قال تعالي:

((قالوا سبحانك لا علم لنا الا ماعلمتنا انك انت العليم الحكيم)) .

صدق الله العظيم

سورة البقرة الآية **(32)**

Dedication

To Soul of my father Hassan Mohamed

To my beloved mother whom made countless sacrifices throughout my life. She continues to give her unconditional support until today.

To my wonderful Husband Yasir my kids Hassan & Haneen & Ranin for the patience and countless support throughout my study.

To my brothers, sisters and extended family for their support and encouragement.

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First and famous I will like to thank the almighty Allah for the strength that keeps us standing and for the hope that keeps us believing that this achievement would be promising and with GOD everything is possible.

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Special thanks and the greatest debt goes to my husband **Dr. Yasir Shareef Sadig.**

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List of Abbreviations

Abbreviation	Full meaning
ACC	American college of cardiology
AHA	American heart association
AMI	Acute myocardial infarction
AV	Aortic valve
Av	Atrioventricular
BNP	B-type natriuretic peptide
CCF	Congestive cardiac failure
CHF	Congestive heart failure
CK-MB	Creatine kinase myocardium bound
COPD	Chronic obstructive pulmonary disease
CT	Computerized tomography
CVA	Cerebrovascular accident
DCM	Dilated cardiomyopathy
ECG	Electrocardiogram
EKG	Electrocardiogram
EF	Ejection fraction
GE	General electric
HF	Heart failure
HTN	Hypertension
IVC	Inferior vena cava
IV	Interventricular
JVP	Jugular vein pressure
LA	Left atrium
LAD	Left anterior descending
LCA	Left coronary artery
LVEF	Left ventricular ejection fraction
LV	Left ventricle
MRI	Magnetic resonant imaging
MR	Mitral regurgitation
NYAH	New York heart association

PLVEF	Preserved left ventricular ejection fraction
QRS	QRS wave
RA	Right atrium
RCA	Right coronary artery
RV	Right ventricle
SA	Sino-atrial
STE	ST segment elevation
SVC	Superior vena cava
TEE	Trans-esophageal echo
TTE	Trans-thoracic echo
T6	Thoracic vertebra 6

Abstract

The objective of the study was to evaluate the blood flow among heart failure patients by using echocardiography.180 patients (62 female and 118 male) with heart failure sign were enrolled. Their ages ranged between (20 – 85) years. The study was conducted at Khartoum state in Echocardiography departments of Omdurman Military Hospital and Sudan Heart Center during the period from November 2014 to September 2017. Two-dimensional, M-mode and Doppler echocardiograms were recorded using ultrasound machine HDI 4000 scanner (Philips Medical Systems) equipped with a commercially available 3-13 MHz linear transducer with color and power Doppler capability.

The incidence of the heart failure was high among the age group mean 60.56 years that means the risk of heart failure increase with age. In view of the importance of Ejection fraction by using 2D echo, the results showed that mean EF 38.94%(mnimum15% –maximum 60%).The left ventricular ejection fraction volume represents that the maximum was 60% and this was normal value unless the patient had right side failure or diastolic dysfunction which is called heart failure with preserved left ventricular ejection fraction (PLVEF) or “diastolic” heart failure. The hypertension is predominant cause of heart failure in Sudan (36. 1%).The common finding of the study was left ventricular systolic dysfunction LVSD which represent about 121 patients 67.7%. This study found that the correlation is significant at the 0.01 level. between the left ventricular internal diastolic dimeter LVIDD, left ventricular internal systolic dimeter LVISD and ejection fraction EF equal -.376 (negative) inversely proportional.

MR was severe in 23 (12.8%), moderate in 29 (16.1%), mild in 69 (38.3%), trivial in 25 (13.9%), and absent in 28 (15.6%). The remaining was mitral stenosis. In

addition, the severity of MR correlated with the severity of systolic dysfunction. Ejection fraction (EF) reduced with the severity of the MR. small color flow jets are seen in roughly 38% of study sample and therefore large color flow jets are seen in roughly 12.8% of study sample. The incidence of mild regurgitation tends to increase with age.

مستخلص البحث

الهدف من هذه الدراسة هو تقويم انسياب الدم بالنسبة لمرضى فشل القلب باستخدام تصوير القلب بالموجات فوق الصوتية . اشتملت الدراسة على عدد 180 مريضاً بفشل القلب (منهم 62 من الاناث و118 من الذكور . تراوحت أعمار المرضى بين 20 – 85 عاماً . اجريت الدراسة في ولاية الخرطوم بقسمي تصوير القلب بالموجات فوق الصوتية في مستشفي امدرمان العسكرى ومركز السودان للقلب ، خلال الفترة بين نوفمبر 2014 وسبتمبر 2017 . تم إجراء فحصيين ثنائى الأبعاد بالموجات فوق الصوتية أحدهما بطريقة أم (M–mode) والآخر بطريقة دوبلر . باستخدام جهاز تصوير (HDI 4000) ، من شركة فيلبس ، مزود ببرجام خطى (3–13 MHz) ، ذى إمكانيات تلوين .

كان حدوث فشل القلب عالياً بين متوسط العمر 10,56 عاماً ، وهذا يعنى أن خطر الإصابة بفشل القلب يزداد بإزدياد العمر . ومن خلال أهمية النسبة المفرغه (المطروحة) من الدم ، أوضحت الدراسة أن متوسط النسبة (المطروحة) 38,94 (الحد الأدنى 15 والحد الأعلى 60) . أوضح حجم النسبة المطروحة من البطين الأيسر أن الحد الأدنى كان 60% ، وهى نسبة طبيعية إلا إذا كان المريض يشكو من فشل الجزء الأيمن من القلب أو خللاً في الضغط الأنبساطى والذى يطلق عليه فشل القلب الناتج عن خلل في نسبة الدم المطروح من البطين الأيسر أو فشل القلب (الانبساطى) . يعتبر إرتفاع ضغط الدم السبب الأساسى في حدوث فشل القلب في السودان 36,1%) . كان السبب الأساسى حسب هذه الدراسة هو خلل وظيفة الضغط الانقباضى في البطين الأيسر ، والذي ظهر في 121 مريضاً من عينة الدراسة 67,7%) . أوضحت الدراسة أن الإرتباط كان ذا أهمية عند المستوى 5.51 ، بين القطر الإنبساطى الداخلي للبطين الأيسر والقطر الإنقباضى الداخلي للبطين الأيسر ، وأن نسبة الدم المطروح تساوى 376– (قيمة سالبة) – نسبة معكوسة كانت نسبة الـ MR إرتجاع الدم في الصمام ثنائى الشرفات حاده عند 23 مريضاً (12,8%) ، ومتوسط عند 29 مريضاً (16,1%) وبسيطة عند 69 مريضاً (38,3%) ، وضعيفة جداً عند 25 مريضاً 13,9%) ، ولا توجد عند 28 مريضاً 15,6% . وكان بقية المرضى يعانون من ضيق الإكليكى , أوضحت الدراسة أيضاً أن هناك علاقة إرتباط بين شدة الـ MR إرتجاع الدم في الصمام ثنائى الشرفات وشدة الخلل الوظيفى الإنقباضى ، وتقل نسبة الدم المطروح مع شدة الـ MR إرتجاع الدم في الصمام ثنائى الشرفات ،

وظهرت تدفقات صغيرة ملونة في عدد 38% تقريباً من عينة الدراسة ، بينما شكلت التدفقات الكبيرة 12,8% من عينة الدراسة . أوضحت الدراسة أيضاً أن حدوث حالات القلب البسيط يزداد مع زيادة العمر .

Chapter One
Introduction

1.1 Introduction

Heart failure is a syndrome with symptoms and signs caused by cardiac dysfunction. (Hogg et al., 2004). In the 1995 World Health Organization definition of heart failure states that "cardiac failure is an inability of the heart to deliver blood (and therefore oxygen) at a rate commensurate with the requirements of the metabolizing tissues at rest or during light exercise. This leads to characteristic systemic pathophysiological responses (neural, hormonal, renal and others), symptoms and signs. (Strobeck J.E et al., 1985) Although many conditions can cause heart failure (coronary artery disease, hypertension, cardiomyopathies, valvar and congenital heart disease, arrhythmias, pericardial disease, myocarditis, pulmonary hypertension, and cardio toxic substances—including alcohol), the predominant cause of heart failure in the western world is ischemic heart disease. Fox K F, et al., 2001**.** Echocardiography uses high-frequency ultrasound to evaluate the heart and great vessels. The examination provides a dynamic rendition of cardiac great vessel anatomy and, when combined with the Doppler technique, yields information regarding cardiac and great vessel blood flow (hemodynamics) as well. Because of the high frame rates inherent in ultrasonography, echocardiography can image the heart in a dynamic real-time fashion, so that the motion of cardiac structures can be reliably evaluated. Echocardiography is useful in assessing ventricular function, valvar heart disease, myocardial disease, pericardial disease, intracardiac masses, and aortic abnormalities. With Doppler technology, cardiac chamber function, valvar function, and intracardiac shunts frequently seen in congenital heart disease can be assessed. Combined Doppler echocardiography is a commonly performed procedure

because it is relatively inexpensive and widely available, provides a wealth of information, is noninvasive, has no risk of ionizing radiation, and can also be performed at the bedside in critically ill patients. Furthermore, the results are immediately available because no special post examination image processing is required. However, this technique is technically challenging and requires a great deal of operator expertise. Also, a small percentage of patients have poor acoustic windows that can severely degrade image quality. This disadvantage can be obviated by placing the sonographic probe in the esophagus, a procedure called trans esophageal echocardiography (TEE). Trans esophageal echocardiography yields consistently excellent images of the heart and great vessels, but involves a small amount of discomfort and risk to the patient. More recently, echocardiography has been combined with stress-testing modalities to assess inducible myocardial ischemia using wall motion analysis of left ventricular function (Soongswang J.,2000). Valvular regurgitation has long been recognized as an important cause of morbidity and mortality. Although the physical examination can alert the clinician to the presence of significant regurgitation, diagnostic methods are often needed to assess the severity of valvular regurgitation and remodeling of the cardiac chambers in response to the volume overload state (Zoghbi et al., 2003). Echocardiography with Doppler has recently emerged as the method of choice for the non-invasive detection and evaluation of the severity and etiology of valvular regurgitation (Bonow et al., 1998). The examination provides a dynamic interpretation of cardiac great vessel anatomy and, when combined with the Doppler technique, yields information regarding cardiac and great vessel blood flow (hemodynamics) as well. Because of the high frame rates inherent in ultrasonography, echocardiography can image the heart in a dynamic real-time fashion, so that the motion of cardiac structures can be reliably evaluated. Echocardiography is useful in assessing ventricular function, valvar

heart disease, myocardial disease, pericardial disease, intracardiac masses, and aortic abnormalities. With Doppler technology, cardiac chamber function, valvar function, and intracardiac shunts frequently seen in congenital heart disease can be assessed. However, this technique is technically challenging and requires a great deal of operator expertise. Also, a small percentage of patients have poor acoustic windows that can severely degrade image quality. This disadvantage can be avoided by placing the sonographic probe in the esophagus, a procedure called Trans-esophageal echocardiography (TEE).

Trans-esophageal echocardiography yields consistently excellent images of the heart and great vessels, but involves a small amount of discomfort and risk to the patient. More recently, echocardiography has been combined with stress-testing modalities to assess inducible myocardial ischemia using wall motion analysis of left ventricular function (Soongswang et al., 2000). The present study aimed to evaluate the anatomy of mitral valve and the blood flow in the mitral valve as well as the mechanism of mitral regurgitation by using high frequency ultrasound.

1.2 Problem of study:

Heart failure is a very serious illness that can cause life-threatening complications and significantly limit life span. It is the leading cause of hospitalization in people older than 65. The difficulty in clinical evaluation in the detection of significant heart valve diseases main problems. In addition to the complication during cardiac catheterization for the assessment of heart valves disease severity and lack of knowledge for experimental information related to Doppler echocardiography findings in patients with HF.

1.3 Objectives:

1.3.1 General Objective:

The main objective of this study is to determine the changes in cardiac blood flow in the patients with Heart failure by medical Doppler echocardiography.

1.3.2 Specific Objectives:

- To determine the changes in cardiac valves blood flow abnormalities, in high risk patients to predict early and late onset cardiac failure by medical Doppler echocardiography.
- To measure Ejection fraction(EF) to estimate of heart function.
- To determine the left systolic and diastolic dimeter of the heart.
- To determine the causes of the heart failure.
- To determine the relation between age, gender to HF.

1.4 Outlines of Thesis:

- Chapter one was deal with introduction, problems statement and the objectives. Chapter two was highlight the literature review, Anatomy, physiology and pathology of cardiovascular circulation in addition to Normal & abnormal sonographic features of it and physical characteristic of echocardiography machine and techniques. Chapter three was showed the materials and methods. Chapter four was concern with reviewing of results. Chapter five will show the following sections: Section 1: Discussion. Section 2: Conclusion. Section 3: Recommendations. References Appendices.

Chapter Two
Theoretical Back Ground and
Literature review

2.1 Anatomy of the heart:

The heart is a cone-shaped organ about the size of a loose fist. It is located within the mediastinum and extends from the level of the second rib to about the level of the sixth rib. Although many people think that the heart is found in the left side of the chest, the heart is located only slightly left of the midline of the body. The heart is bordered laterally by the lungs, posteriorly by the vertebral column, and anteriorly by the sternum. Inferiorly, the heart rests on the diaphragm (Kathryn-2008).

The heart, slightly larger than a clenched fist, is a double, self-adjusting, suction and pressure pump, the parts of which work in unison to propel blood to all parts of the body. Right side of the heart (right heart) receives poorly oxygenated (venous) blood from the body through the SVC and IVC and pumps it through the pulmonary trunk to the lungs for oxygenation (Keith-2006).

The left side of the heart (left heart) receives well-oxygenated (arterial) blood from the lungs through the pulmonary veins and pumps it into the aorta for distribution to the body. The heart has four chambers: right and left atria and right and left ventricles. The atria are receiving chambers that pump blood into the ventricles (the discharging chambers). The synchronous pumping actions of the heart's two atrioventricular (AV) pumps (right and left chambers) constitute the cardiac cycle. The cycle begins with a

period of ventricular elongation and filling (diastole) and ends with a period of ventricular shortening and emptying (systole). (Keith-2006).

2.1.1 The wall of the heart chamber:

- Endocardium, a thin internal layer (endothelium and sub endothelial connective tissue) or lining membrane of the heart that also covers its valves.
- Myocardium, a thick, helical middle layer composed of cardiac muscle.
- Epicardium, a thin external layer (mesothelium) formed by the visceral layer of serous pericardium.

The walls of the heart consist mostly of thick myocardium, especially in the ventricles. When the ventricles contract, they produce a wringing motion because of the double helical orientation of the cardiac muscle fibers (Keith-2006).

2.1.2 The apex of the heart:

Is formed by the inferolateral part of the left ventricle. Lies posterior to the left 5th intercostal space in adults, usually approximately 9 cm (a hand's breadth) from the median plane. remains motion less throughout the cardiac cycle. Is where the sounds of mitral valve closure are maximal (apex beat), the apex underlies the site where the heartbeat may be auscultated on the thoracic wall (Keith-2006).

2.1.3 The base of the heart:

Is the heart's posterior aspect (opposite the apex), is formed mainly by the left atrium, with a lesser contribution by the right atrium. Faces posteriorly toward the bodies of vertebrae T6-T9 and is separated from them by the pericardium, oblique pericardial sinus, esophagus, and aorta, extends superiorly to the bifurcation of the pulmonary trunk and

inferiorly to the coronary groove, receives the pulmonary veins on the right and left sides of its left atrial portion and the superior and inferior vena cava at the superior and inferior ends of its right atrial portion (Keith-2006).

2.1.4 The surfaces of the heart:

The four surfaces of the heart are the:

Anterior (sternocostal) surface, formed mainly by the right ventricle, diaphragmatic (inferior) surface, formed mainly by the left ventricle and partly by the right ventricle; it is related mainly to the central tendon of the diaphragm, right pulmonary surface, formed mainly by the right atrium, left pulmonary surface, formed mainly by the left ventricle; it forms the cardiac impression of the left lung and the SVC enters its right side. Posterior to the aorta and pulmonary trunk and anterior to the SVC, this border forms the inferior boundary of the transverse pericardial sinus (Keith-2006).

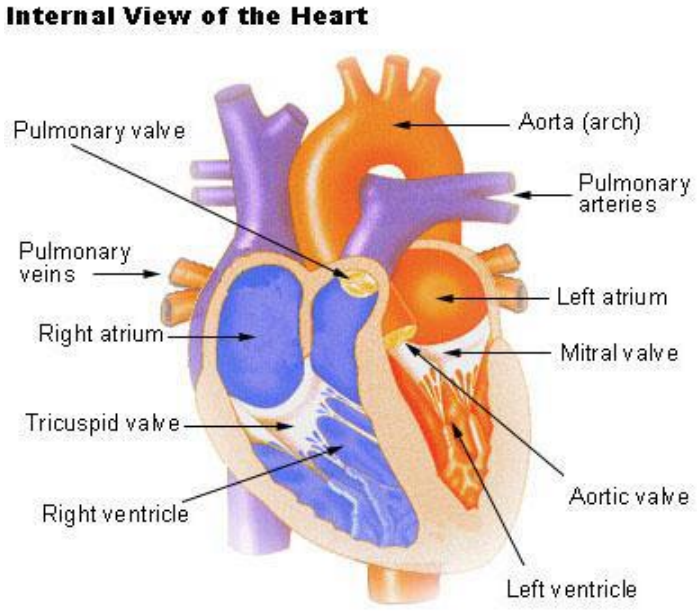

Figure (2.1): Gross anatomy of the heart. (F. Buba-2008).

2.1.5 Right Atrium:

The right atrium forms the right border of the heart and receives venous blood from the SVC, IVC, and coronary sinus. The ear-like right auricle is a conical muscular pouch that projects from this chamber like an add-on room, increasing the capacity of the atrium as it overlaps the ascending aorta (Richarch-2000).

2.1.6 Right Ventricle:

The right ventricle forms the largest part of the anterior surface of the heart, a small part of the diaphragmatic surface, and almost the entire inferior border of the heart. Superiorly it tapers into an arterial cone, the conus arteriosus (infundibulum), which leads into the pulmonary trunk. The interior of the right ventricle has irregular muscular elevations (trabeculae cornea). A thick muscular ridge, the supraventricular crest, separates the ridged muscular wall of the inflow part of the chamber from the smooth wall of the conus arteriosus, or outflow part. The inflow part of the ventricle receives blood from the right atrium through the right AV (tricuspid) orifice located posterior to the body of the sternum at the level of the 4th and 5th intercostal spaces. The tricuspid valve guards the right AV orifice. The bases of the valve cusps are attached to the fibrous ring around the orifice (Richarch-2000).

2.1.7 Left Atrium:

The left atrium forms most of the base of the heart. The valve less pairs of right and left pulmonary veins enters the smooth-walled atrium. In the embryo, there is only one common pulmonary vein, just as there is a single pulmonary trunk. The wall of this vein and four of its tributaries were incorporated into the wall of the left atrium, in the same way that the sinus venous was incorporated into the right atrium (Richarch-2000).

2.1.8 Left Ventricle:

The left ventricle forms the apex of the heart, nearly all its left (pulmonary) surface and border, and most of the diaphragmatic surface because arterial pressure is much higher in the systemic than in the pulmonary circulation.

The interior of the left ventricle has Walls that are two to three times as thick as that of the right ventricle (Richarch-2000).

The mitral valve has two cusps, anterior and posterior. The mitral valve is located posterior to the sternum at the level of the 4th costal cartilage. Each of its cusps receives tendinous cords from more than one papillary muscle (Richarch-2000).

2.1.9 Vasculature of the Heart:

The blood vessels of the heart comprise the coronary arteries and cardiac veins, which carry blood to and from most of the myocardium. The blood vessels of the heart are affected by both sympathetic and parasympathetic innervations (Keith-2006).

2.1.9.1 Arterial Supply of the Heart:

The coronary arteries, the first branches of the aorta, supply the myocardium and epicardium. The right and left coronary arteries arise from the corresponding aortic sinuses at the proximal part of the ascending aorta. The coronary arteries supply both the atria and the ventricles; however, the atrial branches are usually small and not readily apparent in the cadaveric heart. The ventricular distribution of each coronary artery is not sharply demarcated. (Stary-1990). The right coronary artery (RCA) arises from the right aortic sinus of the ascending aorta and passes to the right side of the pulmonary trunk, running in the coronary groove. Near its origin, the RCA usually gives off an ascending sinuatrial nodal branch The RCA then descends in the coronary groove and gives off the

right marginal branch, which supplies the right border of the heart as it runs toward the apex of the heart the RCA supplies the right atrium, most of right ventricle, part of the left ventricle (the diaphragmatic surface), part (usually the posterior third) of the IV septum. The SA node (in approximately 60% of people), the AV node (in approximately 80% of people) (Keith-2006).

The left coronary artery (LCA) arises from the left aortic sinus of the ascending aorta, passes between the left auricle and the left side of the pulmonary trunk, and runs in the coronary groove the LCA divides into two branches, the anterior IV branch (left anterior descending (LAD) branch) and the circumflex branch. The anterior IV branch passes along the IV groove to the apex of the heart (Fig 2.2) (Keith-2006).

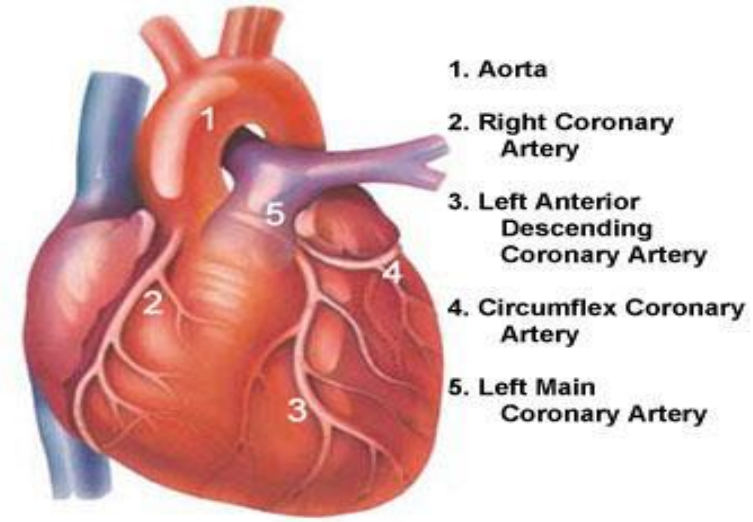

Figure (2.2): Arteries of the heart (F. Buba-2008).

2.1.9.2 Venous Drainage of the Heart:

The heart is drained mainly by veins that empty into the coronary sinus and partly by small veins that empty into the right atrium. The coronary sinus, the main vein of the heart, is a wide venous channel that runs from left to right in the posterior part of the coronary groove. The coronary sinus receives the great cardiac vein at its left end and the

middle cardiac vein and small cardiac veins at its right end. The left posterior ventricular vein and left marginal vein also open into the coronary sinus (Keith-2006).

The great cardiac vein is the main tributary of the coronary sinus. Its first part (anterior interventricular vein) begins near the apex of the heart and ascends with the anterior IV branch of the LCA. The great cardiac vein drains the areas of the heart supplied by the LCA. The middle cardiac vein (posterior IV vein) accompanies the posterior interventricular branch (usually arising from the RCA), and a small cardiac vein accompanies the right marginal branch of the RCA (Keith-2006).

Some cardiac veins do not drain via the coronary sinus. Several small anterior cardiac veins begin over the anterior surface of the right ventricle; cross over the coronary groove, and usually end directly in the right atrium sometimes they enter the small cardiac vein. The smallest cardiac veins are minute vessels that begin in the capillary beds of the myocardium and open directly into the chambers of the heart, chiefly the atria. Although called veins, they are valve less communications with the capillary beds of the myocardium and may carry blood from the heart chambers to the myocardium (Fig 2.3) (Keith-2006).

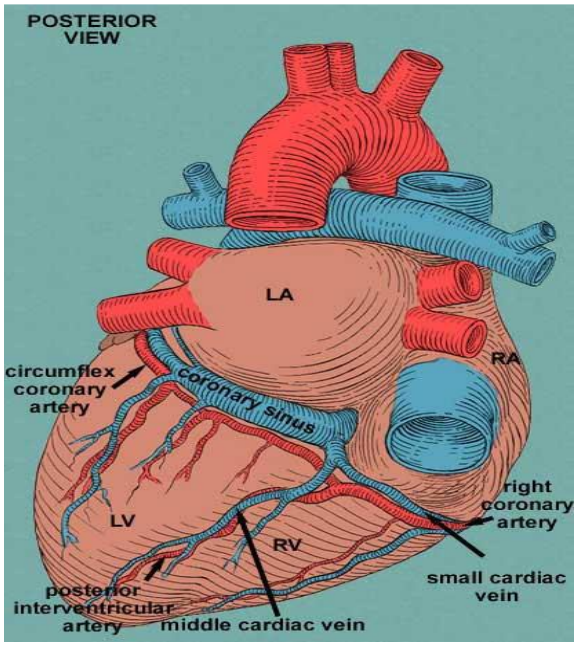

Figure (2.3): Venues drainage of the heart (F.Buba-2008).

2.1.10 Lymphatic Drainage of the Heart:

Lymphatic vessels in the myocardium and sub endocardial connective tissue pass to the subepicardial lymphatic plexus. Vessels from this plexus pass to the coronary groove and follow the coronary arteries. A single lymphatic vessel, formed by the union of various vessels from the heart, ascends between the pulmonary trunk and left atrium and ends in the inferior tracheobronchial lymph nodes, usually on the right side (Richarch-2000).

2.1.11 Innervations of the Heart:

The heart is supplied by autonomic nerve fibers from the cardiac plexus which is often quite artificially divided into superficial and deep portions. The sympathetic supply is from presynaptic fibers, with cell bodies in the inter mediolateral cell columns of the superior five or six thoracic segments of the spinal cord, and postsynaptic sympathetic fibers, with cell bodies in the cervical and superior thoracic paravertebral ganglia of the sympathetic trunks. The postsynaptic fibers traverse cardiopulmonary splanchnic nerves

and the cardiac plexus to end in the SA and AV nodes and in relation to the terminations of parasympathetic fibers on the coronary arteries. Sympathetic stimulation causes increased heart rate; impulse conduction; force of contraction; and, at the same time, increased blood flow through the coronary vessels to support the increased activity. (Keith-2006).

The parasympathetic supply is from presynaptic fibers of the vagus nerves. Postsynaptic parasympathetic cell bodies (intrinsic ganglia) are located in the atrial wall and interatrial septum near the SA and AV nodes and along the coronary arteries. Parasympathetic stimulation slows the heart rate, reduces the force of the contraction, and constricts the coronary arteries, saving energy between periods of increased demand. Postsynaptic parasympathetic fibers release acetylcholine, which binds with muscarinic receptors to slow the rates of depolarization of the pacemaker cells and atrioventricular conduction and decrease atrial contractility (Keith-2006).

2.2 Physiology of the heart:

The heart is the muscular organ of the circulatory system that constantly pumps blood throughout the body. Approximately the size of a clenched fist, the heart is composed of cardiac muscle tissue that is very strong and able to contract and relax rhythmically throughout a person's lifetime (William 2003). The human heart is actually two pumps in one. The right side receives oxygen-poor blood from the various regions of the body and delivers it to the lungs. In the lungs, oxygen is absorbed in the blood. The left side of the heart receives the oxygen-rich blood from the lungs and delivers it to the rest of the body (William-2003).

The heart has four separate compartments or chambers. The upper chamber on each side of the heart, which is called an atrium, receives and collects the blood coming to the heart. The atrium then delivers blood to the powerful lower chamber, called a ventricle, which pumps blood away from the heart through powerful, rhythmic contractions (William-2003).

2.2.1 Mechanical events of the cardiac cycle:

2.2.1.1 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 (William-2003).

2.2.1.2 Atrial Systole:

Contraction of the atria propels some additional blood into the ventricles, but about 70% of the ventricular filling occurs passively during diastole. Contraction of the atrial muscle that surrounds the orifices of the superior and inferior vena cava and pulmonary veins narrows their orifices, and the inertia of the blood moving toward the heart tends to keep blood in it; however, there is some regurgitation of blood into the veins during atrial systole (William-2003).

2.2.1.3 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 (William-2003).

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) 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 (William-2003).

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 (William-2003).

2.2.1.4 Early Diastole:

Once the ventricular muscle is fully contracted, the already falling ventricular pressures drop more rapidly. This is the period of pro to diastole. 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 (Arthur-2006).

2.2.2 Electrical Conduction System:

The heart is composed primarily of muscle tissue. A network of nerve fibers coordinates the contraction and relaxation of the cardiac muscle tissue to obtain an efficient, wave-like pumping action of the heart (Fig 2.4) (Arthur-2006).

- Sinoatrial node (SA node)
- Atrioventricular node (AV node)
- Common AV Bundle
- Right & Left Bundle Branches

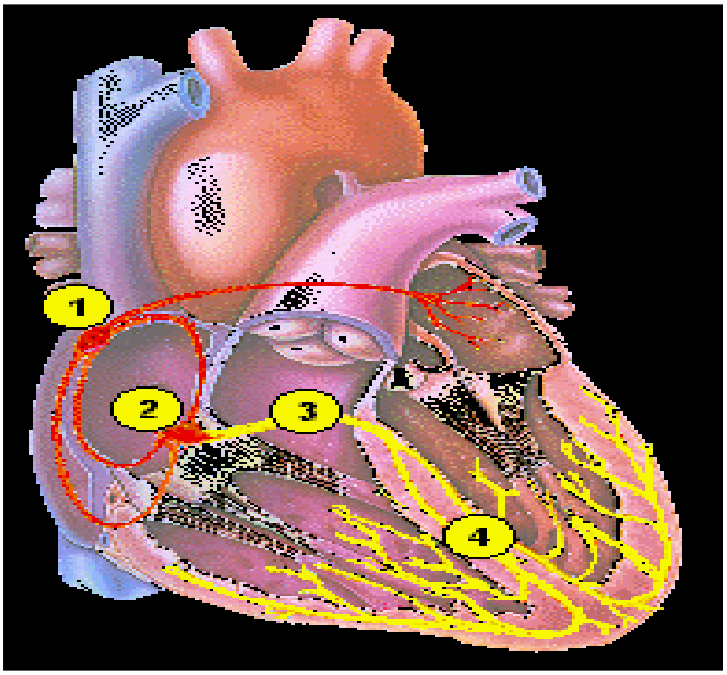

Figure (2.4): Electrical Conduction System of heart (F.Buba-2008).

The Sinoatrial Node (often called the SA node or sinus node) serves as the natural pacemaker for the heart. Nestled in the upper area of the right atrium, it sends the electrical impulse that triggers each heartbeat. The impulse spreads through the atria, prompting the cardiac muscle tissue to contract in a coordinated wave-like manner. The impulse that originates from the sinoatrial node strikes the Atrioventricular node (or AV node) which is situated in the lower portion of the right atrium. The atrioventricular node in turn sends an impulse through the nerve network to the ventricles, initiating the same wave-like contraction of the ventricles (Arthur-2006).

The electrical network serving the ventricles leaves the atrioventricular node through the Right and Left Bundle Branches. These nerve fibers send impulses that cause the cardiac muscle tissue to contract (Arthur-2006).

2.3 Cardiovascular Diseases:

Cardiovascular disease refers to the class of diseases that involve the heart and/or blood vessels (arteries and veins). While the term technically refers to any disease that affects the cardiovascular system, which could be highlighted as atherosclerosis (arterial disease). These conditions have similar causes, mechanisms, and treatments. Most cardiovascular disease arises from the interaction of environmental factors and genetic susceptibility. The contemporary view holds that most clinical cardiovascular diseases result from a complex interplay of genetics and environmental factors that disrupt networks controlling morphogenesis, myocyte survival, biomechanical stress responses, contractility, and electrical conduction (Ramzi-2006).

2.3.1 Heart Failure:

In the heart failure impaired cardiac function fails to maintain a circulation adequate for the metabolic needs of the body despite an adequate blood volume.

The clinical spectrum ranges from asymptomatic cardiac dysfunction through reduced exercise tolerance, in which compensatory mechanisms (ventricular hypertrophy, peripheral vasoconstriction, salt and water retention) maintain tissue perfusion, to stage in which these mechanisms are exhausted and symptoms and signs of heart failure occur at rest (David-2008).

In heart failure, often called congestive heart failure (CHF), the heart is unable to pump blood at a rate commensurate with the requirements of the metabolizing tissues or can do so only at an elevated filling pressure. Although usually caused by a slowly developing intrinsic deficit in myocardial contraction, a similar clinical syndrome is present in some patients with heart failure caused by conditions in which the normal heart is suddenly

presented with a load that exceeds its capacity (e.g., fluid overload, acute myocardial infarction, acute valvular dysfunction) or in which ventricular filling is impaired. CHF is a common and often recurrent condition with a poor prognosis (Ramzi-2006).

Most instances of heart failure are the consequence of progressive deterioration of myocardial contractile function (systolic dysfunction), as often occurs with ischemic injury, pressure or volume overload, or dilated cardiomyopathy. The most frequent specific causes are ischemic heart disease and hypertension. Sometimes, however, failure results from an inability of the heart chamber to relax, expand, and fill sufficiently during diastole to accommodate an adequate ventricular blood volume (diastolic dysfunction), as can occur with massive left ventricular hypertrophy, myocardial fibrosis, deposition of amyloid, or constrictive pericarditis. Whatever its basis, CHF is characterized by diminished cardiac output (sometimes called forward failure) or damming back of blood in the venous system (so-called backward failure), or both (Ramzi-2006)

2.3.1.1 Cardiac hypertrophy:

Pathophysiology and progression to failure.

The cardiac myocyte is generally considered a terminally differentiated cell that has lost its ability to divide. Under normal circumstances, functionally useful augmentation of myocyte number (hyperplasia) cannot occur. Increased mechanical load causes an increase in the content of subcellular components and a consequent increase in cell size (hypertrophy). Increased mechanical work owing to pressure or volume overload or trophic signals (e.g., hyperthyroidism through stimulation of beta-adrenergic receptors) increases the rate of protein synthesis, the amount of protein in each cell, the number of

sarcomeres and mitochondria, the dimension and mass of myocytes and, consequently, the size of the heart. Recent (Ramzi-2006).

The extent of hypertrophy varies for different underlying causes. Heart weight usually ranges from 350 to 600 gm (up to approximately two times normal) in pulmonary hypertension and ischemic heart disease; from 400 to 800 gm (up to two to three times normal) in systemic hypertension, aortic stenosis, mitral regurgitation, or dilated cardiomyopathy; from 600 to 1000 gm (three or more times normal) in aortic regurgitation or hypertrophic cardiomyopathy. Hearts weighing more than 1000 gm are rare.

2.3.1.2 Left sided heart failure:

Left-sided heart failure is most often caused by ischemic heart disease, hypertension, aortic and mitral valvar diseases, and non-ischemic myocardial diseases. The morphologic and clinical effects of left-sided CHF primarily result from progressive damming of blood within the pulmonary circulation and the consequences of diminished peripheral blood pressure and flow (Ramzi-2006).

2.3.1.3 Right sided heart failure:

Isolated right-sided heart failure occurs in only a few diseases. Usually it is a secondary consequence of left-sided heart failure because any increase in pressure in the pulmonary circulation incidental to left-sided heart failure inevitably produces an increased burden on the right side of the heart. The causes of right-sided heart failure must then include all those that induce left-sided heart failure (Ramzi-2006).

Pure right-sided heart failure most often occurs with chronic severe pulmonary hypertension and thus is called Cor Pulmonale. In this condition, the right ventricle is burdened by a pressure workload due to increased resistance within the pulmonary circulation. Hypertrophy and dilation are generally confined to the right ventricle and atrium, although bulging of the ventricular septum to the left can cause dysfunction of the left ventricle. (Ramzi-2006).

2.3.1.4 causes of heart failure:

Causes of heart failure they are due to excessive load on the myocardium, poor myocardium function (contraction or relaxation) or combination of the both mechanisms Table (2:1) (David-2008).

Table 2:1: Causes of heart failure (David-2008).

Left ventricle	**Right ventricle**
Myocardial injury	Pulmonary hypertension
Myocarditis	Increased work load
Ischemia / infarction	Myocardial injury
Cardiomyopathy	Ischemia / infarction
Systemic hypertension	Cardiomyopathy

2.3.1.5 Left ventricular failure:

Left ventricular failure is most commonly due to ischemic heart disease, particularly myocardial infarction, but also to systemic hypertension and aortic and mitral valve disease the failing ventricle dilates which further impairs contraction (David-2008).

2.3.1.6 Right ventricular failure:

Right ventricular failure is usually secondary to left ventricular failure. When the left ventricle fails increased pressure in the left atrium and pulmonary veins leads to pulmonary arteriolar vasoconstriction and resultant pulmonary artery hypertension. Persistent pulmonary hypertension causes right ventricular hypertrophy and eventually failure. When the right ventricle fails, it dilates, stretching of the tricuspid ring results involve incompetence and dilatation of the right atrium (David-2008).

2.3.1.7 Congestive Heart Failure:

Congestive heart failure (CHF), also called congestive cardiac failure (CCF) or just heart failure is a condition that can result from any structural or functional cardiac disorder that impairs the ability of the heart to fill with or pump a sufficient amount of blood throughout the body. It is not to be confused with "cessation of heartbeat", which is known as asystole, or with cardiac arrest, which is the cessation of normal cardiac function in the face of heart disease. Because not all patients have volume overload at the time of initial or subsequent evaluation, the term "heart failure" is preferred over the older term "congestive heart failure". Congestive heart failure is often undiagnosed due to a lack of a universally agreed definition and difficulties in diagnosis, particularly when the condition is considered “mild” (Fig 2.5) (Eric-2013).

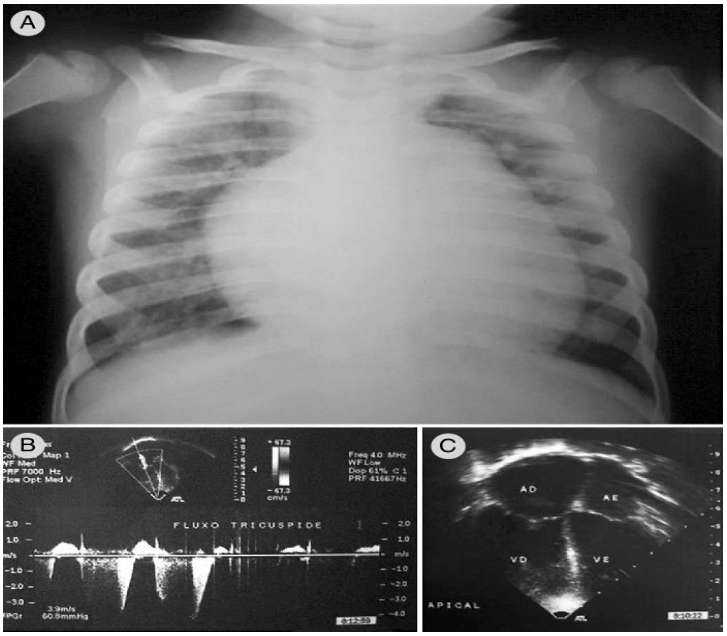

Figure (2.5): Shows 3-year-old child with a history of cardiac complications since birth.

2.3.1.8 Hypertension:

Hypertension or high blood pressure is a medical condition where in the blood pressure is chronically elevated. Persistent hypertension is one of the risk factors for strokes, heart attacks, heart failure and arterial aneurysm, and is a leading cause of chronic renal failure (htt//www.who.int/chp/steps/en).

2.3.1.9 Atherosclerosis:

Atherosclerosis is a disease affecting the arterial blood vessel. It is commonly referred to as a "hardening" or "furring" of the arteries. It is caused by the formation of multiple plaques within the arteries. Arteriosclerosis ("hardening of the artery") results from a deposition of tough, rigid collagen inside the vessel wall and around the atheroma figure (2.6), which increases the stiffness, decreases the elasticity of the artery wall. Atherosclerosis typically begins in early adolescence, is usually found in most major arteries, and yet is asymptomatic and not detected by most diagnostic methods during life, and becomes seriously symptomatic when involving the coronary arteries or cerebral circulation, and is considered the most important underlying cause of strokes, heart

attacks, congestive heart failure and most cardiovascular diseases in general (Fig 2.6) (htt//www.who.int/chp/steps/en).

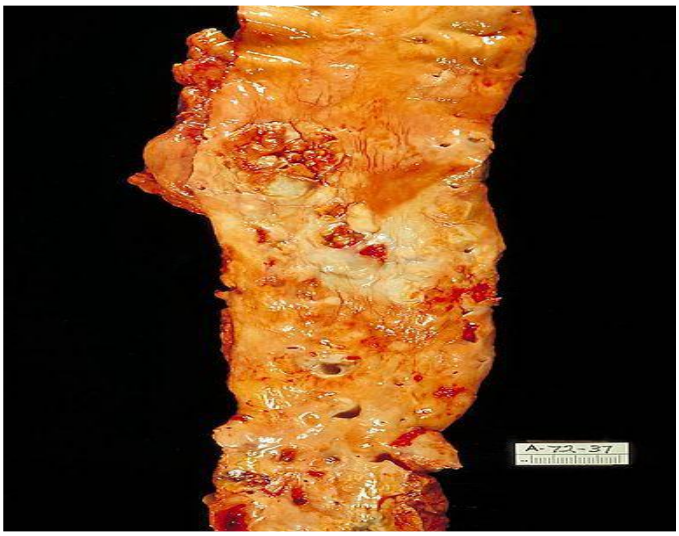

Figure (2-6): Shows severe atherosclerosis of the aorta. Autopsy specimen. (htt //www.who.int/chp/steps/en).

2.3.1.10 Plaque:

Plaque Atheroma or commonly known as plaque is an abnormal inflammatory accumulation of macrophage white blood cells within the walls of arteries. (htt//www.who.int/chp/steps/en).

2.3.1.11 Circulatory Shock:

Circulatory Shock is a severe condition that results from reduced blood circulation. A thrombus, or blood clot, is the final product of the blood coagulation step in hemostasis. It is achieved via the aggregation of platelets that form a platelet plug, and the activation of the humoral coagulation system (i.e. clotting factors). A thrombus is physiologic in cases of injury, but pathologic in case of thrombosis. Preventing blood clots reduces the risk of stroke, heart attack and pulmonary embolism. Heparin and warfarin are often used to inhibit the formation and growth of existing blood clots, thereby allowing the body to shrink and dissolve the blood clots through normal methods. An embolism occurs when

an object (the embolus) migrates from one part of the body (through circulation) and causes a blockage (occlusion) of a blood vessel in another part of the body. Blood clots form the most common embolic material by far: other possible embolic materials include fat globules (a fat embolism), air bubbles (an air embolism), septic emboli (containing pus and bacteria), or amniotic fluid. (htt//www.who.int/chp/steps/en)

2.3.1.12 Stroke:

A stroke, also known as cerebrovascular accident (CVA), is an acute neurological injury whereby the blood supply to a part of the brain is interrupted. Strokes can be classified into two major categories: ischemic and hemorrhagic.

2.3.1.12.1 Ischemic Stroke:

Ischemic stroke, which occurs in approximately 85-90% of strokes, a blood vessel becomes occluded and the blood supply to part of the brain is totally or partially blocked. **Ischemic stroke is commonly divided into thrombotic stroke, embolic stroke, systemic** hypo perfusion (Watershed or Border Zone stroke), or venous thrombosis (htt//www.who.int/chp/steps/en).

2.3.1.12.2. Hemorrhagic Stroke:

A hemorrhagic stroke, or cerebral hemorrhage, is a form of stroke that occurs when a blood vessel in the brain ruptures or bleeds. Like ischemic strokes, hemorrhagic strokes interrupt the brain's blood supply because the bleeding vessel can no longer carry the blood to its target tissue. In addition, blood irritates brain tissue, disrupting the delicate chemical balance, and, if the bleeding continues, it can cause increased intracranial pressure which physically impinges on brain tissue and restricts blood flow into the brain.

There are two types of hemorrhagic stroke: intracerebral hemorrhage, and subarachnoid hemorrhage.

The term "brain attack" has been used to express stroke, and if symptoms of stroke are detected at early on-set, special "clot busting" drugs may be administered(htt//www.who.int/chp/steps/en).

2.3.1.13 Heart Attack

Acute myocardial infarction (AMI or MI), commonly known as a heart attack, a heart attack occurs when the supply of blood and oxygen to an area of heart muscle is blocked, usually by a clot in a coronary artery. Often, this blockage leads to arrhythmias (irregular heartbeat or rhythm) that cause a severe decrease in the pumping function of the heart and may bring about sudden death. If the blockage is not treated within a few hours, the affected heart muscle will die and be replaced by scar tissue. It is the leading cause of death for both men and women all over the world.

2.3.1.14 Angina Pectoris

Angina Pectoris is chest pain due to ischemia (a lack of blood and hence oxygen supply) of the heart muscle, generally due to obstruction or spasm of the coronary arteries (the heart's blood vessels).

2.3.1.15 Aneurysm

An aneurysm (or aneurism) is a localized dilation or ballooning of a blood vessel by more than 50% of the diameter of the vessel and can lead to instant death at any time. Aneurysms most commonly occur in arteries at the base of the brain (the circle of Willis) and in the aorta (the main artery coming out of the heart) - this is an aortic aneurysm. This bulge in a blood vessel, much like a bulge on an over-inflated inner tube, can lead to

death at any time. The larger an aneurysm becomes, the more likely it is to burst. Aneurysms are also described according to their shape: Saccular or fusiform. A saccular aneurysm resembles a small sack; a fusiform aneurysm is shaped like a spindle.

2.4 Examinations of the cardiovascular system:

2.4.1 Inspection:

Inspection by fingers may reveal clubbing (cyanotic heart disease, endocarditis), splinter hemorrhages, Osler ‘s nodes (painful erythematous lesions on the pulps of the fingers) and Jane way lesions (flat erythematous lesions on the palms of the hands). Peripheral infarctions of the digits can be due to emboli from atrial fibrillation or prosthetic heart valves (Eric-2013).

2.4.2 Palpation:

The peripheral pulse reveals the rate and rhythm (pulse volume should be assessed at the carotids). In addition, the character of the pulse may be bounding in aortic regurgitation, giving a water hammer quality. Symmetry of the pulses is important and may be affected by aortic dissection and coarctation (radio-femoral delay). The blood pressure is obtained and a narrow pulse pressure (less than half the diastolic pressure) may be indicative of aortic stenosis. The JVP is examined: elevation occurs with congestive cardiac failure, tricuspid valve disease and pericardial tamponade. The precordium is palpated, and the apex beat located. The apex beat is displaced by conditions that lead to cardiomegaly (congestive heart failure, aortic valve disease, mitral regurgitation). A thrill (palpable murmur) will occur with the associated heart valve disease (Eric-2013).

2.4.3 Auscultation:

The heart is auscultated in the apex with the bell and diaphragm of the stethoscope and then over the second right intercostal space (aortic area), the second left intercostal space (pulmonary area), down the left border of the sternum (for the diastolic murmur of aortic regurgitation) and over the tricuspid area. (Eric-2007).

2.4.4 Peripheral examination:

At the end of the examination it is also important to auscultate the lung bases for pulmonary edema and to examine the liver for hepatomegaly and the legs for peripheral edema (Eric-2013).

2.4.5 Blood tests:

The almost absolute specificity of troponin to cardiac muscle has led to the recent re-definition of myocardial infarction using elevated serum troponin as the primary diagnostic criterion. Troponin is detected in the serum approximately 4–10 hours after the onset of myocardial infarction, peaks at 12–48 hours and remains elevated for 4–10 days.1 CK-MB (myocardium-bound fraction of creatine kinase) is a suitable alternative when troponin assay is not available. CK-MB is elevated 4–8 hours after myocardial infarction, peaks at approximately 12 hours and returns to normal after 2–3 days. CK-MB remains elevated for a much shorter time than troponin, is a useful indicator for early re-infarction and provides an estimate of the size of the infarct.

2.4.6 Electrocardiogram:

The standard 12 lead ECG is obtained by the analysis of electrical current generated by the heart and analyzed in two planes. The first plane is the coronal plane of the limb leads. Each lead is made positive in turn: V1 is in the 4th intercostal space to the right of the sternum, V4 is in the 5th intercostal space to the left of the sternum, and V6 lies in the mid axillary line (Eric-2013).

Grouping of the leads allows the different aspects of the heart to be analyzed. The main regions are the anterior, inferior and left lateral surfaces (Fig 2.7).

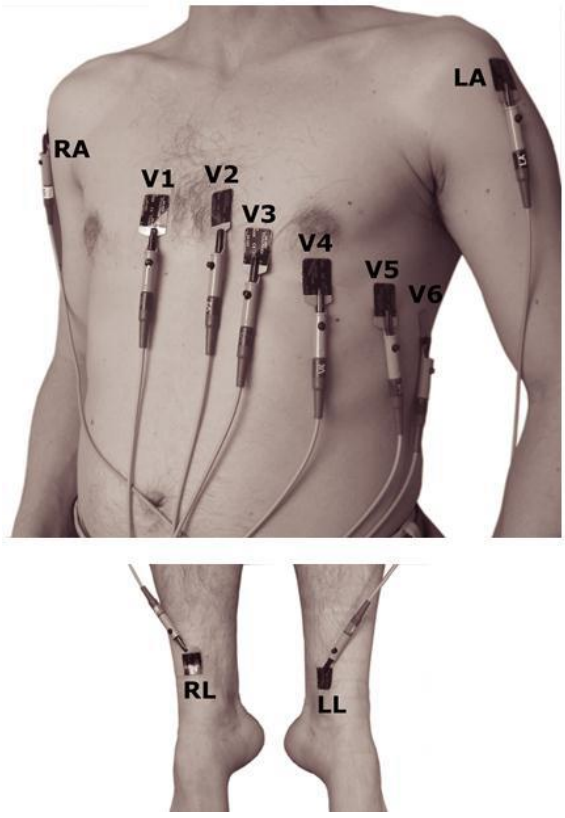

Figure (2.7): Shows Grouping of the leads allows the different aspects of the heart (Eric-2013).

2.4.6.1 P wave:

The P wave (Fig. 2.8) results from atrial depolarization. The current flows from right atrium to left atrium and in a slightly inferior direction; it is therefore positive in the left and inferior leads. The duration of the P wave is 0.12–0.20 seconds (3–5 small squares). A short P wave is usually due to an accessory pathway into the ventricle that bypasses the normal conduction, hence shortening the P wave. Two such common pathways are the bundle of Kent in Wolff–Parkinson–White syndrome and James fibers in Lown–Ganong–Levine syndrome, the pre-excitation syndromes (Eric-2013).

2.4.6.2 QRS complex:

The QRS complex (Fig. 1.3) is caused by depolarization of the ventricles. From a trio-ventricular node, the conducting system (the sites of fastest conduction) comprises the bundle of His, which divides into the right and left bundle branches and finally the terminal Purkinje fibers. The QRS duration is normally less than 0.12 seconds. Left axis deviation is associated with left ventricular enlargement (loosely termed hypertrophy, but the ECG cannot distinguish between myocardial hypertrophy and ventricular dilatation). Right axis deviation is much less common and is associated with right ventricular dilatation (Eric-2013).

2.4.6.3 The ST segment:

The ST segment (Fig. 2.8) represents the time from ventricular depolarization to repolarization. The height of the ST segment is affected by ischemia and infarction. It was previously thought that ST segment elevation was associated

with infarction whilst ST segment depression was associated with ischemia. It is now recognized that either configuration can be associated with infarction and troponin release (Eric-2013).

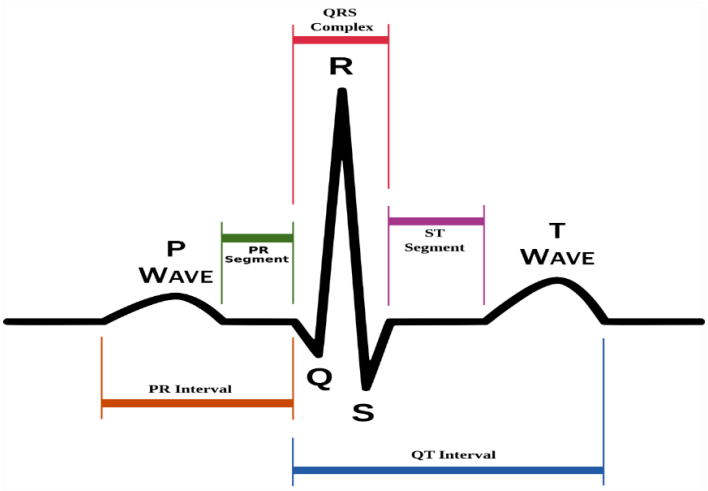

Figure (2.8): Shows Schematic representation of normal ECG.

2.4.6.4 T wave and QT interval:

The T wave (Fig. 2.8) is due to repolarization of the ventricles. Peaked T waves can result from hyperkalemia. The QT interval (from the onset of the Q to the end of the T wave) measures the duration from the start of depolarization to the end of repolarization (Fig.2.8). The normal QT interval varies with the heart rate, therefore the corrected QT interval is normally estimated as QT interval divided by the square root of the R to R interval of a successive beat and is normally less than 0.45 seconds. Drugs (antihistamines, tricyclic antidepressants) and electrolyte abnormalities (hypokalemia, hypocalcaemia) that prolong the QT interval predispose to a form of ventricular tachycardia (torsade de pointes) with a risk of progression to ventricular fibrillation (Eric-2013).

2.4.7 Techniques and normal anatomy:

Imaging of the heart is rendered more complex than usual by factors such as the heart's motion, respiratory motion and also its position in the lower thorax. It is surrounded by tissues of varying density with different abilities to both absorb and scatter radiation. It also overlies the diaphragm and activity can be sometimes difficult to separate from sub-diaphragmatic activity. In females there can also be problems with increased attenuation from overlying breast tissue whilst in males there may be problems related to diaphragmatic attenuation. Imaging of the heart and great vessels has previously been done with plain film, cardiac catheterization, nuclear medicine, and echocardiography as the primary imaging modalities. The recent newer advances in the computed tomography (CT) and magnetic resonance imaging (MRI) technologies, however, have dramatically changed our approach to imaging cardiac disease. CT and MRI, supplemented by CT angiography and MRI angiography, are increasingly replacing the chest film, as well as nuclear and—to some extent—echo imaging as the primary modalities in evaluating heart disease.

2.4.7.1 Echocardiography:

Echocardiography uses high-frequency ultrasound to evaluate the heart and great vessels. The examination provides a dynamic rendition of cardiac great vessel anatomy and, when combined with the Doppler technique, yields information regarding cardiac and great vessel blood flow (hemodynamics) as well. Because of the high frame rates inherent in ultrasonography, echocardiography can image the heart in a dynamic real-time fashion, so that the motion of cardiac structures can be reliably evaluated. Echocardiography is useful in assessing ventricular function, valvar heart disease,

myocardial disease, pericardial disease, intracardiac masses and aortic abnormalities. With Doppler technology, cardiac chamber function, valvular function, and intracardiac shunts frequently seen in congenital heart disease can be assessed. Combined Doppler echocardiography is a commonly performed procedure because it is relatively inexpensive and widely available, provides a wealth of information, is noninvasive, has no risk of ionizing radiation, and can also be performed at the bedside in critically ill patients. Furthermore, the results are immediately available because no special post examination image processing is required. However, this technique is technically challenging and requires a great deal of operator expertise. Also, a small percentage of patients have poor acoustic windows that can severely degrade image quality. This disadvantage can be obviated by placing the sonographic probe in the esophagus, a procedure called trans esophageal echocardiography (TEE). Trans esophageal echocardiography yields consistently excellent images of the heart and great vessels, but involves a small amount of discomfort and risk to the patient. More recently, echocardiography has been combined with stress-testing modalities to assess inducible myocardial ischemia using wall motion analysis of left ventricular function (Fig. 2.9).

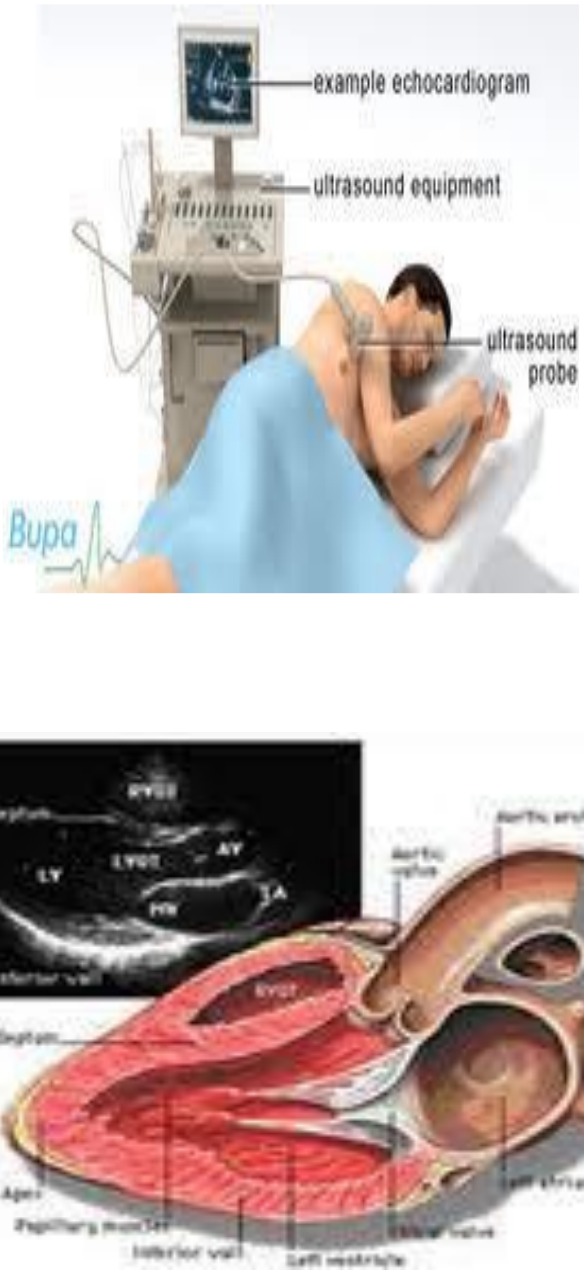

Figure (2.9): Shows Normal transthoracic echocardiogram from a healthy subject. Views are taken from the left mid parasternal region through an inter costal space. The structure closest to the apex of the screen is the chest wall. The mitral valve, separating the left atrium and left ventricle, is partially open in this image from early systole. A, aorta; LA, left atrium; LV, left ventricle; RV, right ventricle.

Echocardiography (echo) – the use of ultrasound to examine the heart – is a safe, powerful, non-invasive and painless technique. Echo is easy to understand as many features are based upon simple physical and physiological facts.

Doppler echocardiography is a noninvasive technique that provides unique hemodynamic information. The accuracy of the results depends, however, on meticulous technique and an understanding of Doppler principles and flow dynamics.

2.4.7.1.1 Viewing the heart:

Echo studies are carried out using specialized ultrasound machines. Ultrasound of different frequencies (in adults usually 2–4 MHz) is transmitted from a transducer (probe) which is placed on the subject's anterior chest wall. This is transthoracic echo (TTE). The transducer usually has a line or dot to help rotate it into the correct position to give different echo views. The subject usually lies in the left lateral position and ultrasound jelly is placed on the transducer to ensure good images. An echo examination usually takes 15–20 min.

2.4.7.1.2 Echo 'windows' and views:

There are a number of standard positions on the chest wall for the transducer where there are 'echo windows' that allow good penetration by ultrasound without too much masking and absorption by lung or ribs.

A number of sections of the heart are examined by echo from these transducers positions, which are used for 2 main reasons:

- There is a limitation determined by the anatomy of the heart and its surrounding structures
- To produce standardized images that can be compared between different studies.

Useful echo information can be obtained in most subjects, but the study can be technically difficult in:

- Very obese subjects
- Those with chest wall deformities
- Those with chronic lung disease (e.g. chronic air flow limitation with

Hyper inflated lungs or pulmonary fibrosis).

Rarely, an echo study is impossible.

A number of 'echo views' are obtained in most studies. 'Axis' refers to the plane in which the ultrasound beam travels through the heart.

2.4.7.1.2.1 Left parasternal window. (2nd–4th intercostal space, left sternal edge).

2.4.7.1.2.1.1 Long-axis view:

Most examinations begin with this view. The transducer is used to obtain images of the heart in long axis, with slices from the base of the heart to the apex. The marker dot on the transducer points to the right shoulder.

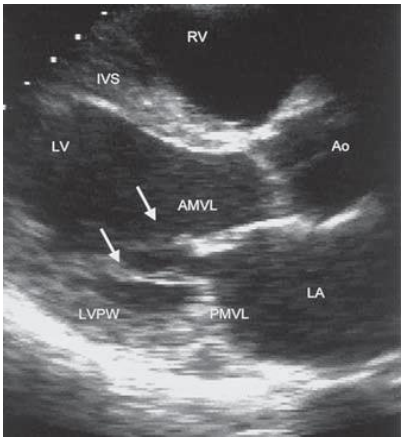

Fig. (2.10): Parasternal long-axis view. Arrows show chordae.

2.4.7.1.2.1.2 Short-axis views

Without moving the transducer from its location on the chest wall and by rotating the transducer through 90º so the marker dot is pointing towards the left shoulder, the heart is cut in transverse(short-axis) sections. By changing the angulation on the chest wall, it is possible to obtain any number of short-axis views, but the standard 4 are at the level of the aortic valve (AV), mitral valve (MV), left ventricular papillary muscles and left ventricular apex.

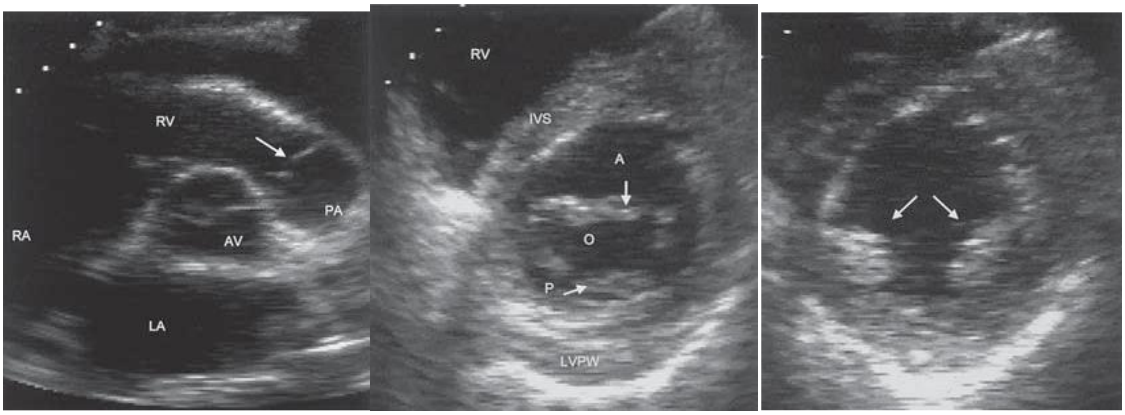

Figure (2.11): Parasternal short-axis views: (a) Aortic valve level. The pulmonary valve is shown (arrow). (b) Mitral valve level. The anterior (A) and posterior (P) leaflets are shown. Mitral orifice (O). (c) Papillary muscles (arrows) level.

2.4.7.1.3 Apical window. (Cardiac apex):

2.4.7.1.3.1 chamber view:

The transducer is placed at the cardiac apex with the marker dot pointing down towards the left shoulder. This gives the typical ‘heart-shaped’ 4-chamber view.

2.4.7.1.3.2 Five-chamber (including aortic out flow):

By altering the angulation of the transducer so the ultrasound beam is angled more anteriorly towards the chest wall, a ‘5-chamber’ view is obtained. The 5th ‘chamber’ is

not a chamber at all but is the AV and ascending aorta. This is useful in assessing aortic stenosis (AS) and aortic regurgitation (AR).

2.4.7.1.3.3 Long-axis and 2-chamber views:

By rotating the transducer on the cardiac apex it is possible to obtain apical long-axis and 2-chamber views which show different segments of the left ventricle (LV).

2.4.7.1.4 Subcostal window.

(Under the xiphisternum) Similar views to apical views, but rotated by 90º. Useful in lung disease, for imaging the interatrial septum, inferior vena cava (IVC) and abdominal aorta. Further windows may be used:

2.4.7.1.5 Suprasternal window. (For imaging the aorta in coarctation).

2.4.7.1.6 Right parasternal window.

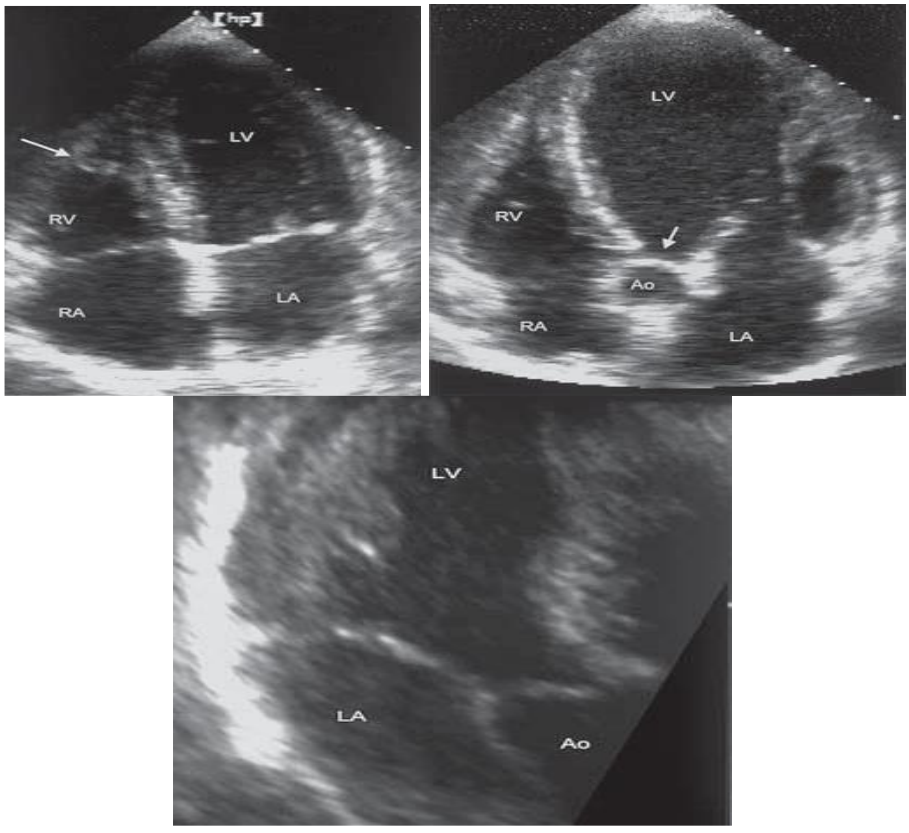

Figure. (2.12): Apical views: (a) Apical 4-chamber view. A moderator band is shown (arrow). This is a normal neuromuscular bundle carrying right bundle branch fibers. (b) Apical 5-chamber view. The aortic valve is shown (arrow). (c) Apical long-axis view.

2.5. General principles of Doppler echocardiogram:

The Doppler principle states that the frequency of reflected ultrasound is altered by a moving target, such as red blood cells. The magnitude of this Doppler shift relates to the velocity of the blood cells, whereas the polarity of the shift reflects the direction of blood flow toward (positive) or away (negative) from the transducer. The Doppler equation

$$\Delta F = \frac{V \times 2F_o \times \cos \theta}{c} \qquad (1)$$

states that the Doppler shift (ΔF) is directly proportional to the velocity (V) of the moving target (ie, blood cells), the transducer frequency (F_o), and the cosine of the angle of incidence (θ) and is inversely proportional to the velocity of sound in tissue (c = 1540 m/s). The Doppler equation can be solved for blood flow velocity as follows:

$$V = \frac{\Delta F \times c}{2 F_o \times \cos \theta} \qquad (2)$$

When solving the Doppler equation, an angle of incidence of 0 or 180 degrees (cosine = 1.0) is assumed for cardiac applications.

Currently, Doppler echocardiography consists of 3 modalities: pulsed wave (PW) Doppler, continuous wave (CW) Doppler, and color Doppler imaging. PW Doppler

measures flow velocity within a specific site (or sample volume) but is limited by the aliasing phenomenon that prevents it from measuring velocities beyond a given threshold (called the Nyquist limit). CW Doppler, on the other hand, can record very high blood flow velocities but cannot localize the site of origin of these velocities along the pathway of the sound beam. Color flow Doppler uses PW Doppler technology but with the addition of multiple gates or regions of interest within the path of the sound beam. In each of these regions, a flow velocity estimate is superimposed on the 2-dimensional (2D) image with a color scale based on flow direction, mean velocity, and sometimes velocity variance.

Doppler echocardiography is used to evaluate blood flow velocity with red blood cells as the moving target. Current ultrasound systems can also apply the Doppler principle to assess velocity within cardiac tissue. The moving target in this case is tissue, such as myocardium, that has higher amplitude of backscatter ultrasound and a lower velocity compared with red blood cells. This new application is called tissue Doppler and can be performed in the PW or the color mode.

Doppler echocardiography has two uses: detection and quantitation of normal and disturbed flow velocities. For detection purposes, all modalities have high sensitivity and specificity. However, color flow Doppler often allows faster detection of abnormal flows and provides a spatial display of velocities in a 2D plane. Quantification of flow velocity is typically obtained with either PW or CW Doppler. Measuring velocity with color Doppler is possible, but the methods are still under development and have not been standardized across different brands of ultrasound equipment. The primary use of PW Doppler is to assess velocities across normal valves or vessels to evaluate cardiac

function or calculate flow. Common applications include measurements of cardiac output (CO) and regurgitant volumes, quantitation of intracardiac shunts, and evaluation of diastolic function.

CW Doppler, on the other hand, is used to measure high velocities across restrictive orifices, such as stenotic or regurgitant valve orifices. These velocities are converted into pressure gradients by applying the simplified Bernoulli equation:(3) pressure gradient = $4V^2$(M Enriquez-1993).

This equation has been demonstrated to be accurate in flow models, animal studies, and in the cardiac catheterization laboratory as long as the velocity proximal to the obstruction does not exceed 1.5 m/s. Common clinical applications include determining pressure gradients in stenotic native valves, estimating pulmonary artery (PA) systolic pressure from the velocity of tricuspid regurgitation (TR), and determining prosthetic valve gradients. The combination of PW and CW Doppler has been used with great accuracy to determine stenotic valve areas with the continuity equation (M Enriquez-1993).

An alternative technique also used for recording high flow velocities is the high pulse repetition frequency (PRF) modification of the PW Doppler. High PRF uses range ambiguity to increase the maximum velocity that can be detected with PW Doppler. Multiple sample volumes are placed proximal to and at the depth of interest. PRF is determined by the depth of the most proximal sample volume, which allows measurement of higher velocities without signal aliasing at the depth of interest. Although the resulting spectral output includes frequencies from each of the sample

volume depths, the origin of the high-velocity signal is inferred from other anatomic and physiologic data, as with CW Doppler.

2.5.1 Recommendations on recording and measurement techniques:

The accuracy of measuring blood cell velocities by Doppler relies on maintaining a parallel orientation between the sound waves and blood flow. Although most ultrasound systems allow correction of the Doppler equation for the angle of incidence, this measurement is difficult to perform accurately because of the three-dimensional orientation of the blood flow. Angle correction is therefore not recommended. The Doppler sound beam should be oriented as parallel as possible to the flow, guided both by the 2D image (sometimes assisted by color flow imaging) and the quality of the Doppler recording. Small (<20 degrees) deviations in angle produce mild (<10%) errors in velocity measurements. Although these errors may be acceptable for low-velocity flows, when Doppler is used to derive pressure gradients even a small error in velocity measurement can lead to significant underestimation of the gradient because of the quadratic relation between velocity and pressure gradient.

2.5.2 PW Doppler:

PW Doppler is used in combination with the 2D image to record flow velocities within discrete regions of the heart and great vessels. Measurements derived from these velocities are used to evaluate cardiac performance (Figure 2.10).

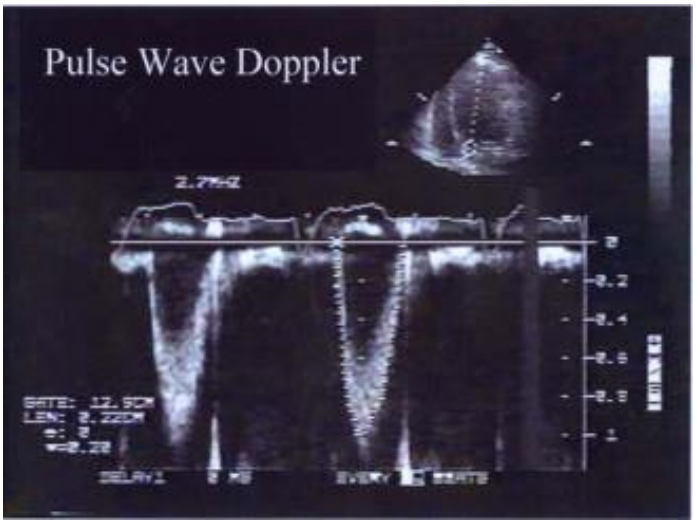

Fig (2.13): PW Doppler recording of left ventricular outflow track velocity obtained from apical window. Because flow is away from transducer, velocities are displayed below baseline. Notice narrow spectral pattern during flow acceleration and deceleration and wider dispersion seen during mid-systole. Degree of dispersion indicates range of blood flow velocities detected within sample volume.

The most common sites are the left ventricular outflow tract (LVOT), mitral annulus and left ventricular inflow (at the tips of the mitral valve leaflets), pulmonic valve annulus and PA, tricuspid valve inflow, and pulmonary veins. The flow volume passing through these sites can be calculated as the product of the velocity-time integral (VTI) and the cross-sectional area (CSA) of the respective site. When recording velocities for flow measurements, the sample volume is placed at the same location as the 2D measurements of CSA. Adjust the sample volume axial length to 5 to 7 mm and set the wall filters at low levels to ensure that lower velocities adjacent to the baseline are recorded for the timing of flow to be measured correctly. The velocities should be recorded over at least 2 or 3 respiratory cycles at a paper or sweep speed of 50 or 100 mm/s; the faster speed is

essential for measurements that require precise time resolution, such as time intervals, integrals, and velocity slopes.

A typical PW Doppler velocity consists of a spectral recording of varying intensity, depending on the acoustic density of the reflected interface, i.e., the mass of blood cells (Fig 2.10). The most dense (or brightest) portion of the spectral tracing represents the velocity of the majority of blood cells, also known as the modal velocity. Likewise, less dense areas depict the velocity of a lesser mass of blood cells. When measuring velocities, use the outer edge of the dense (or bright) envelope of the spectral recording.

2.5.3 CW Doppler:

In contrast to PW Doppler, CW Doppler records the velocities of all the red blood cells moving along the path of the sound beam (Figure2.11).

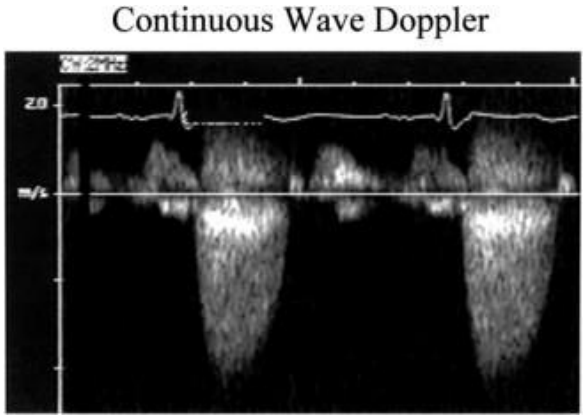

Fig. (2.14) CW Doppler recording of velocity through aortic valve in patient with AS. Transducer position is at apex; thus systolic velocities are displayed below baseline. In diastole, positive mitral inflow velocities can be seen as inflow moves toward transducer. Note wide spectral dispersion of velocities during systole and diastole indicating that Doppler beam is detecting all flow velocities encountered along its course.

Consequently, a CW Doppler recording always consists of a full spectral envelope with the outer border corresponding to the fastest moving blood cells. No simple guidelines guarantee a parallel orientation of the CW beam with blood flow in all instances. However, color flow Doppler can help determine the direction of the jet in a 2D plane, particularly in regurgitant lesions. A non-imaging CW transducer is recommended to search for the highest velocity, particularly in aortic stenosis (AS), in which multiple windows of examination may be required to detect the highest velocity. Measurements of velocities recorded by CW Doppler are always taken from the outer border. The site of origin of a high-velocity jet is inferred from the particular lesion that is being examined. For instance, if the CW beam is directed through a stenotic aortic valve, the outer edge of the recording is assumed to represent the stenotic jet velocity. Therefore, only well-defined envelopes should be used for quantitation of velocities to obviate significant errors. These recommendations are also applied when high PRF is used to record a high-velocity jet, except that high PRF should be used in combination with the 2D image.

2.5.4 Color flow Doppler:

A comprehensive discussion of color flow Doppler is beyond the scope of this document. Nevertheless, the following are some basic recommendations that apply to any ultrasound machine. Processing the multigate Doppler information and creating the color pixels take a certain amount of time; therefore, the larger the area of interest, the slower the frame rate. For this reason, a smaller area of interest should be used and depth settings kept at the lowest possible level that allows visualization of the structure in question. When high-velocity blood flows are analyzed, set the color scale at the maximum allowed for that given depth. Color Doppler gain should be set just below the threshold for noise.

2.5.5 Recommendations relating to specific clinical uses flow measurements and PW Doppler technique:

Flow is derived as the product of CSA and the average velocity of the blood cells passing through the blood vessel or valve orifice during the flow period (Figure2.12), whereas stroke volume (SV) represents the product of CSA and VTI.

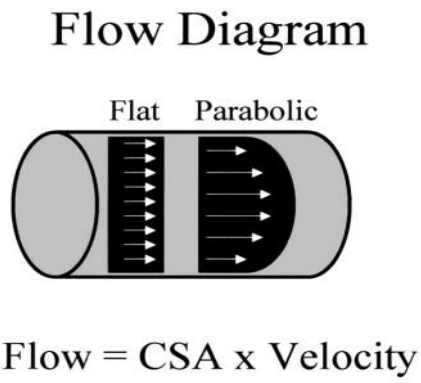

Fig (2.15): Diagrammatic illustration of flow through a vessel showing tow different flow profiles. Flat profile with all cells traveling at same velocity and parabolic profile with cells at center traveling faster than those on the side. At any given time, flow through the vessel represents product of average velocity of all cells multiplied by CSA of the vessel.

When PW Doppler is used, the velocities recorded within the sample volume will be affected by the flow profile. With current instrumentation, assessing flow profile or measuring the average velocity of the blood cells is difficult. Consequently, volume-flow measurements are most accurate when flow is laminar (i.e., all blood cells are moving in the same direction) and the profile is flat. The most important technical factor to ensure accuracy of measurements is to properly match the site of velocity recording with the anatomic measurement of the CSA. (WA Zoghbi-1986). For this reason, it is preferable to use sites where the CSA does not change significantly during the flow period and can be determined accurately from the 2D image and where the flow profile is likely to be

flat. When tracing the velocity to derive a VTI, it is best to trace the outer edge of the most dense (or brightest) portion of the spectral tracing (ie, the modal velocity) and ignore the dispersion that occurs near peak velocity. For patients in sinus rhythm, data from 3 to 5 cardiac cycles may be averaged. However, in patients with irregular rhythms such as atrial fibrillation, 5 to 10 cycles may be required to ensure accuracy of results.

The preferred sites for determining SV and cardiac output (in descending order of preference) are as follows:

- The LVOT tract or aortic annulus
- The mitral annulus
- The pulmonic annulus

The LVOT is the most widely used site (JF Lewis-1984).

SV is derived as (4):SV = CSA × VTI The CSA of the aortic annulus is circular, with little variability during systole. Because the area of a circle = πr^2, the area of the aortic annulus is derived from the annulus diameter (D) measured in the parasternal long-axis view as (5): CSA = $D^2 \times \pi/4 = D^2 \times 0.785$ Image the LV outflow with the expanded or zoom option and place 1 or 2 beats in a cine loop.

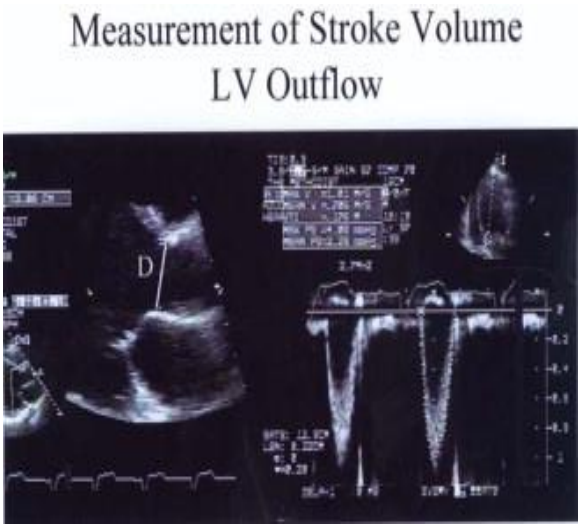

Fig (2.16): Method used in determining systolic flow volume through left ventricular outflow.

This imaging allows a more precise measurement of the annulus diameter during early systole from the junction of the aortic leaflets with the septal endocardium, to the junction of the leaflet with the mitral valve posteriorly, using inner edge to inner edge. The largest of 3 to 5 measurements should be taken because the inherent error of the tomographic plane is to underestimate the annulus diameter. When serial measurements of SV and CO are being performed, use the baseline annulus measurement for the repeated studies because little change in annulus size occurs in adults over time.

The LV outflow velocity is recorded from the apical 5-chamber or long-axis view, with the sample volume positioned about 5 mm proximal to the aortic valve (Figure2.13). The opening click of the aortic valve or spectral broadening of the signal should not be viewed in mid-systole because this means the sample volume is into the region of proximal acceleration. The closing click of the aortic valve is often seen when the sample volume is correctly positioned.

The LVOT method should not be applied when the landmarks needed to measure the annulus diameter cannot be properly visualized or if evidence of LV outflow obstruction exists because the velocities recorded will not be matched to the CSA of the aortic annulus.

Flow across the mitral annulus is measured in the apical 4-chamber view with equation 4 (Figure 2.14)

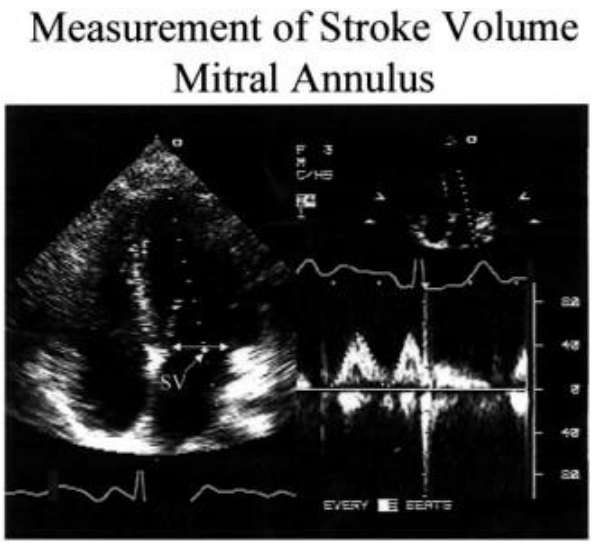

Fig. (2.17): Method used in determining diastolic flow volume through mitral annulus.

Although the mitral annulus is not perfectly circular, applying a circular geometry (equation 5) gives similar or better results than attempting to derive an elliptical CSA with measurements taken from multiple views. The diameter of the mitral annulus should be measured from the base of the posterior and anterior leaflets during early to mid-diastole, one frame after the leaflets begin to close after its initial opening. The sample volume is positioned so that in diastole it is at the level of the annulus. (JF Lewis-1984).

The pulmonic annulus is probably the most difficult of the three sites, mostly because the poor visualization of the annulus diameter limits its accuracy and the right ventricular

(RV) outflow tract contracts during systole. Measure the annulus during early ejection (2 to 3 frames after the R wave on the electrocardiogram) from the anterior corner to the junction of the posterior pulmonic leaflet with the aortic root Figure(2.14). (S Nakatani-1988).

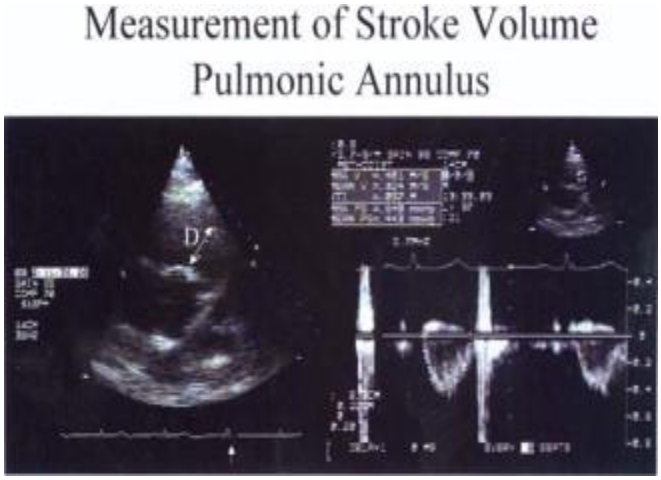

Fig. (2.18): Method used in determining systolic flow volume through pulmonic annulus.

Equations 4 and 5 are used to derive SV and CSA, respectively.

When learning to measure flow volumes across the above sites, make measurements in all three sites in patients without regurgitant lesions or intracardiac shunts because the flow through these sites should be equal. Doing this will develop expertise needed to apply these methods accurately. In regurgitant valve lesions, the forward flow through the regurgitant valve is greater than through the other valves, and the difference between them is equivalent to the regurgitant flow. Regurgitant fraction, an index of severity of regurgitation, is derived as regurgitant flow (in milliliters) divided by the forward flow across the regurgitant valve. In the presence of significant left-to-right intracardiac shunts, flow measurements can calculate the pulmonic to systemic (Qp: Qs) flow ratio. For example, in a patient with an atrial-septal defect, pulmonic flow will be much higher

than aortic flow; the ratio of the two is equivalent to the Qp: Qs ratio. In the best of hands, these calculations may have up to a 20% error.

2.6 Literature review:

2.6.1 Definition and classification of heart failure:

Heart failure is a syndrome with symptoms and signs caused by cardiac dysfunction, resulting in reduced longevity. To establish a diagnosis of heart failure, the European Society of Cardiology guidelines warrant the presence of symptoms and signs, objective evidence of cardiac dysfunction (preferably by echocardiography), and, in case of remaining doubt, a favorable response to treatment directed towards heart failure (Hogg et al., 2004).

World Health Organization definition of heart failure has two components: a clinical and a pathophysiological definition. The pathophysiological definition of heart failure states that "cardiac failure is an inability of the heart to deliver blood (and therefore oxygen) at a rate commensurate with the requirements of the metabolizing tissues at rest or during light exercise. This leads to characteristic systemic pathophysiological responses (neural, hormonal, renal and others), symptoms and signs (Strobeck et al., 1985).

2.6.2 Acute versus chronic heart failure:

Heart failure generally is a chronic condition (chronic heart failure-CHF) in which bouts of worsening symptoms and signs can occur that may require hospitalization or more frequent doctor visits (decompensation of CHF). Alternatively, heart failure may present acutely, with occurrence of severe symptoms and signs within 24 h. Acute heart failure clinically presents in several forms:

- acute pulmonary edema secondary to cardiac dysfunction.
- cardiogenic shock, usually in the setting of an acute coronary syndrome, characterized by hypotension, oliguria, and peripheral vasoconstriction.
- acute worsening (decompensation) of CHF.
- Systolic versus diastolic heart failure, impaired versus preserved left ventricular ejection fraction.

Mc Donough T A et al., 1997 found that heart failure traditionally was seen to result from impairment in ability of the heart to pump sufficient amounts of blood into the circulation during systole—that is, left ventricular systolic dysfunction. Echocardiography is most often employed to assess left ventricular systolic function, an ejection fraction of ⩽40% indicating impaired left ventricular systolic function. Heart failure can also occur in patients with normal left ventricular systolic function in whom higher filling pressures are needed to obtain a normal end-diastolic volume of the left ventricle, so called heart failure with preserved left ventricular ejection fraction (PLVEF) or "diastolic" heart failure.

The occurrence of heart failure with PLVEF has been documented in numerous population-based studies, (Owan et al., 2005) as well as in studies of patients presenting to the hospital with acute pulmonary edema (Redfield et al., 2003). Heart failure with PLVEF is more common in women, at a higher age, and in persons with longstanding hypertension, and carries a better prognosis than heart failure caused by impaired left ventricular systolic function.

Wang T Jet al., 2003 in a recent hospital-based study among 2802 patients admitted with heart failure in Ontario, Canada, reported similar one-year mortality rates in

patients with preserved ejection fraction (>50%) and those with an ejection fraction <40%: 22% versus 26% (p=0.07).

2.6.3 Asymptomatic versus symptomatic left ventricular dysfunction:

Population-based echocardiographic studies have demonstrated that more than 50% of participants with left ventricular systolic dysfunction (generally defined as LVEF <35–40%) have no symptoms or signs of heart failure. Asymptomatic left ventricular systolic dysfunction is found more frequently in person with coronary artery disease (relative risk (RR) 12.5, 95% confidence interval (CI) 4.5 to 33.3), hypertension (RR 3.5, 95% CI 1.4 to 8.5) or an abnormal ECG (RR 7.1, 95% CI 2.8 to 16.7) (Owan T E et al., 2006).

2.6.4 NYHA classification versus AHA/ACC staging:

The New York Heart Association (NYHA) classification has traditionally been used to classify severity of heart failure and indicate prognosis and, thus, to guide patient management. The severity of heart failure ranges from essentially asymptomatic—well treated patients in whom symptoms have been relieved (NYHA I)—to mild (NYHA II, slight limitation in physical activity), to moderate (NYHA III, symptoms while walking on the flat), to severe heart failure (NYHA IV, breathless at rest and essentially housebound). An NYHA III class patient may improve to class II upon initiation of treatment, indicating that the NYHA classification is essentially a functional/symptomatic score, not taking into account the underlying cardiac disorder that will almost inevitably progress.

Levy D. et al., 1996 said that the staged American College of Cardiology/American Heart Association (ACC/AHA) heart failure classification acknowledges that heart failure is

largely preventable (by control of blood pressure and other risk factors), is generally preceded by asymptomatic structural and functional cardiovascular abnormalities, and when present generally progresses.

2.6.5 Occurrence of heart failure:

2.6.5.1 Heart failure in the real world:

Since the review by Cowie et al, which provides an extensive overview of studies published until 1995, numerous papers have been published addressing aspects of the heart failure epidemic from patients in population based cohorts to the highly selected groups of participants in clinical trials. The estimates vary considerably owing to a lack of uniformity in the definition and assessment of heart failure, that can be attributed to the absence of a gold standard for heart failure. MacIntyre K, et al.,2000 demonstrated that non-cardiac conditions (chronic obstructive pulmonary disease (COPD), obesity or a poor physical condition) may mimic heart failure. If heart failure is deemed present on clinical grounds (medical history, signs and symptoms) heart failure with PLVEF (diastolic heart failure) is hard to tell apart from heart failure caused by impaired LVEF without the use of Doppler echocardiography or invasive measurements, although some features may point to heart failure with PLVEF.

2.6.6 Epidemiology of heart failure:

Generally speaking, the prevalence of heart failure can be estimated at 1–2% in the western world and the incidence approaches 5–10 per 1000 persons per year. Estimates of the occurrence of heart failure in the developing world are largely absent. (Suskin N, et al.,2001).

Prevalence: The number of persons having heart failure at a certain moment in time (generally expressed as a percentage).

Incidence: The number of persons newly diagnosed with heart failure during a certain time period (generally expressed as the number of new cases of heart failure per 1000 persons per year—the incidence rate—and sometimes expressed as a proportion of those free from the disease that develops the disease within a specified time period—for example, the five-year cumulative incidence).

2.6.7 Prevalence of heart failure:

Persons younger than 50 years are hardly ever found to have heart failure, but in those older than 50 years the prevalence and incidence increase progressively with age. In a recent US population-based study the prevalence of heart failure was 2.2% (95 CI 1.6% to 2.8%), increasing from 0.7% in persons aged 45 through 54 years to 8.4% for those aged 75 years or older.8 Congestive heart failure was found to be more common in women. These figures are highly comparable to previous studies, such as the Rotterdam study (prevalence of heart failure being 1% in age group 55–64 years, 3% in age group 65–74 years, 7% in age group 75–84 years, and over 10% in those aged ⩾85 years), (Owan T Eet al.,2006).

The first prevalence study to use two-dimensional echocardiography in the population at large took place in Glasgow (1647 participants, 48% men, mean age 50 years), deeming heart failure present if LVEF was <30% in persons having cardiac shortness of breath or using loop diuretics. The prevalence of heart failure was 1.5% (1.6% in men, 1.4% in women). Interestingly asymptomatic left ventricular systolic dysfunction was found to occur very frequently (overall 1.4%, men 2.4%, women 0.5%).

The Rotterdam study (using M-mode echocardiography in a subgroup of 2267 participants, aged 55 years or older) also found left ventricular systolic dysfunction to occur more frequently in men than in women (5.5% vs 2.2%), (Owan T E, Hodge D O, Herges R M. et al.,2006).

Subsequent studies have confirmed these earlier data and confirmed that many persons (more than half) with impaired left ventricular systolic function have no symptoms or signs of heart failure at all.(Redfield M, Jacobsen S J, Burnett J C., Jr *et al* ., 2003).

Roger V L, Weston S A, Redfield M. *et al.*, 2004 Said that only a few epidemiological studies have used echocardiography to investigate diastolic dysfunction specifically, other studies defining diastolic heart failure as symptoms and signs of heart failure in the presence of a normal left ventricular ejection fraction (that is heart failure with preserved left ventricular systolic function).

The MONICA Augsburg study (1274 persons, aged 25 to 75 years) found a high prevalence of diastolic function abnormalities, assessed by Doppler echocardiography, increasing from 2.8% in persons aged 25–35 years to 15.8% in those over 65 years. Interestingly, in participants without left ventricular hypertrophy, hypertension, coronary artery disease, obesity or diabetes, diastolic function abnormalities were largely absent.

2.6.8 Incidence of heart failure:

Reliable estimates of the incidence of heart failure are available from the Rotterdam and Hillingdon heart failure studies. Both studies are population based and used an expert panel to establish the presence or absence of heart failure. In the Hillingdon study the

incidence of heart failure increased from 0.2/1000 person years in those aged 45–55 years to 12.4/1000 person years in those aged >85 years.

In Rotterdam the incidence increased from 2.5/1000 person years (age 55–64 years) to 44/1000 person years (>85 years or older).

Heart failure occurs more frequently in men than in women (15 and 12 per 1000 person years, respectively). (Levy W C, Mozaffarian D, Linker D T. *et al.,2006*).

2.6.9 Hospitalisations for heart failure:

The number of hospitalisations for heart failure (usually expressed as a number per 1000 patients per year) and the change in this measure provide useful information on the epidemic of heart failure. It should be emphasised, however, that hospitalisation rates result from a complex interaction of multiple determinants, including the prevalence, incidence and survival of the disease, referral patterns and treatment possibilities in primary care as well as discharge diagnosis coding practices. Consequently, time trends in hospitalisation rates are often difficult to interpret.

Age adjusted hospitalisation rates increased considerably throughout the western world in the 1980s and early 1990s, as documented by reports from New Zealand, the USA, Sweden, Scotland, and the Netherlands. (Cowie M R, Mosterd A, Wood D A. *et al.*,1997).

Recent reports have suggested that admission numbers have peaked in the 1990s (at least in Scotland, the Netherlands and Sweden) and that the prognosis of hospitalised heart failure patients has improved. (MacIntyre K, Capewell S, Stewart S. *et al.,2000*).

In the Netherlands the total number of hospitalisations for heart failure increased by 72% between 1980 and 1999. Part of this increase is explained by the ageing of the population, the mean age of patients admitted with heart failure increasing from 71.2 to 72.9 years in men and from 75.0 to 77.7 years in women during the same period. After reaching a peak in 1992 (men) and 1993 (women), age adjusted hospitalisation rates for heart failure started to decline by 1.0–1.5% per year. Concomitantly, the average duration of hospitalisation declined from 21.1 days in 1980 to 12.9 days in 1999. In-hospital mortality declined from 18.6% to 13.5%.

2.6.10 Aetiology and risk factors for heart failure:

2.6.10.1 Aetiology of heart failure:

Although many conditions can cause heart failure (coronary artery disease, hypertension, cardiomyopathies, valvular and congenital heart disease, arrhythmias, pericardial disease, myocarditis, pulmonary hypertension, and cardiotoxic substances—including alcohol), the predominant cause of heart failure in the western world is ischaemic heart disease. Fox K F, Cowie M R, Wood D A. *et al.,2001*.

The variation in frequencies of causes of heart failure reported in different studies can be explained by differences in study population, from the highly selected group of participants in clinical trials to relatively unselected participants in population-based studies, differences in definitions, and time differences (the Framingham heart study originated in 1948). In addition, it has become clear that using non-invasive methods the precise aetiology of heart failure cannot always be determined accurately. In the Bromley heart failure study the percentage of heart failure with unknown cause declined from 42% to 10% after nuclear testing and cardiac catheterization, while the percentage of patients

with ischemic heart failure increased from 29% to 52%. Fox K F, Cowie M R, Wood D A. *et al.,2001.*

Risk factors for heart failure:

Various (population-based) studies have addressed risk factors for the occurrence of heart failure. Kenchaiah S, Narula J, Vasan R S.2004.Coronary artery disease notably increases the chance of developing heart failure; in 7–8 years after myocardial infarction up to 36% of patients will experience heart failure, especially those with left ventricular systolic dysfunction documented during admission. Hellermann J P, Goraya T Y, Jacobsen S J. *et al.,2003*

Although the risk of heart failure associated with hypertension (systolic blood pressure >140 mm Hg, or diastolic blood pressure >90 mm Hg, or treatment with antihypertensive medication) is smaller than that associated with myocardial infarction, hypertension contributes considerably to the population burden of heart failure as it occurs more frequently than myocardial infarction. Levy D, Larson M G, Vasan R S. *et al.,1996.*

Obesity (body mass index >30 kg/m2), increasingly present in western societies, doubles the risk of heart failure after adjustment for associated risk factors. Kenchaiah S, Evans J C, Levy D. *et al.,2003.*

Valvular abnormalities, factors indicative of heart disease (left ventricular hypertrophy, left ventricular dilatation), a parental history of heart failure, conventional risk factors (such as smoking, diabetes, obesity), as well extra cardiac conditions (renal dysfunction, obstructive pulmonary disease) all increase the risk of heart failure. Kenchaiah S, Narula J, Vasan R S.,2004.

The relation of obesity, increased cholesterol values and hypertension to cardiovascular morbidity and mortality is undisputed.

In patients with CHF considerable evidence exists that obesity, hypercholesterolemia and hypertension are “protective”. This phenomenon has been termed “reverse epidemiology” for which a comprehensive explanation is still lacking.20 Proposed explanations are the syndrome of cardiac cachexia, reverse causation (the underlying cause of hypotension—that is, pump failure—being detrimental rather than hypotension per se) and time discrepancies among competitive risk factors. The reverse epidemiology does not hold for all conventional risk factors, as quitting smoking is associated with a better prognosis in heart failure patients.Suskin N, Sheth T, Negassa A. et al .,2001.

2.6.10.2 Comorbidity in heart failure

Heart failure, being a common disease in the elderly, should not be viewed in isolation: anemia, cachexia, renal impairment, obstructive sleep apnoea, chronic pulmonary disease and diabetes mellitus are conditions frequently observed in heart failure patients and unfavorably affect prognosis. McMurray J, Pfeffer M A. Heart failure- 2005.

Comorbidity was found to be one of the prime determinants of prognosis in a study of patients admitted with heart failure and contributes to the poor quality of life as perceived by heart failure patients.

The complex interplay between diseases previously perceived as entities on their own, such as COPD, renal disease, obstructive sleep apnoea on the one hand and heart failure on the other, is increasingly acknowledged.

Patients with chronic kidney disease constitute a group of patients at high risk for having or developing cardiovascular disease, including heart failure.

Heart failure in these patients may result from coronary heart disease, longstanding hypertension resulting in concentric hypertrophy, or volume overload due to anemia, fluid overload and arteriovenous fistulas leading to left ventricular dilatation. The prevalence of heart failure increases with severity of renal impairment; approximately 20% of patients with a glomerular filtration rate <30 ml/min/1.73 m2 (not on dialysis) have heart failure.

Along similar lines, heart failure appears to be frequently present in patients with COPD; in a recent Dutch study of 405 patients older than 65 years with a diagnosis of COPD, 20.5% were found to have previously unrecognised heart failure. Rutten F H, Cramer M J, Grobbee D E. et al.,1894.

2.6.11 Prognosis of heart failure:

There is no doubt that the prognosis of heart failure patients remains poor, even in the realm of the development of a myriad of effective pharmacological and non-pharmacological interventions. This is illustrated by the title of a paper on the prognosis of the syndrome: “More malignant than cancer”.

Any doctor treating heart failure patients will confirm that life expectancy in heart failure patients is “reduced” and that sudden cardiac death is a “major” cause of death, that (acute) worsening of CHF occurs “quite often”, leading to “frequent” hospitalizations, and that quality of life in these patients is “impaired considerably”. We included the

quotation marks in the latter sentence to indicate the implicit nature of prognostication in clinical practice.

2.6.12 Mortality and causes of death:

The best impression of the prognosis of the "average" heart failure patient is obtained in population based research, in which *incident* cases of established heart failure are followed up carefully.

Such studies have been carried out in the USA, UK, the Netherlands and Switzerland. Studies including incident cases admitted to hospital show that the mortality rate is relatively high in the first few weeks after the occurrence of heart failure, while after that period the slope of the survival curve shows a much more gradual slope. The 30-day mortality is reported to lie around 10–20%.

Both in the US (Framingham heart study) and in England (Hillingdon heart failure study) 1-year survival following the onset of heart failure was 70%. Five years after the occurrence of heart failure only 35% of Framingham participants were still alive.

Survival rates in a study on prevalent cases of heart failure from the Netherlands (Rotterdam study) were more favorable: 1, 2 and 5-year survival rates of 89%, 79% and 59%, respectively, reflecting a threefold increase in the risk of death compared to the age and gender matched population at large. The 1-year survival rate in the study from Switzerland was 77%. A more recent report from the Rotterdam study including cases of *incident* heart failure, and allowing for inclusion of patients hospitalized for heart failure as incident cases, produced lower 1, 2 and 5- year survival rates of 63%, 51% and 35%, respectively. The importance of the disease spectrum is further illustrated by the

comparison with survival rates from hospitalized-based studies. In contrast, survival rates from (placebo-treated) participants from the large randomised trials tend to underestimate mortality, since participants in these trials are usually not representative of the "average" heart failure patient.

2.6.13 Prevention:

2.6.13.1 Lifetime risk of heart failure:

The incidence figures from the Framingham heart study and Rotterdam study have been used to provide estimates of the life time risk of developing heart failure. The overall chance that a 40-year-old person develops heart failure during the rest of his/her life is 21%. In hypertensive persons (systolic blood pressure >160 mm Hg and/or diastolic blood pressure >100 mm Hg) this chance is appreciably higher (28%) than in normotensive persons; they have a lifetime heart failure risk of 13%. Lloyd-Jones D M, Larson M G, Leip E P. *et al*.,2003.

2.6.13.2 Prevention of heart failure:

As coronary artery disease and hypertension are the predominant causes of heart failure, prevention of the onset of hypertension and coronary artery disease is key to reducing the burden of heart failure.

Given the high prevalence of hypertension in western societies, the impact of antihypertensive treatment may well be larger than that of adequate treatment of acute coronary syndromes. Levy D, Larson M G, Vasan R S. *et al.,1996.*

Chapter Three
Materials and Methods

3.1 Materials:

3.1.1 Subject:

This was cross-sectional study of 180 Sudanese patients with heart failure sign were enrolled. The study was conducted at Khartoum state in Echocardiography departments of Omdurman Military Hospital and Sudan Heart Center. The exclusion criteria were the patients with age less than 20 years. All those patients were under went echocardiography study to identify the common change in heart blood flow

3.1.2 Area and duration:

The study was conducted at Khartoum state in Echocardiography departments of Omdurman Military Hospital and Sudan Heart Center during November 2014 to August 2017.

3.1.3 Machine used:

Echocardiograms were recorded using ultrasound machine HDI 4000 scanner (Philips Medical Systems) equipped with a commercially available 3-13MHz linear transducer with color and power Doppler capability and GE medical system LOQIC5Expert, made by Yocogama medical systems. LTD–JAPAN – model 2302650, serial number 1028924, manufactured April 2005.

3.1.4 Ethical consideration:

Verbal consent was taken from each patient. It is considered that all information that obtained from the patients was kept as highly security data, and results will not be permitted. The participants were provided with information about the study and any risks that may arise especially when the examination technique was applied.

3.2 Methods:

Echocardiography used the gross anatomy of the heart can be evaluated by two dimensional transthoracic echocardiography (TTE) in para sternal, apical, supra sternal and subcostal position. The standardized planes used are long axis, short axis and four chambers.

3.2.1 Technique used:

TTE by using the long axis view is obtained by placing the ultrasound transducer in the left a picosternal position and provides detailed images of the left ventricle, aorta, left atrium and mitral and aortic valves. Angling the beam towards the right also allows assessment of the right atrium, right ventricle and tricuspid valves. Rotating the transducer by 90 degrees in the clockwise direction produces the short axis view, which allows assessment of the left ventricle, papillary muscle, chordae tendineae and mitral valves. The four-chambers view demonstrates the ventricles, atria and mitral and tricuspid valves. Rotation of the transducer allows two- chambers views of the heart and more detailed assessment of the aorta and aortic valves demonstrated.

The EF and volumes were measured with 2D-biplane Simpson's method, 2D-triplane and 3- dimensional echocardiography (3DE) by two investigators blinded to any clinical data.

By using the protocols of echo which was established by British Society of echocardiography Education Committee which state that for viewing the HF along –axis view is used applying of this view was obtained by put the echo transducer (probe) in the left apicosternal position and provides detailed images of many parts of heart specially the left ventricle, aorta, left atrium and mitral and aortic valves.

3.2.2 Data collection variables

- Age
- Gender
- Weight
- Comorbidity
- EF
- Echo finding
- Heart valves (Mitral. Tricuspid, Aortic, and pulmonary valves)
- LVIDD& LVISD

3.2.3 Data analysis:

Statistical Package for the Social Sciences (SPSS) was used. Categorical data were expressed as percent frequencies, Statistical correlation between continuous variables was tested. All analyses were performed using SPSS version16. Statistical software (SPSS Inc., Chicago, Illinois).

Chapter Four

Results

The following tables and figures presented the results of the study.

Table 4.1: Mean values of the measured parameters in the whole sample Descriptive Statistics.

	N	Minimum	Maximum	Mean	Std. Deviation
Age	180	20	85	60.56	13.169
Weight	180	50	107	80.14	10.666
EF	180	15.00	60.00	38.9444	9.55766
LVDD	180	36	89	56.52	9.538
LVSD	180	23	80	42.68	10.410

Table 4.2: Frequency distribution of gender among the sample of the study.

Gender

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Male	118	56.6	65.6	65.6
	Female	62	34.4	34.4	100.0
	Total	180	100.0	100.0	

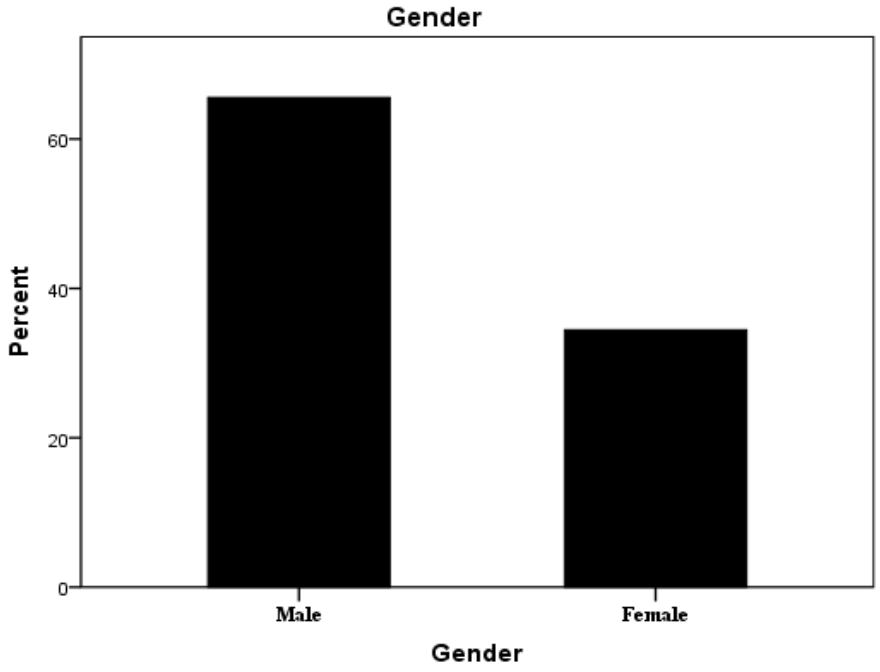

Figure (4.2): Gender distribution.

Table 4.3: Frequency distribution of comorbidity among the sample of the study.

Comorbidity

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Hypertension	65	36.1	36.1	36.1
	Diabetes	7	3.9	3.9	40.0
	I.H.D	39	21.7	21.7	61.7
	R.H.D	6	3.3	3.3	65.0
	D.C.M	2	1.1	1.1	66.1
	Other	7	3.9	3.9	70.0
	All	54	30.0	30.0	100.0
	Total	180	100.0	100.0	

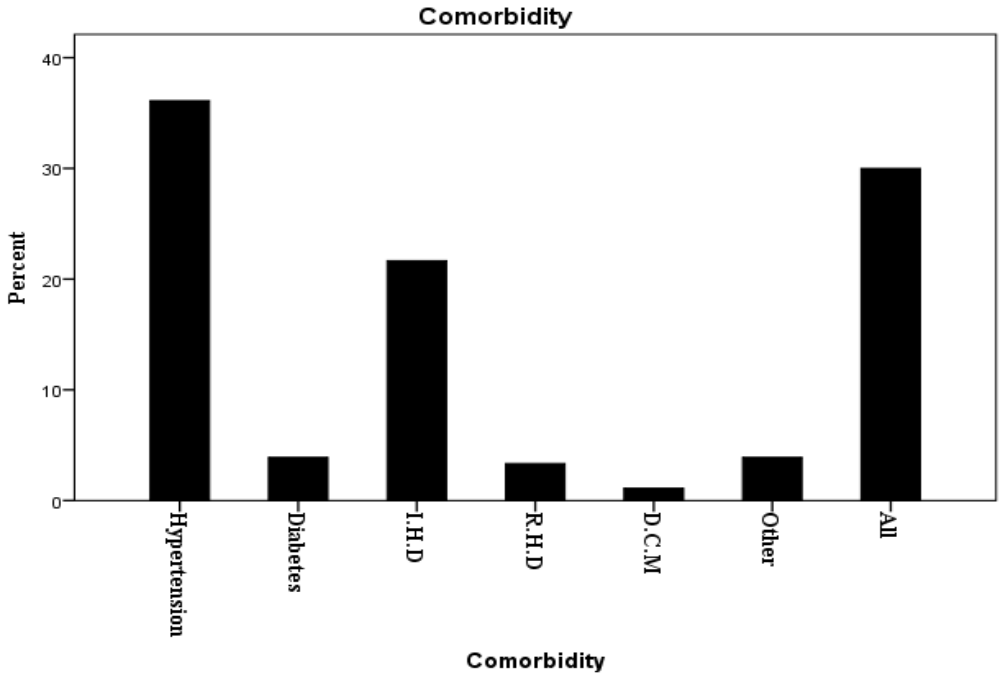

Figure (4.3): Comorbidity distributions.

Table 4.4: Frequency distribution of echo findings among the sample of the study.

Echo Findings

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Mild LVSD	40	22.2	22.2	22.2
	Moderate LVSD	48	26.7	26.7	48.9
	Severe LVSD	33	18.3	18.3	67.2
	DCM	9	5.0	5.0	72.2
	IHD	8	4.4	4.4	76.7
	CCF	5	2.8	2.8	79.4
	Mild LVDD	14	7.8	7.8	87.2
	LVH	6	3.3	3.3	90.6
	RVD	1	.6	.6	91.1
	Moderate LVDD	4	2.2	2.2	93.3
	Severe LVDD	3	1.7	1.7	95.0
	Other	4	2.2	2.2	97.2
	RVDD	3	1.7	1.7	98.9
	14	2	1.1	1.1	100.0
	Total	180	100.0	100.0	

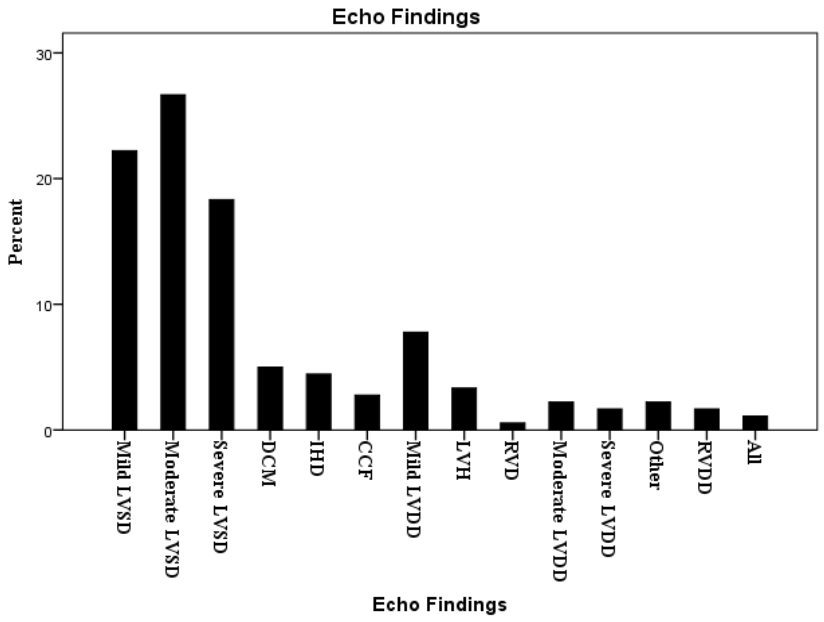

Figure (4.4): Echo findings distribution

Table 4.5: Frequency distributions of echo findings of mitral valve among the sample of study

MV

		Frequency	Percent	Valid Percent	Cumulative Percent
	Normal	28	15.6	15.6	15.6
	Trivial	25	13.9	13.9	29.4
	Mild	69	38.3	38.3	67.8
	Moderate	29	16.1	16.1	83.9
	Severe	23	12.8	12.8	96.7
	Mild Stenosis	3	1.7	1.7	98.3
	Moderate Stenosis	3	1.7	1.7	100.0
	Total	180	100.0	100.0	

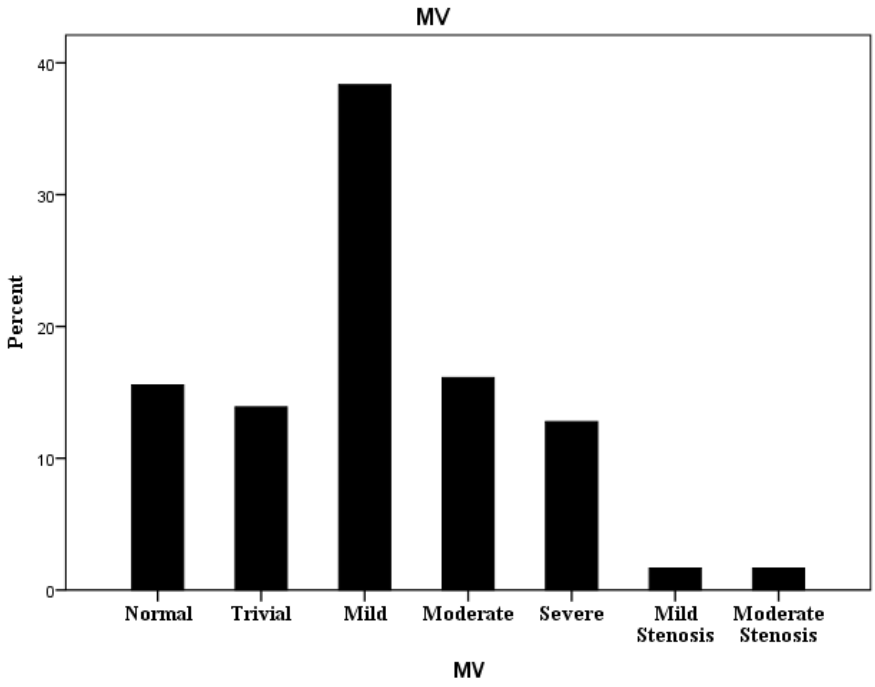

Figure 4.5: Mitral valve findings distributions

Table 4.6: Frequency distributions of echo findings of tricuspid valve among the sample of study.

TRV

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	62	34.4	34.4	34.4
	Trivial	36	20.0	20.0	54.4
	Mild	55	30.6	30.6	85.0
	Moderate	10	5.6	5.6	90.6
	Severe	17	9.4	9.4	100.0
	Total	180	100.0	100.0	

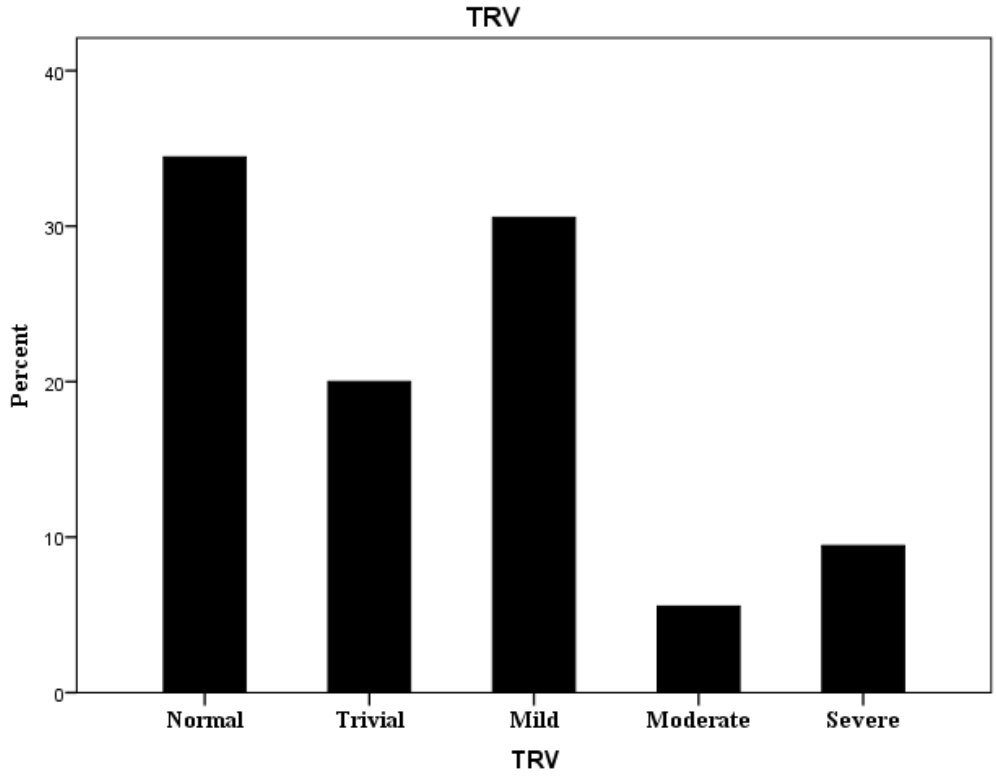

Figure 4.6: Tricuspid valve findings distributions

Table 4.7: Frequency distributions of echo findings of Aortic valve among the sample of study.

AV

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	113	62.8	62.8	62.8
	Trivial	26	14.4	14.4	77.2
	Mild	28	15.6	15.6	92.8
	Moderate	7	3.9	3.9	96.7
	Severe	1	.6	.6	97.2
	Mild Stenosis	2	1.1	1.1	98.3
	Severe Stenosis	3	1.7	1.7	100.0
	Total	180	100.0	100.0	

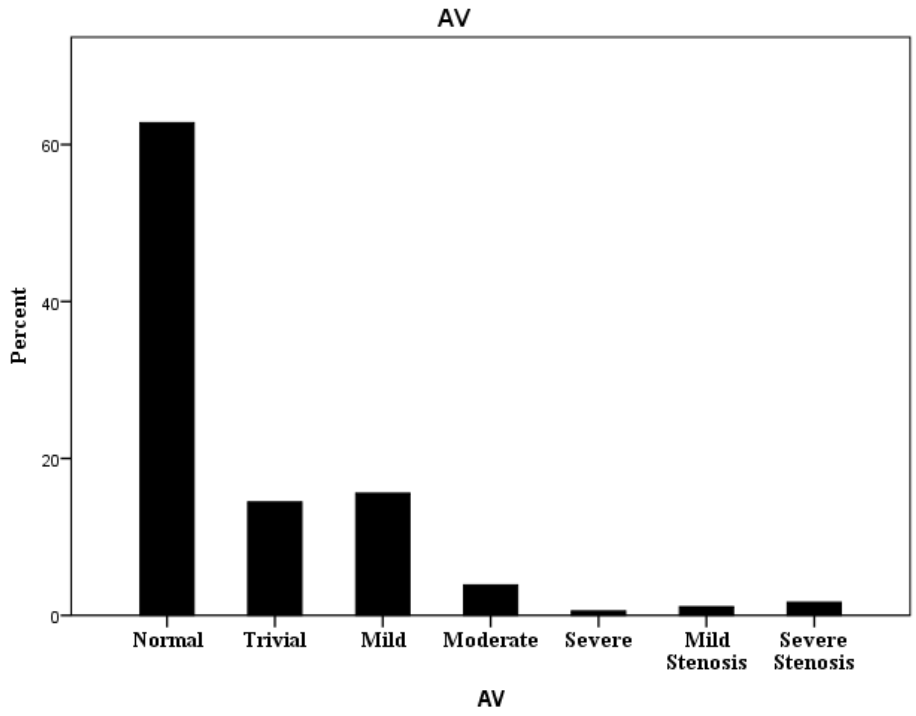

Figure 4.7: Aortic valve findings distributions

Table 4.8: Frequency distributions of echo findings of pulmonary valve among the samples of study.

PV

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	91	50.6	50.6	50.6
	Trivial	9	5.0	5.0	55.6
	Mild	4	2.2	2.2	57.8
	Mild PHT	28	15.6	15.6	73.3
	Moderate PHT	17	9.4	9.4	82.8
	Sever PHT	31	17.2	17.2	100.0
	Total	180	100.0	100.0	

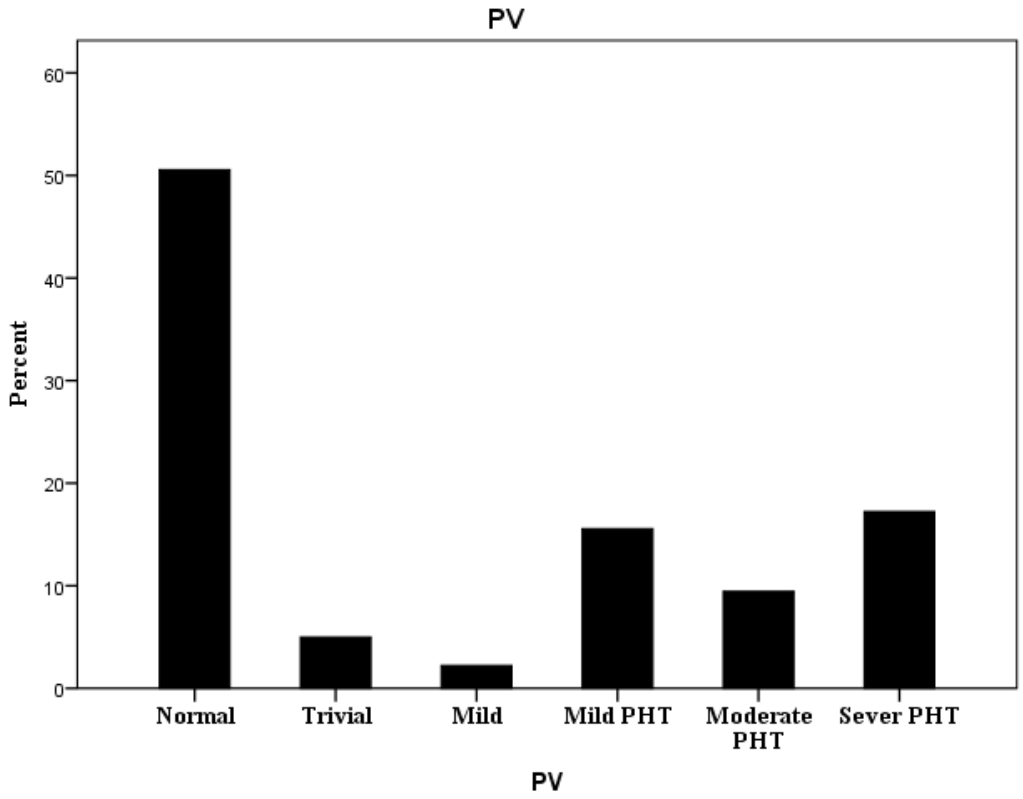

Figure 4.8: Pulmonary valve findings distributions

Table 4.9: Correlation between Mitral valve and Ejection fraction

	MV	N	Mean	Std. Deviation	Std. Error Mean
EF	Trivial	25	40.6000	9.13783	1.82757
	Mild	69	39.4493	9.04976	1.08946
	Moderate	29	34.0000	10.80344	2.00615
	Severe	23	33.6957	8.42310	1.75634

Table 4.10: Cross tabulation of echo findings with mitral valve.

Echo Findings * MV Crosstabulation

			MV							Total
			Normal	Trivial	Mild	Moderate	Severe	Mild Stenosis	Moderate Stenosis	
Echo Findings	Mild LVSD	Count	8	9	17	3	2	1	0	40
		% within Echo Findings	20.0%	22.5%	42.5%	7.5%	5.0%	2.5%	.0%	100.0%
	Moderate LVSD	Count	6	7	18	8	6	0	3	48
		% within Echo Findings	12.5%	14.6%	37.5%	16.7%	12.5%	.0%	6.3%	100.0%
	Severe LVSD	Count	2	4	12	9	6	0	0	33
		% within Echo Findings	6.1%	12.1%	36.4%	27.3%	18.2%	.0%	.0%	100.0%
	DCM	Count	1	1	2	2	3	0	0	9
		% within Echo Findings	11.1%	11.1%	22.2%	22.2%	33.3%	.0%	.0%	100.0%
	IHD	Count	1	0	6	0	1	0	0	8
		% within Echo Findings	12.5%	.0%	75.0%	.0%	12.5%	.0%	.0%	100.0%

	CCF	Count	0	0	3	1	1	0	0	5
		% within Echo Findings	.0%	.0%	60.0%	20.0%	20.0%	.0%	.0%	100.0%
	Mild LVDD	Count	5	3	4	1	1	0	0	14
		% within Echo Findings	35.7%	21.4%	28.6%	7.1%	7.1%	.0%	.0%	100.0%
	LVH	Count	1	0	1	1	2	1	0	6
		% within Echo Findings	16.7%	.0%	16.7%	16.7%	33.3%	16.7%	.0%	100.0%
	RVD	Count	1	0	0	0	0	0	0	1
		% within Echo Findings	100.0%	.0%	.0%	.0%	.0%	.0%	.0%	100.0%
	Moderate LVDD	Count	1	0	0	2	1	0	0	4
		% within Echo Findings	25.0%	.0%	.0%	50.0%	25.0%	.0%	.0%	100.0%
	Severe LVDD	Count	0	0	2	1	0	0	0	3
		% within Echo Findings	.0%	.0%	66.7%	33.3%	.0%	.0%	.0%	100.0%
	Other	Count	0	1	1	1	0	1	0	4
		% within Echo Findings	.0%	25.0%	25.0%	25.0%	.0%	25.0%	.0%	100.0%
	RVDD	Count	2	0	1	0	0	0	0	3
		% within Echo Findings	66.7%	.0%	33.3%	.0%	.0%	.0%	.0%	100.0%
	All	Count	0	0	2	0	0	0	0	2
		% within Echo Findings	.0%	.0%	100.0 %	.0%	.0%	.0%	.0%	100.0%
Total		Count	28	25	69	29	23	3	3	180
		% within Echo Findings	15.6%	13.9%	38.3%	16.1%	12.8%	1.7%	1.7%	100.0%

Table 4.11: Correlation between EF, LVDD and LVSD

		EF	LVDD	LVSD
EF	Pearson Correlation	1	-.376**	-.481**
	Sig. (2-tailed)		.000	.000
	N	180	180	180
LVDD	Pearson Correlation	-.376**	1	.910**
	Sig. (2-tailed)	.000		.000
	N	180	180	180
LVSD	Pearson Correlation	-.481**	.910**	1
	Sig. (2-tailed)	.000	.000	
	N	180	180	180
**. Correlation is significant at the 0.01 level (2-tailed).				

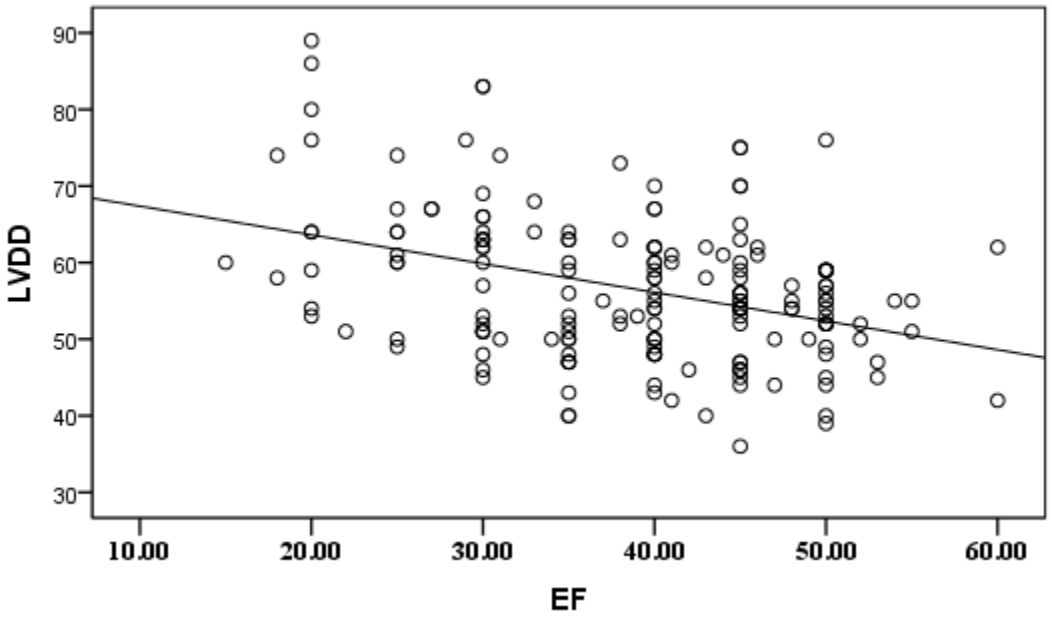

Figure (4.11) shows the correlations between LVIDD and EF

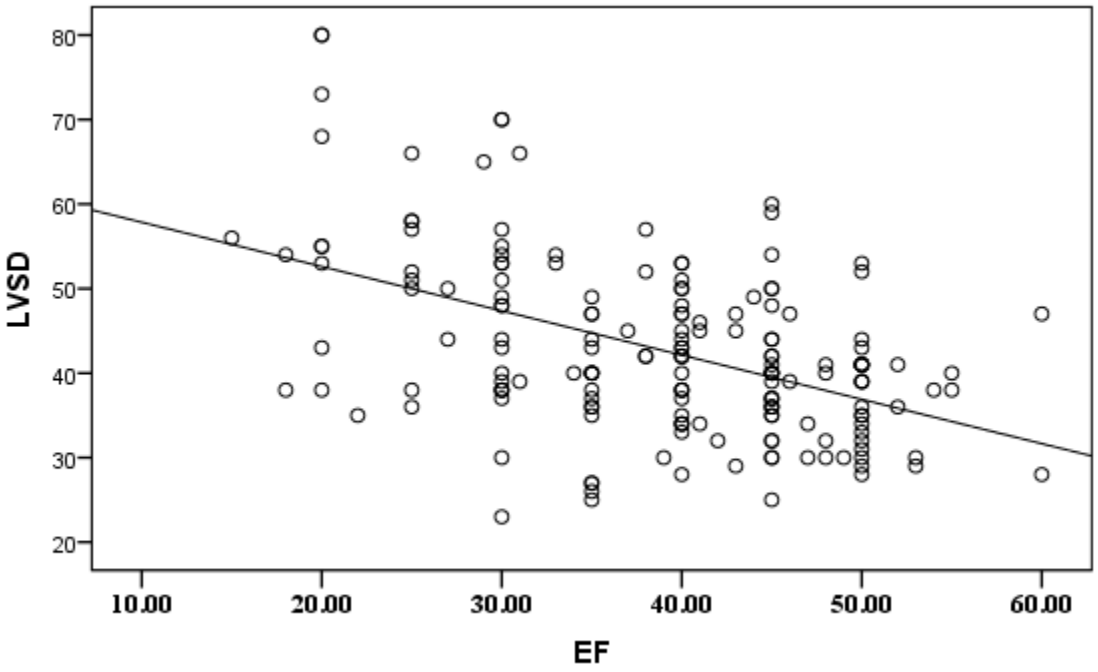

Figure (4.12) shows the correlations between LVISD and EF

Chapter Five

Discussion, Conclusion and Recommendation

5.1 Discussion

The study is conducted on 180 patients with heart failure condition investigated by using echocardiogram, 118 cases (65.6%) were male and 62 cases (34.4%) were female table &figure (4.2), this agrees with study done by (Levy W C, et al) Heart failure occurs more frequently in men than in women (15 and 12 per 1000 person years, respectively). (Levy W C et al.,2006).

The Rotterdam study (using M-mode echocardiography in a subgroup of 2267 participants, aged 55 years or older) also found left ventricular systolic dysfunction to occur more frequently in men than in women (5.5% vs 2.2%). (Owan T E. et al.,2006).

In a recent US population-based study the prevalence of Congestive heart failure was found to be more common in women.

The incidence of the heart failure was high among the age group mean 60.56 and the minimum age was 20 and maximum age was 85 in the sample of study showed in table (4.1) that mean the risk of heart failure increase with age which was same result with comparable to previous studies, such as the Rotterdam study (prevalence of heart failure being 1% in age group 55–64 years, 3% in age group 65–74 years, 7% in age group 75–84 years, and over 10% in those aged ⩾85 years), (Owan T et al.,2006).

In view of the importance Ejection fraction by using 2D echo, the result showed mean EF 38.94(mnimum15 –maximum 60) in table (4.1) the left ventricular ejection fraction volume represent that the maximum was 60% and this was normal value unless the

patient had right side failure or diastolic dysfunction which is called so heart failure with preserved left ventricular ejection fraction (PLVEF) or “diastolic” heart failure. This same result with McDonough et al.,1997 found that Echocardiography is most often employed to assess left ventricular systolic function, an ejection fraction of ⩽40% indicating impaired left ventricular systolic function. Heart failure can also occur in patients with normal left ventricular systolic function in whom higher filling pressures are needed to obtain a normal end-diastolic volume of the left ventricle, so called heart failure with preserved left ventricular ejection fraction (PLVEF) or “diastolic” heart failure.

The occurrence of heart failure with PLVEF has been documented in numerous population-based studies**,** (Owan et al.,2005) as well as in studies of patients presenting to the hospital with acute pulmonary edema. (Redfield M. et al.,2003). Heart failure with PLVEF is more common in women, at a higher age, and in persons with longstanding hypertension, and carries a better prognosis than heart failure caused by impaired left ventricular systolic function. Wang T J. et al.,2003 in a recent hospital-based study among 2802 patients admitted with heart failure in Ontario, Canada, reported similar one-year mortality rates in patients with preserved ejection fraction (>50%) and those with an ejection fraction <40%: 22% versus 26% (p=0.07).

Regarding to comorbidity in table &figure (4.3) the hypertension represent 36.1% followed by 30% of (HTN,DM and IHD) followed by IHD 21.7%that means the hypertension is predominant cause of heart failure in Sudan Although many conditions can cause heart failure (coronary artery disease, hypertension, cardiomyopathies, valvular and congenital heart disease, arrhythmias, pericardial disease, myocarditis, pulmonary

hypertension, and cardiotoxic substances—including alcohol), the predominant cause of heart failure in the western world is ischaemic heart disease **(**Fox K F. et al.,2001).

In table and figure (4.4) the common finding in the study sample was left ventricular systolic dysfunction LVSD which represent about 121 patients 67.7% followed by left ventricular diastolic dysfunction LVDD which represent 21 patients 11.7% followed by dilated cardiomyopathy DCM 5% followed by congestive cardiac failure CCF2.8%.

This result same result with population-based echocardiographic studies have demonstrated that more than 50% of participants with left ventricular systolic dysfunction (generally defined as LVEF <35–40%) have no symptoms or signs of heart failure. Asymptomatic left ventricular systolic dysfunction is found more frequently in person with coronary artery disease (relative risk (RR) 12.5, 95% confidence interval (CI) 4.5 to 33.3), hypertension (RR 3.5, 95% CI 1.4 to 8.5) or an abnormal ECG (RR 7.1, 95% CI 2.8 to 16.7). (Owan et al., 2006***).***

This study found that the correlation is significant at the 0.01 level. between the left ventricular internal diastolic dimeter LVIDD and ejection fraction EF in table (4.11) equal -.376 (negative) inversely proportional.

Also the correlation is significant at the 0.01 level between the left ventricular internal diastolic dimeter LVISD and ejection fraction EF in table 4.11equal -.376 (negative) inversely proportional.

Changes in LV measurements and geometry due to hypertension reflect the dominant underlying hemodynamic alterations associated with blood pressure elevation, Ganau (1992), Matsukubo (1977).

The changes detected by echocardiography showed increase in left ventricular mass which is a consistent feature of hypertension. The justification of those changes is described as structural changes, Berkinst (2001). Heart failure cause changes as cardiac cell hypertrophy but the cardiac myocyte cell number does not increase. In addition, hypertension causes considerable interstitial change and fibroblast proliferation Berkinst (2001). The Framingham study showed the hazard for developing heart failure in hypertensive, thus documenting the importance of assessing left ventricular function in hypertensive heart disease. The conventional way of assessing left ventricular function with echocardiography is via the left ventricular ejection fraction, determined by applying Simpson's method of discs Shahgaldi (2009). Figures (4.11, 4.12) showed the linear Correlation between LVISD, and LVIDD with EF, it showed that as the LV measurements increases the EF decreases.

Similarly, Blendea *et al*., (2007) reported that the alterations in left ventricular long-axis systolic and diastolic function could predict the onset of heart failure. Despite the widespread clinical use of the left ventricular ejection fraction, it should be kept in mind that it is a load-dependent systolic index. From this point of view, it is clearly very important to identify the slightest initial impairment of left ventricular function, using additional indices apart from ejection fraction that are not load-dependent.

Noninvasive assessment of mitral valve filling by echocardiography provides important information about regurgitation and stenosis status in selected subsets of patients.

This study was designed to assess valves for two-dimensional (2D), M-mode and Doppler echocardiographic assessment the blood flow in detection of Changes in Echocardiographic Parameters among heart failure patients.

In table (4.5) the result of mitral valve 69 patients38.3%with mild regurgitation, 29 patients 16.1%with moderate regurgitation,23 patients 12,8% with Sever regurgitation and 3.4%with mitral stenosis that mean the echocardiography is effective in assessment the blood flow in the cardiac valves

In table (4.6) the result of tricuspid valve 62patients 34.4%were normal,36 patients20%with trivial regurgitation,55patients30,6%were mild regurgitation.

In table4.7 the result of aortic valve most of patients were normal 113patients 62.8%.

In table (4.8) the result of pulmonary valve 91 patients 50% were normal, 28 patients15.6% had mild pulmonary hypertension PHT, 17 patients 9.4%moderate PHT and 31patients 17% with sever PHT.

Evaluation of the anatomy of the mitral valve apparatus by 2D echocardiography is critically important in the assessment of severity of mitral regurgitation (MR).

The mitral apparatus includes the leaflets, chordae tendineae, annulus, and the papillary muscles with their supporting left ventricular (LV) walls. Careful evaluation of these structures should be able to define the mechanism of MR and yield clues to its severity. For example, a prominent flail leaflet is usually associated with severe MR. On the other hand, severe MR rarely occurs in the setting of anatomically normal mitral valve and support apparatus. Defining the mechanism of MR may determine whether valve repair is feasible instead of valve replacement. (Stewart WJ, Currie.,1990) (Stewart WJ, Salcedo EE.,1991). In patients with MR in the setting of LV dilatation and/or systolic dysfunction, it is important to determine whether MR is functional (i.e. due to LV dilatation) or primary (i.e. due to an abnormality of the valve apparatus) Table (4.10) showed sever LVSD lead to 36.4% with mild mitral regurgitation,27.3%with moderate mitral regurgitation and 18.2%with sever mitral regurgitation .this functional mitral

regurgitation due to systolic dysfunction .Also patients with LVH (left ventricular hyper atrophy) the severity of regurgitation increase 33.3%. In functional MR, the leaflets are usually tethered by outward displacement of the LV walls and papillary muscles, with or without annular dilation. (Otsuji Y et al., 2008)

Color flow Doppler. Color Doppler flow mapping is widely used to screen for the presence of mitral regurgitation. Importantly, small color flow jets are seen in roughly 38% of study sample table (4.5) and therefore large color flow jets are seen in roughly 12.8% of study sample table (4.5). The incidence of mild regurgitation tends to increase with age. (Yoshida K et al., 1988).

Regurgitant jet area. As a general rule, large jets that extend deep into the LA represent more MR than small thin jets that appear just beyond the mitral leaflets. However, the correlation between jet area and MR severity is poor due to a variety of technical and hemodynamic limitations as noted earlier. (Sahn DJ 1988).

Patients with acute severe MR, in whom blood pressure is low and LA pressure is elevated may have a small eccentric color flow jet area, whereas hypertensive patients with mild MR may have a large jet area. Furthermore, the same regurgitant flow will produce larger or smaller jets depending on the size of the atrium, which has led to indexing for atrial area. (Helmcke F et al., 1987)

The sever regurgitation occurs when the patients had DCM 33,3%, LVH 33,3% followed by moderate LVDD25%and CCF 20%.

The heart is the pump of the circulatory system, so it is reasonable that the increased arterial pressure affects it from the early stages of heart failure and it actually suffers the commonest heart failure related organ damage. Any provoked alterations involving either the anatomy or the functionality of the heart can easily be detected and imaged by

echocardiography, which represents a real-time, quick, reproducible, cheap, and widespread method.

This is why echocardiography is one of the very first examinations that a heart failure patient is recommended to undergo. The echocardiographic assessment of a heart failure patient is performed on two levels: i) an anatomic approach, which includes measurement of the heart cavities (Table 4.2), and ii) a functional approach, which includes assessment of indices of function. Overall, to summarize, a global echocardiographic evaluation of a patient with heart failure should include assessment of the following: a) left ventricular ejection fraction b) left ventricular internal diastolic dimeter c) left ventricular internal systolic diameter d) valvar assessment. Finally, Shortcoming of our study was the limited number of study participants.

5.2 Conclusion

- The incidence of heart failure was high among the age group mean 60,56 years.
- The risk of heart failure increase with age.
- The heart failure common in male than female.
- The HF with preserved left ventricular ejection fraction PLVEF that mean HF with normal ejection fraction.
- The HTN is predominant cause of heart failure in Sudan.
- The common echo finding was LVSD.
- The linear Correlation between LVISD, and LVIDD with EF, it showed that as the LV measurements increases the EF decreases.
- This study attempts to describe the echocardiographic features of Left ventricular among heart failure Sudanese patients. Echocardiography itself could be used for heart disease screening in clinical patients. Echocardiography is a significant tool for the evaluation of heart failure patients.
- Assessing a heart failure patients echocardiographically does not simply represent adherence to a routine examination procedure that has limited clinical value.
- Sever mitral valve regurgitation occur in patients with DCM and LVH.
- This study attempts to simulate the basic clinical echocardiography M-mode& Doppler protocol in heart failure patients.
- Echocardiography with Doppler has become the first line approach to the evaluation and management of valvular heart disease. While 2D echocardiography provides an assessment of valvular structure, mechanism of regurgitation and adaptation to the volume overload state, Doppler allows in the same setting, a comprehensive evaluation of

the severity of regurgitation using qualitative and quantitative methods from Color flow and spectral Doppler.

- Echocardiography is noninvasive assessment of mitral valve filling provides important information about regurgitation and stenosis status and also in assessment of tricuspid, aortic and pulmonary valves.
- Sever PHT is commonest in patients with heart failure.

5.3 Recommendations

From the results of this study the researcher recommended that:

- Echocardiographic investigations should be used as routine for any patient complaining of heart disorders.
- All the Echocardiographic reference parameters that we use to compare during Echocardiographic studies are derived from those defined in the western world. We till date do not have a proper reference range based on studies conducted in Sudan or Africa itself. It is a well-known fact that that the population in Sudan has a very much different genetic and physical make up as compared to the population in the west. The difference in the body size in itself brings into question the reference range that we quote as normal values derived from studies conducted in the west. We have thus made an effort to study a hypertensive population in Sudan, for recording changes of echocardiographic parameters.
- Despite many technical limitations (interobserver variability, low quality imaging in obese patients, obstructive lung disease, etc.)
- This study is small study consisting of randomized populations of people in Khartoum state and the study population cannot be the representation of whole Sudanese population. Relatively the sample size was also small.
- Further studies are recommended to know the relation between internal heart measurements among Sudanese population and other factors such as presence of history and occupation.

References:

Arthur Cgyton, M. D. Text book of medical physiology, Eleventh edition (Igaku-shon/Saunders), 2006,p 150-163.

Berkinst, K. E. and Ball, S. G. 2001. Hypertension essential Hypertension: The Heart and Hypertension Heart; 86:467–475.

Blendea, D., Duncea, C., Bedreaga, M., Crisan, S. and Zarich, S. 2007. Abnormalities of left ventricular long-axis function predict the onset of hypertension independent of blood pressure: a 7-year prospective study. J Hum Hypertens.; 21: 539- 545.

Bonow RO, Carabello B, de LA Jr, Edmunds LH Jr, Fedderly BJ, Freed MD, et al. Guidelines for the management of patients with valvular heart disease: executive summary; a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Patients with Valvular Heart Disease). Circulation 1998;98:1949-84.

Carol M. Rumack, Stephanie R. Wilson, J. William Charboneau, and Deborah Levine, Diagnostic ultrasound (2007) 4th edition published by Elsevier

Carol M. Rumack, Stephanie R. Wilson, J. William Charboneau, and Deborah Levine, Diagnostic ultrasound (2007) 4th edition published by Elsevier

Cowie M R, Mosterd A, Wood D A. *et al* The epidemiology of heart failure. Eur Heart J 199718208–225. [Pub Med] Extensive review of the epidemiology of heart failure.

David Alevison, Robin Reid, Alastair, D Burt, David J Harrison, stewartfleming, 2008,text book of pathology, fourteenth edition, hodder Arnold 2007,p 134-143 44.

Doppler Echocardiography at the US National Library of Medicine Medical Subject Headings (MeSH).

Emanuel LL, Bonow RO. Care of patients with end-stage heart disease. In: Bonow RO, Mann DL, Zipes DP, Libby P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 9th ed. Philadelphia, Pa: Saunders Elsevier; 2011:chap 34.

Eric Lim, Ziad Ali, Kevin Varty, John Wallwork. Philip Poole-Wilson. Section 1.1. Introduction. 1. PART 1A. Diseases of the heart and great vessels ,Ch01-F07260.qxd 5/9/07 45.

F. Buba, B.N. Okeahialam and C.O. Anjorin. The Value of Chest Radiogram and Electrocardiogram in the Assessment of Left Ventricular Hypertrophy among Adult Hypertensives .Journal of Medical Sciences, 2008. 8: 298-301.

Feigenbaum, H. 1981. Echocardiography 3rd edn. Lea &Febiger, hiladelphia

Fox K F, Cowie M R, Wood D A. et al Coronary artery disease as the cause of incident heart failure in the population. Eur Heart J 200122228–236. [PubMed]Study documenting the importanceof vigorously evaluating patients with heart failure to establish the cause of heart failure.

Ganau, A., Devereux, R.B, Roman, M.J, de Simone, G.,Pickering, T.G, Saba, P.S, et al. 1992.Patterns of left ventricular hypertrophy and geometric remodeling in essential hypertension. J Am Coll Cardiol; 19:1550e8.

Hellermann J P, Goraya T Y, Jacobsen S J. et al Incidence of heart failure after myocardial infarction: is it changing over time? Am J Epidemiol 20031571101–1107. [PubMed] Excellent paper documenting that improved survival following myocardial infarction does not contribute to the heart failure epidemic.

Helmcke F, Nanda NC, Hsiung MC, Soto B, Adey CK, Goyal RG, et al. Color Doppler assessment of mitral regurgitation with orthogonal planes. Circulation 1987;75:175-83.

Hogg K, Swedberg K, McMurray J. Heart failure with preserved left ventricular systolic function; epidemiology, clinical characteristics, and prognosis. J Am Coll Cardiol 200443317–327. [PubMed] Excellent review of the epidemiology of heart failure with preserved left ventricular systolic function (“diastolic heart failure”).

Human Physiology/The cardiovascular system, Available at http://www.who.int/chp/steps/en/ (accessed 12 December 2013).

http://www.webmd.com/heart-disease/guide-heart-failure?page=2

Jessup M, Abraham WT, Casey DE, et al. 2009 focused update: ACCF/AHA Guidelines for the Diagnosis and Management of Heart Failure in Adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines: developed in collaboration with the International Society for Heart and Lung Transplantation. Circulation. 2009 Apr 14;119(14):1977-2016. Epub 2009 Mar 26.

JF Lewis, LC Kuo, JG Nelson, MC Limacher, MA Quiñones Pulsed Doppler echocardiographic determination of stroke volume and cardiac output: clinical validation of two new methods using the apical window Circulation, 70 (1984), pp. 425-431.

Kathryn A. Booth ,RN ,BSN total care programming palm coast, FL and Terri D. Wyman Anatomy ,Physiology ,and pathophysiology For Allied Health Published by MC Graw 2008978–0–07–337393–5.

Keith L Moor PHD ,Arthur F. Dalley PHD ,Clinically Oriented Anatomy ,fifth edition , Lippincott Williams & Wilkins 2006,p 141 -165.

Kenchaiah S, Narula J, Vasan R S. Risk factors for heart failure. Med Clin North Am 2004881145–1172. [PubMed] Excellent review of risk factors for the occurrence of heart failure.

Kenchaiah S, Evans J C, Levy D. et al Obesity and the risk of heart failure. N Engl J Med 2003347305–313. [PubMed] First study to document the importance of obesity, adjusted for known risk factors, as a risk factor for heart failure.

Klabunde, RE (2007). "Cardiac Valve Disease". Cardiovascular Physiology Concepts. Richard E. Klabunde. Retrieved 2010-08-06.

Levy D, Larson M G, Vasan R S. *et al* The progression from hypertension to congestive heart failure. JAMA 19962751557–1562. [PubMed] Paper addressing the importance of hypertension vis a vis coronary artery disease in the occurrence of heart failure.

Levy W C, Mozaffarian D, Linker D T. et al The Seattle Heart Failure Model: prediction of survival in heart failure. Circulation 20061131424–1433. [PubMed] Extensively validated model to predict prognosis in heart failure patients

Lloyd-Jones D M, Larson M G, Leip E P. et al Lifetime risk for developing congestive heart failure: the Framingham heart study. Circulation 20031063068–3072.First paper to present lifetime risks of the occurrence of heart failure, including the detrimental effect of hypertension. [PubMed

MacIntyre K, Capewell S, Stewart S. *et al* Evidence of improving prognosis in heart failure: trends in case fatality in 66 547 patients hospitalized between 1986 and 1995. Circulation 20001021126–1131. [PubMed] First paper to report a decline in age adjusted hospitalisation rates for heart failure.

Matsukubo, H., Matsuura, T., Endo, N., Asayama, J. and Watanabe, T. 1977, Echocardiographic measurement of right ventricular wall thickness. A new application of subxiphoid echocardiography. Circulation; 56:278e84.

McMurray J J, Pfeffer M A. Heart failure. Lancet 20053651877–1889. [PubMed] Review of the current state of the art in heart failure.

Mant J, Al-Mohammad A, Swain S, Laramee P. Guideline Development Group. Management of chronic heart failure in adults: synopsis of the National Institute For Health and Clinical Excellence guideline. Ann Intern Med. 2011;155(4):252-259.

Mann DL. Management of heart failure patients with reduced ejection fraction. In: Bonow RO, Mann DL, Zipes DP, Libby P, eds. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine. 9th ed. Philadelphia, Pa: Saunders Elsevier; 2011:chap 28.

M Enriquez-Sarano, KR Bailey, JB Seward, AJ Tajik, MJ Krohn, JM Mays Quan- -titative Doppler assessment of valvular regurgitation Circulation, 87 (1993), pp. 841-848.

Michael Y. M. Chen, Thomas L. Pope, David J. Ott, Basic Radiology, 2nd Edition, 2011, 2004 by The McGraw-Hill Companies, Inc.

Mc Donagh T A, Morrison C E, Lawrence A. *et al* Symptomatic and asymptomatic left-ventricular systolic dysfunction in an urban population. Lancet 1997350829–833.First paper to document the importance of asymptomatic left ventricular systolic dysfunction in the population.

Otsuji Y, Handschumacher MD, Schwammenthal E, Jiang L, Song J, Guerrero JL, et al. Insights from three-dimensional echocardiography into the mechanism of functional mitral regurgitation: direct in vivo demonstration of altered leaflet tethering geometry. Circulation 1997;96:1999-2008.

Owan T E, Redfield M M. Epidemiology of diastolic heart failure. Prog Cardiovasc Dis 200547320–332.Excellent review of the epidemiology of heart failure with preserved left ventricular systolic function ("diastolic heart failure").

Owan T E, Hodge D O, Herges R M. *et al* Trends in prevalence and outcome of heart failure with preserved ejection fraction. N Engl J Med 2006355251–259. [PubMed] First paper to address trends in prevalence and outcomes for heart failure patients with preserved as well as impaired left ventricular systolic function.

Peter Armstrong, Martinl. wastie, 1998, Diagnostic imaging, fourth edition Armstrong Wastie, p 103-120.

Ramzi S.Cotran, M.D., Vinay Kumar, M.D. and Stanley l. Robbins, M.D, applied pathology, fifth edition, Harcourt Brace, 2006.

Redfield M M, Jacobsen S J, Burnett J C., Jr *et al* Burden of systolic and diastolic ventricular dysfunction in the community: appreciating the scope of the heart failure epidemic. JAMA 2003289194–202. [PubMed] Very elegant population based study of the prevalence of both left ventricular systolic and diastolic dysfunction in relation to symptoms and signs.

Richard s. Snell, MD, PHD, 2000, clinical anatomy for medical students, six edition, Lippincott Williams & Wilkins, chapter 3 p95-105.

Roger V L, Weston S A, Redfield M M. *et al* Trends in heart failure incidence and survival in a community-based population. JAMA 2004292344–350. [PubMed] Population-based study on trends in incidence and survival of heart failure.

Rutten F H, Cramer M J, Grobbee D E. et al Unrecognized heart failure in elderly patients with stable chronic obstructive pulmonary disease. Eur Heart J 2005261887–1894. [PubMed] First study to address the under appreciation of heart failure in patients with pulmonary disease.

Sahn DJ. Instrumentation and physical factors related to visualization of stenotic and regurgitant jets by Doppler color flow mapping. J Am Coll Cardiol 1988;12:1354-65.

Shahgaldi, K., Gudmundsson, P., Manouras, A., Brodin, L.A. and Winter, R. 2009. Visually estimated ejection fraction by two dimensional and triplane echocardiography is closely correlated with quantitative ejection fraction by real-time three dimensional echocardiography. Cardiovascular Ultrasound.; 7: 41

Suskin N, Sheth T, Negassa A. et al Relationship of current and past smoking to mortality and morbidity in patients with left ventricular dysfunction. J Am Coll Cardiol 2001371677–1682. [PubMed] Study highlighting the beneficial effect of quitting smoking in patients with heart failure.

Stewart WJ, Currie PJ, Salcedo EE, Lytle BW, Gill CC, Schiavone WA, et al. Intraoperative Doppler color flow mapping for decision-making in valve repair for mitral regurgitation: technique and results in 100 patients. Circulation 1990;81:556-66.

Stewart WJ, Salcedo EE, Cosgrove DM. The value of echocardiography in mitral valve repair. Cleve Clin J Med 1991;58:177-83.

Stary HC. These quence of cell and matrix changes in atherosclerotic lesions of coronary arteries in the first forty years of life .Eur Heart J 1990;11(Suppl.E 3–19.

Suskin N, Sheth T, Negassa A. *et al* Relationship of current and past smoking to mortality and morbidity in patients with left ventricular dysfunction. J Am Coll Cardiol 2001371677–1682. [PubMed] Study highlighting the beneficial effect of quitting smoking in patients with heart failure.

Strobeck J.E., Sonnenblick E.H. (1985) Pathophysiology of Heart Failure. In: Levine H.J., Gaasch W.H. (eds) The Ventricle. Springer, Boston, MA.

S Nakatani, T Masuyama, K Kodama, A Kitabatake, K Fujii, T. KamadaValue and limitations of Doppler echocardiography in the quantification of stenotic mitral valve area: comparison of the pressure half-time and the continuity equation methods Circulation, 77 (1988), pp. 78-85

Wang T J, Evans J C, Benjamin E J. *et al* Natural history of asymptomatic left ventricular systolic dysfunction in the community. Circulation 2003108977–982. [PubMed]First paper to describe the natural history of asymptomatic left ventricular systolic dysfunction

WA Zoghbi, MA Quiñones Determination of cardiac output by Doppler echocardiography: a critical appraisal Herz, 11 (1986), pp. 258-268

WA Zoghbi, KL Farmer, JG Soto, JG Nelson, MA Quiñones Accurate noninvasive quantification of stenotic aortic valve area by Doppler echocardiography Circulation, 73 (1986), pp. 452-459

William f Ganong , review of medical physiology 21st Ed, (Appelton & Lange California) , Section VI , 2003,509 -521

Yoshida K, Yoshikawa J, Shakudo M, Akasaka T, Jyo Y, Takao S, et al. Color Doppler evaluation of valvular regurgitation in normal subjects. Circulation 1988;78:840-7.