ventricular cavity (depending on transducer angulation). The mitral valve pattern is usually seen 6 to 9 cm from the patient's skin surface. It has the greatest amplitude and excursion and can be unquestionably recognized by its "double" or biphasic kick. This is caused by the initial opening of the valve in ventricular diastole and the atrial contraction at end diastole (Sandra,2011) .

2.29.2 Normal Aortic Valve and Left Atrium

To examine the aortic root, semilunar cusps, and left atrial cavity, the transducer should be directed cephalad toward the right shoulder, from the landmark area of the mitral valve. The cardiac sonographer should be able to identify the anterior leaflet of the mitral valve blending with the posterior aortic wall at the same time as the interventricular septum blends into the anterior aortic wall. Often there is a double parallel echo appearance along the anterior and posterior aortic walls, denoting wall thickness. Care should be taken to record both wall echoes to ensure proper measurement of the aortic root dimensions. Adjustment of the near-gain control allows excellent visualization of the anterior wall. The echoes recorded from the aortic root should be parallel, moving anteriorly in systole and posteriorly in diastole (Sandra,2011).

As the transducer is angled slightly medial, two of the three semilunar cusps can be visualized. On M-mode tracings, the right coronary cusp is shown anterior and the non-coronary posterior. When seen, the left coronary cusp is shown in the midline between the other two cusps. The onset of systole causes the cusps to open to the full extent of the aortic root. The extreme force of blood through this opening causes fine flutter to occur during systole. As the pressure relents in the ventricle, the cusps begin to drift to a closed position until they are fully closed in diastole (Sandra,2011).

The chamber posterior to the aortic root is the left atrium, which can recognized by its immobile posterior wall. As one sweeps from the mitral apparatus medially and superiorly , the left ventricular wall blends into the atrioventricular groove and finally into the left atrial wall. Thus the sweep demonstrates good contractility in the left ventricle, with anterior wall motion in systole to the atrioventricular area where the posterior wall starts to move posteriorly in systole, and then to the left atrium, where there is no movement (Sandra,2011) .

Sometimes it is possible to record the left pulmonary vein within the left atrial cavity. This appears as thin, double –walled vessel and can be a problem in determining left atrial measurements. Care should be taken to sweep from the mitral valve to the aortic root and back to the mitral apparatus several times to note the continuity of the posterior ventricular wall with the left atrial wall to avoid confusion. The pulmonary vein never appears continuous with the left ventricular wall (Sandra,2011) .

Other structures posterior to the left atrial cavity that may lead to confusion in the identification of the left atrial wall are the atrial appendage and descending aorta. The left atrial appendage may appear very prominent posterior to the left atrial wall if there is severe enlargement of the left atrial cavity (especially seen in patients with severe mitral valve disease). Real time evaluation with the

transducer in the apical four –chamber position clarifies the atrial appendage as a separate structure. The descending aorta may also be recognized as a parallel pulsating tubular structure posterior to the left atrial cavity. The aorta is not continuous with the left ventricular wall as the left atrial is; thus the cardiac sonographer should be able to distinguish this echo reflection as normal anatomy (Sandra,2011) .

2.29.3 Interventricular septum

The Interventricular septum divides the right ventricle from the left ventricle.. As the transducer is angled slightly inferior and lateral to the mitral valve, the septum moves somewhat anteriorly in early systole and posteriorly at the end of systole and early diastole (Sandra,2011) .

Both sides of the septum should move symmetrically. If they do not, the transducer should be placed more medial on the chest wall or the patient should be rolled into a slightly steeper decubitus position. Echo reflections from the chordate tendineae of the papillary muscle apparatus in the right heart may be mistaken for the right side of the septum, and care should be used to accurately identify the true right side of the septum (Sandra,2011) .

The septum thickens in systole at the midportion of the ventricular cavity. The measurement and evaluation of septal thickness and motion should be made at this point. Normal septal thickness should match that of the posterior left ventricular wall and not exceed 1.2 cm (Sandra,2011) .

2.29.4 Left Ventricle

The determination of left ventricular volume and function may be made with a routine M-mode sweep. The patient is generally examined in the left semidecubitus position to best define septal motion and left ventricular posterior wall motion. The anterior leaflet of the mitral valve should first be located and

then the beam angled slightly inferior and lateral (toward the left hip) to record the left ventricular chamber. Correct identification of this chamber may be made when both sides of the septum are seen to contract with the posterior heart wall. If the septum is not well defined or does not appear to move well, a more medical placement of the transducer along the sternal border with a lateral angulation may permit better visualization of this structure (Sandra,2011).

The three layers of the posterior heart wall-endocardium (inner layer), myocardium (middle layer), and epicardium (outer layer)-should be identified separately from the pericardium. Sometimes it is difficult to separate the epicardium from the pericardium until the gain is reduced. The myocardium usually has a fine scattering of echoes throughout its muscular layer. The endocardium may be a more difficult structure to record, since it reflects a very weak echo pattern. Sometimes the multiple chordate tendieae are difficult to separate from the endocardium, and the posterior wall must be carefully evaluated. The chordae are much denser structures than the endocardium. They generally are shown in the systolic segment along the anterior surface of the endocardium. As the ventricle contracts, the endocardial velocity is greater than the chordae tendieae velocity.

Small pieces of the mitral apparatus seen in the left ventricle ensure that the correct dimension is being evaluated. Posterior papillary muscles are shown near the apex of the ventricle. These appear as a dense, fuzzy echo band and make it difficult to evaluate the posterior wall clearly. If the ventricular volume is to be determined, these muscles are a clue that the transducer is directed too far inferior to the desired point of measurement and the cavity size would be underestimated (Sandra,2011).

2.29.5 Right Ventricle

The right ventricle is the most anterior chamber of the heart. Its anterior wall may be demonstrated with proper near-gain settings adjusted so the first moving echo shown after the immobile main bang and chest wall echoes represents the right ventricular wall. If this echo is not clearly defined, Popp has suggested an arbitrary measurement of 0.5 cm from the last nonmoving echo to serve as the right ventricular wall for right ventricular size determination. Most ventricular measurements are made in the supine position and thus must be slightly adjusted if the patient is examined in an upright or decubitus position (Sandra,2011).

2.29.6 Right Atrium

The right atrium is best seen on the longitudinal, sub-costal, two-dimensional display as the inferior vena cava sternal into. It may also be seen on the parasternal long-axis two-dimensional view as the cardiac sonographer angles the transducer medially from the level of the mitral valve to visualize the right ventricle, tricuspid valve, and right atrial cavity. The apical four-chamber view is another excellent position for evaluating the size of the right atrium (Sandra,2011).

Often fine linear echoes may be recorded within the right atrial cavity, which probably represent remnants of the Chiari network (these linear echoes are located near the interatrial septum) and the Eustachian valve (the valve found at the exit of the inferior vena cava) (Sandra,2011).

2.29.7 Normal Tricuspid Valve

The tricuspid valve is not as easily identified as the mitral valve because of its substernal location in most patients. Recordings are easily made if the right ventricle is slightly enlarged or if the heart is recorded the mitral apparatus; the beam should be angled slightly medially, under the sternum, to record the tricuspid valve. It is fairly easy to identify the whipping motion of the anterior

valve in systole and early diastole. However, the complete diastolic period reveals the pathologic changes of stenosis and regurgitation; careful angulation may allow this phase to be recorded. An alternate method of recording the valve is to locate the aortic root. The transducer beam should sweep inferiorly and medially toward the patient's right foot to record the valve leaflet (Sandra,2011).

Sometimes on M-mode scan it may be confusing to differentiate the tricuspid valve from the pulmonary valve. In the normal person the tricuspid valve is always inferior and medial to the aortic root, whereas the pulmonary valve is superior and lateral to the aorta. The other difference is that the tricuspid valve moves anteriorly with atrial contraction and the pulmonary valve dips, posteriorly (Sandra,2011).

2.29.8 Normal Pulmonary Valve

The pulmonary valve was the last of the four cardiac valves to be adequately visualized by ultrasound. Gramiak and Nanda were the first to document its echographic pattern through the aid of contrast studies. Although it is a semilunar three-cusp valve, only the left or posterior cusp can be adequately demonstrated echocardiographically (Sandra,2011).

A slow sweep from the aortic valve area, laterally and superiorly toward the left shoulder, should allow visualization of the pulmonary valve area. The parallel aortic echoes serve as a landmark in the sweep to the pulmonary valve. The anterior aortic root forms the posterior boundary of the pulmonary valve area. There should be a 2- to 4-cm space beneath the anterior chest wall and in front of this posterior border in which to visualize the pulmonary valve. Gramiak identified these posterior structures as the junction of the right ventricular outflow tract with the pulmonary artery and the atriopulmonary sulcus (with the left atrium posterior) (Sandra,2011).

When this structure complex is identified, small adjustments in beam position and direction usually pass the beam through the left pulmonary valve cusp. The appearance of the cusp is similar to that of the aortic cusp and requires very slight angulations of the beam to demonstrate fully. We have not found it easier to locate this valve in one particular position. Generally we search for the cusp with the patient in a semidecubitus position. With two dimensional capabilities, the optimal view is generally a high-parasternal short-axis view with a slight angulation of the beam toward the left shoulder (Sandra,2011).

Gramiak described the physiologic parameters as shown on the M-mode echocardiogram. At the beginning of diastole, the pulmonary valve is displaced downward and is represented anteriorly on the ultrasound recording. The low transducer position with upward beam angulation, together with the vertical inclination of the pulmonary ring, results in the examination of the valve from below. All elevations of the pulmonary valve in the stream of flow are represented as posterior movements on the echo.Likewise, downward movements are represented by anterior cusp positions on the trace (Sandra,2011).

2.30 Other Methods of Echocardiographic Examination

In a small percentage of the patients scanned the examiner cannot record adequate information from the conventional left sternal approach. This may be a function of lung interference, an unusual angulation of the cardiac structures, or relational pathology surrounding the cardiac structures. Therefore other useful approaches should be used to obtain the echographic information (Sandra,2011).

Suprasternal Approach. The suprasternal technique was first described by Goldberg. A special angulated transducer is placed in the suprasternal notch with the beam directed caudad toward the aortic arch. The transducer beam passes through the left brachiocephalic artery, aortic arch, right pulmonary artery, and

left atrium. This technique has proven useful in the further detection of aneurismal growth, tumor invasion, and in determining accurate great vessel dimensions (Sandra,2011).

Subxiphoid Approach. Chang first described the subxiphoid approach as an alternative method in the evaluation of cardiac structures obscured by lung tissue. The transducer is directed in a cephalic angulation from the subxiphoid approach. Recording can then be made of the left ventricular wall, mitral valve, and aortic valve. Although measurements cannot be obtained from this tangential approach and compared with "normal" measurements from the semidecubitus approach, this method has proven a useful technique in ruling out certain cardiac problems such as valvular disease, pericardial effusion, and tumor formation (Sandra,2011).

2.31 EVALUATION OF THE HEART WITH TWO-DIMENSIONAL ECHOCARDIOGRAPHY

The widespread clinic acceptance of the real-life two-dimensional image has tremendously aided the diagnostic results of a typical echocardiographic examination. Improved transducer design, resolution capabilities, focus parameters, gray-scale differentiation, gain control factors, cine loop functions, and other computer capabilities have aided the cardiac sonographer in the attempt to record consistent, high-quality images from the multiple scan planes necessary to obtain a composite image of the cardiac structures. In addition, most two-dimensional transducers have the combined function of imaging and selecting one crystal to perform and M-mode or a Doppler study simultaneously. Thus the addition of these functions has increased the accuracy of the M-mode and Doppler studies. The ability to actually image the cardiac anatomy has also improved the speed with which an M-mode study can be performed. Most equipment has the capability of computing dimensions of various cardiac structures in either two-dimensional or M-mode. More advanced

echocardiographic equipment has the added feature of combined pulsed-wave (PW) and continuous-wave (CW) Doppler capabilities, with the further ability to actually steer the CW Doppler beam (Sandra,2011).

The recent addition of color-flow Doppler has added a new dimension for the cardiac sonographer in detecting in tracardiac shunt flow, mapping out regurgitant pathways, and in determining obstructive flow pathways (Sandra,2011).

2.31.1 Two-Dimensional Echocardiography

Nomenclature and Image Orientation. The Committee on Nomenclature and Standards in Two-Dimensional Echocardiography of the American Society of Echocardiography recommends the following nomenclature and image orientation standards for transducer location: (Sandra,2011).

Suprasternal Transducer placed in the suprasternal notch.

Subcostal transducer located near body midline and beneath costal margin.

Apical transducer located over cardiac apex (at the point of maximal impulse) (Sandra,2011).

Parasternal transducer placed over the area bounded superiorly by left clavicle, medially by sternum, and inferiorly by apical region (Sandra,2011).

Imaging planes these planes are described by the manner in which the two-dimensional transducer transects the heart (Sandra,2011).

Long axis transects heart perpendicular to dorsal and ventral surfaces of body and parallel with long axis of heart (Sandra,2011).

short axis transects heart perpendicular to dorsal and ventral surfaces of body and perpendicular with long axis of heart.

Four chamber transects heart approximately parallel with dorsal and ventral surfaces of body (Sandra,2011).

2.31.2 The two dimensional examination

A routine two-dimensional examination for the adult and pediatric patient usually begins with the patient in a semileft lateral decubitus position. This position allows the heart to move away from the sternum and closer to the chest wall, thus allowing a better cardiac window (Sandra,2011).

Parasternal long –Axis Views. The parasternal long –axis (PLA) view should be used first in the echographic examination. An attempt should be made to record as many of the cardiac structures as possible (from the base of heart to apex). Generally this is accomplished by placing the transducer slightly to the left of the sternum in about the fourth intercostals space. When the bright echo reflection of the pericardium is noted, the transducer is gradually rotated until a long- axis view of the heart is obtained. If it is not possible to record the entire long axis on a single scan, the transducer should be gently rocked cephalad to caudad in an “ice-pick” fashion to record all the information from the base to the apex of the heart.

The cardiac sonographer should observe the following structures and functions in the PLA view: (Sandra,2011)

1-Composite size of the cardiac chambers

2-Contractility of the right and left ventricles

3-Thickness of the right ventricular wall

4-Continuity of the interventricular septum with the anterior wall of the aorta

5-Pliability of the atrioventricular and semilunar valves

6-Coaptation of the atrioventricular valves

7-Presence of increased echoes on the atrioventricular and semilunar valves

8-Systolic clearance of the aortic cusps

9-Presence of abnormal echo collections in the chambers or attached to the valve orifice

10-Presence and movement of chordal-papillary muscle structure

11-Thickness of the septum and posterior wall of the left ventricle

12-Uniform texture of the endocardium and myocardium

13-Size of the aortic root

Respective M-mode tracings should then be made in these areas:

1. Record the aortic root at the level of the cusp opening.

2.Record the size of the left atrium.

3.Sweep the beam from the aortic root to the mitral valve:

a. Demonstrate right side of interventricular septum to anterior aortic wall continuity.

b. Demonstrate posterior aortic wall to anterior leaflet mitral valve continuity.

c. Show transition from left atrial wall to atrioventricular groove to posterior wall of the left ventricle (Sandra,2011).

4.Record the anterior leaflet of the mitral valve at the tip of the leaflet

5.Record both leaflets of the mitral valve.

6.Record the left ventricle at an area inferior to the papillary muscles.

Parasternal Short-Axis (PSA) View. The transducer should be rotated 90 degrees from the parasternal long-axis view to obtain multiple transverse short-axis views of the heart at particularly these four levels:

1.High PSA view to demonstrate the pulmonary valve, right ventricular outflow tract and aorta:

a. Typical sausage-shaped right ventricular outflow tract and pulmonary artery draped anterior to circular aorta

b. Semilunar cusp thickness and mobility

c. Presence of calcification, extraneous echoes, or both in right ventricle or valve areas

d. Pulmonary valve mobility and thickness

Respective M-mode tracings should then be made in these areas:

1.Record the mobility of the pulmonary cusps.

2- Moderate to high PSA view to demonstrate the right ventricle, tricuspid valve, aortic cusps, coronary arteries, right and left atria:

a. Size of right ventricle and left atrium

b. Presence of mass lesions in right or left atrium

c. Mobility and thickness of tricuspid and aortic valves

d. Continuity of interatrial septum

e. Right ventricular wall thickness

f. Presence of trileaflet aortic valve

Respective M-mode tracings should then be made in these areas:

1.Record the right ventricular outflow tract, aorta with cusps, and left atrial size.

2.Record the right ventricle and tricuspid valve.

3.Mid PSA view to demonstrate the right ventricle, left ventricular outflow tract, and anterior and posterior leaflets of the mitral valve:

a. Size of the left ventricular outflow tract

b. Size of the septum and posterior wall

c. Presence of mass lesions in left or right ventricle

d. Mobility and thickness of the mitral valves

e. Presence of a flutter on the septum or anterior leaflet of the mitral valve or both

f. Systolic apposition of mitral valve leaflets

g. Contractility of septum and posterior wall

Respective M-mode tracings should then be made in these areas:

1-Record the right ventricle, interventricular septum, anterior leaflet of the mitral valve , and left ventricle.

2-Record both leaflets of the mitral valve in the left ventricular cavity

3-Low PSA should demonstrate the right ventricle, left ventricle, and papillary muscles (chordal echoes may also be seen):

a. Contractility of septum and posterior wall of the left ventricle

b. Thickness of the septum and posterior wall

c. Size of the left ventricle

d. Presence or absence of mitral thrombus or other mass

e. Presence or absence of pericardial fluid, constriction, or restriction

f. Presence of increased echo density in posterior wall

g .Number of papillary muscles and their location within the left ventricular cavity

Respective M-mode tracing should then be made in these areas:

1.Record the right ventricle, interventricular septum (chordate tendineae), left ventricle, endocardium, myocardium, epicardium, and pericardium. Reduce gain to show bright pericardial echo reflection.

2.Sweep from left ventricle to mitral valve to aorta.

Apical Views. Two apical views are very useful: the four – chamber view and the apical long –axis vies, or two chamber view. The cardiac sonographer should palpate the patient's chest to detect the point of maximal impulse (PMI). The transducer should then be directed in a transverse plane at the PMI and angled sharply cephalad to record the four chambers of the heart. If there is too much lung interference, then the proper cardiac window has not been found and care should be taken to adjust the patient's position or the transducer position to adequately see all four chambers of the heart. Many laboratories have found it useful to use a very thick mattress with a large hole cut out of the mattress at the

level of the apex of the heart. This allows the transducer more flexibility in recording the four chamber view (Sandra,2011).

This view is excellent for assessing cardiac contractility, size of cardiac chambers, presence of mass lesions, alignment of atrioventricular valves, coaptation of atrioventricular valves, septal or posterior wall hypertrophy, chordal attachments, and the presence of pericardial effusion. It is not a good view to evaluate the presence of an atrial septal defect, since the beam is parallel to the thin foramen ovale and the septum commonly appears as a defect in this view. The subcostal four-chamber view is much better to evaluate the presence of such a defect (Sandra,2011).

Generally no M-mode tracings are made in the apical views: therefore the cardiac sonographer should observe the following structures:

1.Size of the cardiac chambers

2.Contractility of right and left ventricles

3.Septal and posterior wall thickness, contractility, and continuity

4.Coaptation of atrioventricular valves

5.Alignment of atrioventricular valves

6.Presence of increased echoes on valve apparatus

7.Presence of mass or thrombus in cardiac chambers

8.Entrance of pulmonary veins into left atrial cavity

9.Size of left ventricular outflow tract, signs of obstruction, mobility of aortic cusps, absence of subaortic membrane

10.Entrance of inferior and superior vena cava into the right atrium

The apical long-axis view is very useful to evaluate the left ventricular cavity and aortic outflow tract . Once the apical four-chamber view is obtained, the transducers should be rotated 90 degrees to visualize the left ventricle, left atrium, and aorta with cusps. This view permits the cardiac sonographer to

evaluate the wall motion of the posterior basal segment of the left ventricle, the anterior wall, and the apex of the left ventricle. It also permits another view of the left ventricle outflow tract, which may be useful in determining aortic cusp motion or the presence of a subvalvular membrane (Sandra,2011).

Subcostal View. The subcostal view also has multiple windows in the four-chamber and short-axis planes. Many of the views are only available in the pediatric patient (because of the flexible abdominal muscles).The subcostal four-chamber view is generally a useful view in many adults and may serve as an alternate view if the apical four-chamber view is unobtainable. The transducer should be placed in the subcostal space, and with a moderate amount of pressure, angled steeply toward the patient's left shoulder. The plane of the transducer is transverse to visualize the four chambers of the heart (Sandra,2011).

It is usually easy to follow the inferior vena cava into the right atrium of the heart. With careful angulation the interatrial septum may be visualized between the anterior right atrial chamber and posterior left atrial chamber. It usually is more difficult to open up the right ventricular cavity in this view; therefore no size assessment should be made. This view is usually very good to evaluate the presence of pericardial effusion, especially since it surrounds the anterior segment of the right heart (Sandra,2011).

Suprasternal View . In the suprasternal view, the transducer is directed transversely in the patient's suprasternal notch and angled steeply toward the arch of the aorta. This view is only useful if the design of the transducer is small enough to fit well into the suprasternal notch. The patient best prepared if several towels or a pillow is placed under the shoulders in an effort to flex the neck enough to avoid interference with the neck of the transducer and cable. The Patient's head should be turned to the right, again to avoid interference with the cable. With careful angulation, the cardiac structures visualized are the aortic

arch, brachiocephalic vessels, right pulmonary artery, left atrium, and left main bronchus (Sandra,2011).

This view is especially useful in determining supravalvular enlargement of the aorta, coarctation of the aorta, or dissection of the aorta (Sandra,2011).

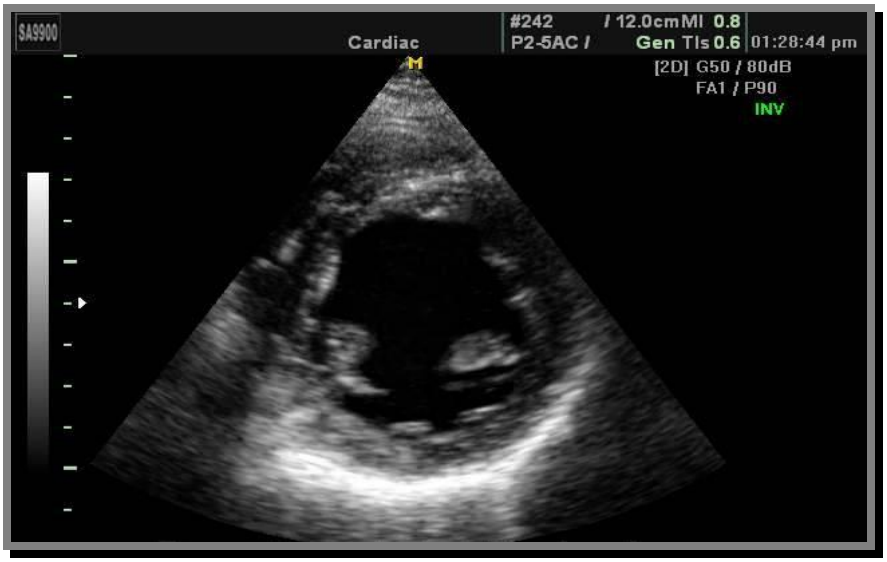

Figure. 2.18 *Parasternal short –Axis Views.*

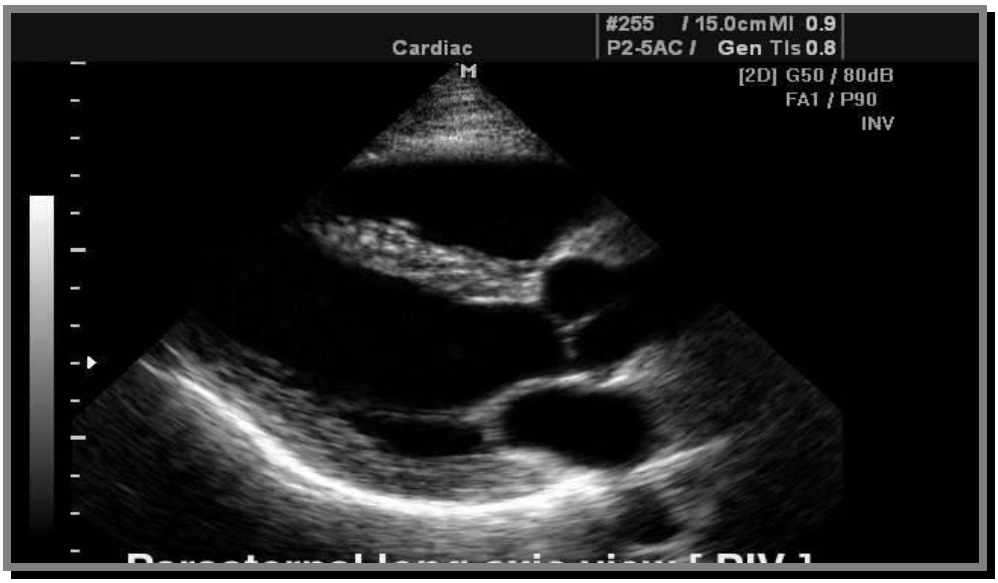

Figure. 2.19 *Parasternal long –Axis Views.*

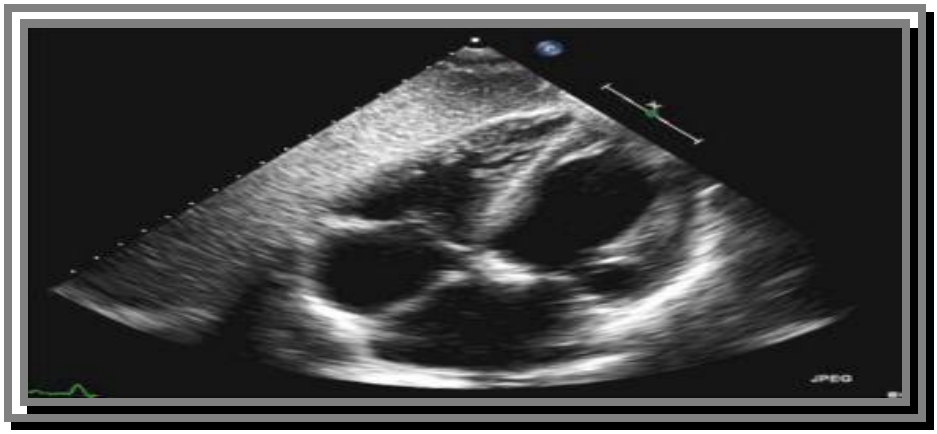

Figure. 2.20 *Subcostal view.*

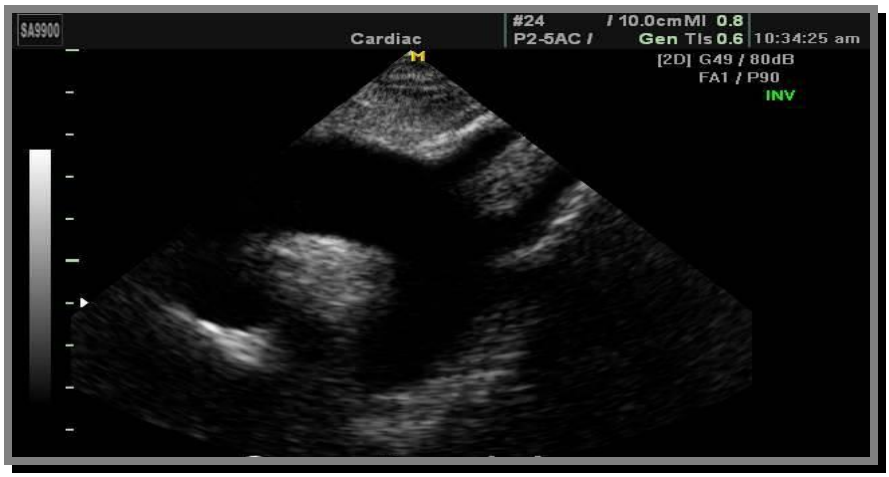

Figure. 2.21 *Suprasternal view.*

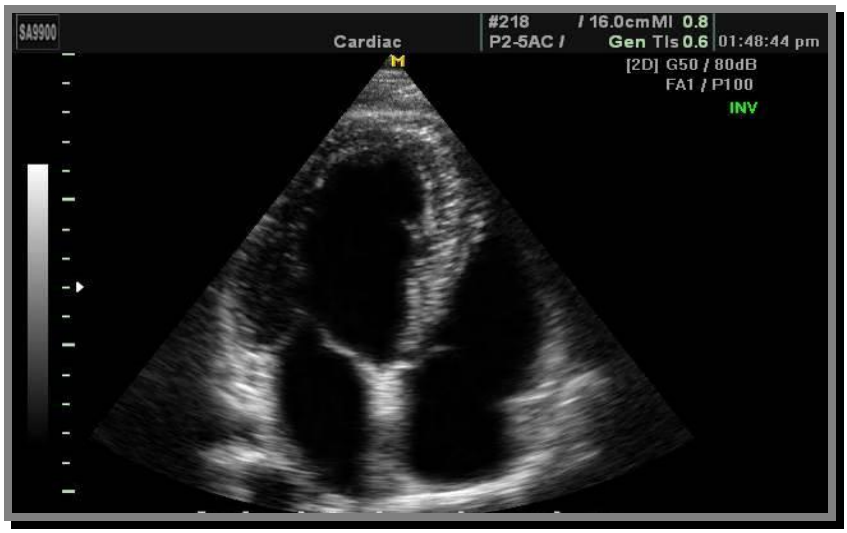

Figure. 2.22 *Apical 4 chamber view.*

2.32 DOPPLER APPLICATIONS AND TECHNIQUE

The Doppler effect, first described by Christian Johann Doppler, is demonstrated on an echocardiogram as red blood cells move from a lower – frequency sound source at rest toward a higher-frequency sound source. The change in frequency is called the *Doppler shift in frequency*, or the *Doppler frequency* (Sandra,2011) .

2.32.1 NORMAL DOPPLER EXAMINATION AND TECHNIQUES

Doppler echocardiography has emerged as a valuable noninvasive tool in clinical cardiology to provide hemodynamic information about the function of the cardiac valves and chambers of the heart. When combined with conventional two-dimensional and M-mode echocardiography, Doppler techniques may be focused to produce specific information on the flows of a particular area within the heart. Advances in Doppler technology have made it possible to provide steerable continuous-wave Doppler along with pulsed-wave Doppler. The ability to be qualitative and quantitative in evaluating valvular function, intracardiac shunts, dysfunction of a prosthetic valve, obstruction of a surgically inserted shunt, and to record normal cardiac blood flow patterns has contributed to the understanding and diagnostic capability of the Doppler technique in cardiology. However, to record this information, the cardiac sonographer and physician should master cardiac physiology and hemodynamics. In addition, the operator must clearly understand Doppler principles, artifacts, and pitfalls to produce a quality study. Although cardiac instrumentation is fundamentally similar to imaging echocardiography, the approach to Doppler especially color Doppler, varies considerably from one company to another; a solid understanding of the instrumentation is necessary to produce a valid examination (Sandra,2011).

2.32.2 Normal Flow Patterns

It is important to understand the relationship between the two-dimensional study and the Doppler-flow study. Real time two-dimensional imaging allows one to assess the cardiac anatomy and function. On the other hand, Doppler-flow analysis allows one to study blood flow rather than cardiac anatomy. The Doppler principle on which this technique is based involves the backscatter of transmitted ultrasonic waves from circulating red blood cells. The difference in frequency between transmitted and backscattered sound waves (Doppler shift) is used to quantify forward or backward blood-flow velocity (Sandra,2011).

2.32.3 Pulsed-Wave Doppler

A PW transducer is constructed with a single crystal that sends bursts of ultrasound at a rate called the pulsed repetition frequency. Sound waves backscattered from moving red blood cells are received by the transducer during a limited time between transmitted pulses. A time-gating device is then used to select the precise depth from which the returning signal has originated, since the signals return from the heart at different times. The pulsed-wave probe is built into the two-dimensional imaging probe. The particular site undergoing Doppler evaluation is referred to as the sample volume. The sample volume and directional line placement of the beam are moved through use of the trackball. The exact size and location of the sample volume can be adjusted at the area of interest. Some instruments have a fixed sample volume size, whereas others allow the operator to select the size that would be appropriate for the particular study. Velocities under 2 m per sec are usually easily recorded without an alias pattern. However, pulsed Doppler is limited in its ability to record high-velocity patterns. The maximum frequency shift that can be measured by a pulsed Doppler system is called the *Nyquist limit*, and is one half the pulsed repetition

frequency. Velocities that exceed this limit are known to produce an aliasing pattern. Normal cardiac structures do not exceed the Nyquist limit and are very easily measured with the pulsed Doppler system (Sandra,2011).

2.32.4 Continuous-Wave Doppler

The continuous-wave probe differs from the pulsed-wave probe in that it requires two crystals. If it is part of a two dimensional imaging transducer, the sample direction can sometimes be steered by use of the trackball. Some instruments have a fixed CW sample direction, which means that the area of interest must be aligned with a stationary line on the screen to obtain the best signal. Since the diameter of the probe is smaller, it allows greater flexibility to reach in between small rib interspaces or to obtain signals from the suprasternal notch. Many patients allow the small independent probe to be angled within their suprasternal notch rather than the bulky imaging probe. The independent probe is often more sensitive and therefore produces better Doppler signals. The audio portion of the Doppler exam becomes a critical factor in this study, since there is no two-dimensional image to guide in the transducer location. Sometimes it is useful to use a combination of both probes to perform the study. Once the proper transducer position is found with the imaging transducer, the angulation and window are marked for proper placement of the CW transducer. The audio sound and spectral wave pattern are then used to guide the correction angulation of the beam for maximum velocity recordings. There is not a particular sample volume site within the CW beam. Velocities are recorded from several points along the linear beam. The techniques has the ability to record maximum velocities without alias patterns. This is especially useful for very-high-velocity patterns (Sandra,2011).

2.33 The Doppler Examination: Techniques and Transducer Position

The Doppler examination is generally performed after a complete two-dimensional study of the cardiac structures. During this conventional study, the operator noted structures that may need special attention during the Doppler examination (e.g., a redundant mitral valve leaflet may indicate the need to search for mitral regurgitation). Throughout the Doppler study, various patient positions and transducer rotations are necessary to place the sample volume parallel to blood flow. The patient should be forewarned about the audio sounds produced by the Doppler signal because some find them quite alarming (Sandra,2011).

2.34 THE NORMAL CARDIAC COLOR-FLOW EXAMINATION

The color-flow mapping (CFM) examination is generally performed after the conventional two-dimensional examination. The advantage of CFM is its ability to rapidly investigate flow direction and movement within the cardiac chambers. Flow toward the transducer is recorded in red, whereas flow away from the transducer is blue. As the velocities increase, the flow pattern in the variance mode turns from a red color to various shades of red, orange, and yellow before it aliases. Likewise the blue color turns to various shades of blue, turquoise, and green before it aliases. Depending on the location of the transducer the flow signals from various structures within the heart appear as different colors. An understanding of cardiac hemodynamics helps the examiner understand the flow patterns. Although normal cardiac flows are difficult to accurately time during the CFM examination because of its slow frame rate, the use of color M-mode (with a faster frame rate) allows one to precisely determine specific cardiac events in correlation with the ECG. The color M-mode is made in the same manner as a conventional M-mode study. The cursor is placed through the area of interest and the flow is evaluated using an autocorrelation technique. The operator must thoroughly understand the color instrument settings to produce a

high-quality image. Familiarity with the color-flow maps provided in the software of the equipment is necessary to understand the alias pattern and turbulent flow parameters (Sandra,2011).

The CFM examination is generally performed in the same planes used for conventional Doppler examination. How-ever, to provide a basic understanding of the color Doppler image, we first describe flow pattern with the conventional two-dimensional views (Sandra,2011).

Table 2.1:Reference limits and values and partition values of left ventricular

	Women				Men			
	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal
Left Ventricle size	3.9–5.3	5.4–5.7	5.8–6.1	≥6.2	4.2–5.9	6.0–6.3	6.4–6.8	≥6.9
IVS thickness	0.6–0.9	1.0–1.2	1.3–1.5	≥1.6	0.6–1.0	1.1–1.3	1.4–1.6	≥1.7
Posterior wall thickness	0.6–0.9	1.0–1.2	1.3–1.5	≥1.6	0.6–1.0	1.1–1.3	1.4–1.6	≥1.7
Ejection Fraction	≥55	45–54	30–44	<30	≥55	45–54	30–44	<30

Table 2.2:Normal Individuals valves velocity (m/sec)

	Children	Adults
Mitral	0.8 – 1.3	0.6 -1.3
Tricuspid	0.5 -0.8	0.3 -0.7
Pulmonary	0.7 – 1.1	0.6 -0.9
Aorta	1.2 -1.8	1.0 -1.7

2.35 Previous Studies

In the realm of echocardiography of myocardial infarction, few authors and ultrasound practitioners have wrote about. Weissman et al, (2000) stated that : the echocardiogram has become an important tool in the evaluation of patients with a wide range of cardiac abnormalities, including acute myocardial infarction (MI). This card will review the role of echocardiography in establishing the diagnosis, location, and extent of MI, in diagnosing mechanical complications of infarction, and in providing prognostic information that is important for risk stratification. The use of transthoracic echocardiography for the evaluation of chest pain in the emergency department and of transesophageal echocardiography in patients with ischemic heart disease are discussed separately. In the study carried out by Elsadig et al, (2010) found that The commonest causes of sudden natural death, which were pneumonia, myocardial infarction and tuberculosis, were different from the literature in which the commonest causes were myocardial infarction followed by pulmonary embolism.

Mehta et al, (2001) indicate significant age-associated differences in clinical characteristics in elderly patients with AMI, which account for some of the age-associated differences in mortality. The practice of grouping older patients together as a single age group may obscure important age-associated differences. In the same realm Novella et al, (2008) indicate that the incidence of fatal and non-fatal myocardial infarction was very high in the elderly population of Madrid. Both incidence and mortality rates increased dramatically with age after 64 years. Rates were higher in men than women at all ages, though gender differences decreased with age. In the study carried out by Shah et al, (2010) found that Of the total 281 patients, 45 (16%) were ≤40 years old (Group-A) and 236 (84%) were older than 40 years (Group-B). There was no significant

difference between the two groups with respect to the risk factors like gender, hypertension and diabetes mellitus. On coronary angiography, the two groups neither differed in the number of totally occluded vessels, nor in the severity of the culprit lesion. There were only 3 patients in group-A (6.7%) and 5 patients in group-B (2.1%) with normal coronaries (p=NS). Majority (60%) of the patients in group-A had no significant disease or single vessel disease while majority (69%) of the patients in group-B had two or more vessels involved (p<0.001). As far as the number of lesions in the coronary arteries is concerned, 62.3% patients had 2 or lesser lesions in group-A while 68.6% patients in group-B had three or more lesions (p=0.001). There was a significant interaction between age and sex with chest pain at presentation, with a larger sex difference in younger than older patients, which became attenuated with advancing age. Multivariable adjusted age-specific odds ratios (ORs) for lack of chest pain for women (referent, men) were younger than 45 years, 1.30 (95% CI, 1.23-1.36); 45 to 54 years, 1.26 (95% CI, 1.22- 1.30); 55 to 64 years, 1.24 (95% CI, 1.21-1.27); 65 to 74 years, 1.13 (95% CI, 1.11- 1.15); and 75 years or older, 1.03 (95% CI, 1.02-1.04) in study by Canto et al (2012).

Woodfield et al, (1997) found that Women do not differ significantly from men with regard to either early infarct-related artery patency rates or reocclusion after thrombolytic therapy or ventricular functional response to injury/reperfusion. Gender was an independent determinant

of 30-day mortality after acute myocardial infarction. In the same field, Arnetz et al, (2008)indicate that younger (<70 years of age) female MI patients placed significantly more value on shared decision-making than younger (<70) men. More than one third of patients would have liked to be more involved in their care during hospitalization and discharge planning, with women significantly more dissatisfied than men. Significantly fewer younger female patients discussed

secondary preventive lifestyle changes with cardiology staff prior to hospital discharge. Also in other study which was about the effect of BMI in occurrence of MI, Yusuf et al, (2005) state that BMI showed a modest and graded association with myocardial infarction (OR 1·44, 95% CI 1·32–1·57 top quintile vs bottom quintile before adjustment), which was substantially reduced after adjustment for waist-to-hip ratio (1·12, 1·03–1·22), and non-significant after adjustment for other risk factors (0·98, 0·88–1·09). For waist-to hip ratio, the odds ratios for every successive quintile were significantly greater than that of the previous one (2^{nd} quintile: 1·15, 1·05–1·26; 3^{rd} quintile: 1·39; 1·28–1·52; 4^{th} quintile: 1·90, 1·74–2·07; and 5th quintiles: 2·52, 2·31–2·74 [adjusted for age, sex, region, and smoking]). Waist (adjusted OR 1·77; 1·59–1·97) and hip (0·73; 0·66–0·80) circumferences were both highly significant after adjustment for BMI ($p<0·0001$ top vs bottom quintiles). Waist-to-hip ratio and waist and hip circumferences were closely ($p<0·0001$) associated with risk of myocardial infarction even after adjustment for other risk factors (ORs for top quintile vs lowest quintiles were 1·75, 1·33, and 0·76, respectively). The population-attributable risks of myocardial infarction for increased waist-to-hip ratio in the top two quintiles was 24·3% (95% CI 22·5–26·2) compared with only 7·7% (6·0–10·0) for the top two quintiles of BMI. In (2006) Wells et al indicate that despite the association between obesity and development of coronary artery disease, obesity does not adversely impact in-hospital outcomes in AMI. However, obesity is associated with AMI at a younger age. Median BMI was 28.6. BMI was inversely associated with crude 1-year mortality (normal, 9.2%; overweight, 6.1%; obese, 4.7%; morbidly obese; 4.6%; P <.001), which persisted after multivariable adjustment. When BMI was examined as a continuous variable, the hazards curve declined with increasing BMI and then increased above a BMI of 40. Compared with patients with a BMI of 18.5, patients with higher BMIs had a 20% to 68% lower

mortality at 1 year. No interactions between age (P=.37), sex (P=.87), or diabetes mellitus (P=.55) were observed. It is result of study done by Emily et al in 2012. The effect of residence on myocardial infarction occurrence has been considered in study carried out by Hassan et al (2009) a total of 7351 patients were hospitalized with an acute MI during the study period. Rates of cardiac catheterization differed across the three groups (MA 45.6%, UA 37.3%, RA 37.3%; P<0.0001), as did mean waiting times (MA 15.0 days, UA 32.1 days, RA 28.7 days) (P<0.0001). After adjusting for differences among patients, residence in either UA or RA emerged as an independent predictor of lower rates of cardiac catheterization (UA: hazard ratio [HR] 0.77, P<0.0001; RA: HR 0.75, P<0.0001), greater waiting times (UA: an additional 14.1 days, P<0.0001; RA: an additional 10.8 days, P<0.0001) and increased long-term rates of readmission (UA: HR 1.24, P=0.0001; RA: HR 1.12, P=0.04).

S. Yusuf et al, (1996) have evaluated the effect of some drugs in myocardial infarction and they found that: The management of patients with AMI should be based on relevant and reliable evidence from large randomized controlled trials. In the acute phase, thrombolysis therapy is supported by such a level of evidence. In the longer term, the efficacy of aspirin, beta-blockers and ACE-inhibitors is similarly supported and they should be adopted for widespread prescription as simple effective and affordable drugs. Their dosages should be tailored to the individual but should pertain as closely as possible to those which were used in the relevant positive clinical trials.

In the same field, Pals et al, (2010) indicated that: Apyrase treatment according to a clinically applicable protocol, with administration of apyrase after induction of ischemia, does not reduce myocardial infarct size or microvascular obstruction. Also in (2011) Banerjee et al state that: For patients with the clinical presentation of AMI within 12 h after symptom onset, early mechanical (PCI) or

pharmacological reperfusion should be performed. Platelet activation and subsequent aggregation play a dominant role in the propagation of arterial thrombosis and consequently are the key therapeutic targets in the management of AMI. Adjunctive therapy with antiplatelets and antithrombotics is essential. A recommendation for routine urgent PCI (within 24 h) following successful fibrinolysis seems to be most practical option.

Osula et al, (2002) have evaluated the etiology of myocardial infarction and they found that: Myocardial infarction in young adults can be broadly divided into two groups, those with angiographically normal coronary arteries and those with coronary artery disease of varying aetiology. There is significant overlap in pathophysiology between these two groups.

Also in the same decade, Jahangir et al, (2012), stated that :Smokers have a higher risk of myocardial infarction than non-smokers, as the odd ratio comes out to be 3.71 which is significant. This raises also that smoking is more harmful with regard to ischemic heart disease, possibly because of constituents of tobacco smoke. Although there are certain aspects of risk factors which must be considered like family history, hypertension, obesity, etc. The implication for tobacco control is clear: that reduced smoking will reduce the leading cause of adult death in Pakistan. Our results add additional impetus in particular to cessation: some 70–80 million males over the age of 30 smoke, with vascular disease, tuberculosis and cancers being the major cause of premature mortality and disability among these men. In same year, Krishna et al, (2012) stated that: The present study concludes that PSL, play an important role in prognosis and in-hospital outcome of AMI; the outcome being unfavorable in diabetes and stress hyperglycemia. Increased caution needs to be maintained in patients of AMI presenting with hyperglycemia, requiring vigilant monitoring, and efficient management of the hyperglycemia.

Konermann et al, (1997) have evaluated the effect of left ventricle size in myocardial infarction and they found that: changes of the left ventricular chamber during the first 6 months following MI are dependent on its size and location, with large anterior infarctions having the worst course. Myocardial wall remodeling is also dependent on infarct size and location, and the volume-mass ratio increases in the presence of large areas of necrosis, indicating the non-compensatory effect of myocardial hypertrophy. However, these changes have no clinical effect during the first half year after MI. In same field Fernandes, et al (2007) indicated that: Indeed, the increase in LV size and resultant change in LV geometry from the normal prolate ellipse to a more spherical shape creates several mechanical burdens for the failing heart.

Poutanen et al, (2003) have evaluated the interventricular septum thickness of myocardial infarction and they found that: The maximal septum thickness by 2DE differed significantly from that obtained with M-mode in these children and adolescents ($p < 0.001$). In agreement analysis between 2DE and M-mode, the bias ± 2 SD was 0.8 ± 1.7 mm. The results obtained by M-mode correlated well with maximal septum thickness ($r = 0.84$, $p < 0.001$). The measurements also correlated well with BSA (maximal thickness, $r = 0.81$; ivsd by M-mode, $r = 0.87$). The mathematical formulae for describing the relation between septum diameter (mm) and BSA (m2) were calculated by linear regression analysis: 2DE septum = $2.638 + 3.256 \cdot$ BSA and M-mode septum (ivsd) = $2.145 + 3.008 \cdot$ BSA. There were no differences in septum thickness between males and females.

Additional study was carried out by Kiyoshi et al, (1971) which was about Posterior wall thickness, they stated that: The time-motion representation of the echogram was used to evaluate the left ventricular posterior wall motion in myocardial infarction. The left ventricular posterior wall echo was obtained in 10 normal subjects and 26 patients with chest pain admitted to a coronary care unit,

including 11 with acute myocardial infarction (group 1), nine with old myocardial infarction (group 2), and six with chest pain of miscellaneous origin (group 3). The total amplitude of posterior wall excursion, left ventricular isometric contraction time, left ventricular systolic ejection time, and mean posterior wall velocity (ratio of posterior wall excursion to ejection time) were measured. By using the measurements of posterior wall excursion, mean posterior wall velocity, and isometric contraction time, group 1 patients were differentiated from those of groups 2 and 3 and from the normal subjects (all $P < 0.01$). The data from patients in groups 2 and 3 were not statistically different from those of normal subjects. The measurements obtained by the time-motion curve of the left ventricular posterior wall echo appear to be of value in the bedside evaluation of acute myocardial infarction.

Somolinos et al, (1987) have evaluated the effects of myocardium infarction in pericardium and they found that: clinical and echocardiographic data were evaluated in 46 patients after acute myocardial infarction (AMI). M-mode echocardiogram was performed 24 and 72 h and 5 days after AMI. Early acute pericarditis (EAP) was clinically recognized in 19 (41%) patients. Pericardial effusion (PE) was detected in 29 (63%) patients. In 23 (50%) patients both anterior and posterior PE was observed, while in six (13%) patients PE was only posterior. An echocardiographic pattern consistent with localized fibrinous pericarditis was detected in 11 (24%) patients. Eighteen (95%) of 19 patients with EAP had PE, and only 11 (40%) of the patients without EAP had PE (p less than .001). We conclude that PE is observed frequently after AMI and that the echocardiographic study can help in the diagnosis of EAP after AMI. In addition to that Hafiz-ur-Rehman et al, (2010) documented that Pericardial effusion was seen in one third of the patients with first acute myocardial infarction. In acute phase of myocardial infarction, the chances of development of pericardial

effusion increases as the time passes. Left ventricular failure was the commonest in-hospital morbidity followed by cardiogenic shock and mitral regurgitation. In-hospital mortality was more in patients with pericardial effusion.

The Valve velocity in MI have also been considered by some authors recently, for instance Evangelista et al, (1996) stated that Mean value of aortic valvular flow VTI (22 +/- 4 cm) was slightly higher than that of pulmonary valvular flow (20 +/- 4 cm). Mean VTI values of left and right ventricular outflow tract and mitral valvular flow were similar (16 +/- 3, 15 +/- 3 and 15 +/- 3 cm, respectively) with an acceptable correlation ($r = 0.76$-0.83). VTI of tricuspid valvular flow was clearly lower than the rest (10 +/- 3 cm; $p < 0.001$). Linear regression analysis showed the VTI to be inversely related to heart rate and age, and mean velocity positively related to heart rate and inversely to age. While VTI remained relatively stable up to the age of 60 and decreased sharply thereafter, mean velocity decreased progressively with age. VTI values were identical for both sexes; however, mean velocity was higher in women up to the age of 60. Also in the same filed, Witt et al, (2011), stated that At day 1, tricuspid annular velocities were significantly reduced in patients with, compared to those without, ST elevation in V4R (11.1 vs. 13.7 cm/sec, 9.4 vs. 13.1 cm/sec and 14.1 vs. 15.9 cm/sec for systolic, early, and late diastolic velocities, respectively; $P < 0.01$). With a cutoff value for a tricuspid annular systolic velocity of 13 cm/sec, sensitivity and specificity for identifying patients with ST elevation in V4R were 89% and 71%, respectively. After 6 months, both tricuspid annular systolic and diastolic velocities in patients with RV infarction had increased significantly, but only tricuspid annular systolic velocity was still reduced compared to HS (12.3 vs. 14.7 cm/sec; $P < 0.01$). In addition to that Hsu et al, (2011) documented that In patients with a first, acute reperfused ST-elevation myocardial infarction without associated RV infarction, RV function may be affected discrepantly

depending on the different infarction sites. In patients with inferior infarction without concomitant RV infarction, only regional RV diastolic dysfunction is observed, whereas the alteration of global RV function is more pronounced in patients with anterior wall infarction.

In addition to that work, McClements et al, (2000) found that in the study about left ventricular ejection fraction For myocardial infarctions of similar size, left ventricular ejection fraction is lower when apical involvement is extensive and the site of infarction is anterior. This site-dependent difference may be related to characteristics specific to the apex. Also, Galema et al, (2007) stated that Forty-eight patients underwent transthoracic echocardiography in secondharmonic mode (SHI) with and without contrast within 5 days after an acute myocardial infarction. LV-EF was determined using the Simpson's biplane method. With contrast intra-observer variability decreased from 12.5+11.5% to 7.0+7.0% (P , 0.001) and inter-observer variability decreased from 16.9+9.9% to 7.0+6.2% (P , 0.001). Bland–Altman analysis confirmed these findings by demonstrating smaller 95% limits of agreement for both the intra- and inter-observer variability when contrast was used. This improvement in intra- and inter-observer variability was seen to a comparable extent in patients with moderate-to-poor and good quality SHI echocardiograms.In same field Kjell et al, (2008) stated that 167 patients had normal LVEF (>50%), 56 intermediate (40%–50%), and 33 reduced (<40%). The mean (SD) KCCQ clinical summary scores were 85 (18), 75 (22), and 68 (21) ($p < 0.001$) in the three groups, respectively. The corresponding EQ-5D Index scores were 0.83 (0.18), 0.72 (0.27), and 0.76 (0.14) ($p = 0.005$) and EQ-VAS scores were 72 (18), 65 (21), and 57 (20) ($p = 0.001$). In multivariable linear regression analysis age $\geq$ 70 years, known chronic obstructive pulmonary disease (COPD), subsequent MI, intermediate LVEF, and reduced LVEF were independent determinants for reduced KCCQ clinical summary score. Female

sex, medication for angina pectoris at discharge, and intermediate LVEF were independent determinants for reduced EQ-5D Index score. Age ≥ 70 years, COPD, and reduced LVEF were associated with reduced EQ-VAS score.

CHAPTER THREE

MATERIALS AND METHODS

3.1 Study design and population:

A prospective, cohort study was carried out in Sudan Heart Center, between July 2012 to June 2014. Study population comprises a total of 250 adults patient who underwent cardiac ultrasound scanning were enrolled in this prospective study. Sudanese participants were included in this study, if they have a global left ventricular systolic dysfunction with an ejection fraction of <50% as determined by clinical assessment of left ventricular function (echocardiography) and received drugs treatment. Participants were excluded if they have surgical treatment.

3.2 Technical Information Identifies :

Echocardiography studies were performed using Esaote echocardiography System (My Lab 50 X Vision, Italy) with Phased array probe 2.5 MHz (serial number of 1028924YM7, manufactured date of February 2010), convex face with small footprint for "peeking" in between rib interspaces. Printing facility issued through the ultrasound-digital graphic-printer (serial number of 3-619-GBI-01 made by Sony Corporation- Japan), 100 V; 1.5 A; and 50/60 Hz.

Because ultrasound cannot be transmitted through air, a dense coupling medium is needed between the transducer and the skin (Starkey, 1993 and Michlovitz, 1996).

To obtain a good image, a fluid medium is needed to provide a link between the transducer and the surface of the patient. This fluid is called an acoustic coupling agent, often referred to as "gel" (Palmer,1995).

Water is not a good coupling agent because it evaporates rapidly owing to the heat of the body: it also runs away over the patient as the transducer is moved. It should only be used in an emergency, when nothing else is available.

Oil, either mineral or vegetable, is a good coupling agent, but when used for a long time may dissolve the rubber or plastic shielding of the equipment. If oil gets on the operator's fingers, as it inevitably will, it might damage the controls of the ultrasound unit (Palmer,1995).

The best acoustic coupling agent is a watersoluble gel. Many are ommercially available, but they are usually expen ive and sometimes difficult to obtain. It is not necessary to use a particular coupling agent with specified eqUipment. even though manufacturers often suggest that this 1s essen tial. Special coupling agents do not give a better image (Palmer,1995).

The coupling agent is best applied using a squeeze bottle, from which it can be squirted onto the patient's skin. This avoids contamination. Any refillable plastic

squeeze bottle is Suitable, but it must be completely clean and dry before it is filled with the coupling agent. If there is an open wound, a skin rash or any other risk of infection, cover the transducer (or the skin) with thin plastic; put coupling agent on both sides of the plastic. The transducer must be cleaned after every patient (Palmer,1995).

The coupling agent should be removed with paper tissues, paper or cloth towels. It must be completely removed to avoid soiling the patient's clothing (Palmer,1995).

Ingredients

Almost any hospital or commercial pharmacy should be able to prepare a suitable gel. All are based on synthetic resins, polymers of acrylic acid or other liquids that become water-soluble when neutralized with an appropriate alkalizing agent.

1. Carbomer. A synthetic high molecular weight polymer of acrylic acid cross linked with allylsucrose and containing 56-68% of carboxylic acid groups. It is a white, fluffy, acidic, hygroscopic powder with a slight characteristic odour. Neutralized with alkali hydroxides or amines, it is very soluble in water, alcohol and glycerol (Palmer,1995).

There are three carbomers: the most suitable is carbomer 940, which forms a clear gel in aqueous and non-aqueous vehicles. If carbomer 940 is not available, carbomer 934 or 941 can be used (Palmer,1995).

However, they may not be quite so easy to mix (as described below) as carbomer 940 (Palmer,1995).

2. EDTA (edetic acid). A white crystalline powder, very slightly soluble in water. Soluble in solutions of alkali hydroXides (Palmer,1995).

3. Propylene glycol. A colourless, odourless. viscous hygroscopic liquid with a slight sweet taste. Density = 1.035-1.037 g/ml (Palmer,1995).

4. Trolamine (triethanolamine). A mixture of bases containing not less than 80% of triethanolamine, with diethanolamine and small amounts of ethanolamine. A clear, colourless or slightly yellow, odourless, viscous hygroscopic liquid. Density = 1.12-1.13 g/ml (Palmer,1995).

Preparation

1. Combine the EDTA with 400 g (400 ml) of water, making sure that it is dissolved, then add the propylene glycol (Palmer,1995).

2. Add the carbomer to the above solution and stir vigorously, if possible with a high speed stirrer, taking care to avoid the formation of indispersible lumps.

3. Wait until the mixture forms a gel and no more bubbles are observed.

4. Mix the trolamine with the remaining water and add the mixture to the gel to make a total of 500 g (Palmer,1995).

5. Stir carefully; do not shake, to prevent the formation of air bubbles in the gel. The recommended formula is not known to irritate healthy skin or stain clothing, and is easy to clean off (Palmer,1995).

This gel may become more liquid when the patient is sweating, because it is affected by a high concentration of salts. This can be avoided by cleaning and drying the skin before applying the gel. If left in direct sunlight, the gel may liquefy. It is incompatible with bivalent or trivalent cations, such as calcium, magnesium and aluminium; if prolonged storage is likely, it is wise to store the gel in the dark. The stability of carbomer is considerably influenced by the pH, which must be maintained between 5 and 10. Outside these limits, the viscosity will fall (Palmer,1995).

a water-based gel provides the highest percentage of acoustic energy transmission compared with other mediums tested (Reid and Cummings ,1973, Ashton *et al*.,1998 and Draper *et al*., 1993).

3.3. Heart Ultrasound Scanning Technique:

Transthoracic echocardiography was performed to all patients. The examination was performed in supine or 30 degrees left lateral decubitus position, , with the left arm raised up above the head. This position brings the heart out toward the

chest wall, displaces the lingula of the left lung out of the way, and opens the intercostal space by spreading the ribs. The transducer is pressed firmly against the chest and moved back and forth slowly. The transducer is moved to different areas over the chest to provide a detailed view of the heart and its structures. At least 4 separate standard transducer positions which allow for different portions of the heart to be visualized in detail. These standard positions are:

Subxiphoid 4-chamber view

Place the transducer-probe in the subxiphoid area directed into the chest and toward the left shoulder. The left lobe of the liver is used as an acoustic window to view the heart.

Parasternal long-axis (PLA) view

The probe should be placed in the parasternal fourth or fifth intercostal space with the transducer indicator directed pointed to the patient's right shoulder, This allows for typical identification of the right ventricle, left atrium, left ventricle, aortic valve, aortic root, aortic outflow tract, and surrounding pericardium.

Parasternal short-axis view

From the PLA position, rotate the probe clockwise 90° such that the probe indicator is pointed toward the patient's left shoulder.This allows for

identification of the left ventricle, right ventricle, and pericardium. In this view, the right ventricle is closer to the surface and appears crescent-shaped, while the left ventricle is deep to the right ventricle and appears circular.

Apical 4-chamber view

If possible, have the patient raise the left arm up over his or her head to try and spread the ribs. Palpate for the cardiac point of maximal impulse (PMI) and place the probe there with the indicator pointed toward the left axilla and the probe in a coronal plane relative to the heart, as shown below, aimed toward the base of the heart. This allows for identification of the left ventricle, right ventricle, left atrium, right atrium, and pericardium.

Direct the transducer-probe up toward the base of the heart. If the probe is directed anteriorly, the left ventricular outflow tract and aortic valve can often be seen; this is known as an apical 5-chamber view.

Septum thickness was measured using the parasternal long-axis view. M-mode measurements were made according to the recommendations of the American Society of Echocardiography. Thus, the line of measuring was perpendicular to the endocardium of the septum and the posterior wall. With the patient in the recumbent position, the transducer was placed in the fourth intercostal space at the left sterna border and directed posteriorly, laterally, and inferiorly to obtain a group of strong echoes from the posterior left ventricular wall.

Figure.3.1 Esaote echocardiography System (My Lab 50 X Vision)

Figure.3.2 Phased array probe 2.5 MHz

Figure.3.3 an acoustic coupling agent, "gel

3.4. Data collection:

Structured questionnaires were administered and physical examinations were done in all cases. Information was obtained about demographic factors, socioeconomic status of the residential neighborhood, drug described, risk factors, and personal and family history of cardiovascular disease. Both weight and height were measured with standardised protocols.

The sonographic data was obtained through direct ultrasound scanning of heart.

3.5. Statistical analysis:

Data were initially summarized into means, standard deviations (SD); mean ±SD and percentages in a form of comparison tables and graphs. Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS Inc., Chicago, IL, USA) version 16 for windows and (P-value) was used for significance.

-Significant difference if $P < 0.05$.

-Highly significant difference if $P < 0.001$.

-Non significant difference if $P > 0.05$.

The smaller the P-value obtained the more significant are the results.

3.6. Ethical considerations:

Special consideration was given to the right of confidentiality and anonymity for all participants. Anonymity was achieved by using number for each participant to provide link between the collected information and the participants.

In addition confidentiality was obtained by making the collected data accessible only to the researcher and the supervisor. Justice and human dignity was considered by teaching the selected participants equally when offering them an opportunity to participate in the research. The participants are free to decide whether to participate or not. The diagnostic tool used to perform the study was ultrasound which is safe and has no known harmful side effects to the participants.

CHAPTER FOUR

RESULTS

Table 4.1: Gender of the patients

Gender	Number of patients	percentage
Male	**168**	**67.2%**
Female	**82**	**32.8%**
Total	**250**	**100%**

The study population comprised of 250 myocardial infarction patients, made up of 168 (67.2%) males and 82 (32.8%) females .

Figure .4.1 *gender of the patients.*

Table 4.2: Distribution of age groups among gender

	Range	Mean	±SD
Age (years)	**22-86**	**61.92**	**±15.1**

Age	Gender				**Total /Percentage (%)**	
	male		female			
22-29	2	***0.8***	1	***0.4***	3	***1.2***
30-37	12	***4.8***	3	***1.2***	15	***6***
38-45	31	***12.4***	8	***3.2***	39	***15.6***
46-53	7	***2.8***	8	***3.2***	15	***6***
54-61	27	***10.8***	11	***4.4***	38	***15.2***
62-69	36	***14.4***	17	***6.8***	53	***21.2***
70-77	37	***14.8***	21	***8.4***	58	***23.2***
78-86	16	***6.4***	13	***5.2***	29	***11.6***
Total	168	***67.2***	82	***32.8***	250	***100***

Their ages ranged from 22 to 86 years, with mean age and standard deviation (SD) of 61.92 ± 15.1 years .

In the population of males, 37 subjects were in the age group (70-77) years, representing 14.8% of the population. The age group (22-29) years was the smallest (.8%) of the population .

In female population, 21 subjects were in the age group (70-77) years, representing 8.4% of the population. The age group (22-29) years was the smallest (.4%) of the population .

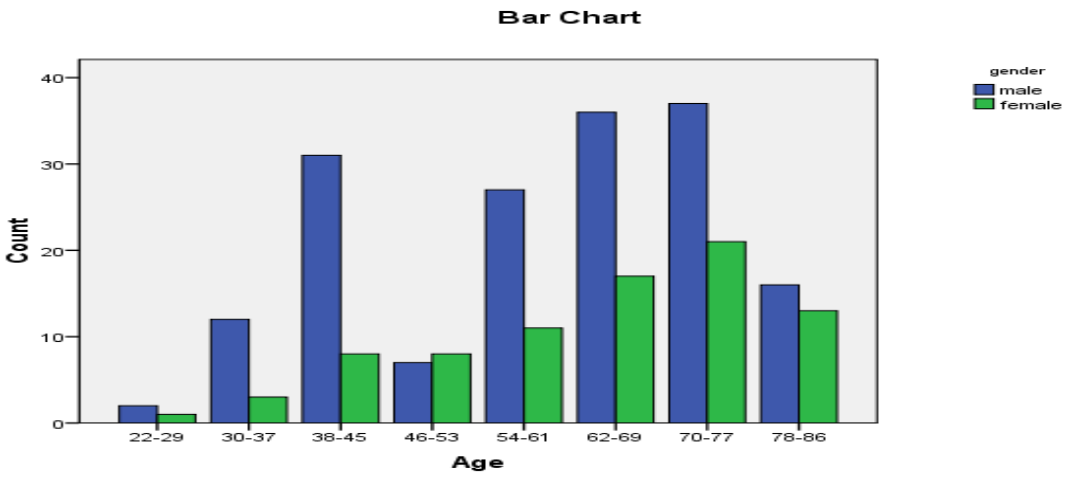

Figure .4.2 *Distribution of age groups among gender*.

Table 4.3: Body Mass Index among patients gender

Body Mass Index	Gender		Total	Percentage (%)	Mean ± SD (years)
	male	female			
Normal (19 - 24)	41(16.4%)	16(6.4%)	57	22.8%	21.64±1.7
Overweight (25 - 29)	65(26%)	25(10%)	90	36.0%	26.97±1.6
Obese (30 -35)	40(16%)	21(8.4%)	61	24.4%	31.98±1.4
Morbidly obese > 35	22(8.8%)	20(8%)	42	16.8%	36.92±1.9
Total	168(67.2%)	82(32.8%)	250	100.0%	28.67±5.4

The mean BMI and standard deviation was 28.67±5.4 kg/m^2.

Of the 250 patients under study, 41 male patients (16.4%) and 16 female patients (6.4%) were normal,65 male patients (26%) and 25 female patients (10%) were overweight, while 40 male patients (16%) and 21 female patients (8.4%) were obese and22 male patients (8.8%) and 20 female patients (8%) had morbidly obese BMI .

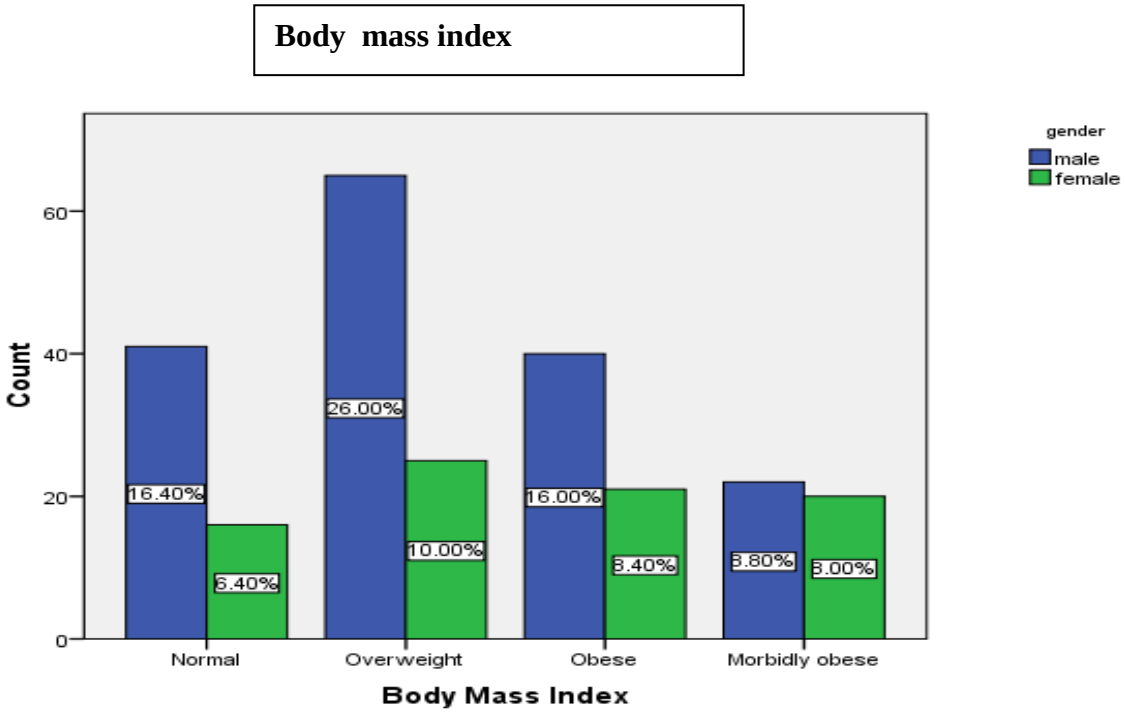

Figure .4.3 *Body Mass Index among patients gender*.

Table 4.4: Residence of patients

Residence	Frequency	Percentage (%)
Metropolitan area	132	52.8
Nonmetropolitan urban area	76	30.4
Rural area	42	16.8
Total	250	100

Place of residence was divided into three groups in which (n = 132; 52.8%) from metropolitan area (MA); (n = 76; 30.4%) from nonmetropolitan urban area (UA); (n = 42; 16.8%) and rural area (RA) .

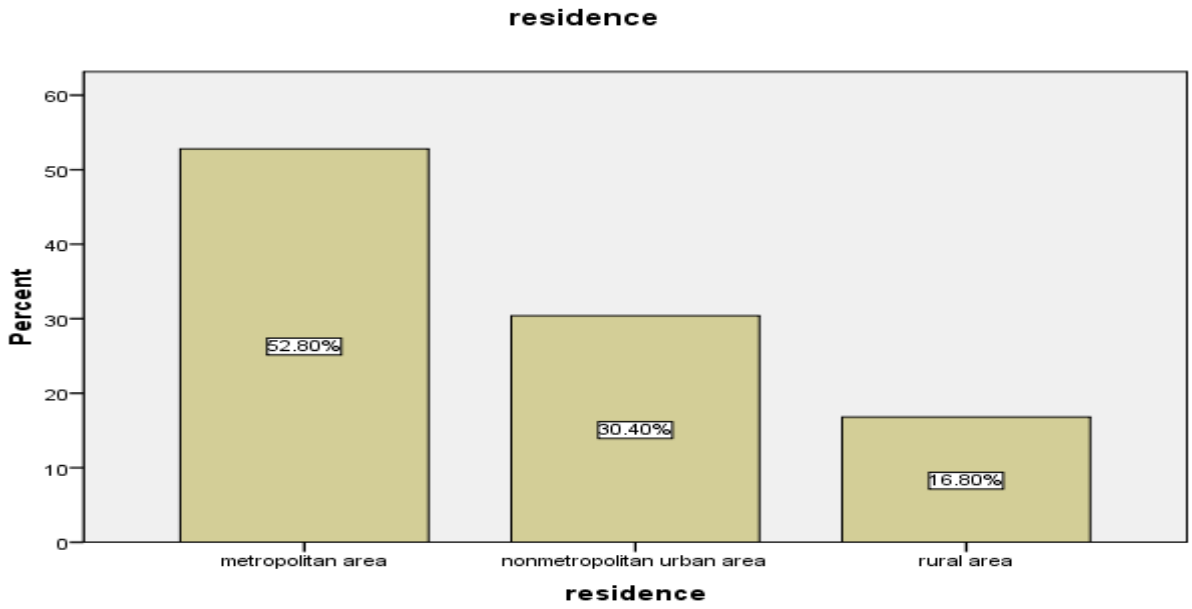

Figure .4.4 *Residence of patients.*

Table 4.5: History of myocardial infarction patients

history * Age * gender					
history	**Age**				
	< 60		**≥ 60**		**Total**
	male	**female**	**male**	**female**	
unknown	6	2	13	5	**26**
family history	7	3	8	8	**26**
cardiovascular disease	10	4	17	9	**40**
Diabetes mellitus	15	2	20	9	**46**
hypertension	14	6	14	12	**46**
obesity	7	7	9	15	**38**
smoking	6	0	22	0	**28**
Total	**65**	**24**	**103**	**58**	**250**

Of the 250 patients under study, 26 patients (10.4%) had unknown reason, 6 males & 2 females under 60 years age; 13 males & 5 females above 60 years age. also 26 (10.4%) had family history 7 males & 3 females under 60 years age; 8 males & 8 females above 60 years age, 40 patients (16%) had cardiovascular disease, 10 males & 4 females under 60 years age; 17 males & 9 females above 60 years age. 46patients (18.4%) had Diabetes mellitus, 15 males & 2 females under 60 years age; 20 males & 9 females above 60 years age. 46 patients (18.4%) had hypertension, 14 males & 6 females under 60 years age; 14 males & 12 females above 60 years age. 38 patients (15.2%) were obese, 7 males & 7 females under 60 years age; 9 males & 15 females above 60 years age. 28 patients (11.2%) were smoking, 6 males under 60 years age; 22 males above 60 years age .

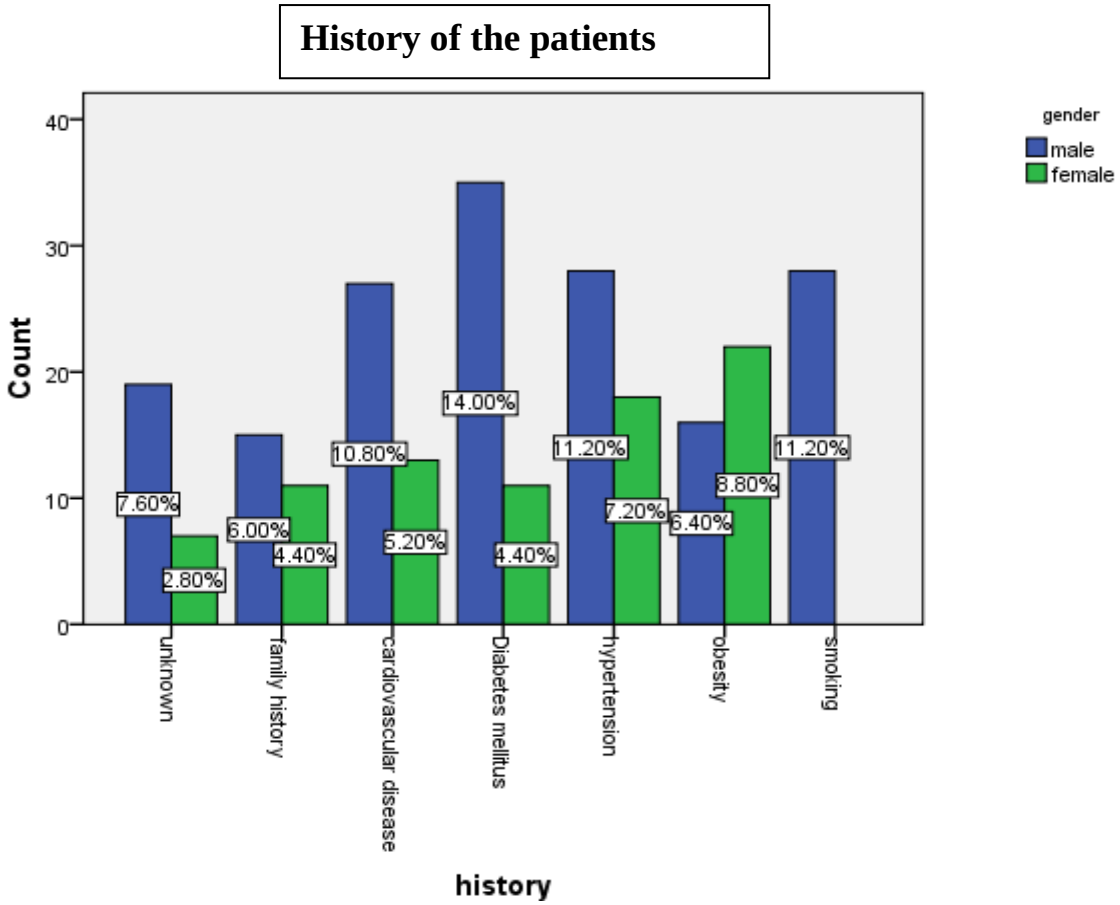

Figure .4.5 *History of myocardial infarction patients.*

Table 4.6: Pattern of left ventricular size in male and female patients

LVED pattern	Males' frequency (%)	Females' frequency (%)	Total No. (%)
Mean, Std. Deviation(cm)	5.1±.86	5.2±.90	
Normal	136(81)	43 (52.4)	179 (71.6)
Mildly abnormal	9 (5.4)	17 (20.7)	26 (10.4)
Moderately abnormal	16 (9.5)	9 (11.0)	25 (10)
Severely abnormal	7 (4.2)	13 (15.9)	20 (8)
Total	168 (100)	82 (100)	250 (100)

The mean and standard deviation of left ventricular size in male were 5.1±.86 cm, and in female were 5.2±.90 .

136 male patients (81%) and 43 female patients (52.4%) had normal size, 9 male patients (5.4%) and 17 female patients (20.7%) had mildly abnormal size,

16 male patients (9.5%) and 9 female patients (11%) had moderately abnormal size while 7 male patients (4.2%) and 13 female patients (15.9%) had severely abnormal size .

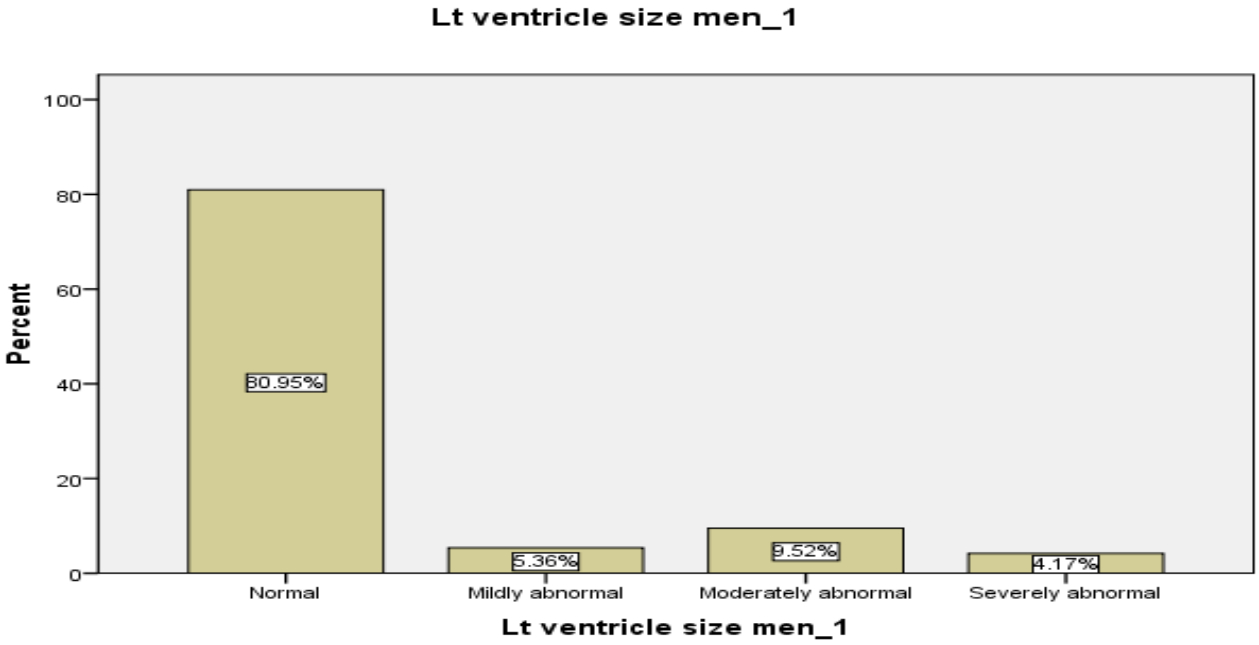

Figure .4.6 a *Pattern of left ventricular size in male patients.*

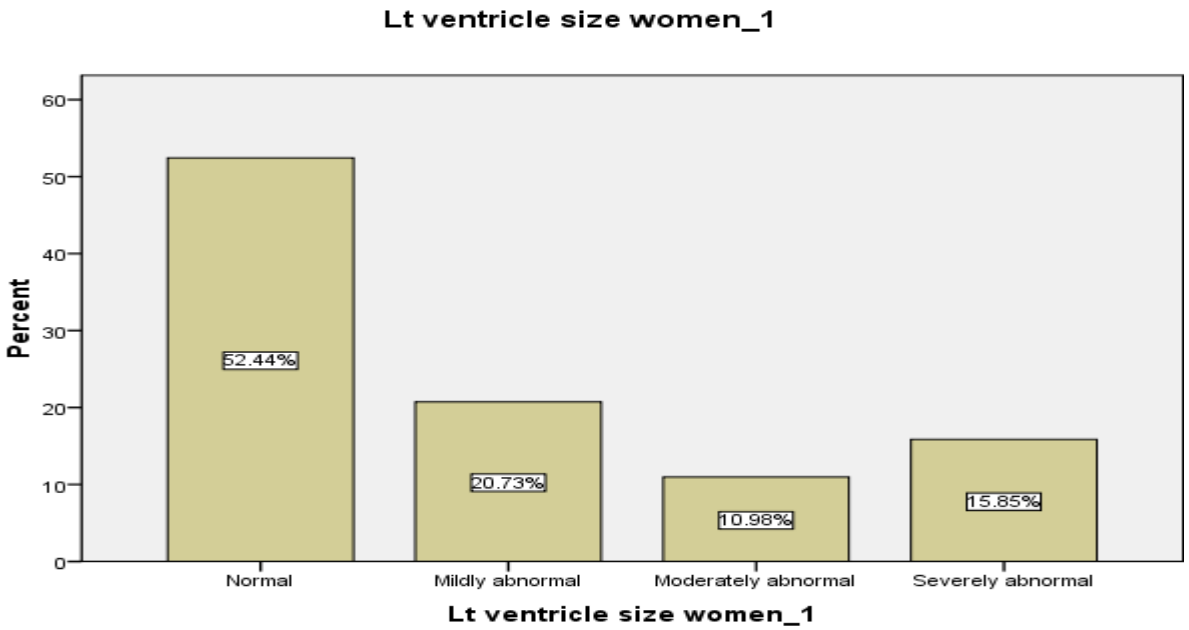

Figure .4.6 b *Pattern of left ventricular size in female patients.*

Table 4.7: Interventricular septum thickness of myocardial infarction patients

IVS thickness	Frequency	Percentage (%)
Mean, Std. Deviation (cm)	.98±.28	
Normal	180	72.0
Mildly abnormal	49	19.6
Moderately abnormal	10	4.0
Severely abnormal	11	4.4
Total	250	100.0

The mean and standard deviation of IVS thickness were .98±.28 cm. Among them 180 patients (72%) had normal thickness, 49 patients (19.6%) had mildly abnormal thickness, 10 patients (4%) had moderately abnormal thickness, while 11 patients (4.4%) had severely abnormal thickness .

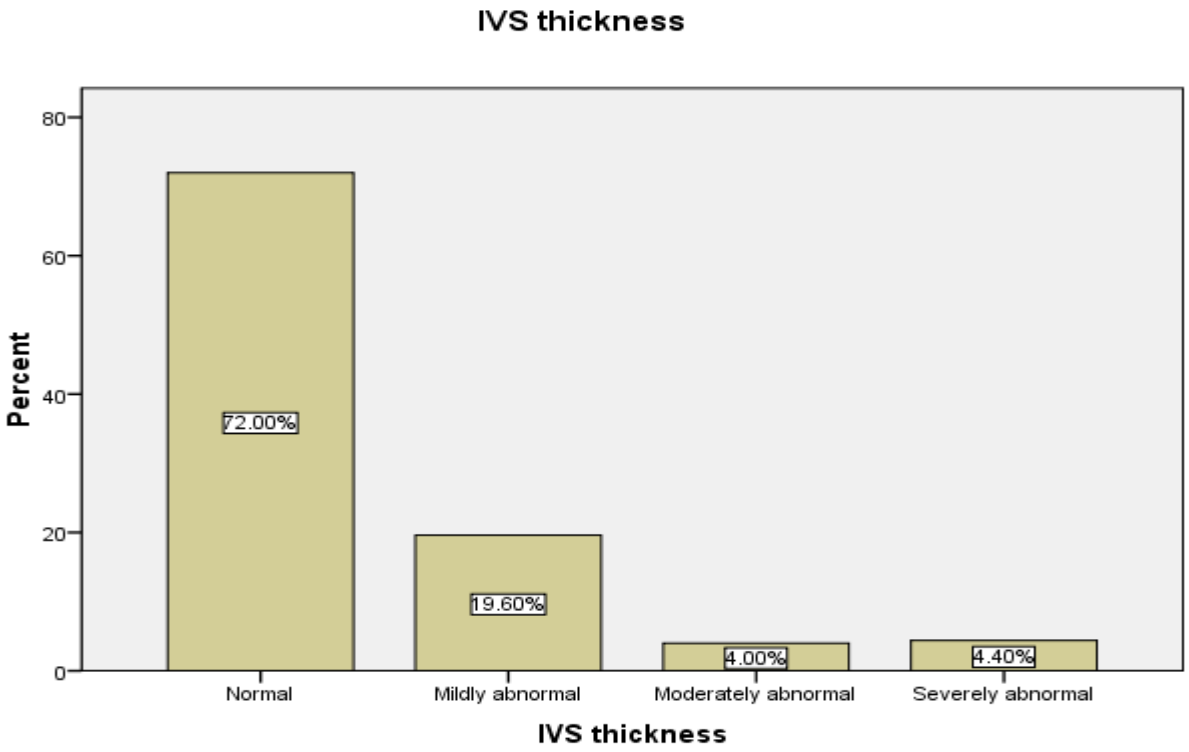

Figure .4.7 *Interventricular septum thickness of myocardial infarction patients.*

Table 4.8: Left ventricular posterior wall thickness of myocardial infarction patients

PW thickness	Frequency	Percentage (%)
Mean, Std. Deviation (cm)	.97±.24	
Normal	192	76.8
Mildly abnormal	45	18.0
Moderately abnormal	5	2.0
Severely abnormal	8	3.2
Total	250	100.0

The mean and standard deviation of PW thickness were .97±.24 cm. Among them 192 patients (76.8%) had normal thickness, 45 patients (18%) had mildly abnormal thickness, 5 patients (2%) had moderately abnormal thickness, while 8 patients (3.2%) had severely abnormal thickness.

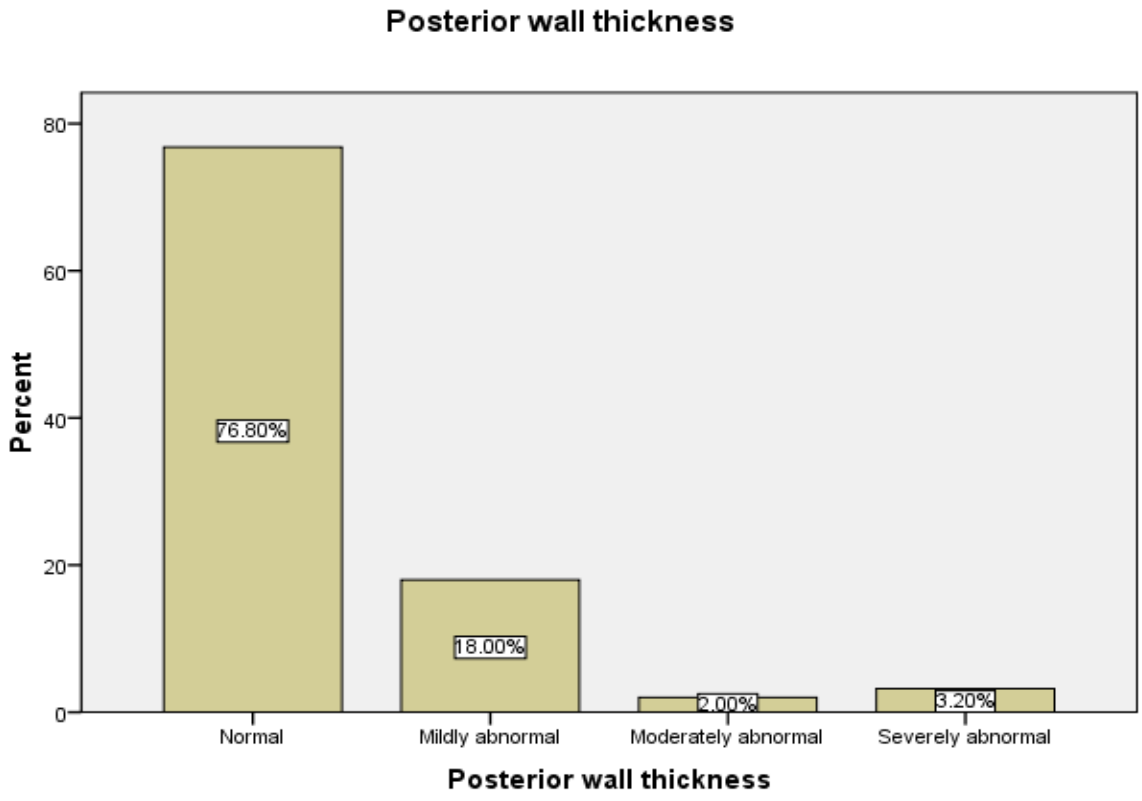

Figure .4.8 *Left ventricular posterior wall thickness of myocardial infarction patients.*

Table 4.9: Mitral valve velocity of myocardial infarction patients

Mitral Valve velocity	Frequency	Percentage (%)
Mean, Std. Deviation(m/sec)	.96±.27	
Normal	234	93.6
Abnormal	16	6.4
Total	250	100.0

The mean and standard deviation of mitral valve velocity were .96±.27 m/sec. Among them 234 patients (93.6%) had normal velocity, while 16 patients (6.4%) had abnormal velocity .

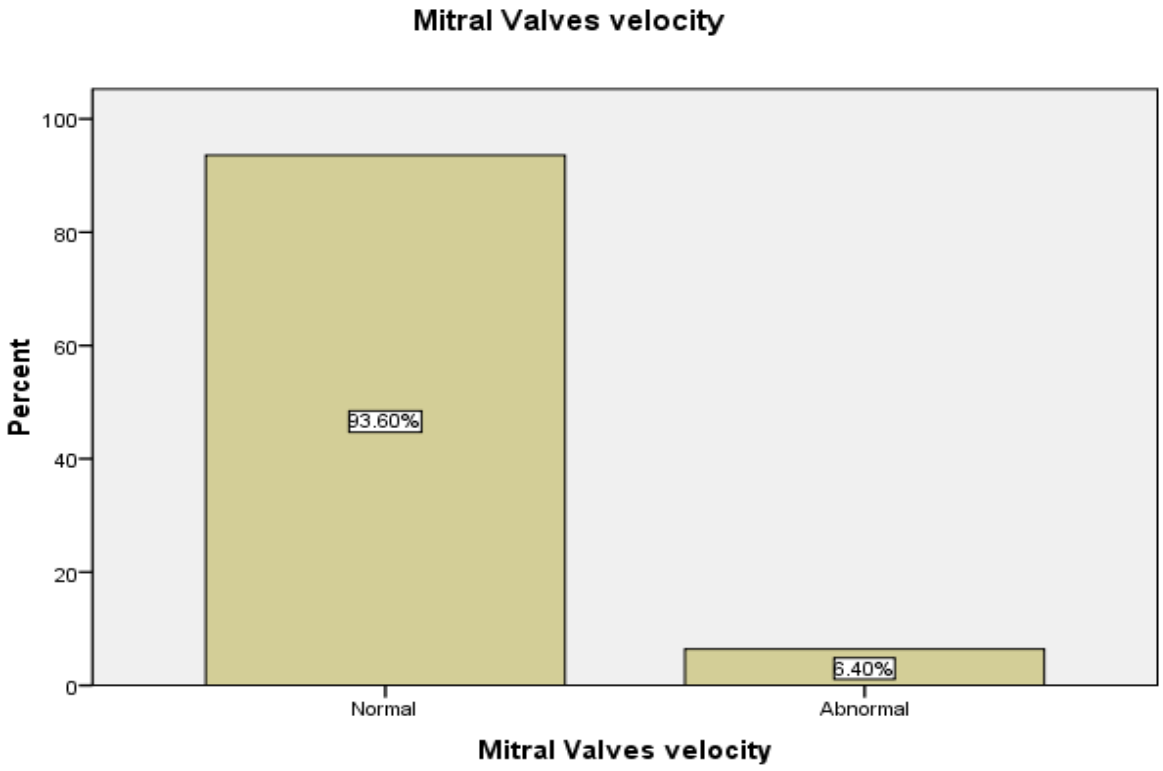

Figure .4.9 *Mitral valve velocity of myocardial infarction patients.*

Table 4.10: Tricuspid valve velocity of myocardial infarction patients

Tricuspid Valve velocity	Frequency	Percentage (%)
Mean, Std. Deviation (m/sec)	.72±.37	
Normal	191	76.4
Abnormal	59	23.6
Total	250	100.0

The mean and standard deviation of tricuspid valve velocity were .72±.37 m/sec. Among them 191 patients (76.4%) had normal velocity, while 59 patients (23.6%) had abnormal velocity.

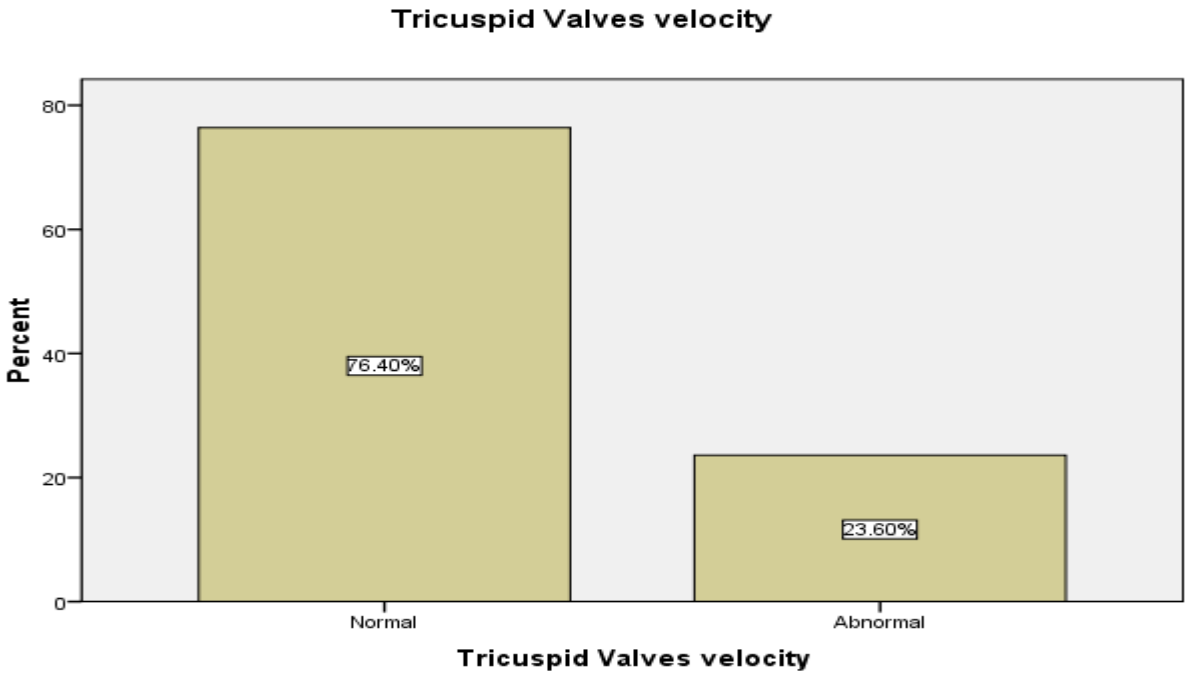

Figure .4.10 *Tricuspid valve velocity of myocardial infarction patients.*

Table 4.11: Pulmonary valve velocity of myocardial infarction patients

Pulmonary Valve velocity	Frequency	Percentage (%)
Mean, Std. Deviation (m/sec)	.79±.15	
Normal	223	89.2
Abnormal	27	10.8
Total	250	100.0

The mean and standard deviation of pulmonary valve velocity were .79±.15 m/sec. Among them 223 patients (89.2%) had normal velocity, while 27 patients (10.8%) had abnormal velocity.

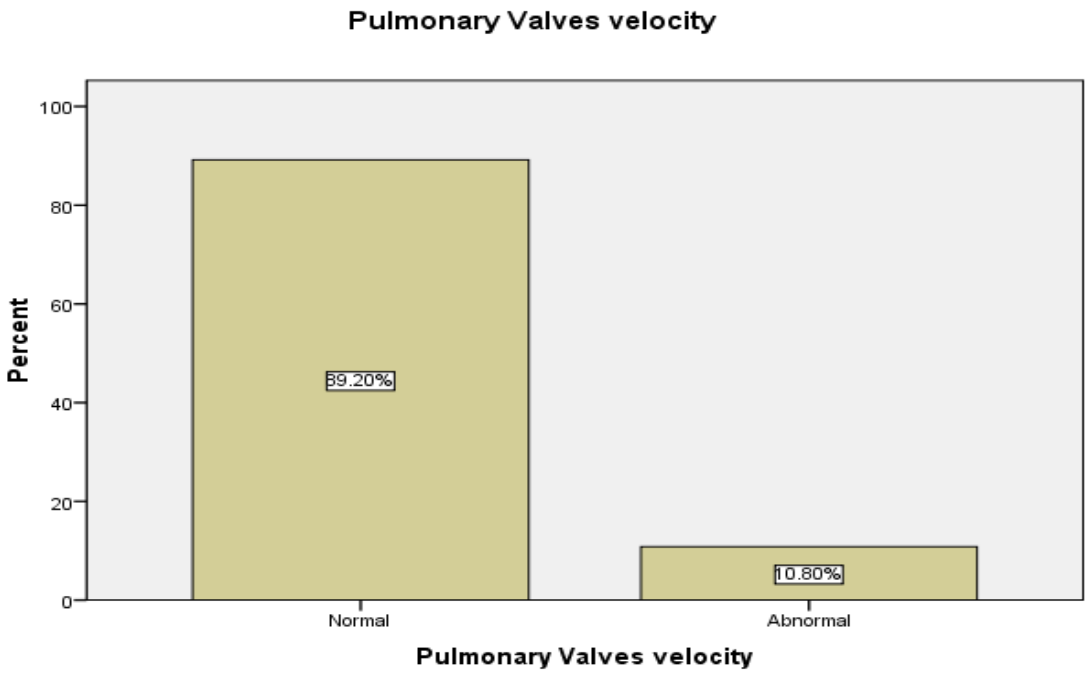

Figure .4.11 *Pulmonary valve velocity of myocardial infarction patients.*

Table 4.12: Aortic valve velocity of myocardial infarction patients

Aortic Valve velocity	Frequency	Percentage (%)
Mean, Std. Deviation (m/sec)	1.44±.45	
Normal	221	88.4
Abnormal	29	11.6
Total	250	100.0

The mean and standard deviation of aortic valve velocity were 1.44±.45 m/sec. Among them 221 patients (88.4%) had normal velocity, while 29 patients (11.6%) had abnormal velocity.

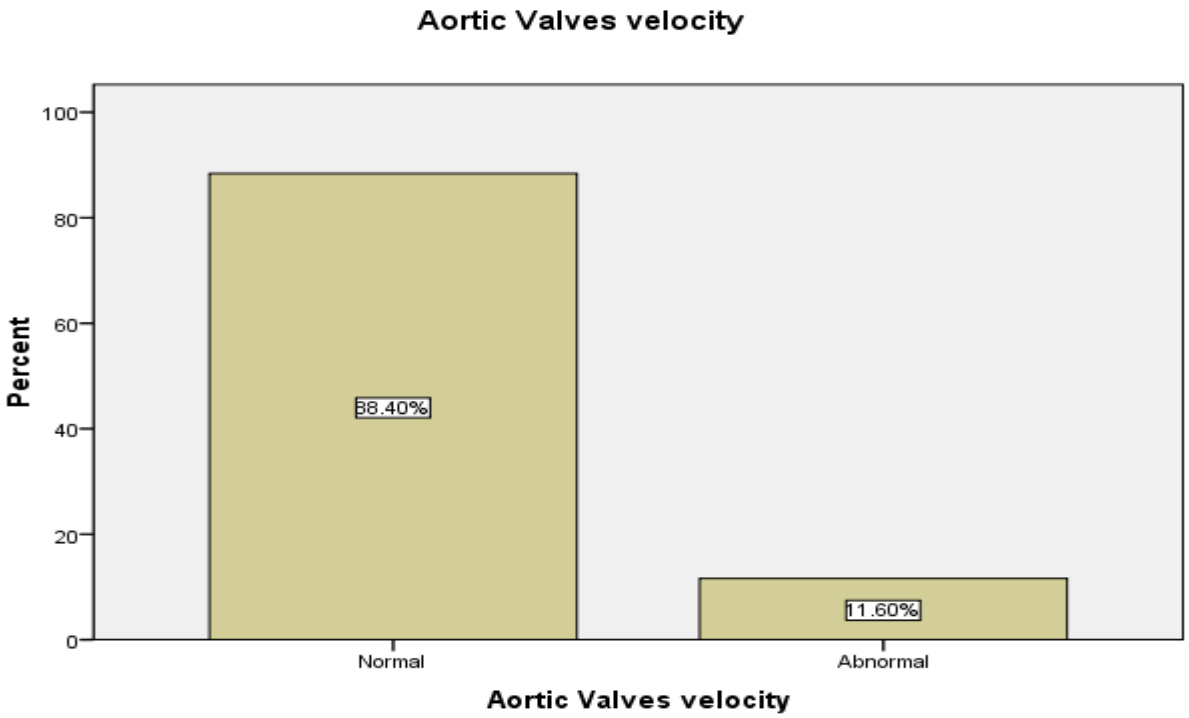

Figure .4.12 *Aortic valve velocity of myocardial infarction patients*.

Table 4.13: Pericardium characteristic of myocardial infarction patients

Pericardium characteristic	Frequency	Percentage (%)
Normal	162	64.8
Pericardial effusion	63	25.2
Acute pericarditis	25	10.0
Total	250	100

162 patients (64.8%) had normal pericardium, 63 patients (25.2%) had pericardial effusion, while 25 patients (10%) had acute pericarditis.

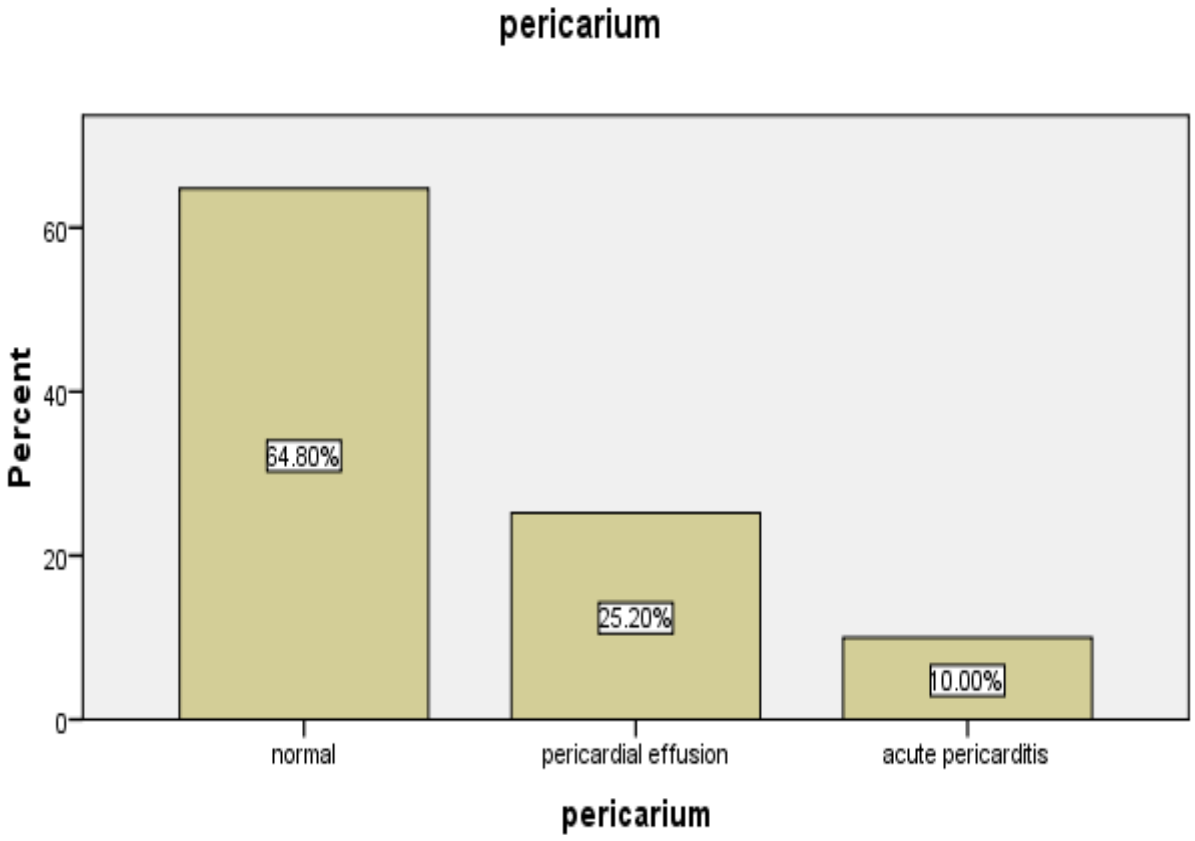

Figure .4.13 *Pericardium of myocardial infarction patients.*

Table 4.14: Ejection Fraction of myocardial infarction patients before used drugs

	Minimum	Maximum	Mean	Std. Deviation
Ejection Fraction % before used drugs	25	49	41.1	3.9

Ejection fraction before used treatment ranged from 25 to 49 with mean and standard deviation 41.1±3.9% .

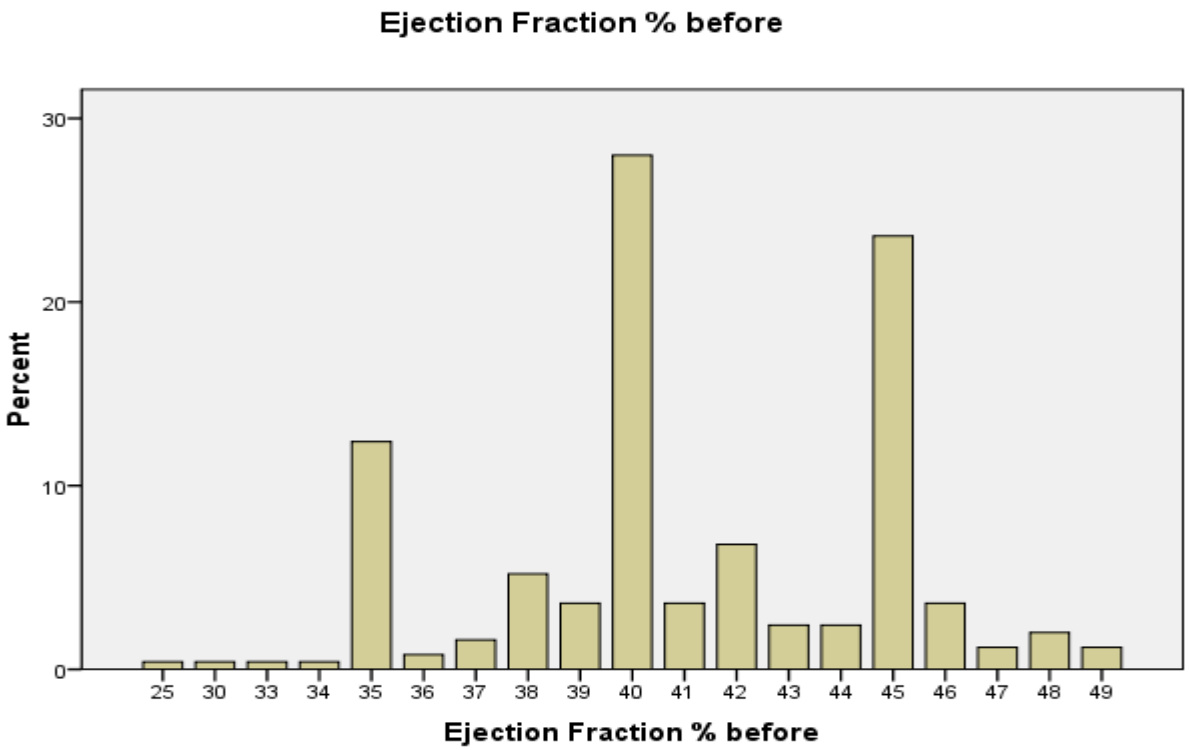

Figure .4.14 *Ejection Fraction of myocardial infarction patients before used drugs.*

Table 4.15: Ejection Fraction of myocardial infarction patients after used drugs

Ejection Fraction		
Range, Mean, Std. Deviation (%)	**25-75(56.8±8.7)**	
Ejection Fraction pattern	**Frequency**	**Percentage (%)**
Normal	176	70.4
Mildly reduced	56	22.4
Moderately reduced	17	6.8
Severely reduced	1	0.4

Ejection fraction after used treatment ranged from 25 to 75 with mean and standard deviation 56.8±8.7 %.

Among them 176 patients (70.4%) had normal ejection fraction, 56 patients (22.4%) had mildly reduced ejection fraction, 17 patients (6.8%) had moderately reduced ejection fraction, while 1 patient (.4 %) had severely reduced ejection fraction.

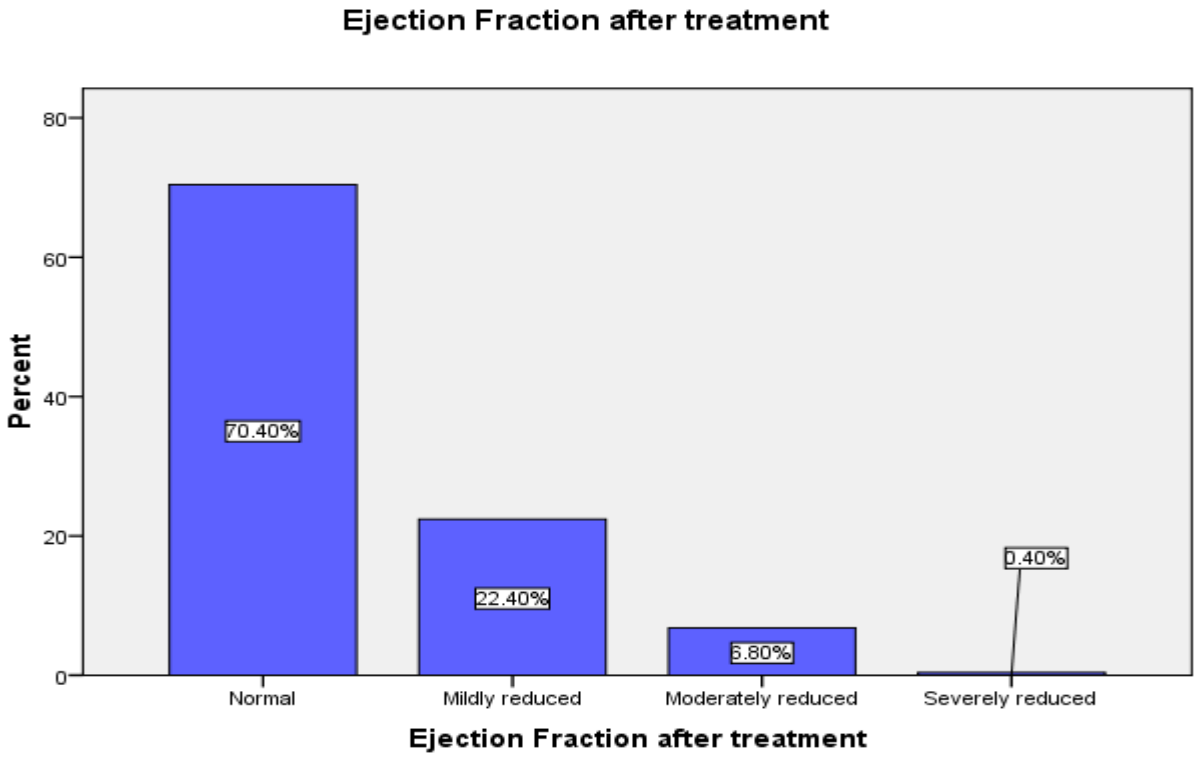

Figure .4.15 *Ejection Fraction of myocardial infarction patients after used drugs.*

Table 4.16: Chi square test of effect of risk factors on myocardial infarction occurrence

Risk Factor	Chi -Square	p-value
Age	68.640	<0.001
Gender	29.584	<0.001
Body mass index	19.344	<0.001
Residence	49.568	<0.001
History	13.536	<0.001

Chi-Square test showed a highly significant difference between the two variables (p <0.001 for all parameters)

Table 4.17: Chi -Square test of the effects of drugs in echocardiographic parameters of myocardial infarction patients

Echocardiographic parameter	Chi- square	p-value
Lt ventricle size	92.416	<0.001
IVS thickness	310.352	<0.001
Posterior wall thickness	373.648	<0.001
Mitral Valve velocity	408.824	<0.001
Tricuspid Valve velocity	69.696	<0.001
Pulmonary Valve velocity	353.288	<0.001
Aortic Valve velocity	147.456	<0.001
pericardium	120.056	<0.001
Ejection Fraction	300.432	<0.001

Chi-Square test showed a highly significant difference between the two variables (p <0.001 for all parameters) .

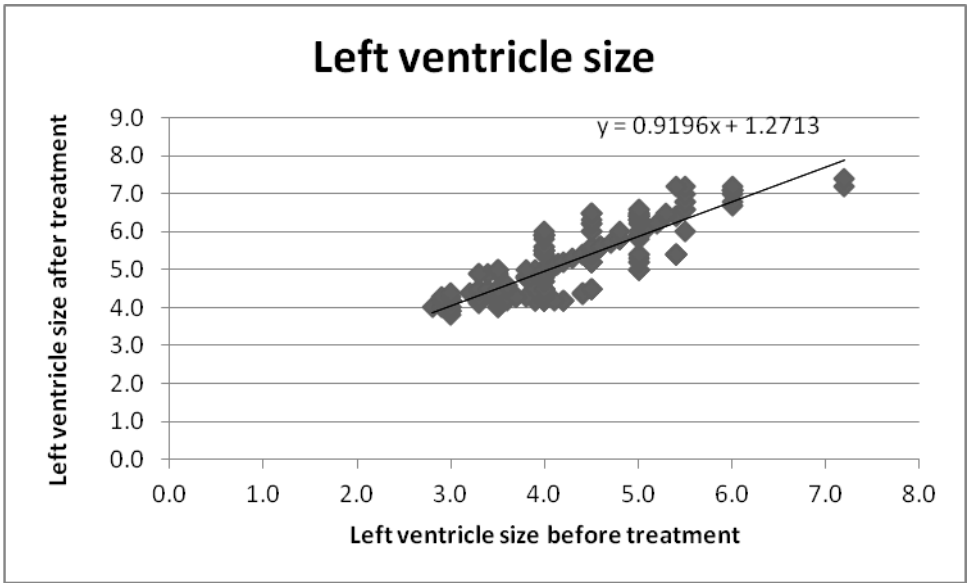

Figure .4.16 *Scatter plot for the relationship between left ventricle size before and after treatment.*

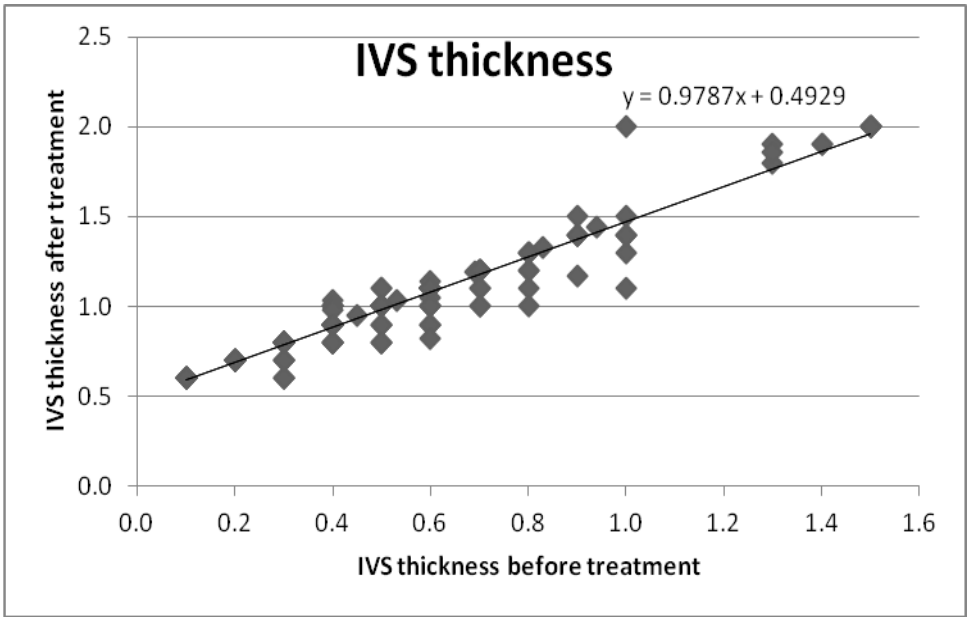

Figure .4.17 *Scatter plot for the relationship between IVS Thickness before and after treatment.*

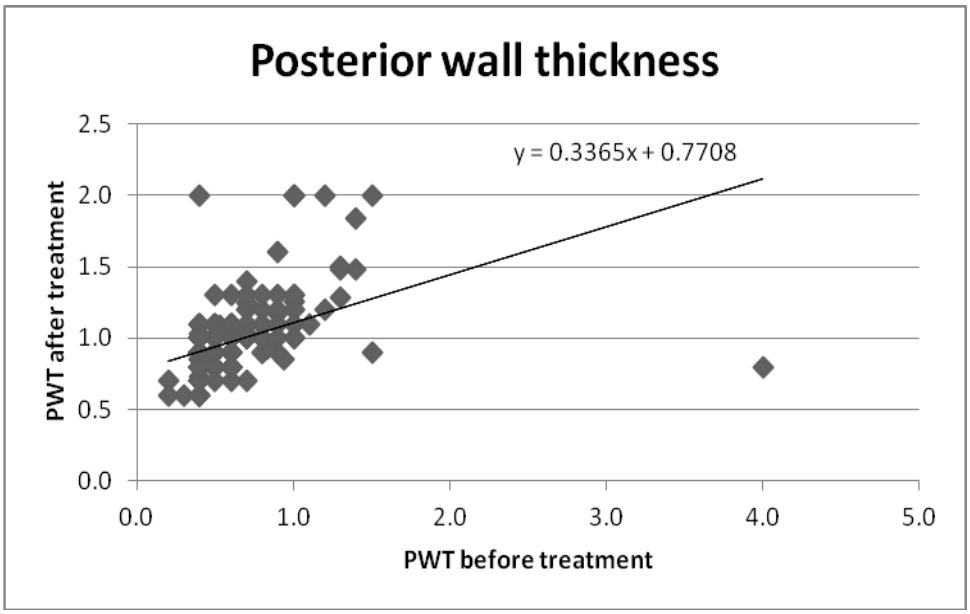

Figure .4.18 *Scatter plot for the relationship between PW Thickness before and after treatment.*

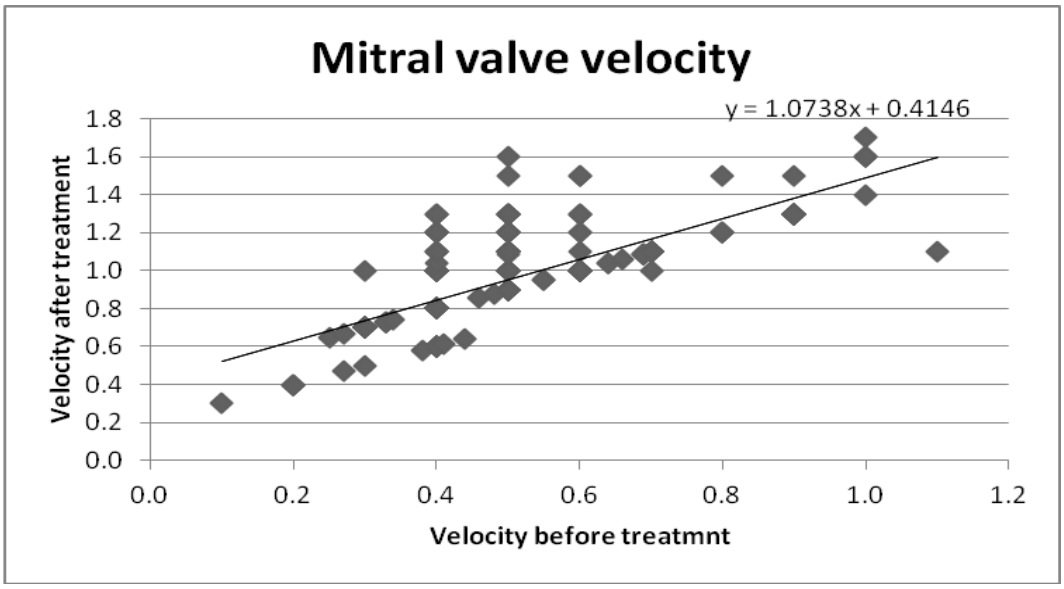

Figure .4.19 *Scatter plot for the relationship between Mitral valve velocity before and after treatment.*

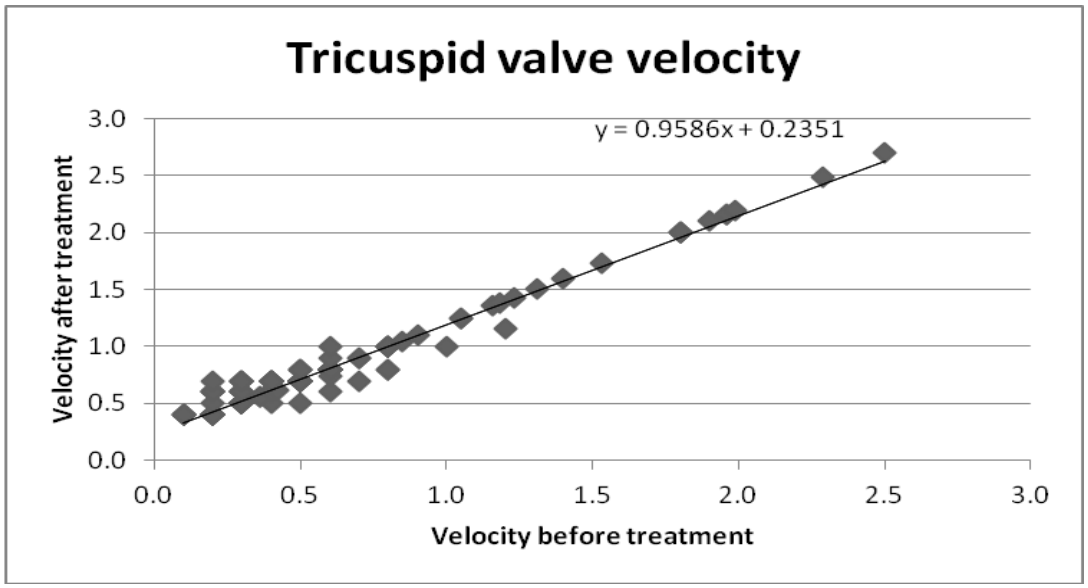

Figure .4.20 *Scatter plot for the relationship between Tricuspid valve velocity before and after treatment.*

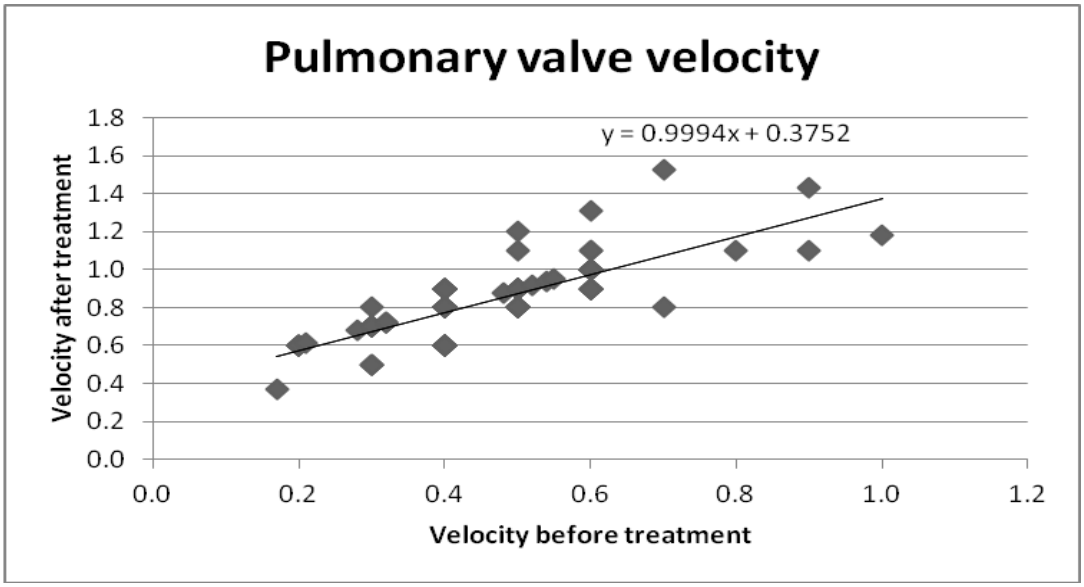

Figure .4.21 *Scatter plot for the relationship between Pulmonary valve velocity before and after treatment.*

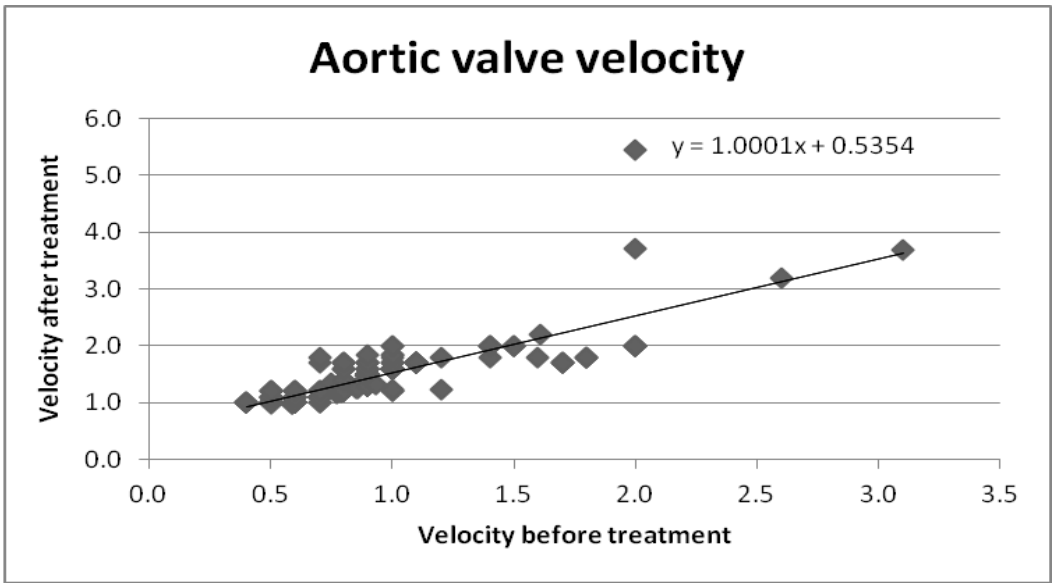

Figure .4.22 *Scatter plot for the relationship between Aortic valve velocity before and after treatment.*

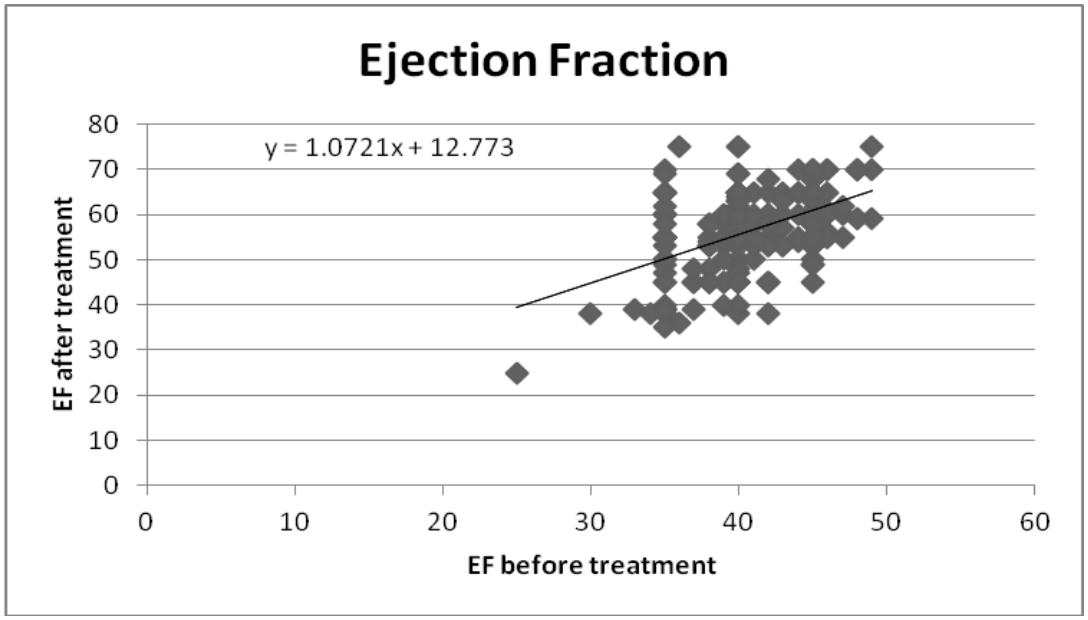

Figure .4.23 *Scatter plot for the relationship between Ejection fraction before and after treatment.*

CHAPTER FIVE

Discussion, Conclusion and Recommendations

5.1 DISCUSSION

Coronary heart disease (CHD) is the leading cause of morbidity and mortality in men as well as women all over the world (Chaudhary and Khan, 2003).The incidence and mortality rates of CHD have been halved in all age groups because of population-wide improvements in the major risk factors, particularly smoking, cholesterol, and blood pressure (Capewell et al., 2000). In fact, the figures in young patients may be lower than actual because of atypical presentation and reluctance to submit themselves for further investigations (Klein and Nathan, 2003).

Two-dimensional echocardiography has recently gained popularity as a noninvasive diagnostic aid in the evaluation of various forms of heart disease. Although M-mode echocardiography is valuable in detecting wall motion changes related to ischemia (Corya et al., 1975), and occasionally in predicting the clinical course of patients after myocardial infarction, two-dimensional echocardiography, with its expanded view in real-time of the left ventricle, has enhanced our ability to evaluate wall motion changes caused by coronary artery disease (Kisslo et al.,1977 and Morganroth et al.,1981).

current study revealed that the percentage of the elderly MI patients were more than in young MI patients, it comprised 188 patients in old group (75.2%), whereas 62 patients in young group (24.8%).This finding was supported by Kam et al., (2002) who found that elderly MI patients were more likely compared to young MI patients. And also by Novella et al., (2008) who indicate that the incidence of fatal and non-fatal myocardial infarction was very high in the elderly population of Madrid. And with Fournier et al., (1996) who stated young patients with AM1 represent 4.1 % of the patients with AM1 admitted to our hospital between June 1986 and December 1992.

This study found that the percentage of male patients was more than female patients, 67.2% males and 32.8% females. This finding was in agreement with Farah et al.,(2008) who reported that out of the one hundred (100) Sudanese patients presented with acute MI; males were about twice the females (69% vs. 31%).And with Aubeidia (2006) who state that Most of the sample in the study was males; they represent about 72% of our study, while females were 28%. Also with Elbasheer (2013) who reported that CAD is a predominantly male disease in Sudanese patients as is the case of the rest of the world.

On other hand, current study revealed that BMI has significant difference.

This finding was in agreement with Jousilahti et al.,(1996) who reported that obesity is a risk factor for AMI, but is also strongly associated with other factors in the development of coronary heart disease, such as hypertension and diabetes mellitus, as well as dyslipidaemia and inflammation, and with Aubeidia (2006) who report the result of overweight among myocardial infarction patients still high.

This study found that percentages of overweight and obese were higher in male patients than female patients. This finding was in agreement with Kragelund et al., (2005) in first part who reported that the prevalence of overweight (BMI 25–29.9 kg/m2) and obesity (BMI>30 kg/m2) were 48% and 13% in males and 31% and 13% in females.

Current study found that percentage of the patients from metropolitan area (MA) were more than nonmetropolitan urban area (UA) and rural area (RA). This finding was in agreement with (Chauhan &Aeri,2013) who found Data also suggests that although the prevalence rates of CVD in rural population will remain lower than that of urban populations. And with Krisela et al.,(2005) who stated that Reports from various African countries document a changing pattern and spectrum of CVD and their risk factors, especially in urban areas. In contrast to the above studies Pandey et al.,(2010) reported that interpretation of results shows 70% of MI cases were from rural population of Jhalawar.

Cardiovascular diseases, Diabetes mellitus, hypertension, obesity and smoking were common causes of myocardial infarction in this study. Cardiovascular diseases more common in age above 60 year in both genders, Diabetes mellitus was common in males under and above 60 year age. Hypertension was more common in above 60 year in both genders and also in male under 60 year. Obesity was common in female above 60 year. Smoking was common in male age above 60.

These findings were in agreement with (Gonzalez et al.,1996 and Gonzalez et al.,1992) who reported that the incidence rate of MI accords with the high prevalence of classic cardiovascular risk factors among this population, such as diabetes, hypertension, and dyslipidemia. Also Machete et al. (1997) found that hypertension and diabetes mellitus are less frequent in younger MI patients than in older MI patients. In 2003; Majahalme showed that more hypertensive patients with acute coronary syndrome (ACS) were women, older, and had more co-morbidities. And with Abu-Ali, (2003) who reported that diabetes increases the risk for AMI attack rate, incidence, casefatality, recurrence and mortality and is an important contributor to all AMIs in middle-aged people. Must &Strauss (1999) had reported that obesity in childhood/adolescence has many negative health consequences, which have been divided into the categories of immediate

physical and social, intermediate (cardiovascular risk factor levels, persistent obesity into adulthood), and long-term (adult morbidity and mortality).
In contrast to current study, Sytkowski et al., (1996) reported that smoking is the risk factor of CHD that occurs most frequently in young MI patients, affecting 76% to 91% of all young MI patients, while in older MI patients the prevalence drops to approximately 40%. Moreover. Barbach et al., (1995) found the actual number of cigarettes smoked per day is significantly higher in young patients as compared to older patients.

current study found that patients with normal left ventricular size, Interventricular septal thickness and Posterior wall thickness had highest percentage. The patients in the study by Kasner et al., (2007) exhibited normal LV chamber size, increased myocardial mass, and a high ratio of mass to volume. Rawat and Satyal, (2003) reported that present study showed relatively increased thickness of IVS and LVPW in untreated patients with marked difference in older patients consistent with other studies. In contrast to the above studies Dąbrowski et al., (2012) who state that the most important finding of our study was the confirmation of the hypothesis that in addition to the reduction of gradient in the LVOT there was also a decrease in IVS thickness also outside the MI area and the free wall of the LV.

On other hand, this study revealed that mitral, tricuspid, pulmonary and aortic valve velocity within standard normal limits indicated by the international literature in most cases. These findings were in agreement with Garot et al.,(1999) who reported that our data confirmed that systolic and diastolic myocardial regional velocities, as assessed by colour M-mode Doppler tissue imaging, were significantly reduced in the infarct region when compared with the corresponding walls in healthy subjects. In addition Witt et al., (2011), stated that at day 1, tricuspid annular velocities were significantly reduced in patients with, compared to those without, ST elevation myocardial infarction. Also Bergeron et al., (1975) reported that this study suggests that mitral valve diastolic velocity follows a characteristic triphasic pattern after acute myocardial infarction. In addition, an initial slow diastolic velocity with a subsequent increase prior to discharge from the hospital was noted.

current study found that patients with normal pericardium had highest percentage then patients with pericardial effusion and last patients who had acute pericarditis. Percentage of pericardial effusion and acute pericarditis in this study were (25.2%, 10% respectively). This finding was supported by Hafiz-ur-Rehman et al., (2010) who reported that the number of patients having PE increases during in-hospital stay as 4.5% of our patients had PE at the time of admission but on day four it was 15%, same trend was reported by Toth et al., (1997) (8% on Day 0, 24% on Day 7). In addition to that Wilansky (1991) who state that the

incidence of post infarction pericardial effusion as detected by use of 2-dimensional echocardiography is reported to be from 25% to 28%. This finding was supported by Pierard et al., (1986) who reported that "the results of this study show a 26% incidence of pericardial effusion after acute myocardial infarction as determined by two-dimensional echocardiography". In contrast to above Somolinos et al., (1987) stated that early acute pericarditis (EAP) was clinically recognized in 19 (41%) patients. Pericardial effusion (PE) was detected in 29 (63%) patients.

The results of this study show significant increase in left ventricle size, IVS thickness, PW thickness, mitral valve velocity,tricuspid valve velocity, pulmonary valve velocity, aortic valve velocity and ejection fraction after received drugs ($p<0.001$); the increase rate for variables was 0.92 cm,0.98 cm,0.34 cm, 1.07 m/sec, 0.96 m/s, 1.00 m/sec, 1.00 m/sec and 1.07% respectively .Mean of ejection fraction after used treatment were 56.83±8.7 % (70.4%) had normal ejection fraction, (22.4%) had mildly reduced ejection fraction, (6.8%) had moderately reduced ejection fraction and (.4 %) had severely reduced ejection fraction. This finding was in agreement with Amiel *et al*., (2012) who reported that mean LVEF was 54 ± 19%. According to TTE-derived LVEF values, 8 patients had an increased LVEF, 51 patients had a normal LVEF, 14 patients had a moderately reduced LVEF and the remaining 11 patients had a severely reduced LVEF. And also supported by Anane *et al*., (2012) who found that a significant increase in the LVEF occurred after treatment with the β-blockers, and with Van Campen *et al*.,(1998) who reported that in almost all studies, significant increase in the LVEF occurred after treatment with beta-blockers. And confirmed by Meyer *et al*.,(1994) who reported that aspirin as an anti-platelet agent has well established role in the setting of acute myocardial infarction and has been clearly documented to reduce mortality alone by 23%. S. Yusuf *et al*., (1996) found that the management of patients with AMI should be based on relevant and reliable evidence from large randomized controlled trials. In the acute phase, thrombolysis therapy is supported by such a level of evidence. And also with Werter *et al.*,(1992) who stated that in addition to reducing mortality, aspirin also reduces strokes and myocardial infarction. The exact mechanisms of the benefit of aspirin is not known. Recent data suggest that facilitation of coronary recanalization occurs with aspirin use after successful thrombolysis.Also supported with In the longer term, the efficacy of aspirin, beta-blockers and ACE-inhibitors is similarly supported and they should be adopted for widespread prescription as simple effective and affordable drugs. Their dosages should be tailored to the individual but should pertain as closely as possible to those which were used in the relevant positive clinical trials.

5.2 CONCLUSION

This study had been carried out in echocardiography department of Sudan Heart Center in Khartoum-Sudan.

The aim of this study was to measure the predisposing factors of MI (V.S), BMI, left ventricle size IVS thickness, posterior wall thickness, pericardium, valves velocity and ejection fraction, to predict the occurrence of MI in young, to correlate between the age and MI incidence, to determine the incidence of MI in gender, to test the response of MI to treatment. Two hundred and fifty subjects were included in this study.

All subjects of the study were subjected to the following:

- History taking and complete clinical examination.
- Calculation of BMI.
- Echocardiogram.

current study confirm that echocardiography is a valuable tool for evaluation of patients with myocardial infarction.
As regard age; this study showed that the elderly patients were more than young patients. Majority of patients are in above 70 age group. MI in young is more common in males than females.
As regard gender of MI; current study found that men show higher rates than women at any age.
As regard treatment of BMI, this study found that BMI shows significant association with myocardial infarction risk worldwide. BMI is a useful statistical tool to track the body size trends in a population.
As regard MI risk factors; current study revealed that traditional risk factors including smoking, cardiovascular disease, Diabetes Mellitus, hypertension and family history were significantly higher in old MI patients than in young MI patients

As regard echocardiograhical findings, current study showed that the means and range for the measurements of left ventricular diastolic dimension, IVS thickness, posterior wall thickness, pericardium and valves velocity found in this survey were within standard normal limits indicated by the international literature. PE is observed frequently after AMI and that the echocardiographic study can help in the diagnosis of EAP after AMI.

As regard treatment of AMI, this study showed that the AMI is one of the treatable cardiac disease depending on how the individual will act immediately when the symptoms arises. The health personals should keep in mind about the patient's condition and always be concerned about support, counseling and their coping ability. Keeping the patients informed about the disease preventive measures should be taken into consideration always. The study has shown that the ability of overcoming the disease differs between young patients and elderly. Even though, the prognosis of AMI in elderly is poor, it is important to be concerned about prevention.
The management of patients with AMI should be based on relevant and reliable evidence from large randomized controlled trials. In the acute phase, thrombolysis
therapy is supported by such a level of evidence. In the longer term, the efficacy of aspirin, beta-blockers and ACE-inhibitors is similarly supported and they should
be adopted for widespread prescription as simple effective and affordable drugs.

This study has demonstrated that a significant increase in the LVEF occurred after treatment with the beta blockers. However, this type of analysis did not allow an accurate statistical comparison of the various beta-blockers, for which a meta-analysis with data for individual patients is needed. Nevertheless, the differences in improvement were small and, importantly, mean the baseline LVEF values for the different beta-blocker studies were almost identical.

Again this study has shown that the association of LVEF and mortality changes substantially across the full spectrum of LVEF. This information clarifies the role of LVEF in advanced heart failure patients and will help clinicians provide better estimates of prognosis. In addition, understanding that arrhythmias and worsening heart failure contribute to substantial mortality observed in patients with reduced LVEF may help target future interventions in this population.

Therefore these results may reflect the true burden of MI and risk factor in the community at large. The need of the hour would be to increase the awareness of risk factor for MI among general public. Those at risk could be asked to modify their lifestyle, bring out dietary changes and increase in physical activity. The challenge is to develop appropriate strategies to prevent CAD and promote healthy lifestyles in rural communities. If the growing epidemic of CAD is to be reversed, clinicians, healthcare organizations, policy-makers and communities must work together to translate evidence into action.

5.3 RECOMMENDATIONS

☞Young people with positive family history or smoking must be screened prior to their first AMI to improve primary prevention.

☞ Knowing the prevalence of various modifiable risk factors may help in planning appropriate secondary preventive programs to target the different age groups. Emphasis for the elderly population should be more targeted at better control of hypertension, while for the young population.

☞Patients with a suspected heart attack have a right to expect prompt diagnosis, pain relief, resuscitation and, if indicated, reperfusion treatment.

☞Patients with suspected or confirmed myocardial infarction should be cared for by staff trained and experienced in modern coronary care.
☞ Appropriate facilities for post discharge follow-up, rehabilitation and secondary prevention should be available.
☞ Patients and their associates should be informed of how to recognise and respond to a further heart attack.
☞ Cardiologists, in association with emergency care physicians and health authorities, should ensure that an optimal system for the care of heart attack patients is operative in their area.
☞ There should be a policy for smoking cessation. This must consist of a continuing programs run by health professionals that not only encourages patients to stop, but endeavours to maintain cessation.
☞ Health authorities should encourage the training of the public in basic cardiopulmonary resuscitation techniques and the ambulance personnel in basic and advanced life support.
☞As diet is one of the major risk factors of myocardial infarction, it is essential to find the most effective diet protocol and supply it to myocardial infarction patients in order to lose their weight, this can be done cooperated with qualified dietitian.
☞It is important for patients to follow physical activities hourly every day; this must be followed up by health promoters, public health specialist, physicians and cardiologists.
☞Rehabilitation programs for all patients survive a heart attack (MI), to prevent the complications of the heart attacks.
☞ Further studies are needed to understand the factors affecting response of myocardial infarction to certain drugs.

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APPENDICES

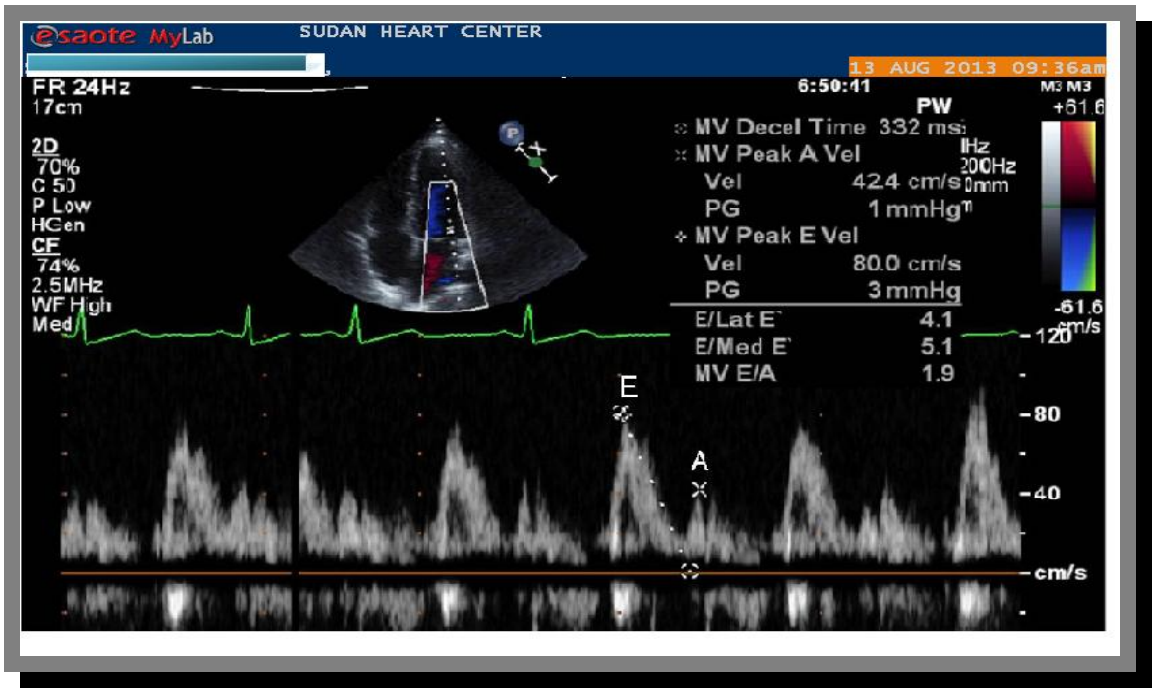

Figure .5.1 *Normal Mitral Valve Inflow Pattern.*

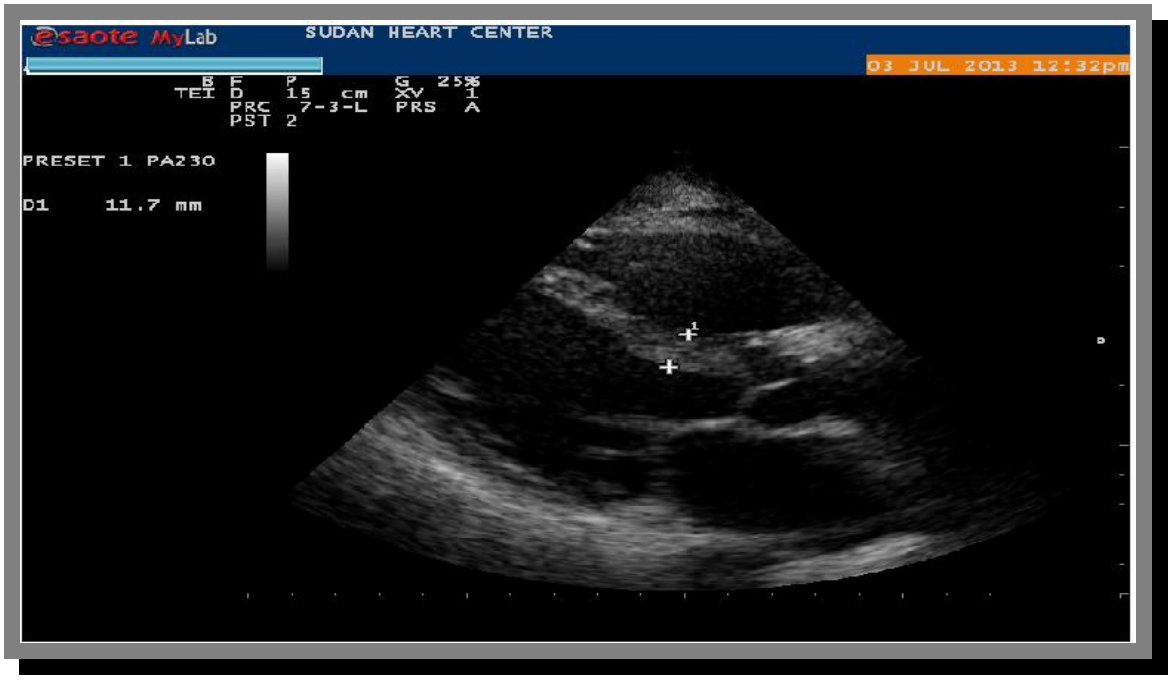

Figure .5.2 *Parasternal long axis shows IVS in mildly abnormal(11.7mm).*

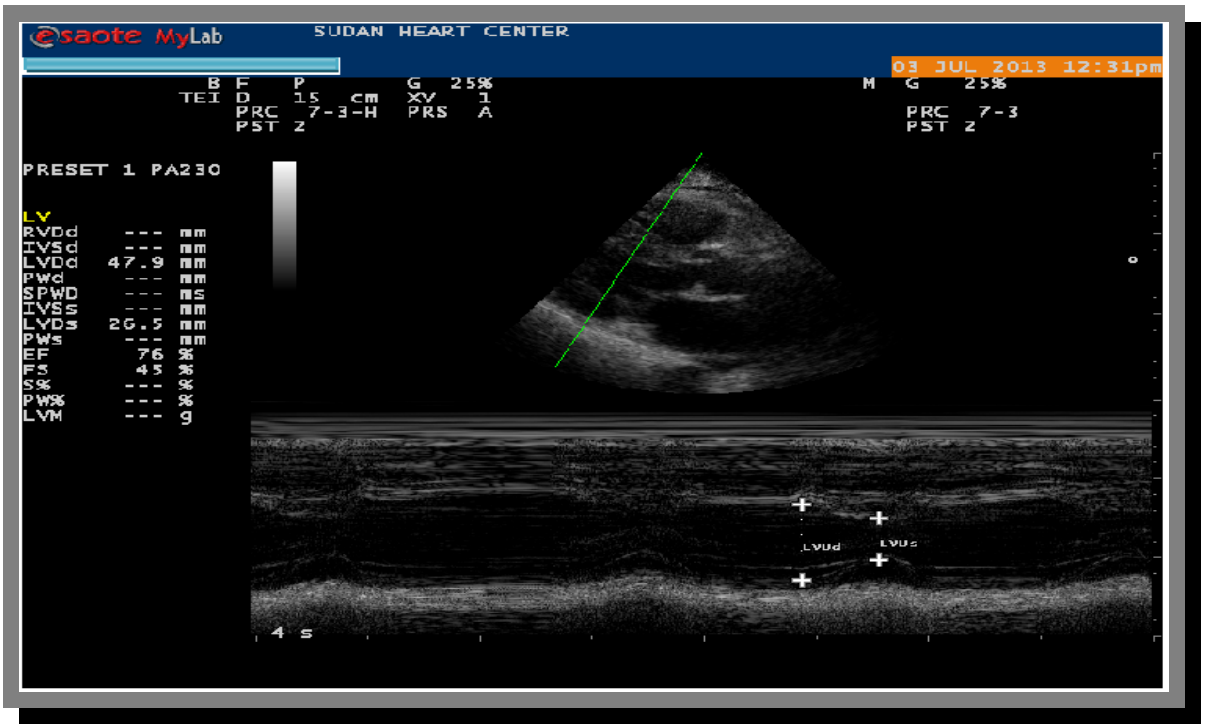

Figure .5.3 *Same patient the LV diastolic measurement is 4.79 cm and the systolic measurement is 2.65 cm and EF76%.*

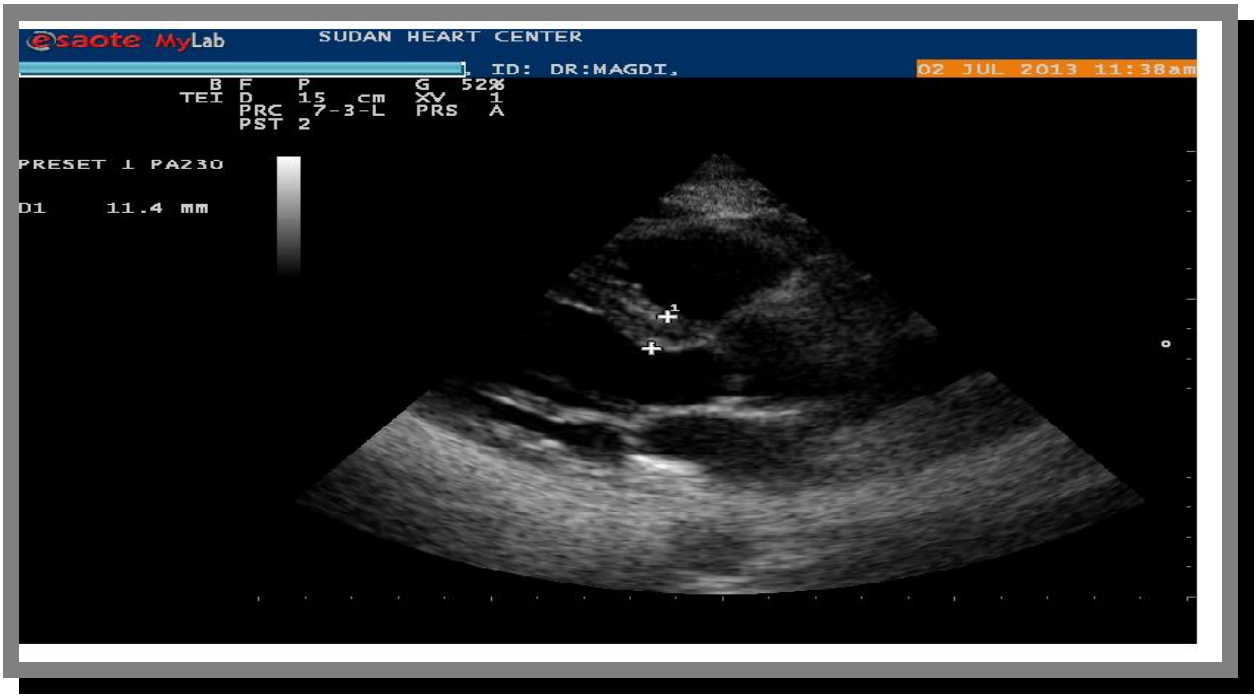

Figure .5.4 *Parasternal long axis shows IVS in mildly abnormal(11.4mm).*

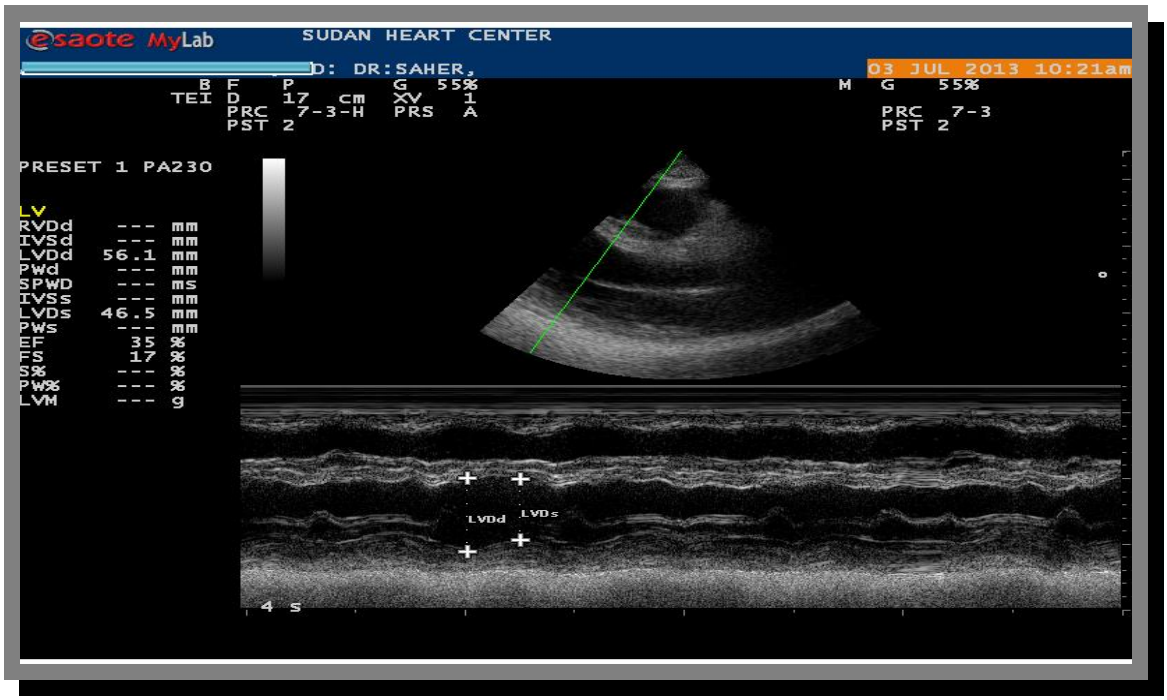

Figure .5.5 *In the M-mode below, the LV diastolic measurement is 5.61cm and the systolic measurement is 4.65 cm, both of which are normal.*

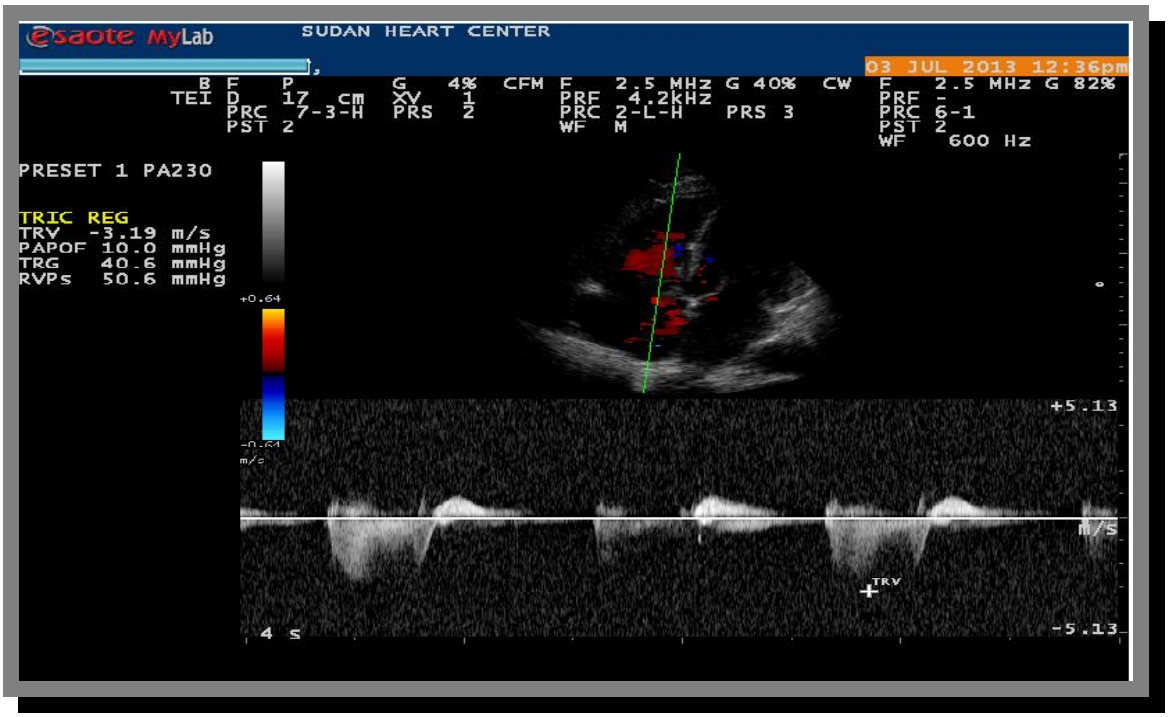

Figure .5.6 *Continuous wave Doppler signal of tricuspid with a peak velocity of approximately 3.19 m/s.*

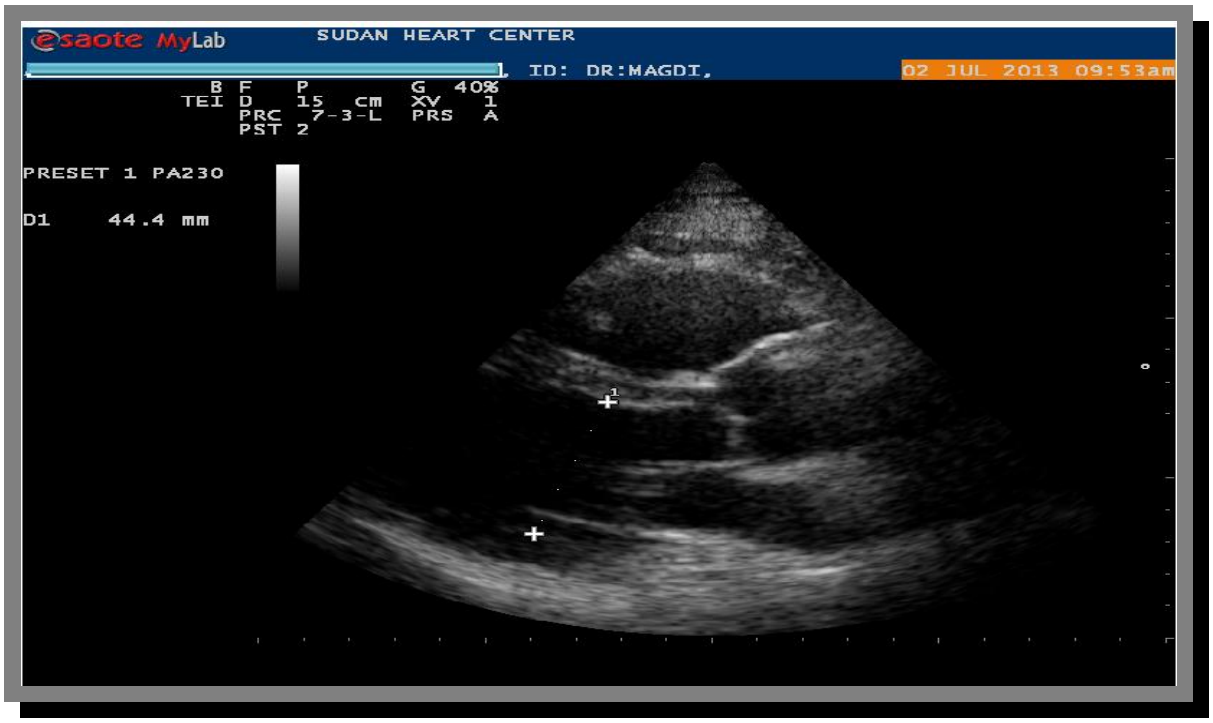

Figure .5.7 *The LV diastolic measurement is 4.44cm.*

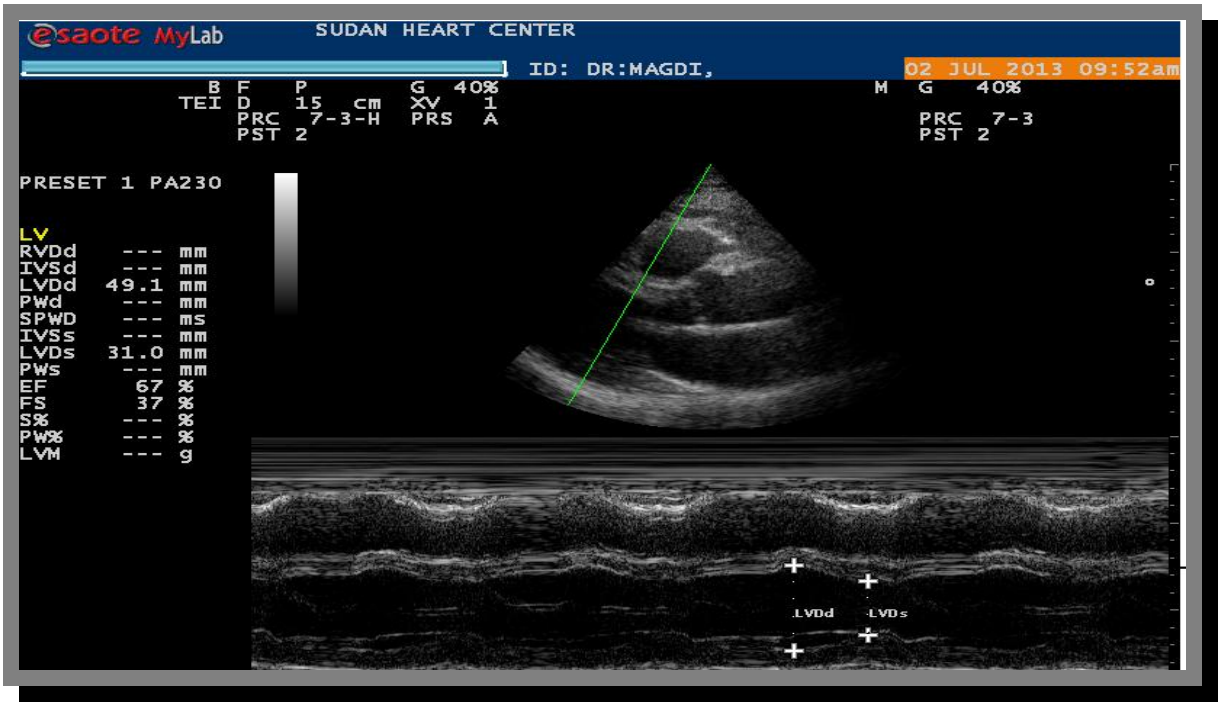

Figure .5.8 *In the M-mode the LV diastolic measurement is 4.91cm and the systolic measurement is 3.10 cm,EF 67% all of which are normal.*

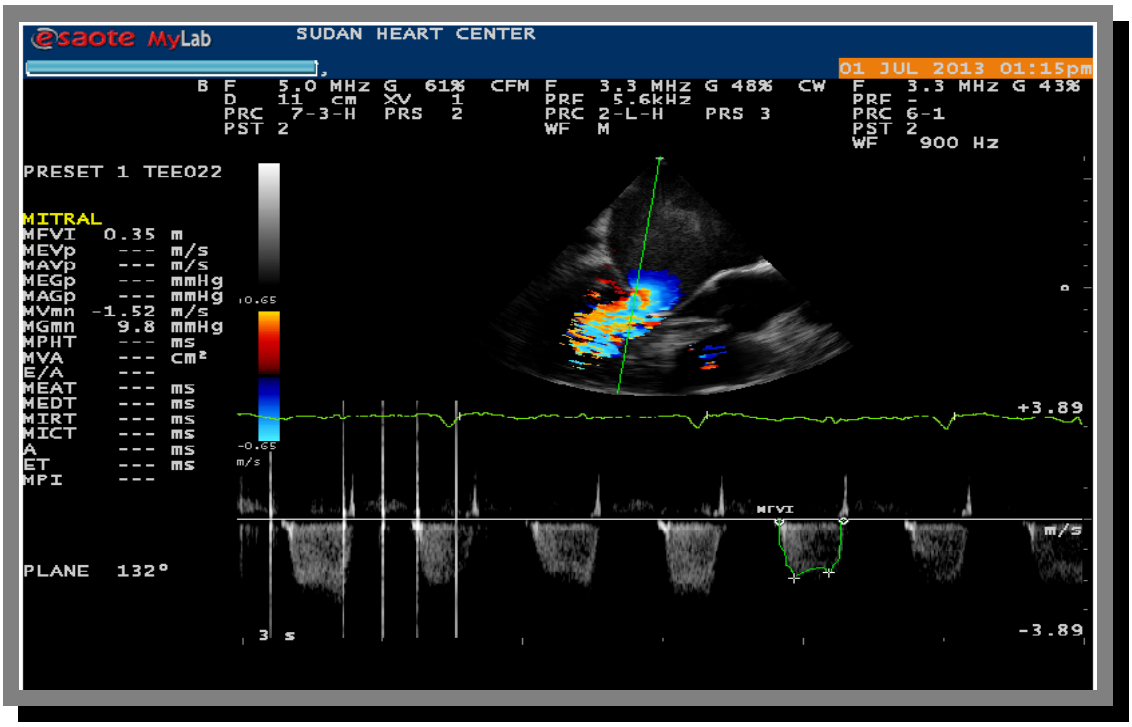

Figure .5.9 *Continuous wave Doppler signal of mitral with a peak velocity of approximately 1.52 m/s.*

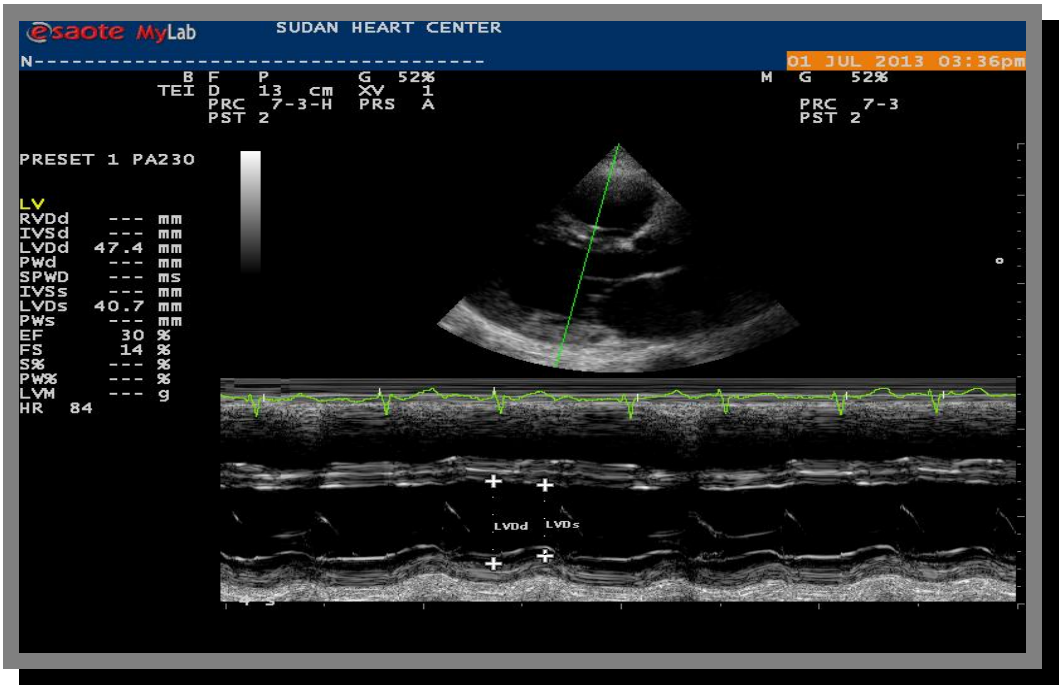

Figure .5.10 *In the M-mode below, the LV diastolic measurement is 4.74 cm and the systolic measurement is 4.07cm and moderately abnormal EF (30%).*

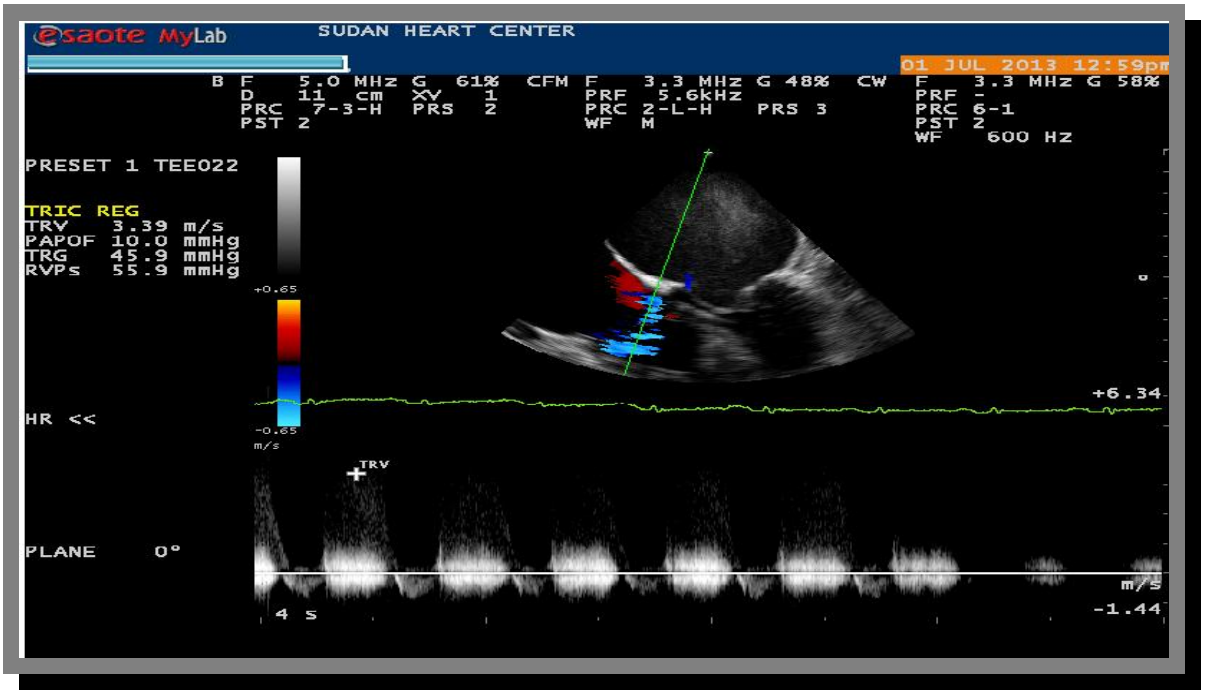

Figure .5.11 *Continuous wave Doppler signal of tricuspid regurgitation with a peak velocity of approximately 3.39 m/s.*

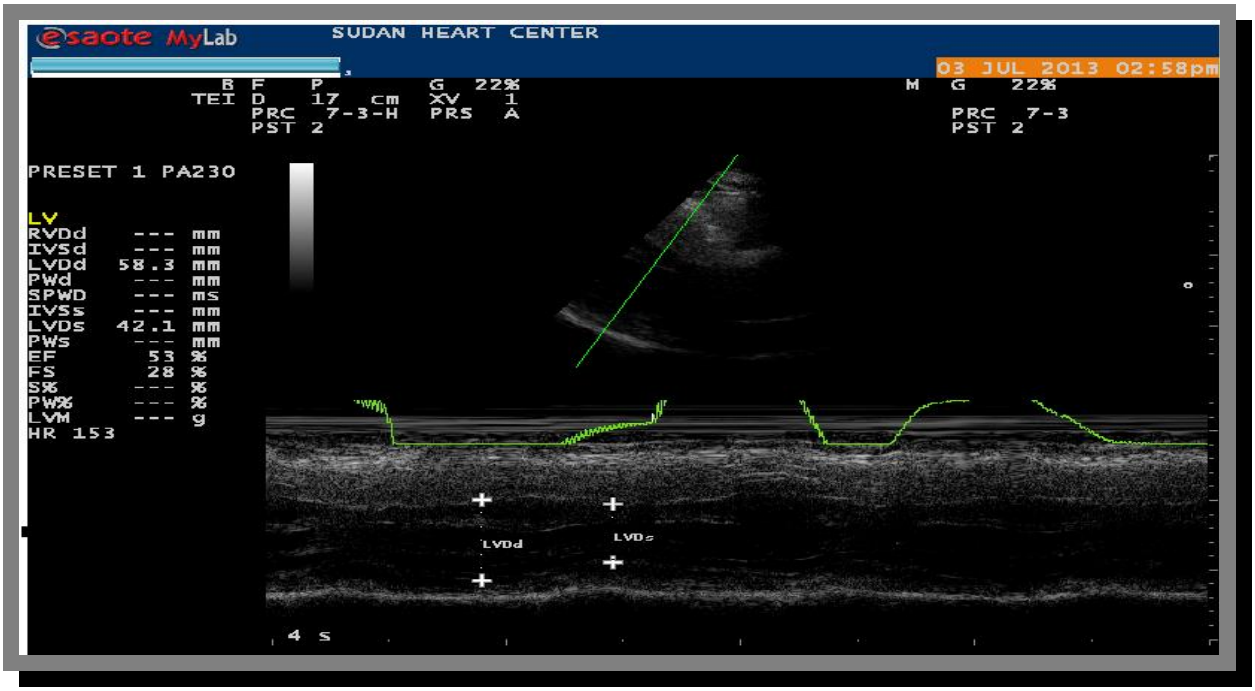

Figure .5.12 *In the M-mode the LV diastolic measurement is 5.83cm and the systolic measurement is 4.21cm, both of which are normal and with mild abnormal EF(53%).*

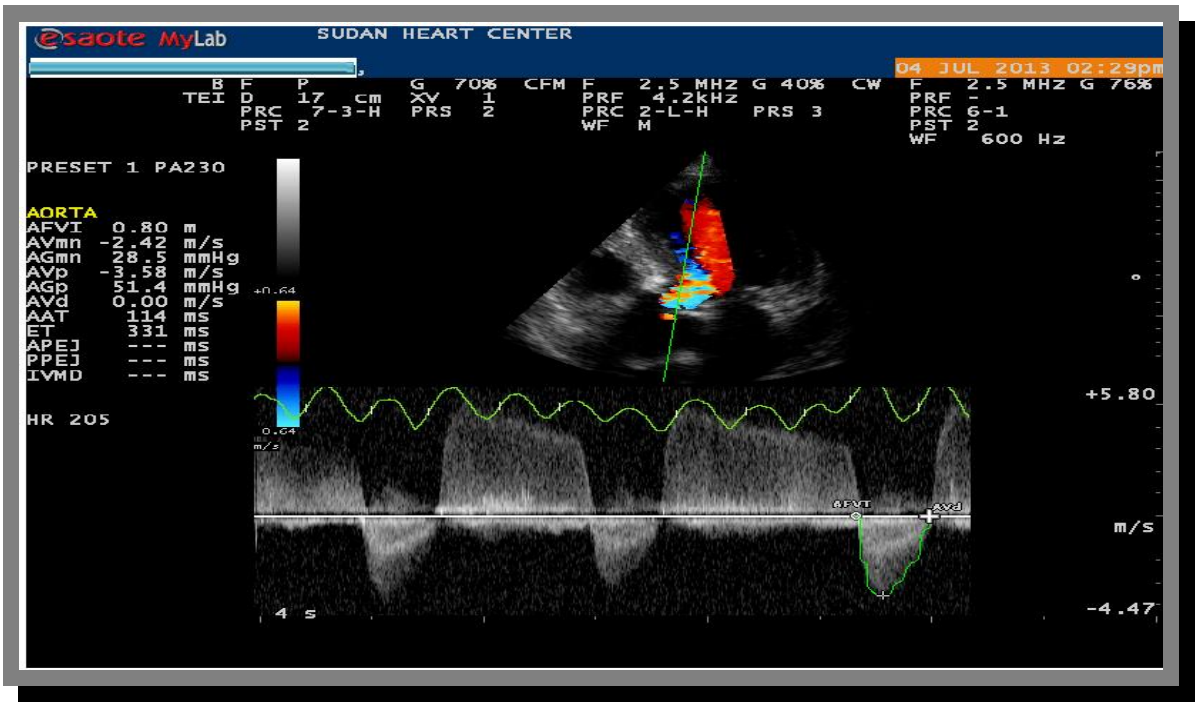

Figure .5.13 *Continuous wave Doppler signal of aorta with a peak velocity of approximately 2.42 m/s.*

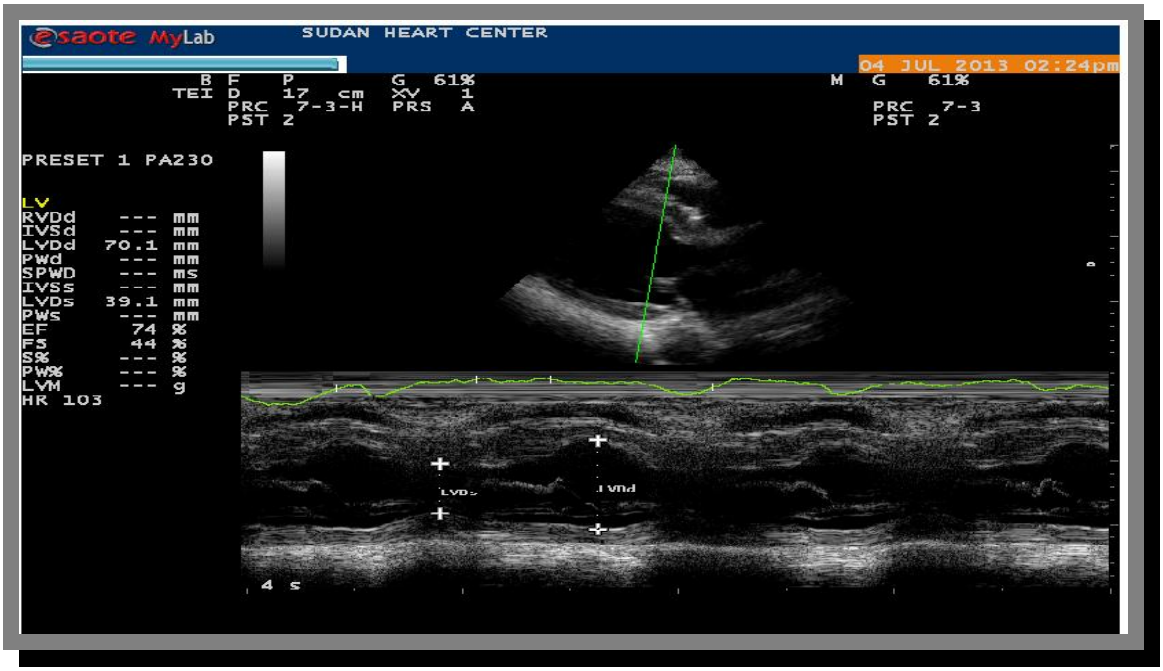

Figure .5.14 *M-mode echocardiogram showing the LV diastolic and the systolic measurement and EF all of which are normal.*

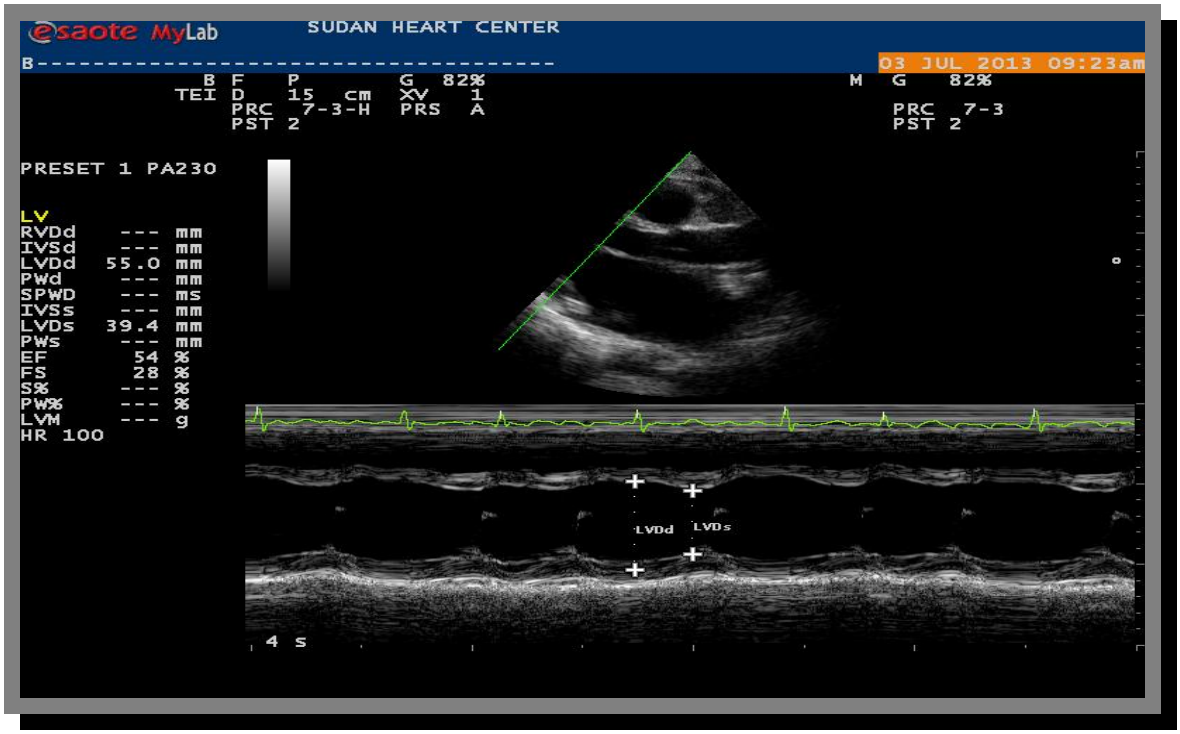

Figure .5.15 *Normal LV diastolic and systolic measurement and mild abnormal EF.*

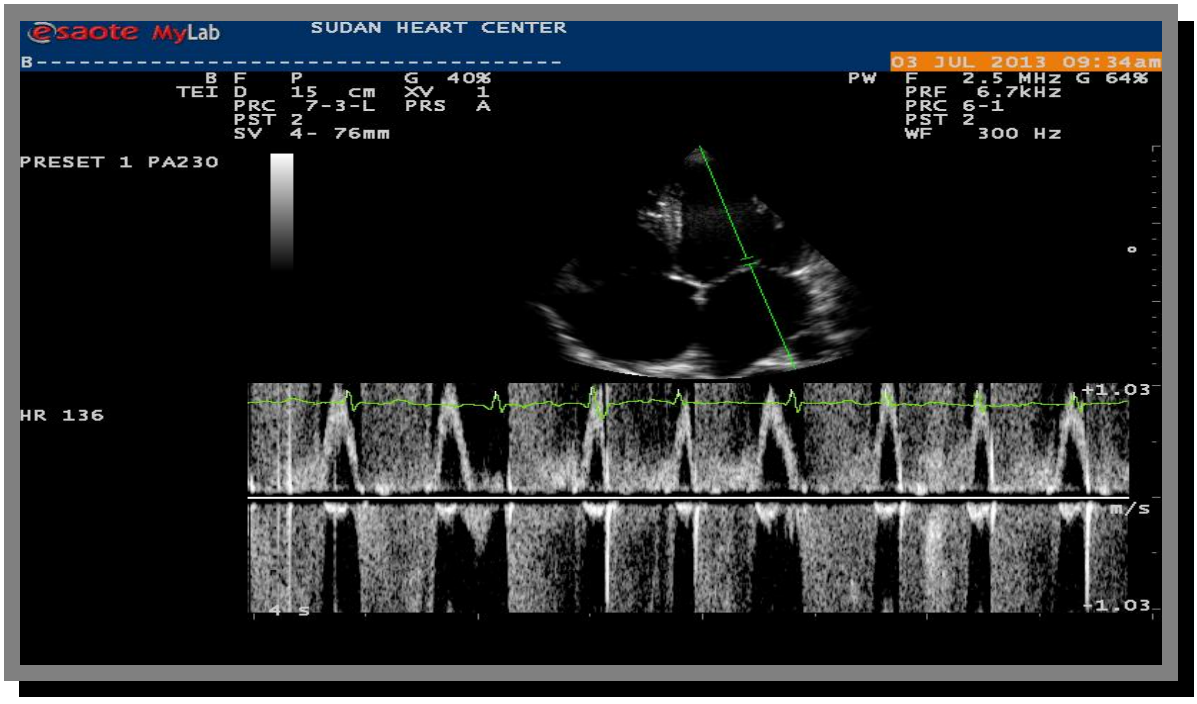

Figure .5.16 *pulse wave Doppler signal of mitralvalve.*

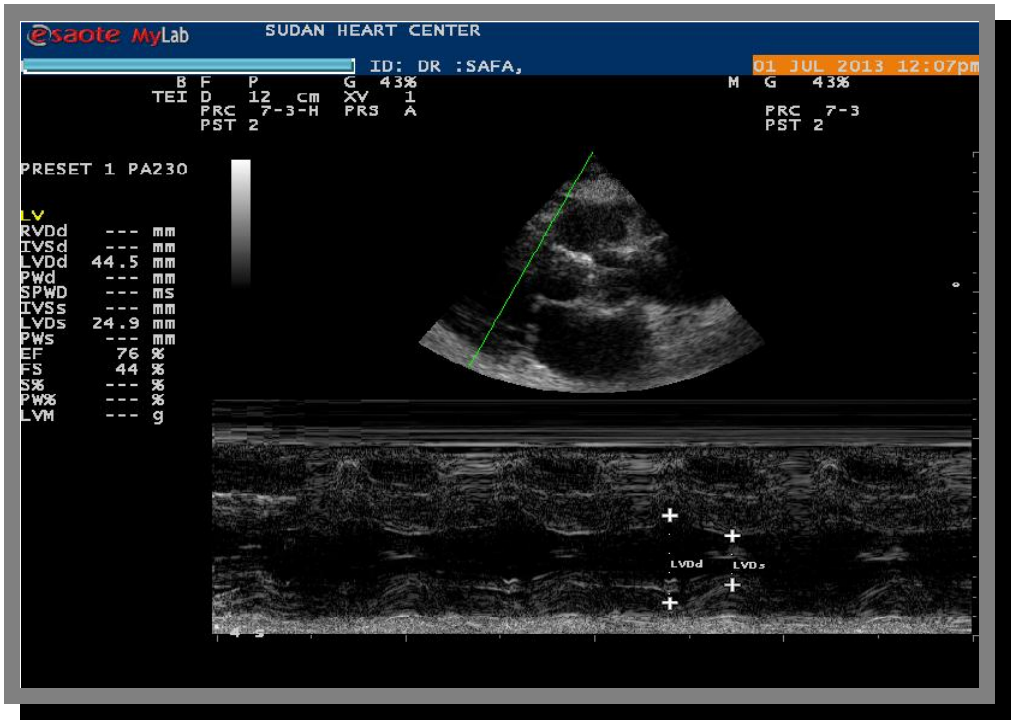

Figure .5.17 *Normal LV diastolic , systolic measurementa and normal EF.*

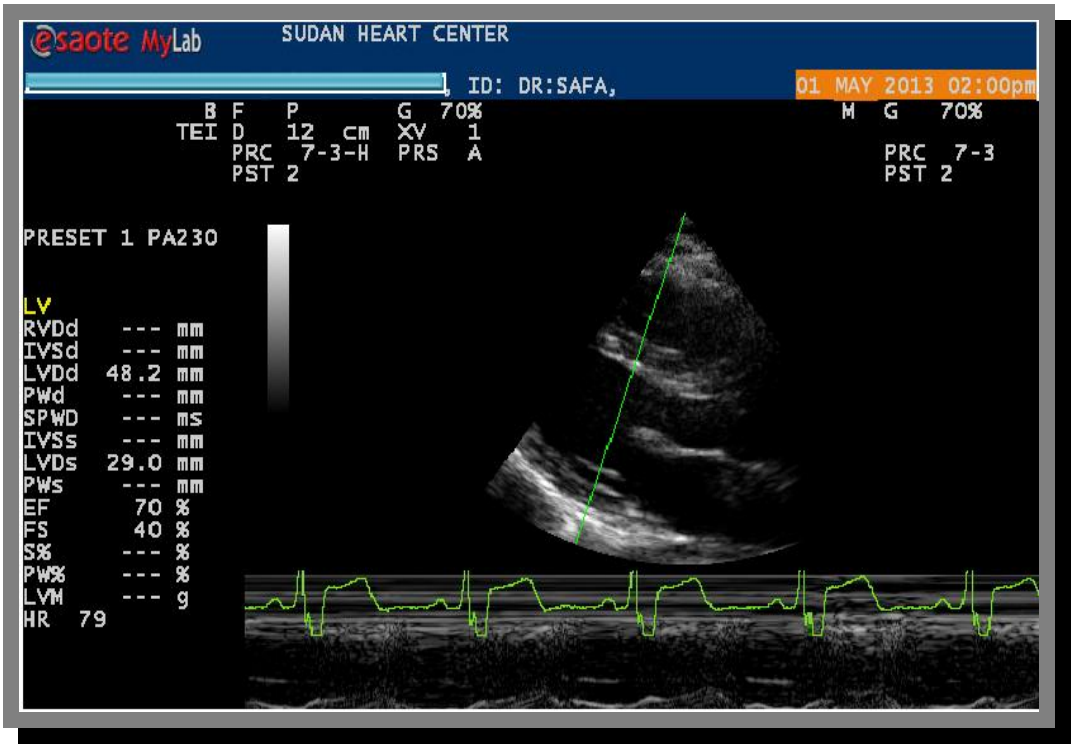

Figure .5.18 *In the M-mode the LV diastolic measurement is 4.82cm , the systolic measurement is 2.90 cm and EF 70%, all of which are normal.*

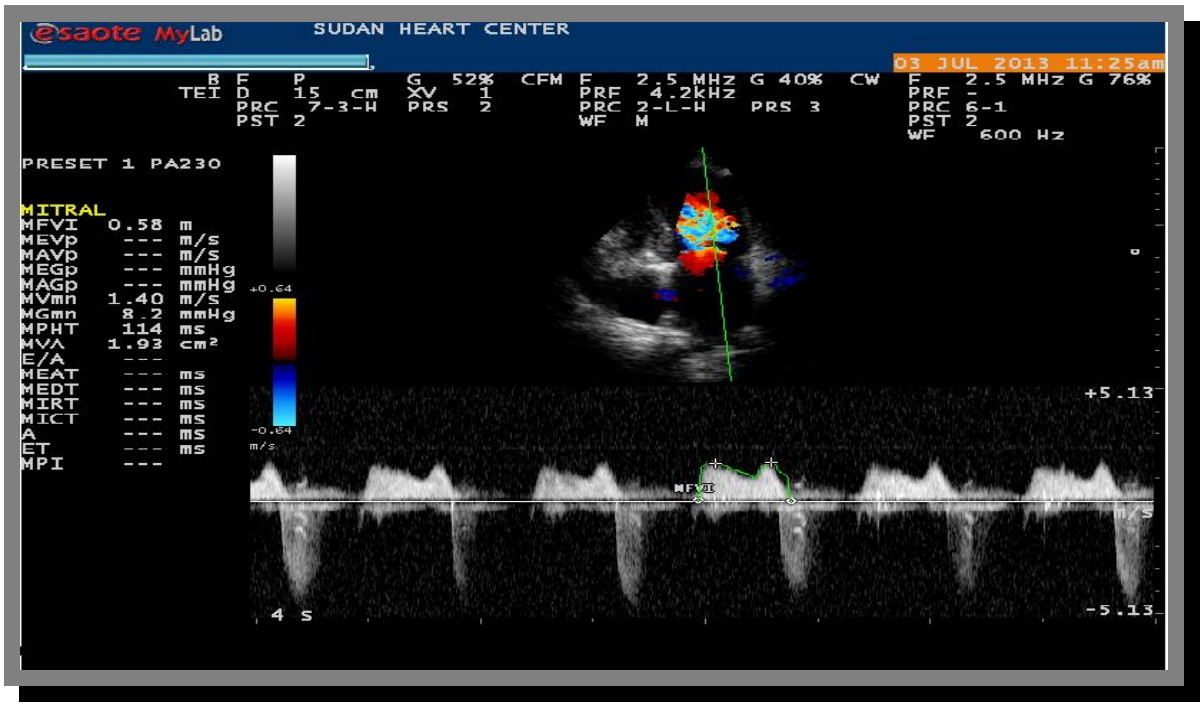

Figure .5.19 *Continuous wave Doppler signal of mitral with a peak velocity of approximately 1.4 m/s.*

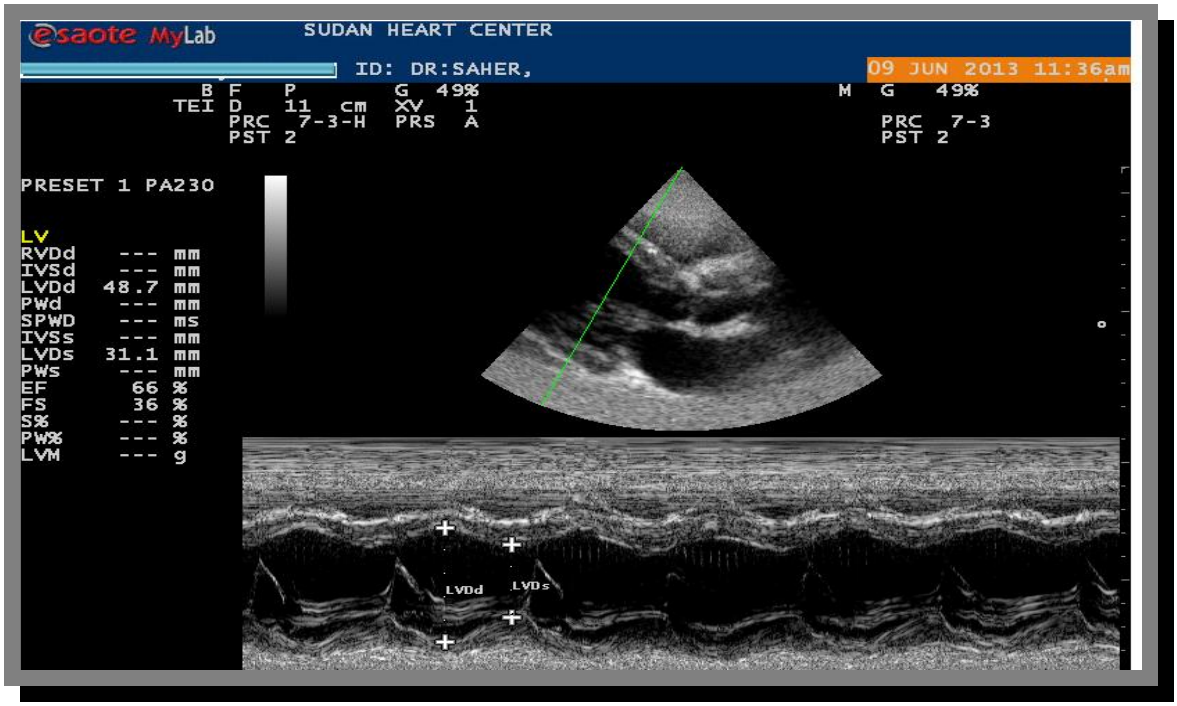

Figure .5.20 *M-mode, shows normal LV parameters.*

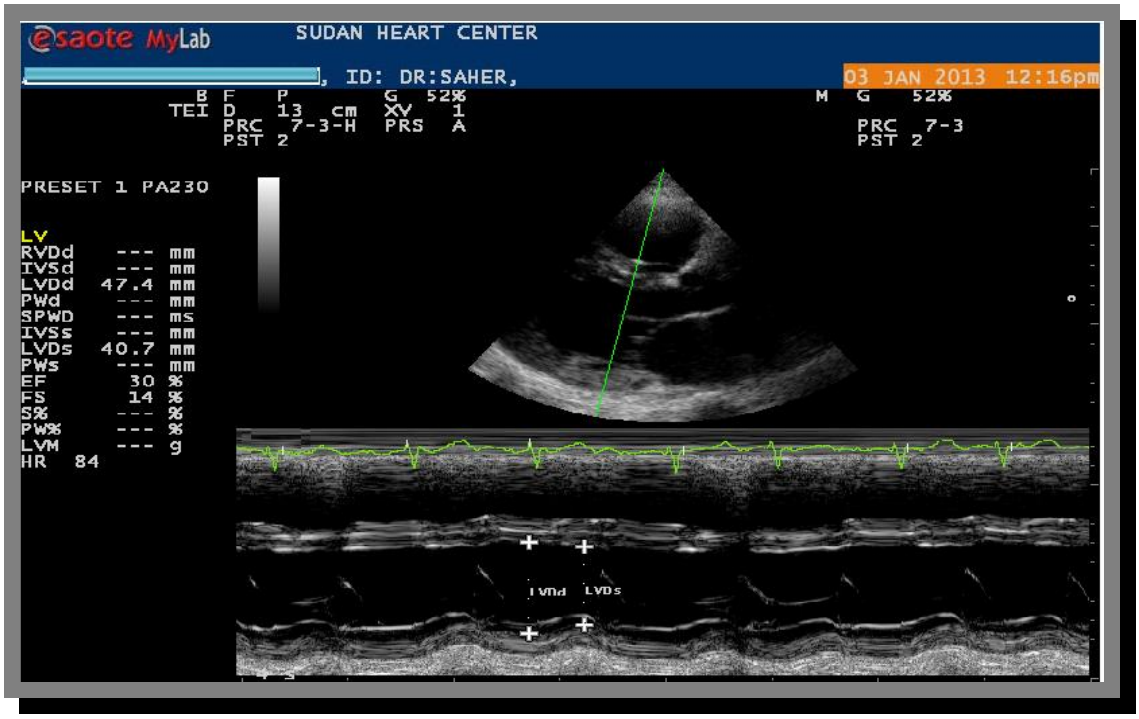

Figure .5.21 *Normal LV diastolic and systolic measurement and mild abnormal EF.*

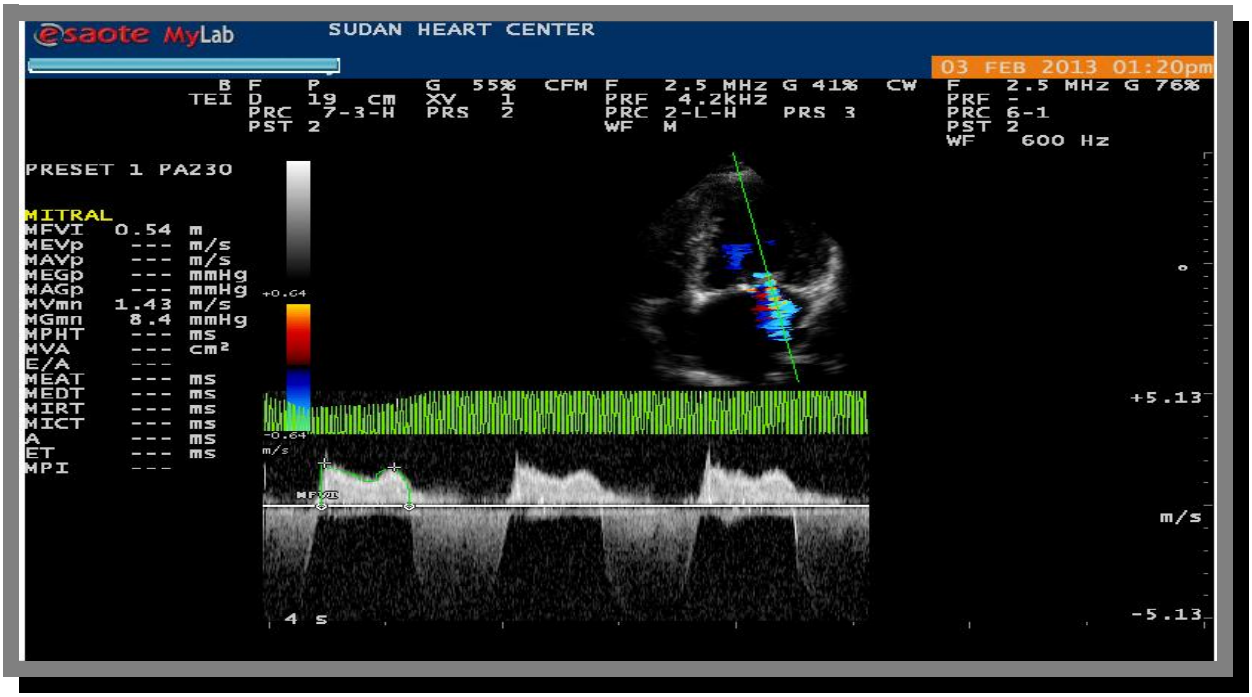

Figure .5.22 *Continuous wave Doppler signal of mitral with a peak velocity of approximately 1.43 m/s.*

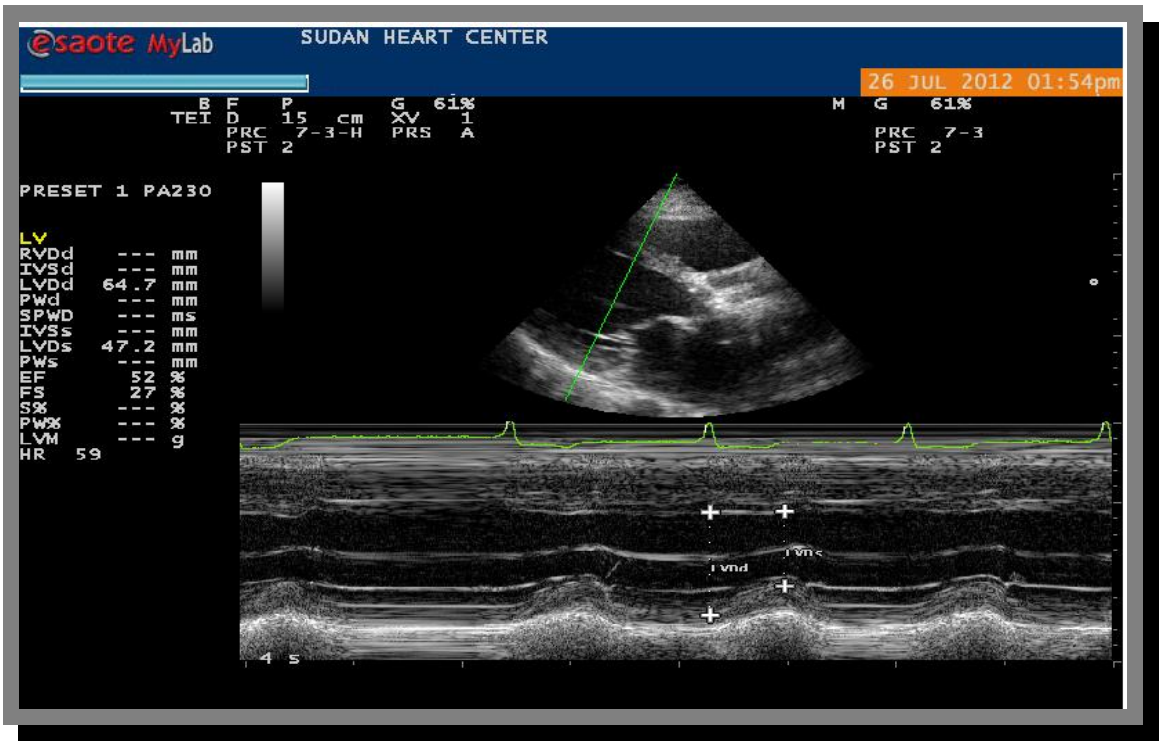

Figure .5.23 *Normal LV diastolic and systolic measurement and mild abnormal EF.*

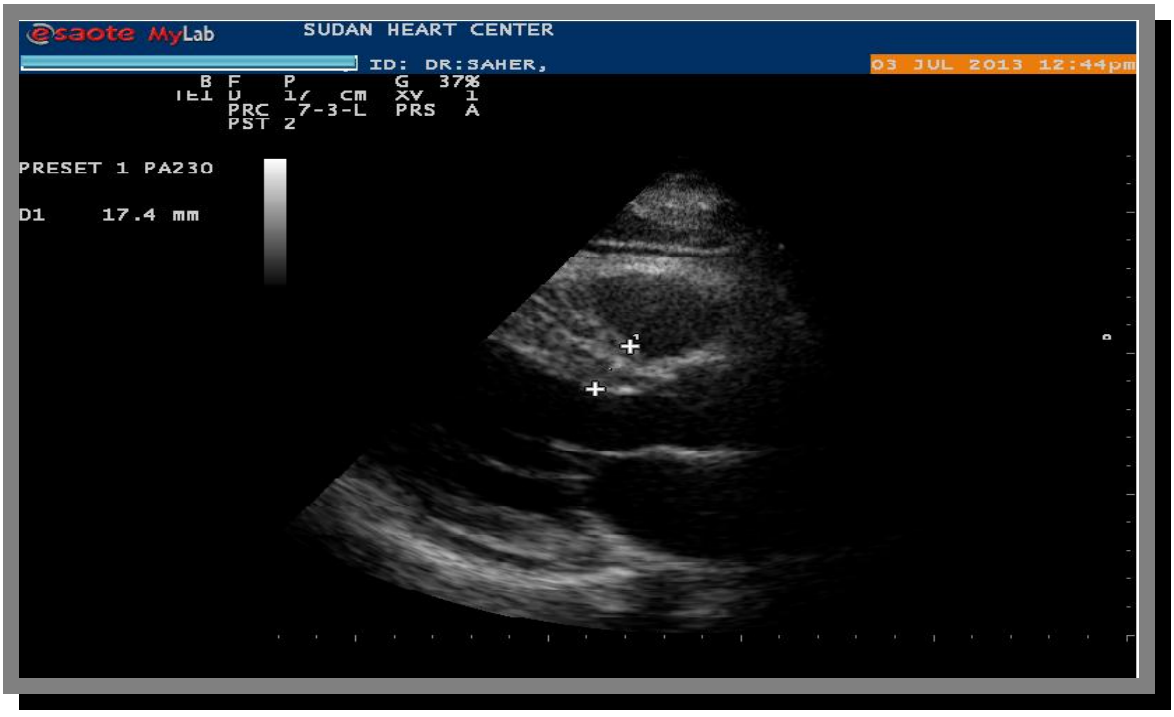

Figure .5.24 *The interventricular septum is seen in this view with 1.74 cm.*

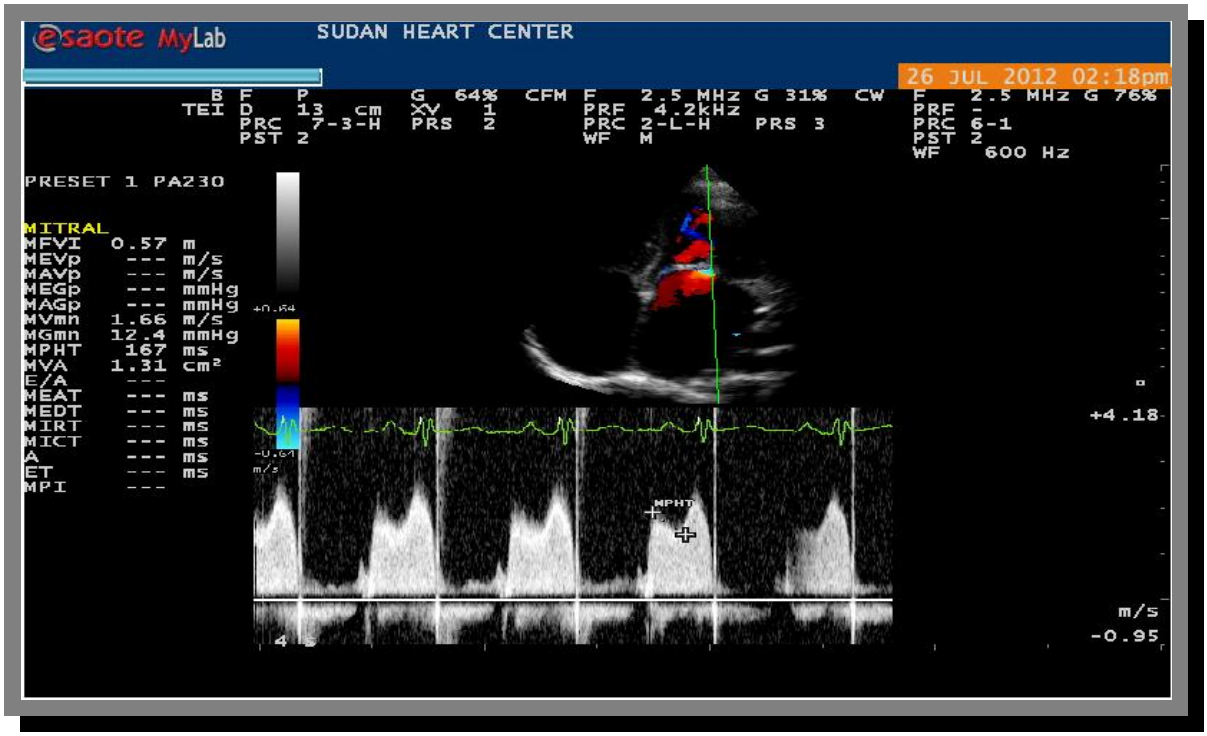

Figure .5.25 *Continuous wave Doppler signal of mitral with a peak velocity of approximately 1.66 m/s.*

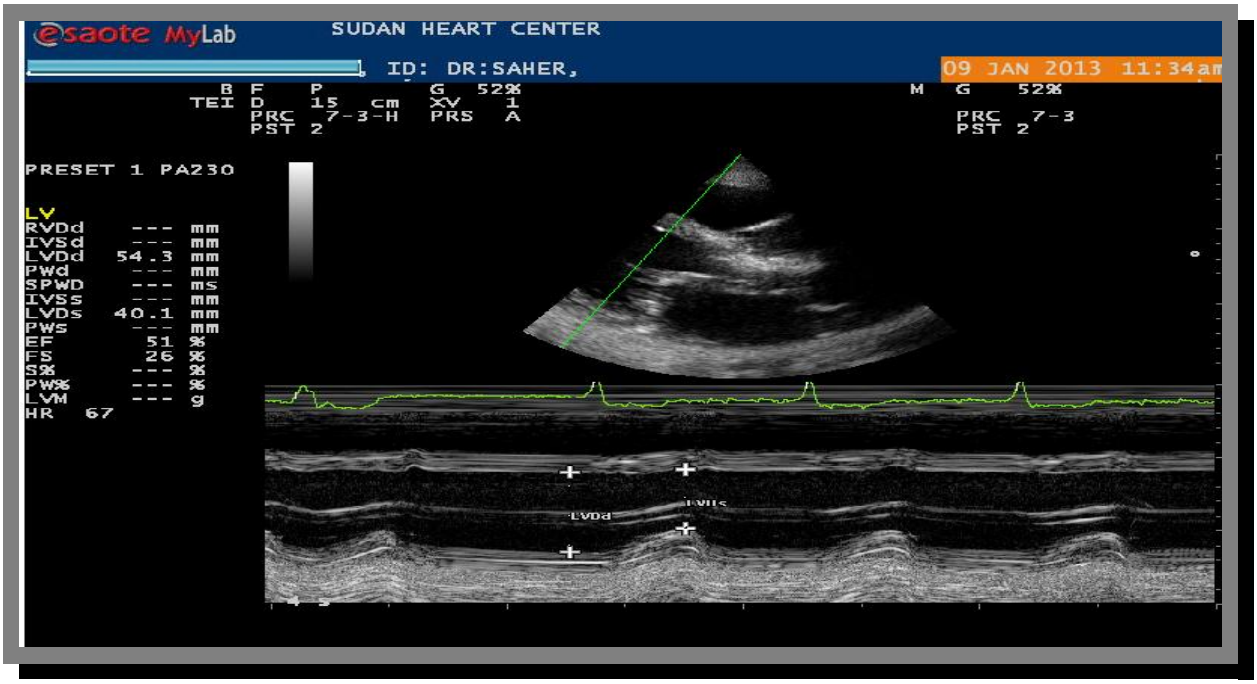

Figure .5.26 *Normal LV diastolic and systolic measurement and mild abnormal EF.*

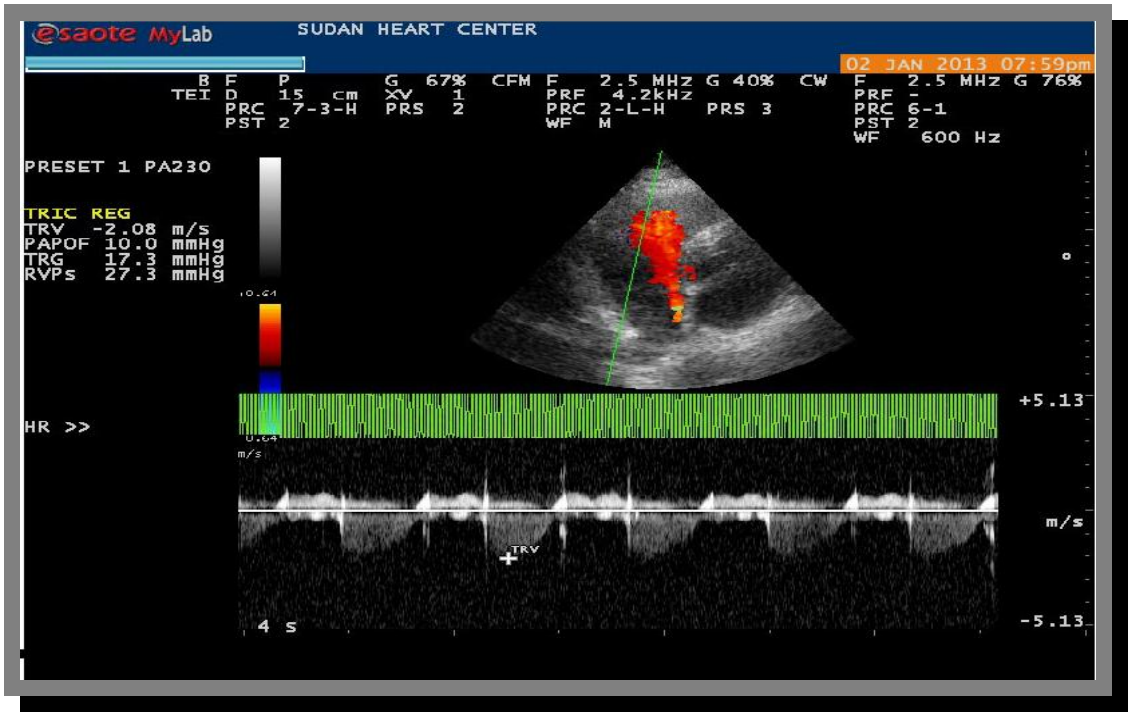

Figure .5.27 *Continuous wave Doppler signal of tricuspid regurgitation with a peak velocity of approximately 2.08 m/s.*

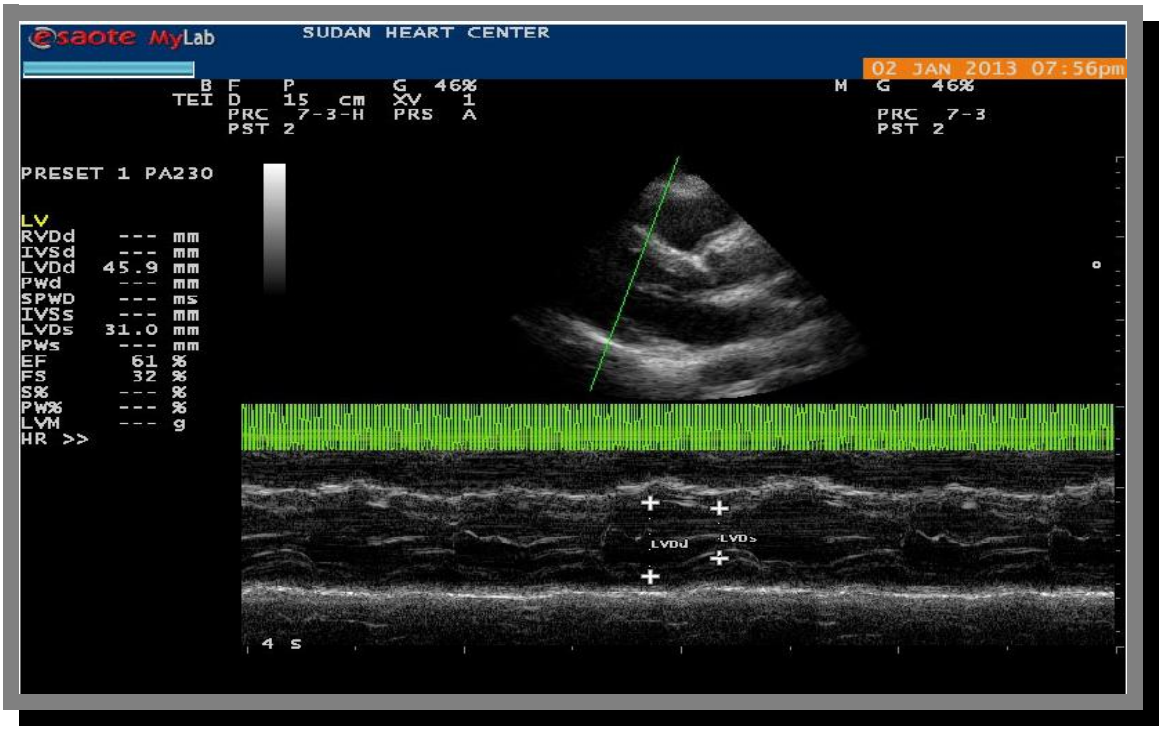

Figure .5.28 *M-mode, shows normal LV parameters.*

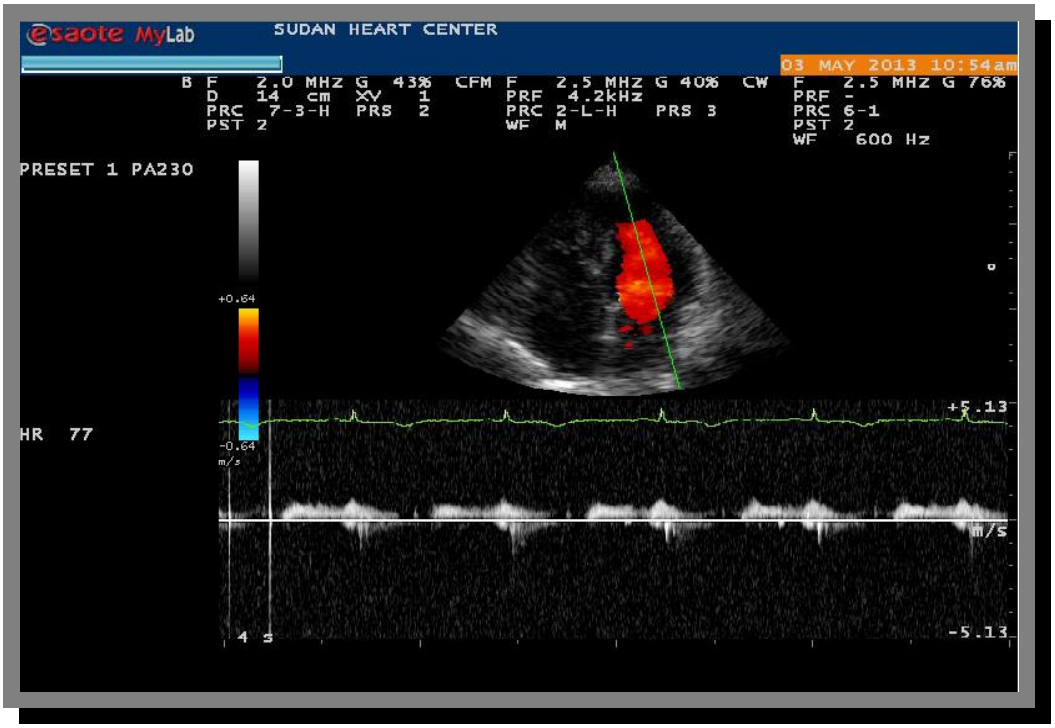

Figure .5.29 *Continuous wave Doppler assesses the blood flow in of mitral valve.*

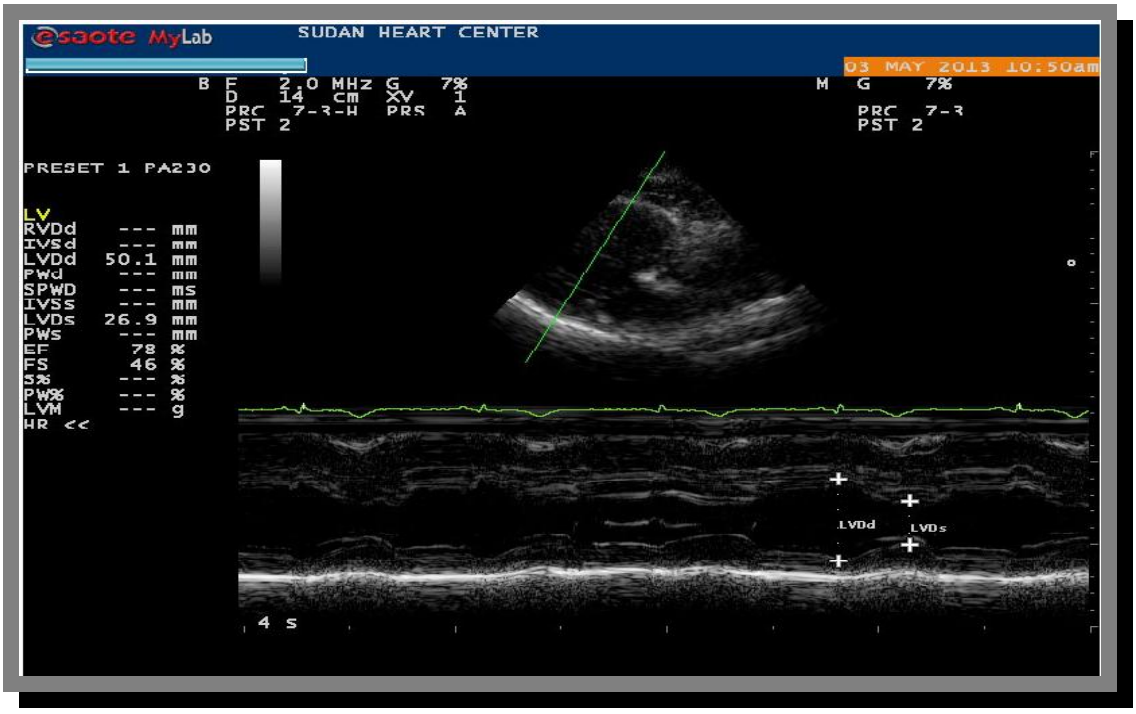

Figure .5.30 *Normal LV diastolic and systolic measurement and mild abnormal EF.*

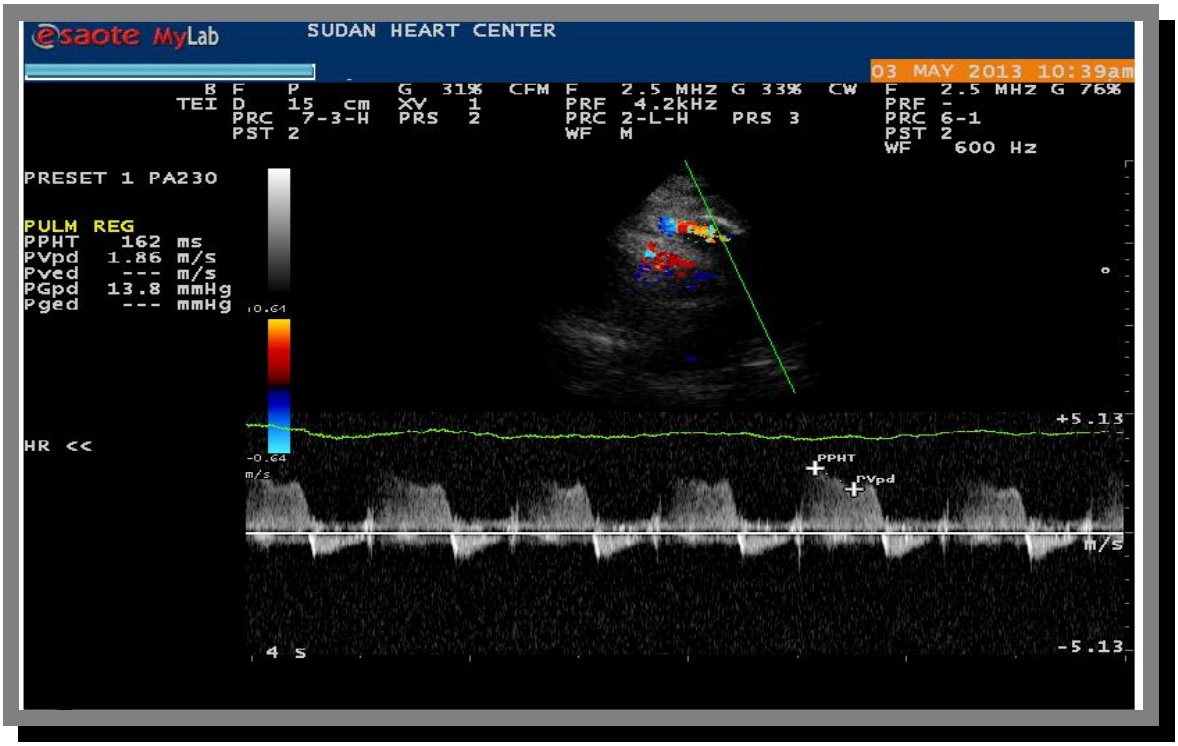

Figure .5.31 *Continuous wave Doppler signal of pulmonary regurgitation with a peak velocity of approximately 1.86 m/s.*

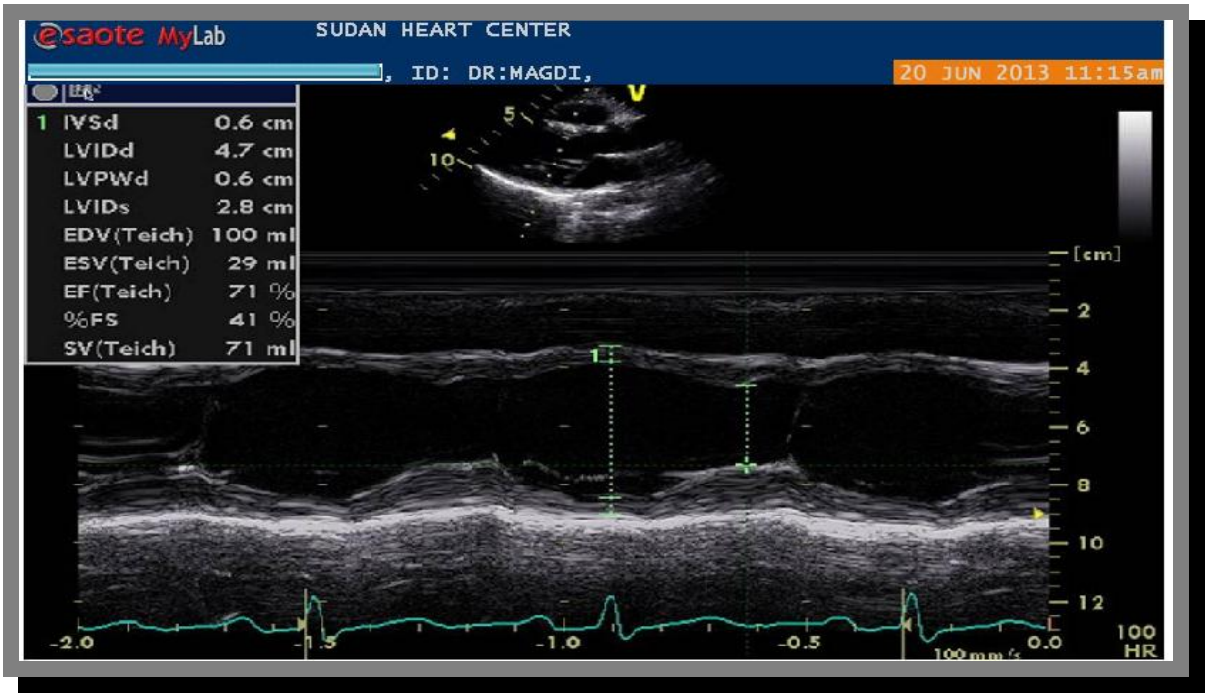

Figure .5.32 *M-mode, shows normal LV parameters*.

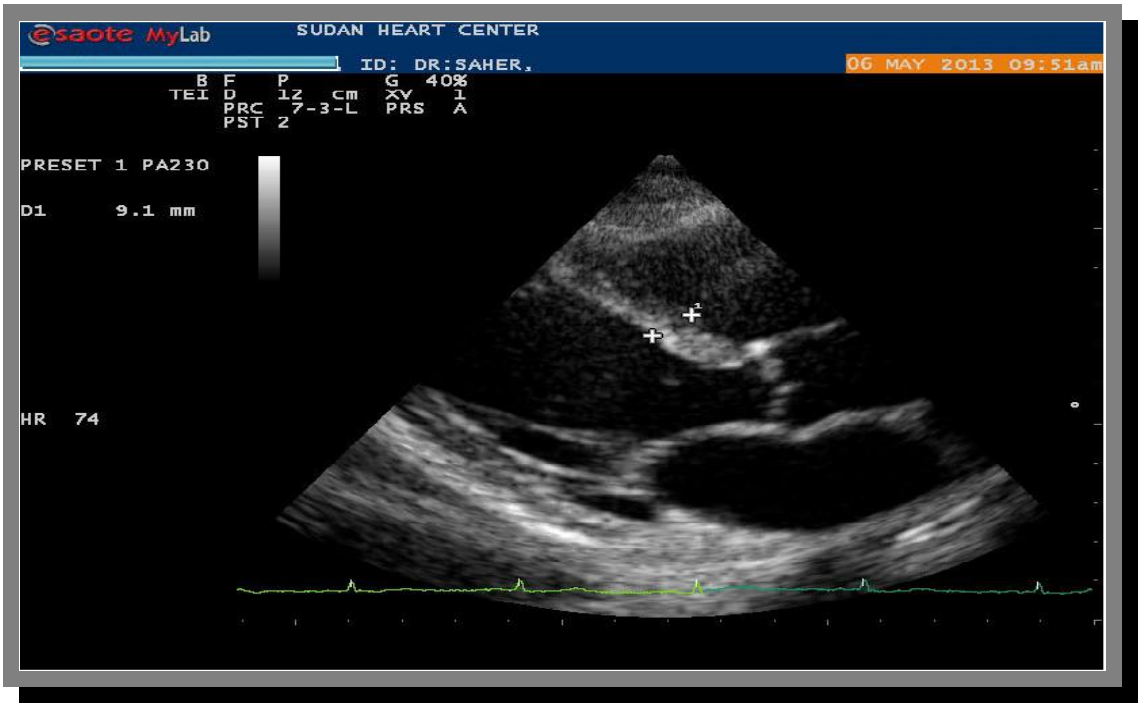

Figure .5.33 *The Interventricular septum with normal range.*

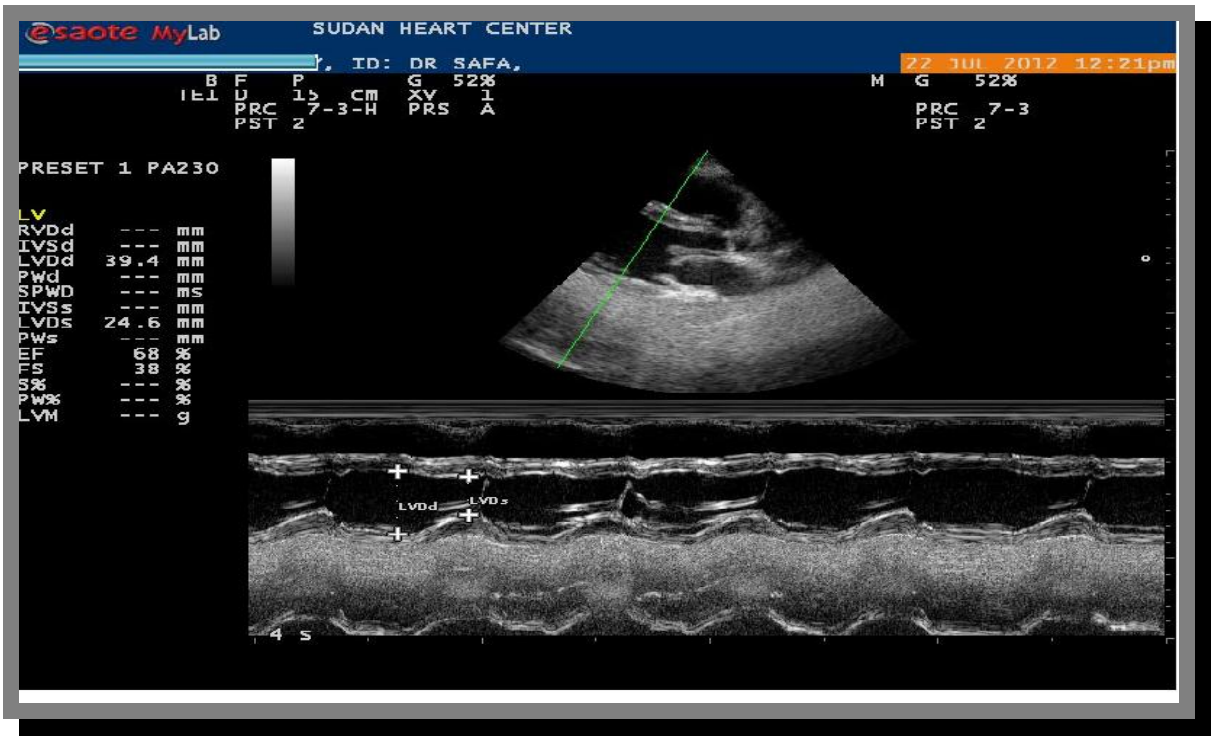

Figure .5.34 *M-mode, shows normal LV parameters*.

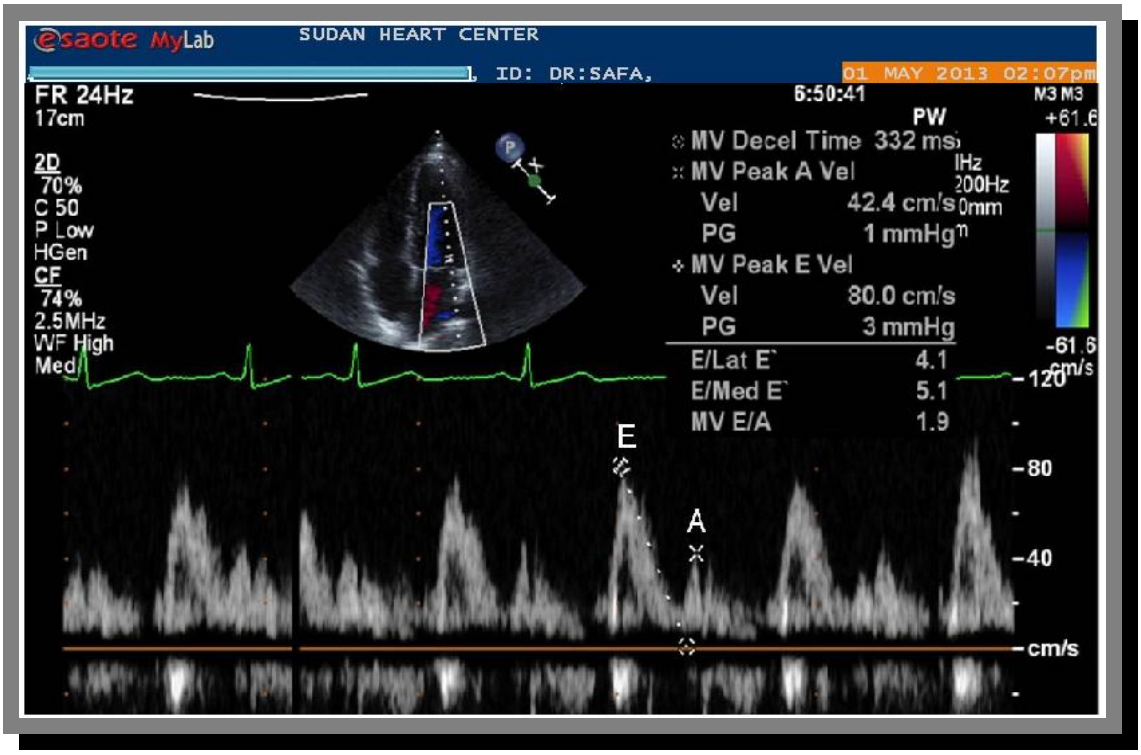

Figure .5.35 *pulse wave Doppler signal of mitral valve.*

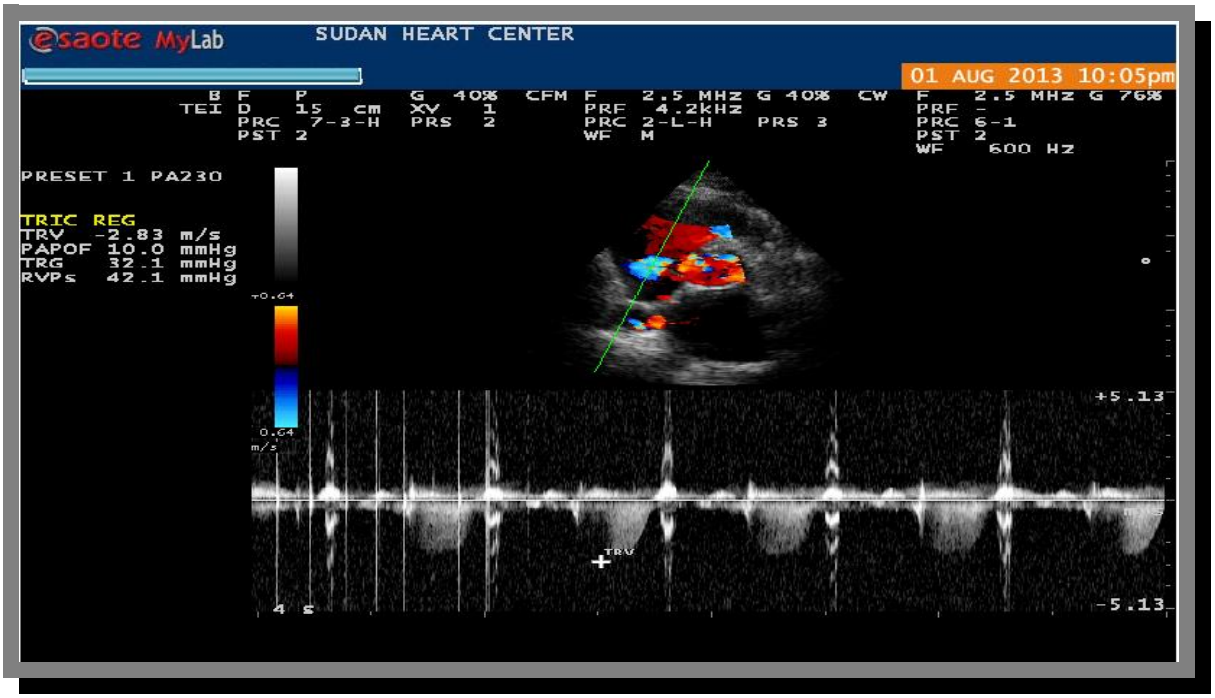

Figure .5.36 *Continuous wave Doppler signal of tricuspid regurgitation with a peak velocity of approximately 2.83 m/s.*

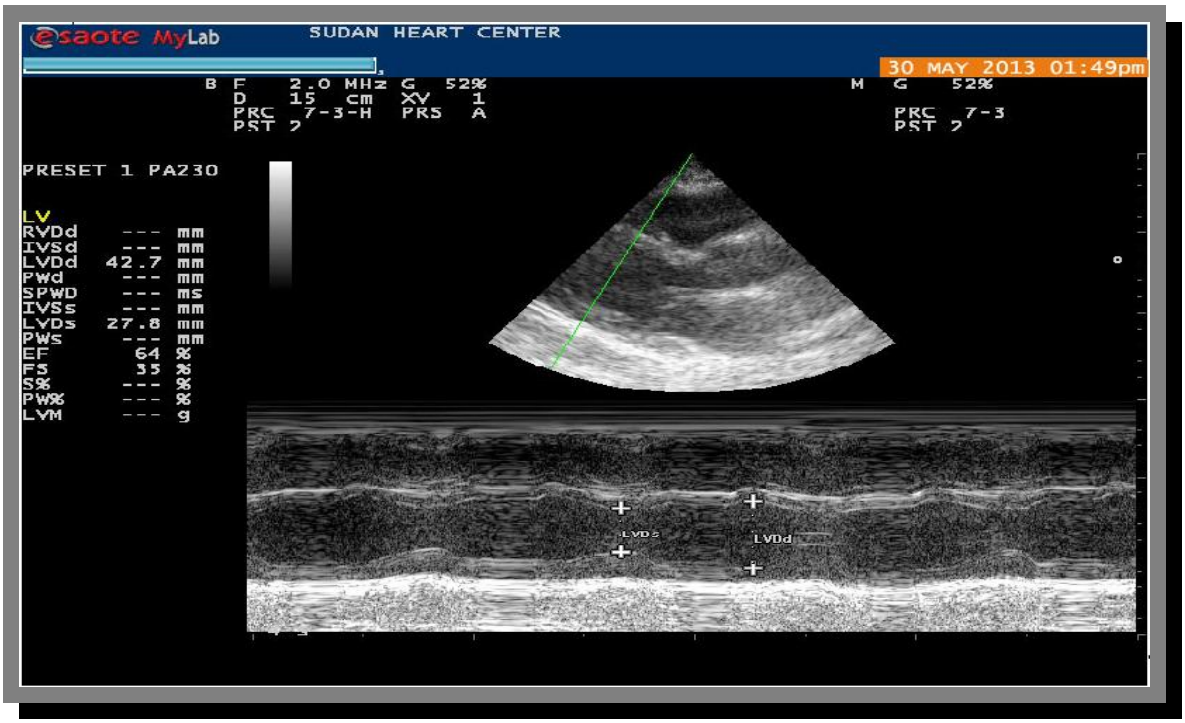

Figure .5.37 *In the M-mode, the LV diastolic measurement is 4.27cm and the systolic measurement is 2.78cm, both of which are normal.*

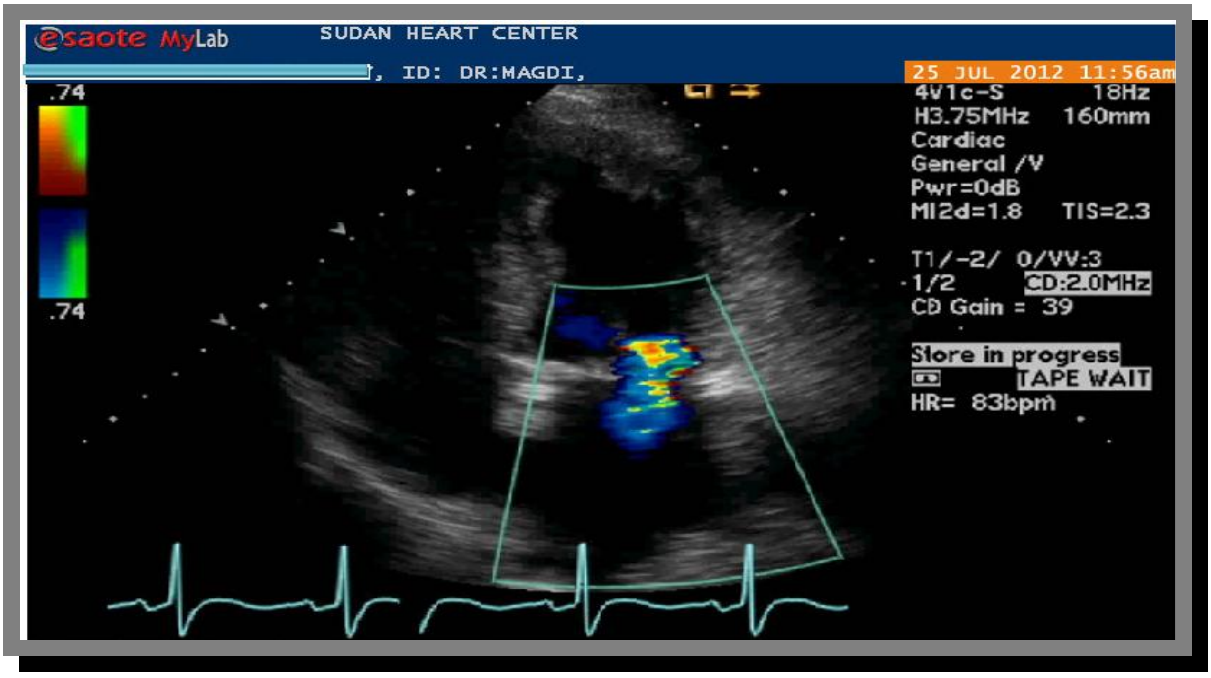

Figure .5.38 *Use of color Doppler shows the presence of tricuspid valve (left) and mitral valve (right) regurgitation.*

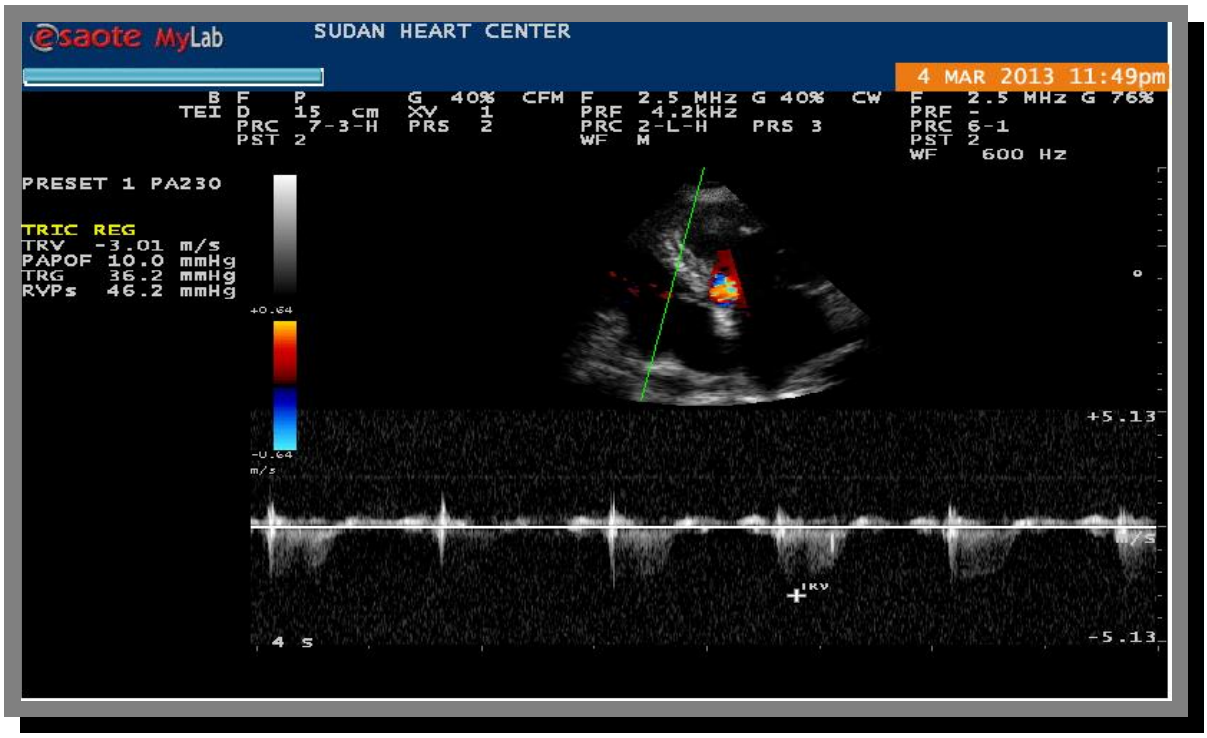

Figure .5.39 *Continuous wave Doppler signal of tricuspid regurgitation with a peak velocity of approximately 3.01 m/s.*

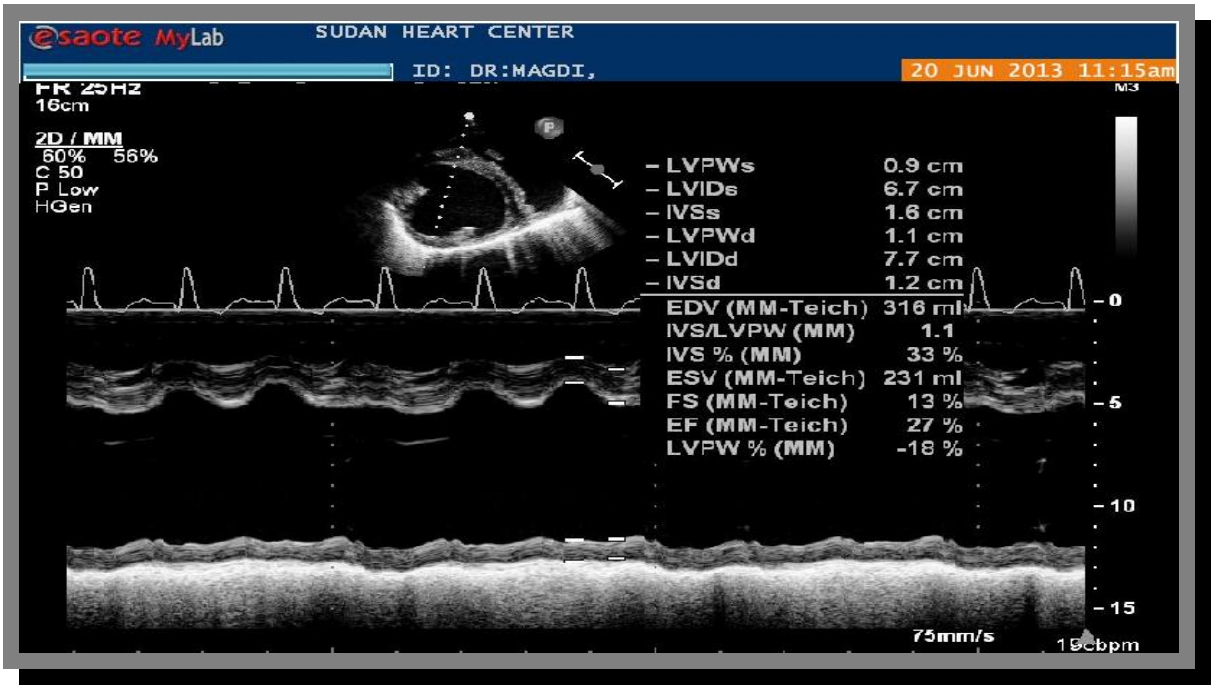

Figure .5.40 *The interventricular septum and posterior wall of the left ventricle (LV) are seen in this view.*

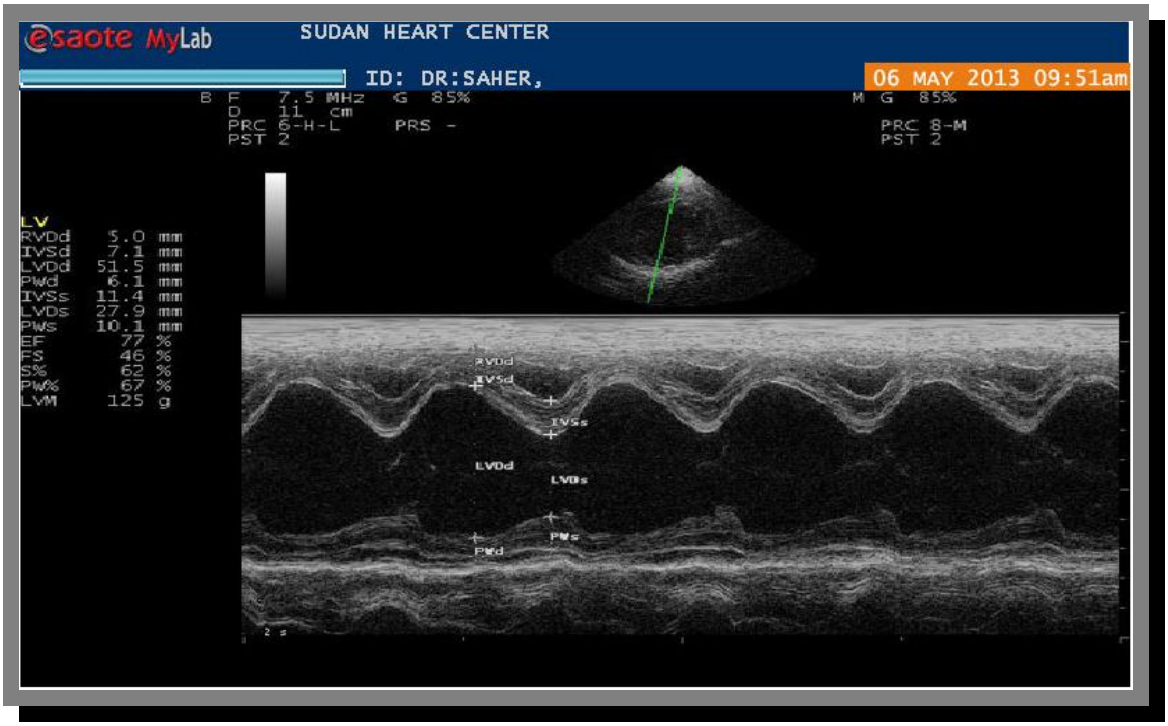

Figure .5.41 *M-mode, shows normal LV parameters.*

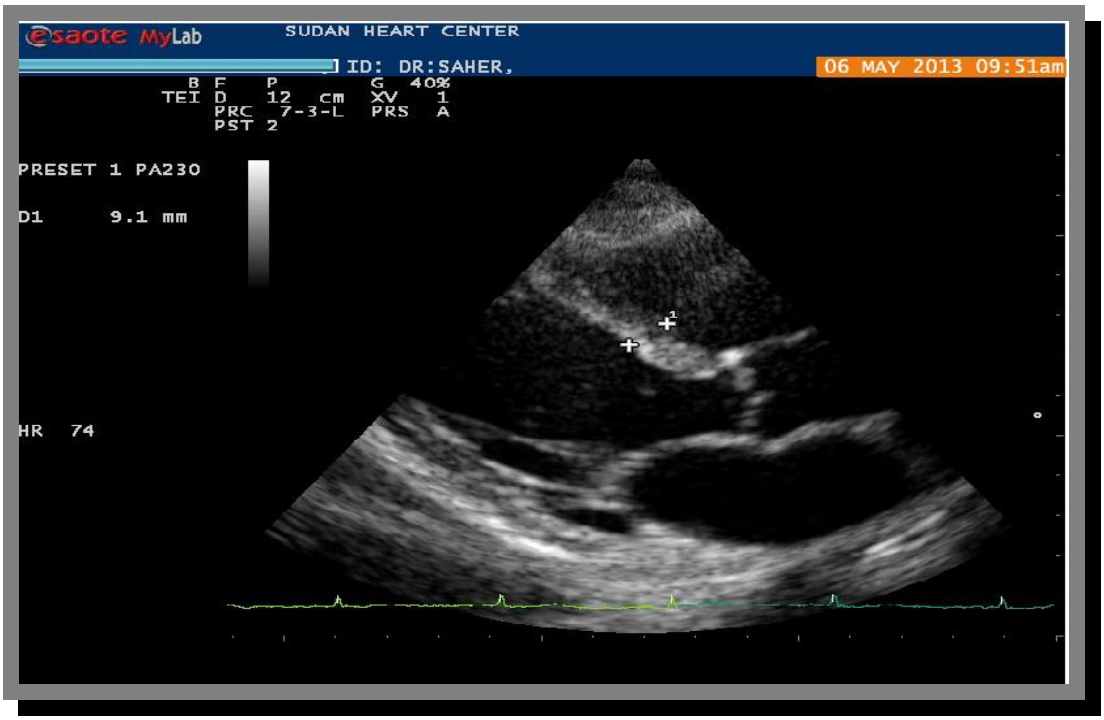

Figure .5.42 *Apical four-chamber view. The posterior interventricular septum is seen in this view.*

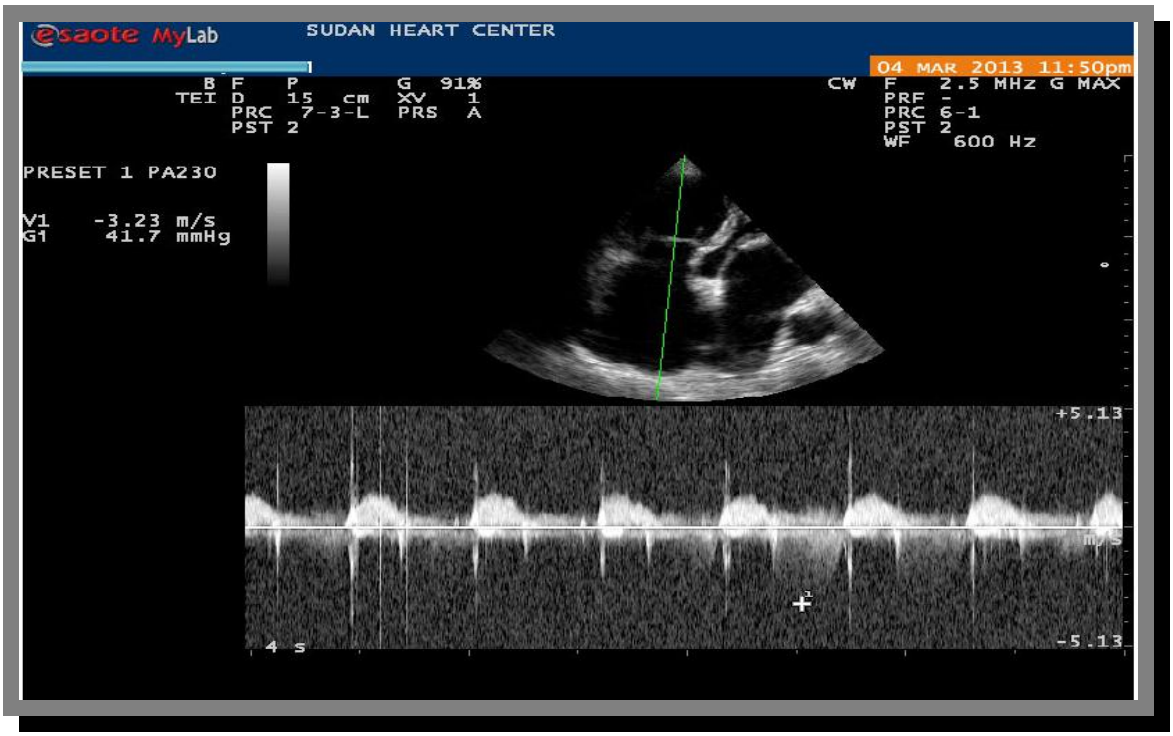

Figure .5.43 *Continuous wave Doppler signal of mitral with a peak velocity of approximately 3.23 m/s.*

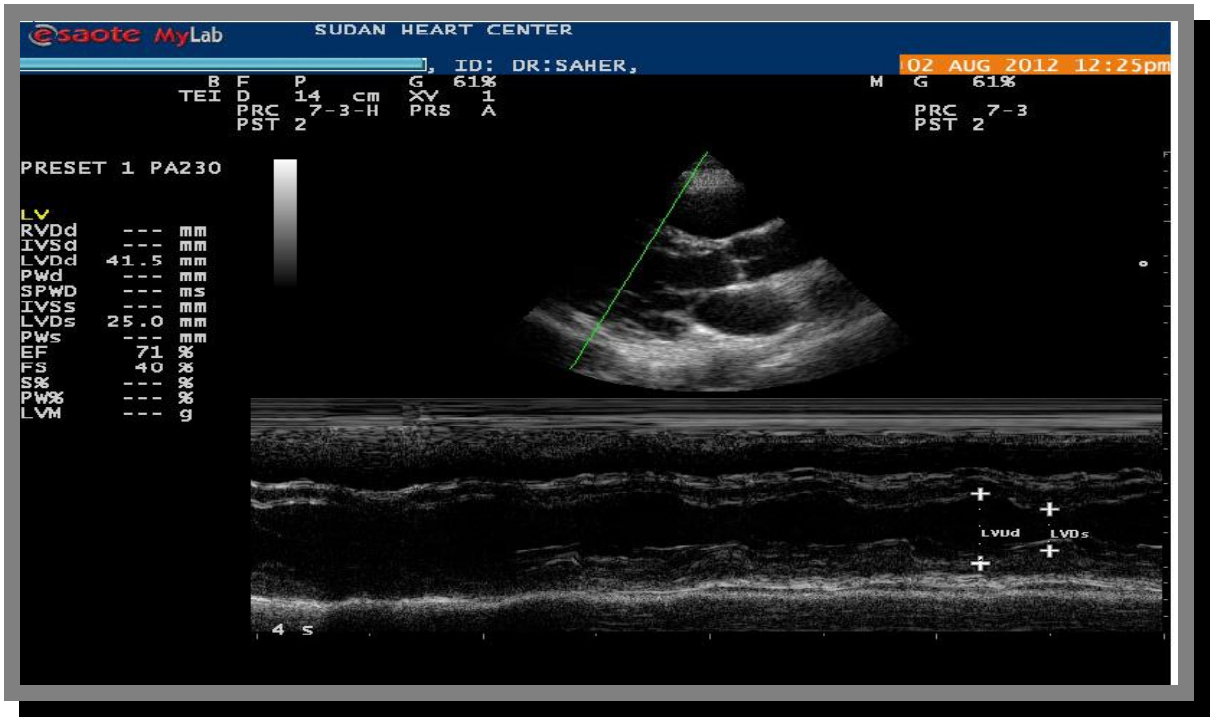

Figure .5.44 *M-mode, shows normal LV parameters.*

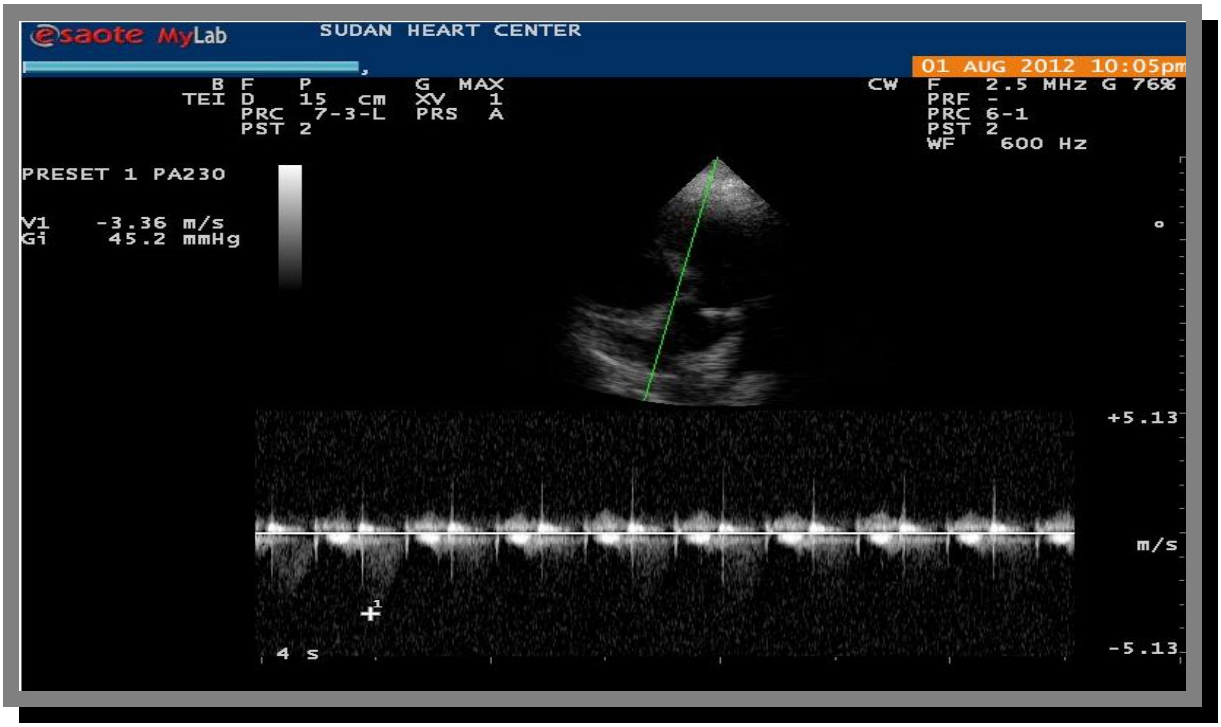

Figure .5.45 *Continuous wave Doppler signal of mitral with a peak velocity of approximately 3.36 m/s.*

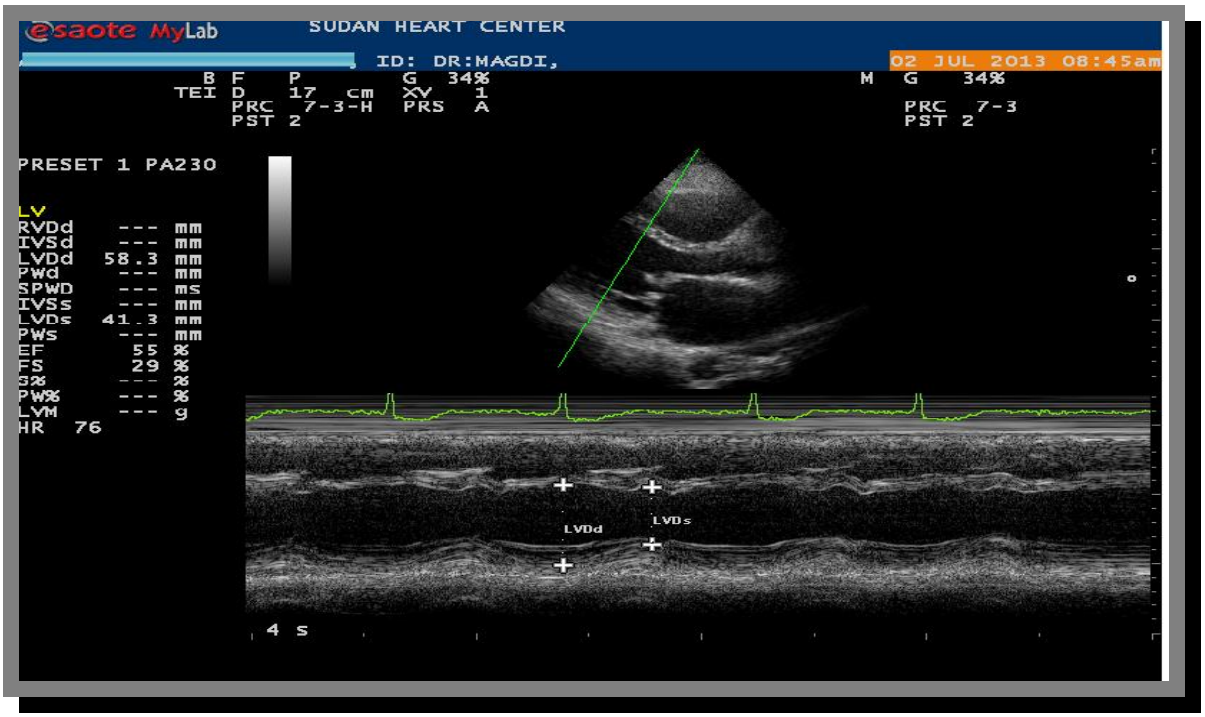

Figure .5.46 *M-mode, shows normal LV parameters.*

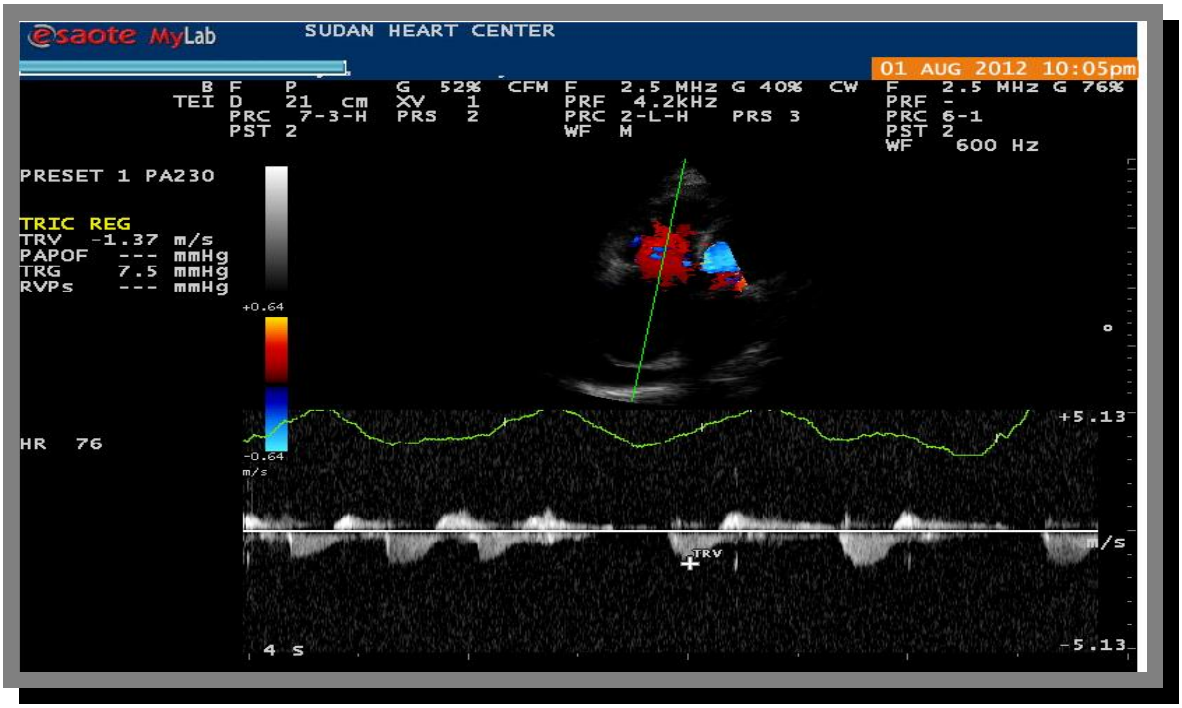

Figure .5.47 *Continuous wave Doppler signal of tricuspid regurgitation with a peak velocity of approximately 1.37 m/s.*

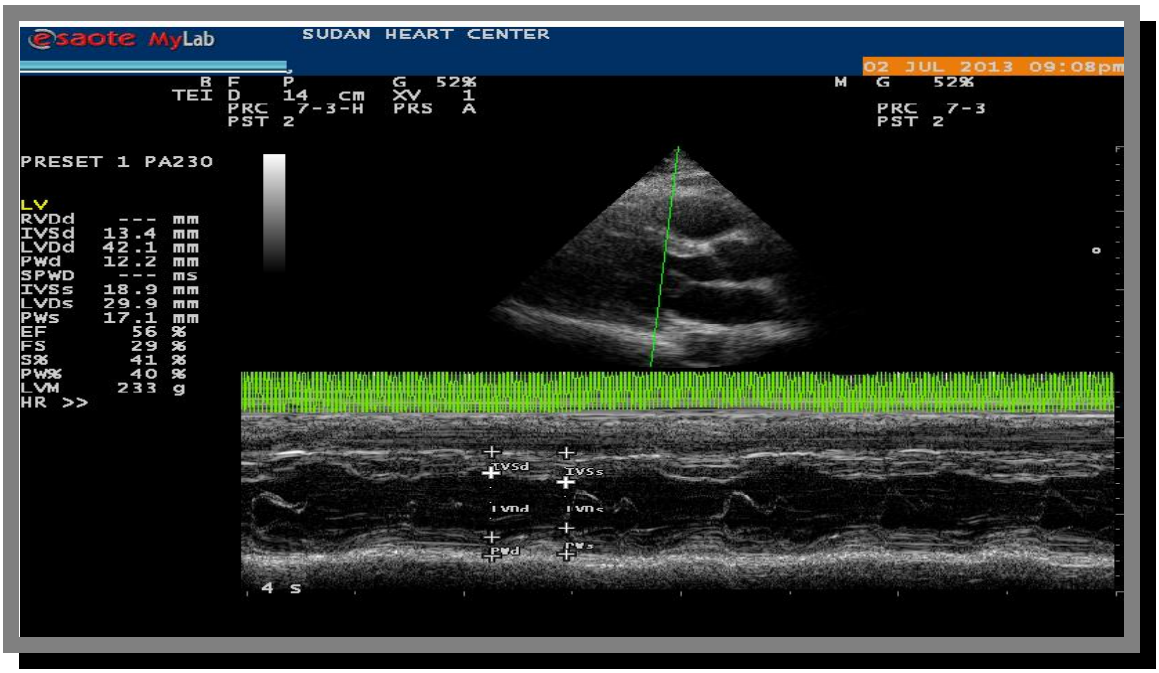

Figure .5.48 *M-mode, shows normal LV parameters.*

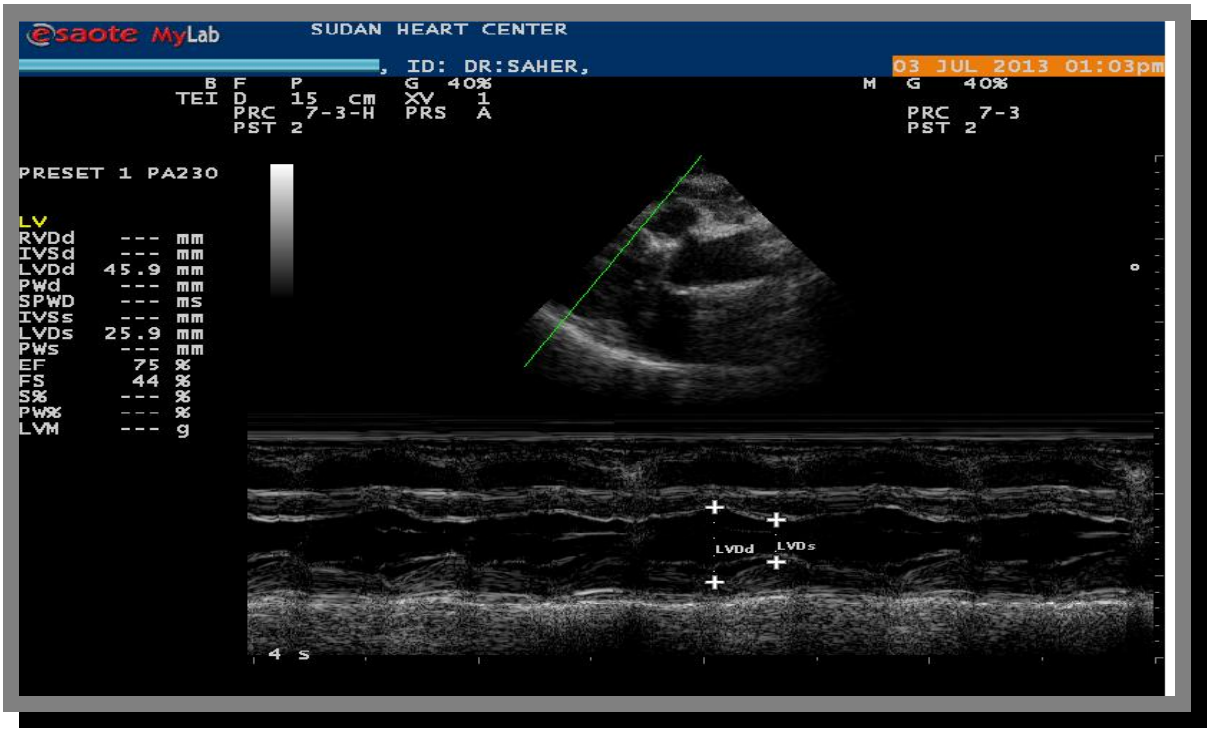

Figure .5.49 *M-mode, shows normal LV parameters.*

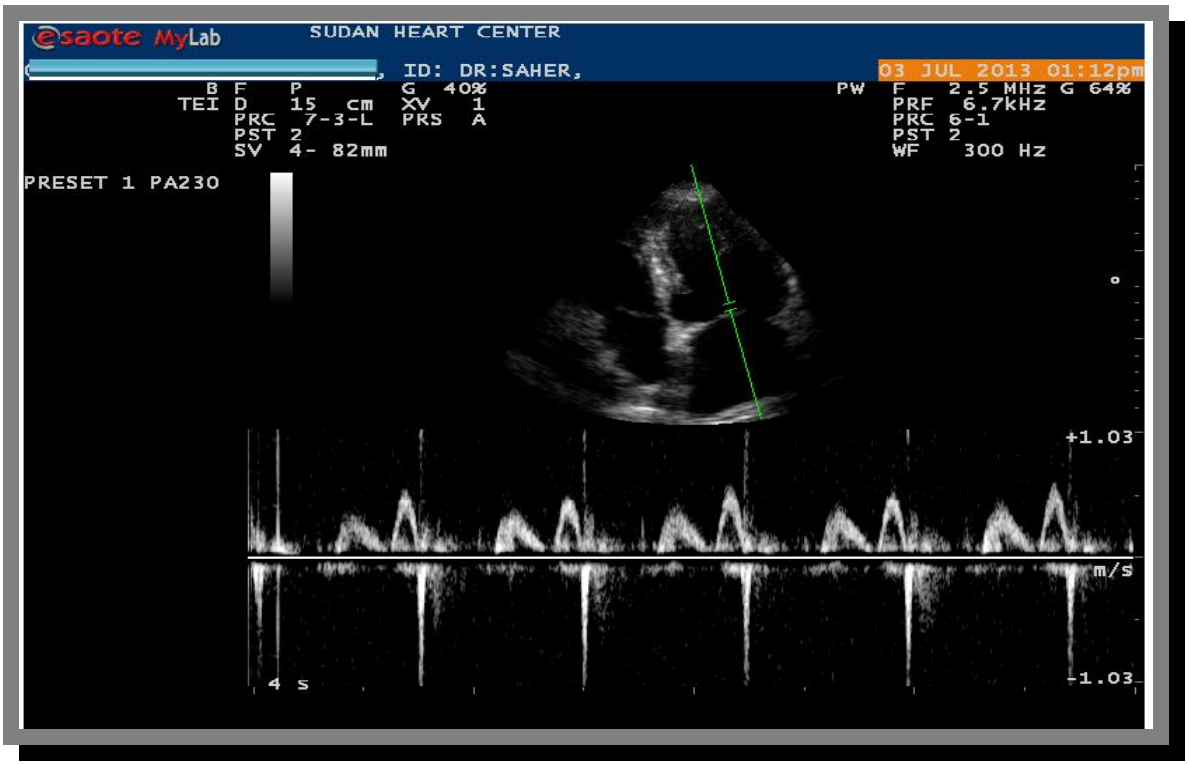

Figure .5.50 *Pulse wave doppler assesses the mitral valve.*