Sudan University of Science and Technology

Collage of Graduate Studies

Impact of Hypertension on Heart using Echocardiography

أثر ارتفاع ضغط الدم علي القلب باستخدام الموجات الصوتيه للقلب

Thesis Submitted for Partial Fulfillment of M.Sc Degree in Medical Diagnostic Ultrasound

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الآيه

بسم الله الرحمن الرحيم

قال تعالى :

﴿وَقُلْ رَبِّ زِدْنِي عِلْمًا﴾

صدق الله العظيم

الآية [114] سورة طه

Dedication

This Research dedicated

To Soul of my Father

To my Mother

Who taught me how to make my future

They have given me the drive and discipline to tackle and task with enthusiasm and determination.

To

My family

Husband, son and daughters

To My

Brothers

Sisters

Acknowledgement

I would like to express my deepest gratitude to my supervisor Dr. Babiker Abdelwahab for giving opportunity to carry out my work for trusting me with assignment of this very interesting subject , I would like to thank my collage in Collage Medical Radiological Science and especially they are in Ultrasound master patch 19, 20

My truthful to Dr Adel Abdalla ultrasound specialist in echocardiography and Dr/ Abdeen

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Abbreviations

Abbreviations	Full name
2D	Two Dimensions
G	Gram
TEE	Trans Oesophageal
IVC	Inferior Vena Cava
SVC	Superior Vena Cava
AV	Atrio Ventricular
MM	Millimetres
SN	Sino atrial Node
A-V node	Atrio Ventricular node
S1	First Heart Sound
S2	Second Heart Sound
S3	Third Heart Sound
S4	Fourth Heart Sound
IHD	Ischemic Heart Disease
LDL	Low Density Lipoprotein
CHD	Congestive Heart Disease
ECG	Echocardiogram
3D	Three Dimensions
SC	Subcostal
SSN	Supra Sternal Node
PLAX	Parasternal Long Axis
HCU	Hand Carried Ultrasound
SAX	Short Axis
4C	Four Chamber
CW	Continues Wave
MPA	Main Pulmonary Artery
PWD	Power Doppler
Ao	Aorta
Ees	End Systolic
TDI	Tissue Doppler Imaging
RV	Right Ventricle
Ae	Atrial elasticity
SPSS	Statistical Package for Social Science
LVH	Left Ventricle Hypertrophy

BP	Blood Pressure
LVIDd	Left ventricle Internal at end Diastole
EF	Ejection Fractions
LVRT	Left Ventricle Internal at end Diastole
IMP	Index of Myocardial

Abstract

This is descriptive study cross-sectional study aim to study the impact of hypertension in heart, using Vivid 3 General Electric USA with probe 3s in Asia hospital, the study had 60 hypertension patients aged between 35-85 years old, affected age between 68-78, all subject are examined in transthoracic echocardiography and pulsed Doppler through apical 4 chamber to assess the flow pattern through mitral valve. The study found that male 35 percentage 58.3% more affected than female 25 percentage 41.7% the affected age 68-78 years of 24 patients from all patients percentage 40%. The study found that the duration of hypertension from 1-35 years. 36 patient had control this The important finding according to the effects of hypertensions that left ventricular hypertrophy in 38 patients percentage 62.7%, these patients have Diastolic Dysfunctions. Aortic root affected in 5 patients percentage 8.3%, mitral regurgitations in 15 patients percentage 25%, Aortic regurgitations in 6 patients percentage 10%, Tricuspid regurgitations in 8 patients percentage 13.3%, Inter ventricular septal thickening in 16 patients 26.7%, left ventricular wall thickening in 17 patients percentage 28.3%. The study found that sclerotic mitral valve and aortic valve in 36 patients percentage 60%, hypertensive heart disease in 11 patients (18.3%). Interventricular hypertrophy in 4 patients percentage 6.7% , the study found that 38 patients have different heart disease due to hypertension. The study conclude that hypertension when it uncontrolled it lead to different heart abnormalities and lead to many complications in heart champers and

blood supply heart or body. The study recommended other studies with large sample and other imaging modalities can show good result.

مستخلص ألدراسة

هذه دراسة وصفيه تحليليه هدفت ألي درأسه تأثير مرض ضغط الدم علي القلب باستخدام جهاز فيفيدو (من شركه اميريكيه) ببروب قوته 3 ميغاهيرز, أجريت هذه ألدراسة بمستشفي أسيا وكانت العينة عبارة عن 60 مريض بضغط الدم تتراوح أعمارهن بين 35-85 سنه وكان العمرالموثر يتراوح بين68 -78 ومتوسط الأعمار تقريبا هو 73 سنه واجري الفحص لجميع المرضي عبر ألصدر باستخدام دوبلار وان عدد الرجال المصابين بمرض ارتفاع ضغط الدم 35 مريض بنسبه 58.3 والنساء المصابات عددهم 25 بنسبه 41.7 وكانت مده الأصابه بهذا المرض تتراوح بين 1-25 عاما ووجدت الدراسة أن 36 مريض لديهم ضغط منتظم منهم 32(53.3%) يعالجون بصوره منتظمة , 24 ليس لديهم وسائل تحكم في المرض ومن أهم نتائج الدراسة حسب تأثير ضغط الدم المرتفع علي القلب إن البطين الأيسر متضخم في 38 مريض بنسبه (62.7%) كلهم لديهم ضعف انبساطي ووجدت الدراسة إن ألتأثير في الأورطه في 5 مرضي بنسبه 8.3% , ; وفي الصمام التاجي رجوع الدم في 15 مريض بنسبه 25%, ورجوعه في الأورطه في 6 مرضي بنسبه 10%, ورجوعه في الشريان ثلاثي الشرف في 8 مرضي بنسبه 13.3% . كما وجدت ألدراسة 16 مريض بنسبه 26.7% لديهم زيادة غير طبيعيه في السمك بين البطينين والأذينين, 17 مريض بنسبه 28.3% لديهم زيادة في سمك البطين ألأيسر . كما نتج عن تشخيص هذه النتائج أن تصلب صمام الأورطه والصمام ألتاجي وجد في 36 مريض بنسبه 66% و مرض ضغط الدم في القلب في 11 مريض بنسبه 18.3, 8 منهم لديهم تضخم في البطين الأيسر و زيادة اسمك بين البطينين في4 (6.7 %). خلصت ألدرسه 38 مريض لديهم امرض مختلفة لها علأقه وطيدة بارتفاع

ضغط الدم منها إمراض في غرف القلب والصمامات والأوعية الدموية المغذية للقلب, كما أوصت ألدراسة بعمل درأسه بعينه اكبر واستعمال وسائل تشخص مختلفة ألمقارنه بين نتائج هذه الدراسات.

Chapter one

Introduction

1-1Introduction

The heart is a hollow muscular organ, lying in a connective tissue space (*mediastinum*) between the vertebral column and the sternum. The heart is the motor of the circulation, by its pumping action it maintains a steady blood flow. The blood circulates through a closed system of elastic pipes, the vascular system, which can be divided into arteries, which lead blood away from the heart and distribute it, Capillaries, in which the exchange of substances takes place, Veins, which return the blood to the heart and Lymph vessels, which serve the transport of fluids and immune cells Irrespective of their oxygen content, all blood vessels leading away from the heart are called arteries, and all blood vessels that lead toward the heart are called veins. For instance, the pulmonary artery that leads from the heart to the lungs carries oxygen-poor blood, and the pulmonary veins, leading from the lungs to the heart, carry oxygen-rich blood. Similarly, the umbilical arteries carry oxygen-poor blood, while the blood in the umbilical veins is rich in oxygen. (Adolf, 2005)

Hypertension is raised blood pressure and the term is applied to both the arterial and venous circulations – arterial hypertension and venous hypertension. Hypotension, low blood pressure in the systemic arterial circulation, can cause damage if blood flow is also low. Infarction is tissue Hypertension is raised pressure in any vascular bed, for example pulmonary hypertension, portal hypertension ., hypertension is usually synonymous with systemic arterial hypertension. Its main clinical importance is as a major risk factor in the cardiovascular diseases due to atherosclerosis .(Murries 2008).

Hypertensive heart disease so that heart is target organ response to arterial hypertension. Left ventricular hypertrophy represents an important predictor for cardiovascular events. Myocardial fibrosis, a common end point in hypertensive heart disease, has been linked to the development of left ventricular hypertrophy and diastolic dysfunction. Echocardiography is clinically useful in the detection of left ventricular hypertrophy and the assessment of diastolic function. Thus, advances in cardiac imaging provide comprehensive, noninvasive tools for imaging left ventricular hypertrophy, diastolic dysfunction, myocardial fibrosis and ischemia observed in hypertensive heart disease (Janardhanan, 2014)

Echocardiography, or cardiac ultrasonography, uses ultrasound echoes reflected from the heart to generate images of cardiac structure and function .In two dimensional (2D) or cross-sectional echocardiography, the transducer scans through the anatomical scan plane. The reflected ultrasound echoes are processed and displayed in real time as a pie-shaped scan sector. Doppler echocardiography displays blood flow velocities as a color flow map or as a spectrum of velocities during the cardiac cycle Figure .Modern echocardiography instruments (Bulwer, 2011)

`1-2Problems of the study

Patients and doctors not awarded by the effect of hypertensions in heart especially that had no control of this disease.

1-3Objectives:

1-3-1General objective

To Assess the Impact of Hypertension in Heart using echocardiography

1-3-2Specific objectives:

*To assess patients age

* To assess patients, gender

* To know duration of hypertension and control of medications

* To assess echocardiography finding in heart after hypertension

* To correlate between finding and hypertension

1-4 Significant of the study:

It helps in early detection of any abnormality in heart according to the effect of hypertension and help in important of medications

1-5 Over view of the study:

This study consist of five chapter

Chapter one introduction, problem, and objective

Chapter two anatomy . physiology, ultrasound measurement of thyroid and previous studies

Chapter three materials and method

Chapter four result

Chapter five discussion, conclusion and recommendations

Chapter Two

Theoretical background

2-1 Heart Anatomy:

The heart grows physiologically proportionally to body mass. The relationship is maintained from the fetus to the adult, and is equally true for all mammalian species. As a general rule, the heart weight can be estimated from the lean body mass. However, by multiplying the weight expressed in kilograms by 0.50% for a man, and 0.40% for a woman (because woman have a lower lean body mass), an approximation of the normalized heart weight can be determined. Physiological heart weight is maximized at 400-450 g for males, and 350- 400 g for females, regardless of body size. **(Schünke, 2004).** Heart is divided into pumping chambers dependent on blood flow moving between two separate circulatory circuits, with the timing of blood movement within the chambers occurring within fractions of a second. This movement must be coordinated by a neural network (e.g. conduction system), heart valves, vascular supply, connective tissue and muscle cells. The heart is dependent on maintaining its own nutrition, while at the same time supplying oxygenated blood to the entire body. During the process of pumping, chamber contraction affects the supply of blood to the tissue performing the pumping, sometimes with adverse consequences. There must be an adequate flow of oxygenated blood to the heart cells, because they have very high metabolic requirements. Although myocytes are organized as individual cells and do not form a syncytium, they must 'talk' to each other electrically and functionally. The cells must each contract, but this contraction must then be coordinated so that groups and layers of cells are shortening sequentially within a very short period of time. Otherwise, this would lead to hyperkinesias (decreased

contraction), dyskinesia (abnormal contraction), or akinesia (absence of contraction). Detailed descriptions are available of the complex structural and functional anatomy of the heart; but most of such works do not provide adequate explanations for acquired Pathophysiology or contractile dysfunction **(Chung, 2001).**

2-1-13Heart Orientation

Although standard terminology suggests that the heart is oriented in a left-right direction, this is misleading. The heart is actually positioned so that the right ventricle is anterior, and lies just below the sternum. The major portion of the left ventricle lies posterior and lateral to the right ventricle, with the apex composed of the left and right ventricles obliquely pointing to the left. The right atrium is superior and posterior to the right ventricle. The left atrium is the most superior and posterior structure, lying as it does immediately anterior to the esophagus and vertebral column. This provides an explanation for why trans-esophageal echocardiography (TEE) is such a superior technique for visualizing the mitral valve; the TEE transducer introduced into the esophagus, is immediately adjacent to the valve. Another
important consequence of the anatomical orientation is the effect of trauma. Blunt trauma to the chest wall (e.g. seat belt or steering wheel injury) may lead to contusion of the anterior surface of the heart below the sternum, or the right ventricle. If the trauma is more lateral and to the left, it may contuse the anterior surface of the left ventricle, **(ELLIS, 2006).**

2-1-1Annulus

The fibrous ring that supports each of the four cardiac valves. The annulus for the tricuspid, mitral, and aortic valves is virtually a continuous structure arranged like a pretzel. The pulmonic valve annulus is somewhat separate from the other three (it arises from distinctly independent embryologic tissue,

e.g. bulbus cordis). The valve base inserts into the annulus with interdigitating connective tissue fibers. The annulus also anchors the base of the heart through connective tissue attachment fibers that extend into the muscle of the ventricle. Sections of the base of the heart just below the annulus typically reveal fibrous tissue; however, this should not be interpreted as pathological scar since it represents normal anatomy. The dilatation may pull the valve base away from the center channel through which blood flows, and where there is closure with valve leaflet/cusp coaptation. (**ELLIS, 2006)**.

2-1-2Capillary loops

The myocardial microcirculation is an end-vascular system, which means that as the coronary arteries progressively branch into smaller ramifications, they reach a point where they no longer inter-connect. This lack of collaterals appears to occur when the vessels are approximately 25-50 microns in diameter, or at a level when they are pre-capillary arterioles. At that point, they give rise to an arcade of capillaries that do not inter-connect with adjacent arcades derived from other arterioles. The capillaries, usually 4-6 in number, provide oxygenated blood to individual myocytes looping around them, with the afferent arm draining into venules and then larger venous channels (Gray's, 2004).

2-1-3 Chordae tendineae

The fine fibrous bands that extend from the tips of the papillary muscles to the atrio-ventricular heart valves. The chordae branch at least 3 times and become smaller between the papillary muscle tip, known as the myotendon junction, and the valve. In total, there are approximately 120 chordae at the valve level. They insert on the undersurface of the valve, at the commissural junction, and at the free edge. With contraction of the ventricle, pressure within the cavity increases thereby forcing the leaflets of the mitral and

tricuspid valves toward the midline. There is concurrent papillary muscle contraction, which pulls the chordae toward the mid portion of the ventricular chamber (Gray's, 2004) .

2-4-1The heart chamber:

Heart has four chambers Similarly, right atrium, right ventricle, left atrium, left ventricle; the left atrium and the left ventricle together are sometimes referred to as the left heart. (ELLIS, 2006).

2-1-4-1 Right Atrium

Receives venous blood from the systemic circuit from the heart muscle itself. Three major vessels empty into the right atrium, superior vena cava (SVC) which drains blood from the head, upper limbs, and superior regions of the trunk, inferior vena cava (IVC) which drains blood from the lower limbs and trunk, coronary sinus drains blood from the heart wall. The inter atrial septum forms a wall between the right and left atria (ELLIS, 2006).

.2-1-4-2Right Ventricle

Receives deoxygenated venous blood from the right atrium. An Interventricular septum forms a wall between the right and left ventricles. Papillary muscles on the internal wall surface cone-shaped, muscular projections anchor chordae tendineae attach to the cusp of the right AV valve and prevent entering and flipping into the atrium when contracting (ELLIS, 2006).

2-1-4-3Left Atrium

Once gas exchange occurs in the lungs, the oxygenated blood travels through the pulmonary veins to the left atrium. Smooth posterior wall of the left atrium contains openings for approximately four pulmonary veins they are, two left pulmonary veins, two right pulmonary veins. And this atrium has pectinate muscles along its anterior wall as well as an auricle. (ELLIS, 2006).

2-1-4-4 Right Ventricle

As described in the organization of the myocardium, the right ventricle is a thin-walled structure with several poorly defined layers of myocardium. The free-wall and septum are heavily trabeculated. The free wall is often infiltrated by fibroadipose tissue that extends between the muscle fibers from the epicardial fat towards the endocardium. usually due to persistent or acquired pulmonary hypertension. right ventricle only has to pump blood to the nearby lungs (Gray's, 2004)

2-1-4-5 Left Ventricle

Largest of the four heart chambers its wall is typically three times thicker than the right ventricular wall. Requires thick walls in order to generate enough pressure to force the oxygenated blood from the lungs into the aorta and then through the entire systemic circuit. Trabeculae carneae in the left ventricle are more prominent. Two large papillary muscles attach to the chordae tendineae that help support the left AV valve. At the superior end of the ventricular cavity, the aortic semilunar valve marks the end of the left ventricle and the entrance into the aorta(Gray's, 2004) .

2-1-4-6 Apex, ventricle

A generally disregarded area of the heart, but one that may have important functional significance. It is composed of the most dependent portions of both left and right ventricles, with the myocardial fibers spiraling towards the tip of the chamber. The muscle and the associated connective tissue matrix (see below) come together as a button-like structure at this point. In fact, during normal contraction there is dimpling of the apex (dimples in the face are the result of connective tissue attachments between skeletal muscle and the dermis) (Gray's, 2004)

1-2-4-7Interventricular Septum

The atrioventricular septum separates the left heart from the right heart, giving two functional units. Consists of plate of fibrous connective tissue between atria and ventricles The septum has a relatively smooth endocardial surface on the left ventricular outflow tract, whereas, the right ventricular surface of the septum is diffusely trabeculated (Malouf, 2008) .

2-1-6 Coronary Arteries

Muscular blood vessels lined by endothelium with a thin intima composed of 1-2 connective tissue cells at birth, and separated from the relatively thick media by an internal elastic lamella. The media usually has two obliquely oriented layers of smooth muscle. The outer coat of the artery is adventitia, or connective tissue that provides both innervations and blood supply to the coronary vessels (vasa nervorum and vasa vasorum, respectively). The diameter of the normal epicardial coronary artery, prior to its branching and entering the myocardium is usually 2-3 mm, but it can be variable. (Gray's, 2004)

2-1-7 Coronary Ostia

The two openings for the left and right coronary arteries, usually located centrally at the upper portion of the sinus of Valsalva, approximately parallel to the free edge of the aortic valve. The location is variable. High insertion in the aortic wall has been associated with sudden cardiac death, presumably secondary to transient decrease of coronary flow. Lower insertion within the sinus of Valsalva may make the ostia more susceptible to stenosis by conditions that affect the aortic valve (Gray's, 2004)

2-1-8 Ductus Arteriosus

It represents one of the physiologic shunts of the fetal circulation (see also foramen ovale). It is the arterial connection between the left pulmonary artery and the aorta just distal to the origin of the left subclavian artery. The ductus allows for a bypass of the high-resistance pulmonary circulation, and the passage of oxygenated blood in the pulmonary artery into the aorta. It begins to close shortly after birth, and completes the closure within the first weeks of life. Persistence of a patent ductus arteriosus may lead to severe, irreversible pulmonary hypertension, if it is not closed. In the newborn with certain congenital heart defects, the closure of the ductus arteriosus generally precipitates cardiovascular collapse. **(Chung, 2012)**

2-1-9 Endocardium:

The endothelial covering of the entire inner surface of the heart. This includes the cardiac chambers, both surfaces of the valves, and the chordae tendineae. The endocardium has functions similar to endothelium in vessels (the heart is really just a big muscular vessel). It can react to hormones (e.g. prostaglandins), and it has secretory capability with release of endothelin and nitric oxide. **(Chung, 2012)** .

2-1-10Foramen Ovale / Fossa Ovalis:

Another one of the normal fetal circulation shunts (see ductus arteriosus). It refers to the opening in the mid-portion of the inter-atrial septum that allows oxygenated blood returning to the right heart from the placenta, to cross into the left atrium. The foramen is covered by a membrane, the septum secundum, which grows down on the left atrial side during embryogenesis, and overlies a separate membrane. During fetal development, when pressures are higher in the right atrium, there is flow from right to left, even when the membrane has completed its development. Following birth, when pressures become higher on the left side, the septum secundum is forced against the foramen. Usually, the membrane fuses to the tissue, and there is an oval depression in the atrial wall. **(Chung, 2012)** .

2-1-11 Jelly, Cardiac

The relatively a cellular layer forming the wall of the embryologic cardiac tube, separating endothelial cells internally from an external layer of mantle cells. This tube forms the primitive ventricular tube and bulbus cordis **(Schünke, 2004)** .

2-1-12 Koch, Triangle

The triangle of Koch is the area defined inferiorly by the septal leaflet of the tricuspid valve inserting into the annulus, posteriorly by the coronary sinus, and superiorly by the ridge-like tendon of Todaro**.** The triangle is significant because it contains the atrio-ventricular node. **(Chung, 2012)** .

2-1-13 Lambl's Excrescence

A small papillary frond-like vegetation that is found on the closing edge of valve cusps and leaflets. It is typically seen in the central closing edge of the aortic valve cusps. The excrescence resembles a fine thread-like structure. Microscopically, they have a fibrous core, and a surface of endothelial cells. They are thought to arise from localized trauma to the valve from repetitive

closure, with organization of the resulting fibrin and platelet vegetation. They are related to the much less common, and larger, organized vegetation called papillary fibroelastoma **(Schünke, 2004)** .

2-1-14Membranous Septum

The round or oval-shaped membranous area immediately below the aortic valve annulus. There are a number of congenital defects of this structure, particularly in association with endocardial cushion defects; however, isolated membranous Septal defects also occur. The membranous septum is the anatomic landmark where the atrio-ventricular conduction tissue progresses through the annulus at the base of the septum, and then branches into left anterior and posterior divisions **(Schünke, 2004)** .

2-1-15 Nuclei, Myocyte

Myocytic nuclei are relatively large, round to oval-shaped structures at birth. During embryogenesis, mitotic division of nuclei and cells can be easily appreciated. Shortly after birth, mitoses become exceedingly rare. For years it has been assumed that myocytes are terminally differentiated cells, with no new cell proliferation after birth. Recent evidence suggests that limited numbers of cells may proliferate in adulthood around areas of injury (e.g. healing myocardial infarction). It is known that myocyte nuclei may divide without cytokines is, thereby giving rise to myocardial cells with 2 nuclei. This is common with increasing age, particularly in association with myocardial hypertrophy **(Schünke, 2004)** .

2-1-16 Organization, Myocardium

As discussed in **connective tissue matrix,** the myocardium is composed of obliquely oriented layers that ‘wrap’ around the ventricular cavity. This is particular obvious in the left ventricle, with three layers; whereas, the thinner, low-pressure right ventricle generally has two less well-defined layers. Since

the layers in the left ventricle are oriented obliquely to each other, sectioning through the ventricular wall reveals groups of myocardial cells parallel to each other, but oriented at approximately 45 degrees to adjacent parallel bundles of cells. Thus, a section across the left ventricle wall has myocytes oriented longitudinally, obliquely, and in cross-section. These layers are attached to each other by connective tissue matrix fibers as previously discussed. Contraction of the ventricle takes place in a screw-like manner, as the obliquely oriented layers shorten in a curvilinear pattern from apex to base. Upon diastolic relaxation, since the layers and the individual myocytes are interconnected, and because there are spring-like connective tissue fibers running along the axis of the ventricle, negative pressure develops enhancing diastolic filling by 'sucking' blood from the atrialcavities. (Faller, 2004)

2-1-17Papillary Muscle

There are two papillary muscles in the left ventricle, an anterolateral and a posteromedial. They are broad, finger-like projections into the cavity that enlarge with ventricular hypertrophy, and therefore may contribute to a decreased cavity volume in association with conditions such as hypertension. The papillary muscles also have a terminal coronary circulation with little or no collaterals. The chordae from both papillary muscles fan out to support both leaflets of the mitral valve. The chordae from the 2 right ventricular papillary muscles support the anterior and middle leaflet. The septal leaflet chordae insert directly into the ventricular septum without a papillary muscle. In the right ventricle, the papillary muscles are less prominent, appearing more like hypertrophied trabeculae (Faller, 2004)

2-1-18 Pericardium

A thin fibrous sac that envelops the heart and extends along the ascending aorta to the brachiocephalic artery. Thus, rupture of the ascending aorta (often

as a result of aortic dissection) can lead to pericardial tamponade. The pericardium is structurally considered as 2 surfaces: a visceral layer made up of mesothelial cells overlying the pericardial fibroadipose tissue, and a parietal layer with an inner lining of mesothelial cells, a fibrous body, and an outer layer that blends with the connective tissue of the mediastinum. The mesothelial cells secrete a small volume (usually 10-20 cc) of thin serous fluid that serves to lubricate pericardial and cardiac movement. The pericardial sac is drained by lymphatic channels. Increased secretion (e.g. associated with inflammation), or reversal of lymphatic flow (e.g. associated with elevated pressure in the venous and lymphatic system that occurs in right-sided congestive heart failure) leads to increased fluid volume in the pericardial sac. (Faller, 2004)

2-1-2 Valves

The four valves have a similar microscopic appearance, despite their different gross anatomy. Each is surfaced entirely by endocardium. Beneath the endocardial layer, there is a fibrous layer (fibrosa) of variable thickness, which increases with age. The fibrous layer is also thicker in the left-sided valves, than those on the right side. The central region of the valve is composed of a myxoid layer with scattered spindled and stellate connective tissue cells, and loose mucopolysaccharide (spongiosa). There is expansion of both the fibrous and the myxoid layer in fibro myxomatous valve degeneration with a proportionally greater increase in the myxoid zone. Moreover, there are relatively severe changes seen in association with hereditary connective tissue conditions such as Marfan's syndrome, or Ehlers- Danlos syndrome. (Faller, 2004)

2-1-2-1Tricuspid Valve

Although structurally considered to have three leaflets, the tricuspid valve frequently has a diffusely scalloped appearance with numerous small leaflets. This appearance is enhanced if associated with fibromyxomatous valve disease (e.g. ‘floppy’ mitral valve, or mitral valve prolapse). (Faller, 2004)

2-1-2-2Pulmonary Valve

Structurally it is similar to the aortic valve, with 3 cusps. The sinuses behind the cusps are shallower than the sinuses of Valsalva. Compared to the aortic valve, the pulmonary valve is rarely susceptible to degeneration, inflammation, or infection; presumably because it is not chronically damaged by a high pressure system. The valve is the site of congenital anomalies particularly that associated with Tetralogy of Fallot. **(STEPHEN, 2002)**

2-1-2-3Mitral Valve

The only valve with two leaflets. The mitral valve has an unusual configuration, with marked asymmetry of the leaflets. The anterior leaflet is a broad, shield-shaped structure that fills approximately two-thirds of the valve orifice, but only one-third of the annular circumference. It extends much deeper into the ventricular chamber than the posterior leaflet. The latter is a shallow, usually scalloped leaflet that has a broad annular attachment, but comprises only one-third of the orifice area. Together, the two leaflets have approximately equal surface areas. Both are tethered on their undersurfaces by chordae tendineae and two papillary muscles, which maintain them in a flat orientation relative to the annulus during ventricular

systole. When seen from the atrial surface, the two leaflets have a curvilinear convex-concave apposition, with the convex surface from the anterior leaflet; resembling a ‘fish-mouth’. The mitral valve orifice is large, thereby allowing a high volume of blood to be passed into the ventricle at low pressure. The commissural attachments of the two leaflets are poorly defined areas of

junction, but important sites for inflammation (e.g. rheumatic heart disease) and infection **(STEPHEN, 2002)** .

2-1-2Aortic Valve

The three cusped valve separating the outflow tract of the left ventricle from the aorta. The cusps attach to the annulus, and insert as thin fibrous bands surfaced by endothelial cells (endocardium) into the aortic wall. These fibrous bands, immediately adjacent to each other but separated by less than
mm, form 3 commissure. **(STEPHEN, 2002)**

2-1-24 Ultrastructure

The ultrastructural appearance of the myocardium is complex and would take an entire atlas to describe. Several unique features will be outlined here. Myocardial cells are generally elongated, with lateral branches that attach at each end and at the end of the branches to adjacent cells by electron-dense intercalated disks. The sarcolemma or myocardial cell membrane is lined by a basal lamina. Invaginations of the sarcolemma at the level of the Z-band extend deeply into the myocyte along with a thinner basal lamina. This is called the T-tubule which permits access of electrolytes into the myocyte where there is interaction at the sarcoplasmic reticulum (a series of fine tubules that extend along the contractile fibrils) to release or uptake calcium necessary for myocyte contraction and relaxation. The contractile unit is known as the sarcomere. It includes myosin, actin, and a number of associated fibrils. The margins of the sarcomere are the lateral Z Band into which the thick myosin filaments attach.. (**ELLIS, 2006)**.

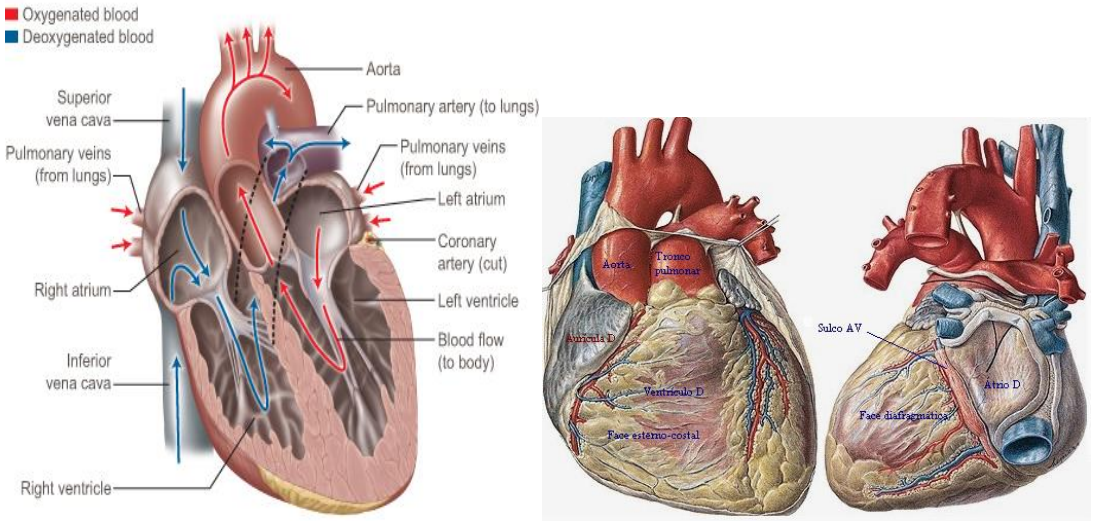

Figure (2-1) Heart anatomy outer and inner **ELLIS (2006)**

2-2Physiology

2-2-1Physiology of Cardiac Muscle

The heart is composed of three major types of cardiac muscle: *atrial muscle*, *ventricular muscle,* and specialized *excitatory* and *conductive muscle* fibers. The atrial and ventricular types of muscle contract in much the same way as skeletal muscle, except that the duration of contraction is much longer. Conversely, the specialized excitatory and conductive fibers contract only feebly because they contain few contractile fibrils; instead, they exhibit either automatic rhythmical electrical discharge in the form of action potentials of the action potentials through the heart, providing an excitatory system that controls the rhythmical beating of the heart (Bers, 2003) .

2-2-2 Conduction System

In addition to the atrio-ventricular node with the bundle of His and conduction bundles, and the sino-atrial node (see below), the conduction system includes

Purkinje fibers that course in the immediate subendocardium of the two ventricles. They serve as a fan-like projection of
conductive fibers that rapidly transmit generated electrical impulses from the A-V node to the myocardium. (Bers, 2003) .

2-2-3 Connective Tissue Matrix

The myocardium is completely invested by a fine mesh-like network of connective tissue fibers that is virtually invisible with standard histologic sections, but can be visualized with special staining techniques or electron microscopy. This connective tissue wraps around single myocytes, attaches individual myocytes laterally, envelops groups of these cells, and maintains connections between myocyte layers in the ventricular wall. Although this skeletal framework is important for maintaining myocyte shape, it is critical for normal ventricular function. (Guyton, 2006) .

2-2-4 Atrio-ventricular node

A modified mass of myocardial cells that serves as the electrical pacemaker of the ventricles. Situated in the lower inter-atrial septum, it lies immediately superior to the tricuspid valve and its annulus and the mitral valve and its annulus. Blood supply is provided by a branch of the right coronary artery in most individuals, or a branch of the circumflex artery in the minority of cases. Thus disease of either vessel may lead to ischemic damage to the node, with the potential for conduction block. The node sends a bundle of fibers into the ventricles (the bundle of His), by penetrating the annulus of the tricuspid and mitral valves. (Guyton, 2006).

2-2-5 Sinoatrial Node

An elongated, fusiform collection of modified cardiac muscle cells and connective tissue lying immediately below the epicardium at the junction of

the superior vena cava and the right atrial wall. The node is supplied by a branch of the right coronary artery or a branch of the left circumflex coronary artery in about equal numbers of people. Degeneration of the node may occur with ischemia, inflammation, or in association with increased atrial pressure (Guyton, 2006) .

2-2-6 Bundle of His and conduction bundles

The bundle of His and conduction fibers are composed of modified cardiac muscle tissue. The bundle of His crosses through the annulus at the base of the cardiac septum. Upon reaching the ventricle, it divides into left and right divisions. The left division skirts around the membranous septum, where it then divides further into an anterior and posterior branch. The right bundle branch extends over the base of the septum into the right ventricle, where it runs superficially in a muscle known as the moderator band between the ventricular wall and the anterior papillary muscle. All of the conduction bundles are susceptible to damage or disruption, because they run superficially just below the endocardium. The bundle of His may be interrupted if the annulus develops degenerative calcification, or if it is affected by infection spreading from endocarditis of an adjacent heart valve.

Left ventricular septal ischemia or infarction, high in the base around the membranous septum, may lead to left anterior or left posterior hemi-block. More proximal infarction may completely damage the bundle, leading to left, right, or complete heart block. Surgery for congenital heart disease, at or around the membranous septum may affect the bundle tissue in the surrounding myocardium. (Guyton, 2006)

2-2-7 Commissure

The commissures of the aortic valve have been described above; they are comparable for the pulmonic valve. The commissures of the atrioventricular

valves are less well defined. The leaflets insert broadly into the annulae along a curved base; at the junction between one leaflet and the adjacent leaflet, they form a commissure. This is a potential site for damage. Inflammation, thrombus, and calcification may scar the commissure and prevent complete opening of the valve. (Bers, 2002) .

2-2-8**Physiologic Anatomy of Cardiac Muscle**

Cardiac muscle has typical myofibrils that contain *actin* and *myosin filaments* almost identical to those found in skeletal muscle; these filaments lie side by side and slide along one another during contraction in the same manner as occurs in skeletal muscle. But in other ways, cardiac muscle is quite different from skeletal muscle, as we shall see*;* they are actually cell membranes that separate individual cardiac muscle cells from one another. That is, cardiac muscle fibers are made up of many individual cells connected in series and in parallel with one another. At each intercalated disc the cell membranes fuse with one another in such a way that they form permeable "communicating" junctions (gap junctions) that allow almost totally free diffusion of ions. Therefore, from a functional point of view, ions move with ease in the intracellular fluid along the longitudinal axes of the cardiac muscle fibers, so that action potentials travel easily from one cardiac muscle cell to the next, past the intercalated discs. The heart actually is composed of two syncytium: the *atrial syncytium* that constitutes the walls of the two atria, and the *ventricular syncytium* that constitutes the walls of the two ventricles. The atria are separated from the ventricles by fibrous tissue that surrounds the atrioventricular (A-V) valvular openings between the atria and ventricles. Normally, potentials are not conducted from the atrial syncytium into the ventricular syncytium directly through this fibrous tissue. Instead, they are conducted only by way of a specialized conductive system called the *A-V*

bundle, a bundle of conductive fibers several millimeters in diameter. This division of the muscle of the heart into two functional syncytium allows the atria to contract a short time ahead of ventricular contraction, which is important for effectiveness of heart pumping (Bers, 2002) .

2-2-7-1 Action Potentials in Cardiac Muscle

The *action potential* recorded in a ventricular muscle fiber, averages about 105 millivolts, which means that the intracellular potential rises from a very negative value, about -85 millivolts, between beats to a slightly positive value, about +20 millivolts, during each beat. After the initial *spike*, the membrane remains depolarized for about 0.2 second, exhibiting a *plateau* as shown in the figure, followed at the end of the plateau by abrupt repolarization. The presence of this plateau in the action potential causes ventricular contraction to last as much as 15 times as long in cardiac muscle as in skeletal muscle. At least two major differences between the membrane properties of cardiac and skeletal muscle account for the prolonged action potential and the plateau in cardiac muscle. First, the *action potential of skeletal muscle* is caused almost entirely by sudden opening of large numbers of so-called *fast sodium channels* that allow tremendous numbers of sodium ions to enter the skeletal muscle fiber from the extracellular fluid. These channels are called “fast” channels because they remain open for only a few thousandths of a second and then abruptly close. At the end of this closure, repolarization occurs, and the action potential is over within another thousandth of a second or so. In cardiac muscle, the action potential is caused by opening of two types of channels. the same fast sodium channels as those in skeletal muscle and, another entirely different population of slow calcium channels, which are also called calcium-sodium channels. This second population of channels differs from the fast sodium channels in that they are slower to open and, even more important,

remain open for several tenths of a second. The second major functional difference between cardiac muscle and skeletal muscle that helps account for both the prolonged action potential and its plateau is this: Immediately after the onset of the action potential, the permeability of the cardiac muscle membrane for potassium ions *decreases* about fivefold, an effect that does not occur in skeletal muscle. When the slow calcium-sodium channels do close at the end of 0.2 to 0.3 second and the influx of calcium and sodium ions ceases, the membrane permeability for potassium ions also increases rapidly; this rapid loss of potassium from the fiber immediately returns the membrane potential to its resting level, thus ending the action potential (Brutsaert, 2003) .

The velocity of conduction of the excitatory action potential signal along both atrial and ventricular muscle fibers is about 0.3 to 0.5 m/sec, or about 1/250 the velocity in very large nerve fibers and about 1/10 the velocity in skeletal muscle fibers. The velocity of conduction in the specialized heart conductive system—in the Purkinje fibers—is as great as 4 m/sec in most parts of the system, which allows reasonably rapid conduction of the excitatory signal to the different parts of the hear (Brutsaert, 2003) .

2-1-7-2Refractory Period of Cardiac Muscle:

Cardiac muscle, like all excitable tissue, is refractory to restimulation during the action potential. Therefore, the refractory period of the heart is the interval of time, as shown to the left in Figure 9–4, during which a normal cardiac impulse cannot re-excite an already excited area of cardiac muscle. The normal refractory period of the ventricle is 0.25 to 0.30 second, which is about the duration of the prolonged plateau action potential(Brutsaert, 2003).

2-2-8 The Cardiac Cycle

The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called the *cardiac cycle.* Each cycle is initiated by spontaneous generation of an action potential in the *sinus node*. This node is located in the superior lateral wall of the right atrium near the opening of the superior vena cava, and the action potential travels from here rapidly through both atria and then through the A-V bundle into the ventricles. Because of this special arrangement of the conducting system from the atria into the ventricles, there is a delay of more than 0.1 second during passage of the cardiac impulse from the atria into the ventricles. This allows the atria to contract ahead of ventricular contraction, thereby pumping blood into the ventricles before the strong ventricular contraction begins. Thus, the atria act as *primer pumps* for the ventricles, and the ventricles in turn provide the major source of power for moving blood through the body's vascular system (Bers, 2002).

2-2-9Function of the Ventricles as Pumps

2-2-9-1Filling of the Ventricles.

During ventricular systole, large amounts of blood accumulate in the right and left atria because of the closed A-V valves. Therefore, as soon as systole is over and the ventricular pressures fall again to their low diastolic values, the moderately increased pressures that have developed in the atria during ventricular systole immediately push the A-V valves open and allow blood to flow rapidly into the ventricles,. This is called the period of rapid filling of the ventricles. The period of rapid filling lasts for about the first third of diastole. During the middle third of diastole, only a small amount of blood normally flows into the ventricles; this is blood that continues to empty into the atria from the veins and passes through the atria directly into the ventricles. During the last third of diastole, the atria contract and give an additional thrust to the

inflow of blood into the ventricles; this accounts for about 20 per cent of the filling of the ventricles during each heart cycle (Bers, 2002) .

2-2-10-1-1Period of Isovolumic (Isometric) Contraction:

Immediately after ventricular contraction begins, the ventricular pressure rises abruptly, as shown in Figure 9–5, causing the A-V valves to close. Then an additional 0.02 to 0.03 second is required for the ventricle to build up sufficient pressure to push the semilunar (aortic and pulmonary) valves open against the pressures in the aorta and pulmonary artery. Therefore, during this period, contraction is occurring in the ventricles, but there is no emptying. This is called the period of isovolumic or isometric contraction, meaning that tension is increasing in the muscle but little or no shortening of the muscle fibers is occurring (Bers, 2002) .

2-2-9-1-2 Period of Ejection

When the left ventricular pressure rises slightly above 80 mm Hg (and the right ventricular pressure slightly above 8 mm Hg), the ventricular pressures push the semilunar valves open. Immediately, blood begins to pour out of the ventricles, with about 70 per cent of the blood emptying occurring during the first third of the period of ejection and the remaining 30 per cent emptying during the next two thirds. Therefore, the first third is called the period of rapid ejection, and the last two thirds, the period of slow ejection (Kass, 2002) .

2-2-9-1-3Period of Isovolumic (Isometric) Relaxation

At the end of systole, ventricular relaxation begins suddenly, allowing both the right and left Interventricular pressures to decrease rapidly. The elevated pressures in the distended large arteries that have just been filled with blood

from the contracted ventricles immediately push blood back toward the ventricles, which snaps the aortic and pulmonary valves closed. For another 0.03 to 0.06 second, the ventricular muscle continues to relax, even though the ventricular volume does not change, giving rise to the period of isovolumic or isometric relaxation. During this period, the intraventricular pressures decrease rapidly back to their low diastolic levels. Then the A-V valves open to begin a new cycle of ventricular pumping (Kass, 2002).

2-2-10End-Diastolic Volume, End-Systolic Volume, and Stroke Volume Output:

During diastole, normal filling of the ventricles increases the volume of each ventricle to about 110 to 120 milliliters. This volume is called the end-diastolic volume. Then, as the ventricles empty during systole, the volume decreases about 70 milliliters, which is called the stroke volume output. The remaining volume in each ventricle, about 40 to 50 milliliters, is called the end-systolic volume. The fraction of the end-diastolic volume that is ejected is called the ejection fraction— usually equal to about 60 per second (Kass, 2002) .

2-2-11Function of the Ventricles as Pumps

2-2-11-1Filling of the Ventricles:

During ventricular systole, large amounts of blood accumulate in the right and left atria because of the closed A-V valves. Therefore, as soon as systole is over and the ventricular pressures fall again to their low diastolic values, the moderately increased pressures that have developed in the atria during ventricular systole immediately push the A-V valves open and allow blood to flow rapidly into the ventricles, as shown by the rise of the left *ventricular volume curve*. This is called the *period of rapid filling of the ventricles.* The period of rapid filling lasts for about the first third of diastole. During the

middle third of diastole, only a small amount of blood normally flows into the ventricles; this is blood that continues to empty into the atria from the veins and passes through the atria directly into the ventricles. During the last third of diastole, the atria contract and give an additional thrust to the inflow of blood into the ventricles; this accounts for about 20 per cent of the filling of the ventricles during each heart cycle (Kass, 2002) . .

2-2-11-2Emptying of the Ventricles During Systole Period of Isovolumic (Isometric) Contraction:

Immediately after ventricular contraction begins, the ventricular pressure rises abruptly, causing the A-V valves to close. Then an additional 0.02 to 0.03 second is required for the ventricle to build up sufficient pressure to push the semilunar (aortic and pulmonary) valves open against the pressures in the aorta and pulmonary artery. Therefore, during this period, contraction is occurring in the ventricles, but there is no emptying. This is called the period of *isovolumic* or *isometric contraction,* meaning that tension is increasing in the muscle but little or no shortening of the muscle fibers is occurring (Kass, 2002) .

2-2-11-3Period of Ejection

When the left ventricular pressure rises slightly above 80 mm Hg (and the right ventricular pressure slightly above 8 mm Hg), the ventricular pressures push the semilunar valves open. Immediately, blood begins to pour out of the ventricles, with about 70 per cent of the blood emptying occurring during the first third of the period of ejection and the remaining 30 per cent emptying during the next two thirds. Therefore, the first third is called the period of rapid ejection, and the last two thirds, the period of slow ejection. (Kass, 2002) .

2-2-11-4 Period of Isovolumic (Isometric) Relaxation.

At the end of systole, ventricular relaxation begins suddenly, allowing both the right and left intraventricular pressures to decrease rapidly. The elevated pressures in the distended large arteries that have just been filled with blood from the contracted ventricles immediately push blood back toward the ventricles, which snaps the aortic and pulmonary valves closed. For another 0.03 to 0.06 second, the ventricular muscle continues to relax, even though the ventricular volume does not change, giving rise to the period of isovolumic or isometric relaxation. During this period, the intraventricular pressures decrease rapidly back to their low diastolic levels. Then the A-V valves open to begin a new cycle of ventricular pumping (Kass, 2002) . .

2-2-11-5 End-Diastolic Volume, End-Systolic Volume, and Stroke Volume Output:

During diastole, normal filling of the ventricles increases the volume of each ventricle to about 110 to 120 milliliters. This volume is called the *end-diastolic volume.* Then, as the ventricles empty during systole, the volume decreases about 70 milliliters, which is called the *stroke volume output.* The remaining volume in each ventricle, about 40 to 50 milliliters, is called the *end-systolic volume.* The fraction of the end-diastolic volume that is ejected is called the *ejection fraction*— usually equal to about 60 per cent. When the heart contracts strongly, the end-systolic volume can be decreased to as little as 10 to 20 milliliters. Conversely, when large amounts of blood flow into the ventricles during diastole, the ventricular end diastolic volumes can become as great as 150 to 180 milliliters in the healthy heart. By both increasing the end-diastolic volume and decreasing the end-systolic volume the stroke volume output can be increased to more than double normal (Despopoulos, 2003) .

2-11-6 Aortic Pressure Curve

When the left ventricle contracts, the ventricular pressure increases rapidly until the aortic valve opens. Then, after the valve opens, the pressure in the ventricle rises much less rapidly, because blood immediately flows out of the ventricle into the aorta and then into the systemic distribution arteries (Despopoulos, 2003) .

2-11-7Diastole and Systole

The cardiac cycle consists of a period of relaxation called *diastole*, during which the heart fills with blood, followed by a period of contraction called *systole.* The *stroke work output* of the heart is the amount of energy that the heart converts to work during each heartbeat while pumping blood into the arteries. *Minute work output* is the total amount of energy converted to work in 1 minute; this is equal to the stroke work output times the heart rate per minute. Work output of the heart is in two forms. First, by far the major proportion is used to move the blood from the low-pressure veins to the high-pressure arteries. This is called volume-pressure work or external work.

Second, a minor proportion of the energy is used to accelerate the blood to its velocity of ejection through the aortic and pulmonary valves. This is the kinetic energy of blood flow component of the work output. Right ventricular external work output is normally about one sixth the work output of the left ventricle because of the six fold difference in systolic pressures that the two ventricles pump (Despopoulos, 2003) .

2-2-12Heart Sound (Guyton2006) :

There are four sound result from turbulence of blood with in the heart during closure of valves or rapid filling of ventricles.

2-2-12-1The first heart Sound (S1):

Caused by closure of AV valves, occurs at the beginning of ventricular systole or as the start of isovolumetric contraction phase and it low pitched and more prolonged than S2.

2-2-12-2 The second hear sound (S2):

Caused by closer of semilunar valve and occurs at the start of ventricular diastole or as isovolumetric relaxation phase it high pitch and takes less time than S1. It may split into two components, aortic S2 and pulmonary S2 in normal state closure of aortic valve precedes closure of pulmonary valve.

2-2-12-3Third heart sound (S3):

Caused by rapid ventricular filling

2-2-12-4 Fourth heart sound (S4):

It abnormal sound it know as atrial sound, it caused by atrial systole in patients suffering from hypotension. (Merghani 2013)

2-3Pathogy

2-3-1Hypertension:

Cardiovascular diseases are major leading health problems in the world, and hypertension is a major risk factor for cardiovascular diseases and stroke which have significantly higher morbidity and mortality In 95% of cases of hypertension there is no detectable cause; such patients are said to have primary or essential hypertension. In the remaining cases, hypertension is secondary to an underlying condition, often renal disease, alcohol misuse or occasionally, an endocrine disorder (Murries, 2006)

2-3-1-1Benign Hypertension

Benign hypertension causes, by IHD, heart failure, stroke, acceleration of renal disease, malignant hypertension. Benign hypertension is usually asymptomatic and most cases are discovered when pressure is measured at a

routine medical examination, often in middle age. Blood pressure rises slowly over many years, usually only to moderately high levels, for example 180/100mmHg. The elderly may develop a form with disproportionately high systolic pressure, systolic hypertension that is probably due to arterial disease. Benign hypertension affects the heart and arteries of all sizes. The main target organs are the heart, brain and kidneys. The commonest complication is IHD including heart failure, accounting for about 60% of deaths, and another 30% die of stroke. Benign hypertension causes changes in the kidney, and hypertension is a major aggravating factor in patients with renal diseases. However, renal failure is uncommon and only affects those at the most severe end of the spectrum. Increased pressure load causes hypertrophy of arteries and the heart. The muscle hypertrophy normalizes the workload of individual cardiac myocytes and arterial smooth The changes of arteriosclerosis which ensue are more severe and occur earlier than the age changes . These permanent structural changes in the arterial tree perpetuate hypertension regardless of its aetiology. The longitudinal and circumferential stretching of arteries, if severe, may cause dilatation of the aortic root and rarely, leakage (incompetence) of the aortic valve (Murries, 2008) .

Longstanding hypertension aggravates atherosclerosis and contributes to the development and rupture of aneurysms. In the heart, left ventricular hypertrophy impairs diastolic function; the thickened ventricle is stiffer due to both increased muscle mass and interstitial fibrosis. As the ventricle is only perfused during diastole, coronary artery. In addition, people with hypertension are likely to have coronary atherosclerosis. In addition, the increased oxygen demand of the hypertrophied left ventricle contributes to ischaemia, even at rest. Thus, left ventricular hypertrophy causes increased

mortality, proportional to the degree of hypertrophy, from cardiac arrhythmias and myocardial infarction.(Thorner, 2002) .

2-3-1-2Malignant Hypertension

Malignant hypertension is defined clinically as hypertension (diastolic blood pressure _130 mmHg) together with retinal changes of bilateral flame-shaped haemorrhages and/or papilloedema. In about 50% of cases malignant hypertension develops without a preceding history of hypertension; fewer than 5% of patients with benign essential hypertension develop the malignant phase. It is commoner in the black races and in those with secondary hypertension, especially if caused by renovascular disease. Malignant hypertension usually affects younger hypertensives; *de novo* cases usually present at 30–40 years (Thorner, 2002) .

Patients with malignant hypertension are ill. If they survive the usual complications of raised blood pressure – heart failure and cerebral haemorrhage – renal failure becomes universal and is the commonest cause of death in untreated cases. (Murries, 2008) .

2-3-1-3Pathology and Pathogenesis of the Arterial Lesions:

Failure of auto regulation in the kidney transmits increased pressure to Glomerular causing fibrinoid necrosis and micro aneurysms of Glomerular capillaries. The pressure also causes fibrinoid necrosis of afferent Glomerular arterioles which may rupture or be associated with luminal thrombosis resulting in small infarcts. Thrombosis causes damage to red blood corpuscles – microangiopathic haemolytic anaemia. the incidence of hypertension and six times the mortality. Females have a higher incidence of hypertension, but mortality in males is 1.5–2 times the mortality of females (**GEORGE, 2009)**

2-3-1-4 Secondary Hypertension

Renal disease is 10 times commoner than all other causes together. Hypertension is caused by disorders of the renal blood vessels (renovascular hypertension), many renal diseases (renal parenchymal hypertension) or simply to loss of nephrons in renal failure (**GEORGE, 2009)**

2-3-2 Ischemic Heart Disease (**GEORGE 2009)**:

Atherosclerosis is the leading cause of morbidity and mortality in developed countries around the world, the major risk factors are **cigarette** smoking, Hypertension, Dyslipidemia, Diabetes mellitus, and Family history. Minor risk factors include are Male gender, Obesity, Hyperhomo cysteinemia and Increased levels of low-density lipoprotein (LDL). (**GEORGE, 2009)**

2-3-3Acute Coronary Syndrome/Unstable Angin

The pathologic lesion of unstable angina is atherosclerotic plaque rupture, which leads to thrombosis and incomplete occlusion of an epicardial coronary artery**.** The presentation of acute coronary syndrome is similar to acute myocardial infarction. (**GEORGE, 2009)**

2-3-4 Acute Myocardial Infarction

Symptoms depend on the location and severity of the infarction, the most common presentation is acute chest pain (angina). Ischemia may cause cardiogenic shock, congestive heart failure (CHF), and pulmonary edema.

If critical conductive areas are ischemic, then an arrhythmia can result, such as ventricular tachycardia or fibrillation. Ischemia of the papillary muscles can lead to acute mitral valvular insufficiency and CHF, it can present as sudden cardiac death. Arrhythmia can occur at presentation or as a late complication due to conduction system disturbance, or myocardial damage resulting in scarring and electrical impulse reentry. Altered **contractility** with pump failure due to myocardial ischemia, especially when large amounts of myocardium are ischemic, such as with left main coronary artery disease.

Mitral valve insufficiency due to papillary muscle dysfunction can lead to acute CHF. Myocardial rupture can occur, usually in non-hypertrophic myocardium, 3–7 days after an infarct due to weakening of the infarcted tissue, so that Right ventricular infarction can occur when the right coronary or posterior descending artery are involved in a posterior wall infarct, sometimes presenting with hypotension and elevated jugular venous pressure. **(Lind, 2001)** .

2-3-5 Chest Pain (Lind 2001):

The presence of specific physical findings can help in the formation of a useful differential diagnosis and guide therapy are Inspiratory crackles may indicate pulmonary edema, A new murmur may be indicative of valvular insufficiency, An S3 gallop indicates left ventricular dysfunction, A pleural or pericardial rub can point to pleuritis or pericarditis, It is important to remember that a normal physical examination does not exclude potentially serious. The main causes of chest pain is Ischemia can only be ruled out by serial ancillary tests (serum markers, electrocardiogram (ECG), stress test, and angiography) **(GEORGE, 2009)**.

2-3-5Ischemic Heart diseases:

Ischemic heart disease and acute coronary syndromes are the leading cause of cardiac morbidity and mortality, and knowledge of the cardiac blood supply is a requisite for optimal assessment of echocardiographic findings. This is especially true for wall motion assessment. Ventricular walls that demonstrate abnormal movement and thickening, a kinetic, dyskinetic, aneurysmal, or thinned (scarred) walls, most commonly do so because of insults to the corresponding coronary artery supply. **(GEORGE, 2009)**

2-3-6Left ventricular hypertrophy:

Is common among patients with hypertension, with an estimated prevalence of more than 20% depending on the measure used, and is an important independent risk factor for cardiovascular complications **(Sandstorms 2001).** Blood pressure reduction reduces cardiovascular morbidity and morbidity in patients with documented left ventricular hypertrophy. Left ventricular mass measurement by transthoracic 2D echocardiography is a valid, serially reproducible and clinically applicable modality with high inter-study variability. With meticulous technique under study conditions, inter-study variability can be reduced to less than 34g and to as little as 25g (10%) with the newer 3D transthoracic echocardiography technique (Dahlof 2002) Nevertheless, echocardiographically derived left ventricular mass measurements remain too difficult to accurately reproduce in the day-to-day clinical setting to allow clinically relevant therapeutic regression of ventricular hypertrophy to be tracked in a given patient. Furthermore, prospective randomized trial data specifically assessing the outcomes associated with and cost effectiveness of routine echocardiography are still lacking is therefore the preferred technique. (Devereux, 2004)

2-3-7Valves diseases:

Infection of the valve tissue (endocarditis) can affect these connections thereby causing leaflet or cuspal dehiscence, or it can lead to an infection of the annulus itself (e.g. valve ring abscess). The proximity of the annulae of the 3 valves means that infection can spread readily from one to the other **(GEORGE, 2009)** .

2-3-7-1Aortic valve:

Dilatation of the root of the aorta, the area immediately superior to the valve, may occur with connective tissue disease (e.g. Marfan’s syndrome),

atherosclerosis, or inflammation, and may lead to aortic valvular insufficiency. **(GEORGE, 2009)** .

2-4 Imaging modalities used in diagnosis of heart diseases:

2-4-1 Echocardiography:

Echocardiographic images are acquired by placing the transducer (with acoustic coupling gel) at specific locations on the chest or abdominal wall they are; Left parasternal position or window, Left apical position or window, Subcostal (SC), and Suprasternal (SSN) position or window. Individual patient characteristics and the clinical indications for the examination will dictate whether all windows are used, or if additional nonstandard windows should be exploited. In adults with congenital heart disease or following chest wall surgery, use whatever windows are available that provide optimal visualization of the desired views. The right parasternal window (R-PLAX) mirrors that of the left. It can provide optimal assessment of peak blood flow velocities in patients with aortic stenosis because of its more parallel alignment with left ventricular and aortic outflow tracts. **(GEORGE, 2009)**

A comprehensive transthoracic echocardiographic examination requires assessment using multiple views or perspectives. Composite views deliver a three dimensional (3D) perspective, and as a general rule, this is the requisite standard for accurate assessment of cardiac structure and function. However, the air-filled lungs and the bony chest wall are the greatest obstacles to the transmission of ultrasound beam. This imposes the need for “windows” to avoid the lungs and bony ribs, thereby optimizing visualization of cardiac structures . Although these echocardiographic windows are a useful starting point, the final “decider” is to use those window(s) that provide optimal visualization of the cardiac structures of interest. Experienced cardiac sonographer rapidly scan the chest wall using broad transducer strokes to get

a quick impression of which windows most readily yield the best views, The standard echocardiographic windows or transducer positions. Because the bony chest wall and lungs act as major obstacles to ultrasound beam transmission, paths that minimize such interference serve as echocardiographic imaging "windows" to the heart. The limitations imposed by the bony chest wall do not apply to echocardiography in the fetus and the new born infant. (Devereux, 2002)

2-4-1-1Examination Protocol:

The comprehensive transthoracic echocardiography examination begins at the left parasternal window, followed by the apical, subcostal, and suprasternal windows (Devereux, 2002)

2-4-1-2Clinical Value of Echocardiography

Echocardiography is the most widely used cardiac imaging technique. It has a clinically proven role in the diagnosis and management of patients with suspected or established cardiovascular disease. Echocardiography is the most versatile cardiac imaging technique. It can provide a wide range of clinically useful information in a variety of settings, and at less cost, with more rapid results, as compared to other cardiac imaging techniques Highly portable battery-powered instruments, known as hand-carried ultrasound (HCU), can be used outside the hospital setting in locations such as community clinics, resource-poor environments, ambulances, and aircraft (even spacecraft). Up-and-coming pocket-sized devices have been dubbed as the "ultrasound stethoscopes" of the future **(GEORGE, 2009)** .

2-4-1-3Anatomical Versus Echocardiographic Image Planes:

The major axis of the heart, the long axis of the left ventricle (LV), is obliquely positioned and typically rotated ~60° away from the median plane in normal adults. This axis projects along a line extending from the right mid-clavicle to

the palpable LV apical impulse *(upper left)*. This major cardiac axis serves as the anatomical reference for the standard echocardiographic imaging planes: long-axis (LAX), short-axis (SAX), and four-chamber (4C) planes *(bottom right)*. The heart, pericardium, and great vessels occupy the middle mediastinum—the portion of the chest cavity that lies in the midline between the two lungs and inferior to the palpable manubriosternal angle . The lungs enwrap the heart (and pericardium) laterally and over much of its anterolateral and posterolateral surfaces. Superiorly the great vessels—superior vena cava (SVC), ascending aorta, aortic arch and branches, and the main pulmonary artery (MPA) and bifurcation—enter and exit. Inferiorly, the heart sits on the diaphragm, through which passes the inferior vena cava (IVC) as it enters the floor of the right atrium (Ibsen, 2002) .

2-4-1-4The heart's anterior or sternocostal surface is related to these structures:

the sternum and the costal cartilages (ribs). Posteriorly, the heart's left atrium, which receives a pair of right and left pulmonary veins, lies immediately anterior to the esophagus (Ibsen, 2002) ..

2-4-1-5Echocardiographic imaging plane:

long-axis (LAX), short-axis (SAX), or four-chamber (4C). Cardiac structures or regions of interest are, left ventricular inflow outflow, right ventricular inflow, or aortic valve level. At each window, the normal examination protocol is to perform are 2D examination **to** Optimize and acquire each view. Obtain linear and volumetric measures where applicable. Assess normal and abnormal. cardiac structure and function as the examination proceeds. Confirm findings in subsequent views as the examination proceeds. Use this modality to time cardiac events and structures of interest. Perform linear and derived measurements where applicable, Visualize "angiographic" blood flow

velocities and flow patterns within cardiac chambers, the great vessels, and across heart valves . **Spectral pulsed wave (PW) and continuous-wave (CW) Doppler examination to** Quantify blood flow velocities within cardiac chambers, the great vessels, and across heart valves . **Tissue Doppler imaging (TDI):** PW TDI to the mitral annulus to quantify myocardial tissue velocities at specific regions . (Ibsen, 2002) .

2-4-1-6 Ultrasound contrast agents:

Use where indicated, e.g., to improve endocardial The normal sequence of the adult transthoracic examination is as, **Left Parasternal Views:** Parasternal long-axis (PLAX); right ventricular (RV) inflow 6 RV outflow; parasternal short-axis (PSAX) views, **Apical Views:** Apical 4-chamber (A4C); apical 2-chamber (A2C); apical long-axis (ALAX) or apical 3-chamber (A3C) views, **Subcostal Views:** Subcostal 4-chamber (SC-4C); inferior vena cava (IVC); abdominal aorta (Abd. A) views and **Suprasternal Notch Views (SSN):** Suprasternal long-axis view of the aortic arch, Left parasternal window, transducer scan planes, and views. From the left parasternal position, a family of long-axis and short-axis views are obtained by sweeping (or angling) the transducer along the cardiac long axis and short axis as shown The following standard parasternal views are obtained(Ibsen, 2002) .

Parasternal long-axis (PLAX) view of the left ventricular (LV) inflow and outflow tracts . Parasternal long-axis (PLAX) view of the right ventricular (RV) inflow tract, hereafter called the RV inflow view. Parasternal long-axis (PLAX) of the right ventricular (RV) outflow tract, hereafter called the RV outflow view . The parasternal short-axis (PSAX) views—at multiple short-axis levels, beginning with the PSAX view at the level of the aortic valve (PSAX-AVL), at the level of the pulmonary artery bifurcation (PSAXPAB),

the level of the mitral valve (PSAX-MVL); the mid-LV level or papillary muscle level (PSAX-PML), and at the level of the LV apical segments (PSAX-apical level), including the apical cap of the LV. The PLAX view is where the adult transthoracic examination begins. This is because the primarily landmark cardiac structures—the right ventricle (RV), left ventricle (LV), aortic root (Ao), and left atrium (LA), and the mitral and aortic valves––can be readily aligned along the cardiac long axis in the PLAX view.(Ibsen, 2002)

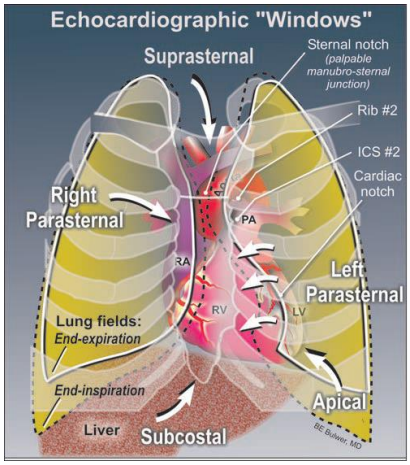

Figure (2-2) Land marks of echocardiography , (Ibsen, 2002)

For the parasternal window, the patient lies in the left lateral position with the left arm behind head. The acoustic window is situated in the fourth intercostal space just to the left of the sternum. (Thomath, 2006).

For the apical view (with the patient once again in the left lateral position) the beam is directed from the apical impulse. For the suprasternal window the patient lies in the supine position. The beam is directed from the suprasternal notch toward the aortic arch, For the subcostal window (with the patient once again in the supine position), the heart is imaged from below. Parasternal long-axis view: Coming from the fourth intercostal space just left of the sternum (the window/orifice allowing free access past the lung is merely the size of a

postage stamp) the transducer is aimed perpendicularly toward the spine , (Ibsen, 2002)

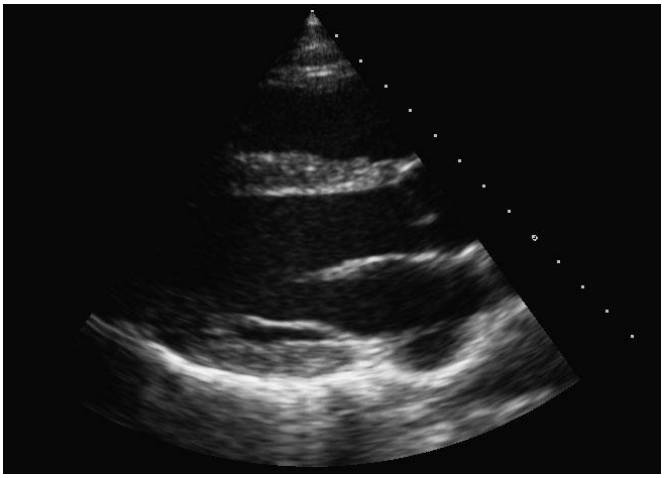

Figure (2-3) The right ventricle is displayed at the top and the left cardiac structures below (Ibsen 2002)

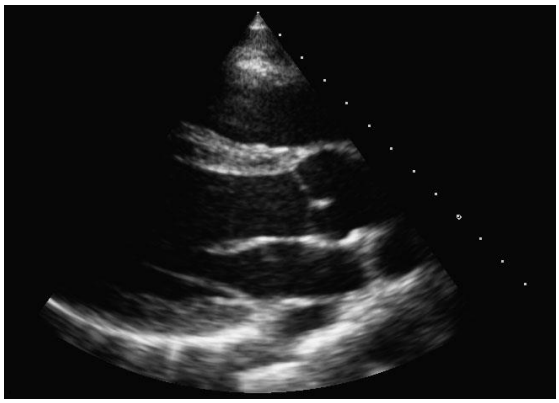

Figure (2-4) The aortic bulb seen just at the right of the center of the image can be used to check orientation; beneath it is the mitral valve and to its left is the left ventricle (Devereux, 2002) .

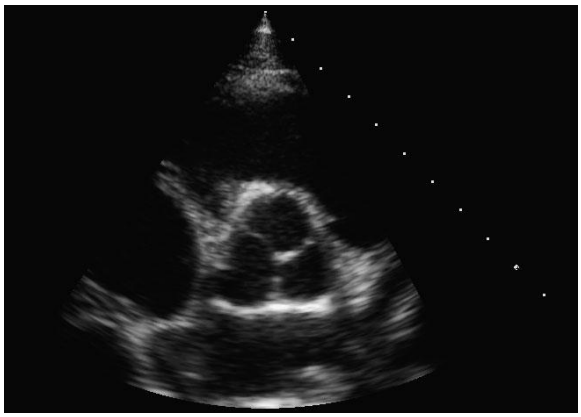

Figure (2-5) Parasternal short axis at the level of the aortic valve (Bulwer, 2011)

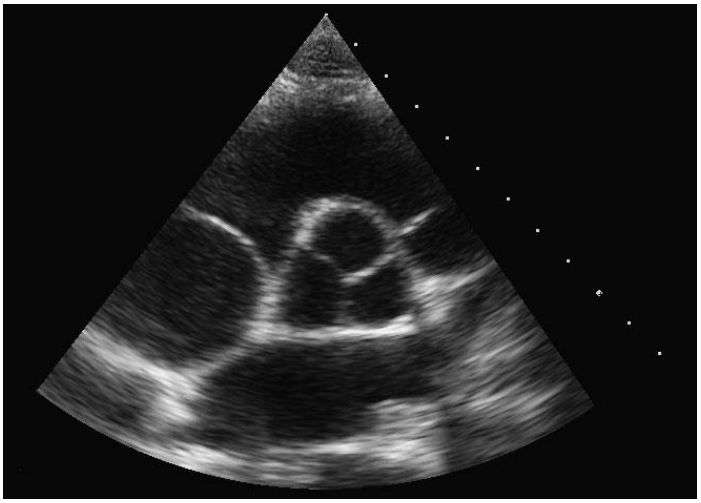

Figure (2-6) Short-axis parasternal view of the aortic valve in the center surrounded by adjacent right cardiac structures (Bulwer, 2011)

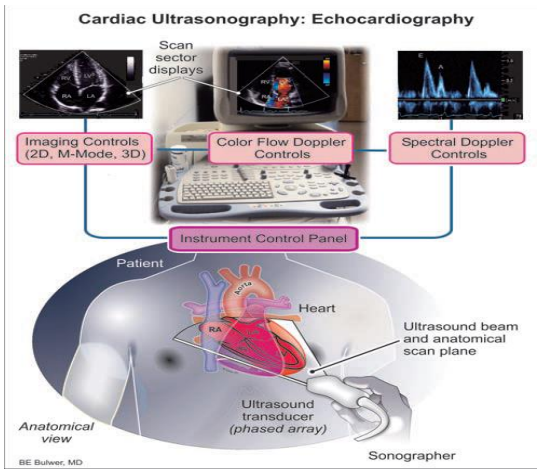

Figure (2-7) echocardiography—To create the ultrasound image, ultrasound must be transmitted, reflected, processed, and displayed (Bulwer, 2011)

2-4-2Transoesophageal and stress echo and other echo techniques:

The echo techniques described so far have used ultrasound directed from the chest wall – transthoracic echo (TTE). The oesophagus in its mid-course lies posterior to and very close to the heart and ascending aorta and anterior to the descending aorta. An echo technique exists for examining the heart with a transducer in the oesophagus – transoesophageal echo (TOE). In some countries, the abbreviation used is TEE. This uses a transducer mounted upon a modified probe similar to those used for upper gastrointestinal endoscopy and allows examination of the heart without the barrier to ultrasound usually provided by the ribs, chest wall and lungs. By advancing the probe tip to various depths in the oesophagus and stomach, manoeuvring the tip of the transducer and by altering the angle of the ultrasound beam with controls placed on the handle, a number of different views of the heart can be obtained (Devereux, 2002) .

2-5 Previous studies:

Hong-Won (2014) et al they study in Left ventricular (LV) twist is usually influenced by LV hypertrophy resulting from hypertension or vascular stiffness. Vascular stiffness would increase arterial elastance (Ea), whereas LV end-systolic stiffness (Ees) could be influenced by LV hypertrophy. Therefore, in hypertensive patients, we assessed the extent to which ventricular–arterial coupling (VAC; Ea/Ees) affects LV twist, which may be a compensatory mechanism for systolic dysfunction. They used 28 Hypertensive patients (n = 128) and healthy controls (n = 40) underwent conventional and speckle tracking echocardiography including LV twist. Ea and Ees were estimated noninvasively by echocardiography. Patients were divided into 3 tertiles according to the twist angle. Univariate and multivariate

regression analyses were performed to test the influence of VAC on twist. They found that Patients in the lowest LV twist tertile had larger LV end-systolic volume, lower ejection fraction, lesser mid-wall fractional shortening (MWFS), and higher LV mass index (LVMI), compared to those with the highest tertile. They showed the lower septal tissue Doppler velocity, and global longitudinal and circumferential strain. With regard to VAC, Ea was similar among 3 groups, but Ees was significantly decreased in patient with lower tertile, resulting in increased VAC (1.1 ± 0.2 vs. 0.9 ± 0.1 vs. 0.7 ± 0.1, $P < 0.001$). While LV twist showed significant correlations with Ees, MWFS, and LVMI, VAC ($\beta = -14.92$, $P < 0.001$) was most associated with twist in a multivariate analysis.

LV twist was significantly associated with VAC in accordance with LV function; LV twist and VAC decreased progressively as LV systolic function deteriorated, while being enhanced during the well-compensated phase.

Możdżan et al 2013 Long-lasting arterial hypertension causes left ventricular hypertrophy (LVH) and impairs left ventricular diastolic function. The aim of this study to compare echocardiographic parameters between hypertensive patients defined as dippers and non-dippers during ambulatory blood pressure (BP) monitoring. Data were collected from the medical records of 76 treated patients with essential hypertension who underwent ambulatory BP monitoring and transthoracic echocardiographic examination at the Department of Cardiology during a period of 14 months (from April 2009 to June 2010). Treatment was individualized and based on lifestyle and pharmacological measures. Diuretics (43%), β-blockers (34%), angiotensin-converting enzyme inhibitors (ACEI – 86%), angiotensin receptor blockers (ARB – 26%) and calcium channel blockers (38%), alone or combined, were

the antihypertensive drugs most frequently used. Hypertensive subjects can be divided into 2 groups, dippers and non-dippers, according to the presence or absence of a nocturnal fall of blood pressure of more than 10%, measured as mean arterial pressure (MAP). The MAP was defined as MAP = 2/3 × DBP + 1/3 × SBP. Inclusion criteria were well-controlled hypertension with preserved left ventricular ejection fraction (EF ≥ 50%) and exclusion criteria were: severe hypertension (systolic BP (SBP)] ≥ 210 mm Hg and/or diastolic BP (DBP) ≥ 115 mm Hg), secondary hypertension, significant kidney disease, valvular heart disease, heart failure and history of ischaemic heart disease. Among the patients with preserved left ventricular ejection fraction (EF ≥ 50%), 35 non-dippers (60 ±12 years, 24 males) and 26 dippers (57 ±13 years, 16 males) matched for age, sex, and body mass index were enrolled in this study. The control group consisted of 25 healthy individuals (53 ±12 years, 13 males). Echocardiographic and arterial pressure parameters were compared between dippers and non-dippers. As the result of analysed 61 consecutive subjects with treated hypertension undergoing 24-h BP monitoring and transthoracic echocardiographic examination and included in the study patients with preserved left ventricular ejection fraction (EF ≥ 50%). Echocardiographic and arterial pressure parameters were compared between the group classified as dippers (n = 26, 57 ±13 years, 16 males) and non-dippers (n = 35, 60 ±12 years, 24 males) according to present or absent decrease of BP during the night > 10%. Echocardiographic data compared between both groups and control subjects without hypertension. The main finding that Dippers had lower average systolic, diastolic and mean arterial pressure during the night hours but did not differ according to the mean pressure calculated from a 24-hour period. All echocardiographic parameters were similar in dippers and non-dippers. All patients with arterial

hypertension presented with larger dimension of both ventricles and left atrium, thicker left ventricular walls, higher LV mass and mass index and preserved EF and E/A ratio as compared with normotensive controls. Normal geometry, concentric remodeling and eccentric hypertrophy were similarly distributed in both groups. Concentric hypertrophy was more prevalent in non-dippers as compared to the dippers (71.4% vs. 38.5%, $p < 0.043$). The concentric type of LVH is the prevalent pattern in non-dippers. Non-dipping blood pressure pattern may be responsible for the development of left ventricular concentric hypertrophy secondary to hypertension.

Ayoub et al 2014 Early detection of subclinical left ventricular (LV) systolic dysfunction in hypertensive patients is important for the prevention of progression of hypertensive heart disease. We studied 60 hypertensive patients (age ranged from 21 to 49 years, the duration of hypertension ranged from 1 to 18 years) and 30 healthy controls, all had preserved left ventricular ejection fraction (LVEF), detected by two-dimensional speckle tracking echocardiography (2D-STE). There was no significant difference between the two groups regarding ejection fraction (EF) by Simpson's method. Systolic velocity was significantly higher in the control group, and global longitudinal strain was significantly higher in the control group compared with the hypertensive group. In the hypertensive group, 23 of 60 patients had less negative global longitudinal strain than −19.1, defined as reduced systolic function, which is detected by 2D-STE (subclinical systolic dysfunction), when compared with 3 of 30 control subjects. 2D-STE detected substantial impairment of LV systolic function in hypertensive patients with preserved LVEF, which identifies higher risk subgroups for earlier medical intervention.

Gerdts 2008 We assessed LV geometric patterns on baseline and annual echocardiograms as time-varying predictors of the primary composite endpoint (cardiovascular death, stroke, and myocardial infarction) in 937 hypertensive patients with LV hypertrophy during 4.8 years losartan- or atenolol-based treatment in the Losartan Intervention for Endpoint reduction in hypertension (LIFE) echocardiography substudy. LV geometry was determined from LV mass/body surface area and relative wall thickness in combination. At end of the study, 52% of patients with initial LV hypertrophy had normal geometry ($P < 0.001$). In particular, concentric remodelling was reduced by 82% and concentric LV hypertrophy by 84%. Development of LV hypertrophy was seen in <5%. In Cox regression analyses including LV geometric patterns as time-varying variables and adjusting for treatment, Framingham risk score, race, and time-varying systolic blood pressure, the patterns independently predicted higher risk of primary composite endpoints [HR 2.99 (1.16–7.71) for concentric remodelling, HR 1.79 (1.17–2.73) for eccentric hypertrophy, and HR 2.71 (1.13–6.45) for concentric hypertrophy; all $P < 0.05$]. In hypertensive patients with ECG LV hypertrophy, in-treatment LV geometry by echocardiography adds information on risk of cardiovascular events. Hypertension, Left ventricular geometry, Left ventricular hypertrophy, Left ventricular (LV) hypertrophy is a cardinal manifestation of preclinical cardiovascular disease that strongly predicts cardiovascular events in hypertensive patients as well as in the general population

Hussein 2015 Diabetes (DM) and hypertension (HT) cause changes in cardiac performance. Long-term diabetes and hypertension can lead to changes in cardiac contractility, reduced left ventricular efficiency and heart failure. The

aim of this study is to evaluate the effect of the coexistence of diabetes mellitus and hypertension on left ventricular myocardial performance and structural changes. The study involved 45 patients with essential hypertension and type 2 diabetes (14 males and 31 females, their mean age was 53.28 ± 13.28 years), and 45 healthy subjects (10 males and 35 females, their mean age was 48.11 ± 13.07 years) as a control group. Transthoracic echocardiography was done for all patients. The echocardiographic measurements included: left ventricle internal diameter at end diastole (LVIDd), left ventricle internal diameter at end systole (LVIDs), peak velocity of early transmitral flow (E), peak velocity of late transmitral flow (A), ejection fraction (EF%), isovolumic relaxation time (IVRT), isovolumic contraction time (IVCT) and ejection time (ET) from which the index of myocardial performance (IMP) was calculated. Results revealed a significant change in the ratio (E/A) between patients and controls (-32.45%) with p value < 0.05, and the change in (LVIDd) and (LVIDs) between patients and control groups were (4.61%) and (0.754%) respectively with insignificant p value. The change in IMP was (44.65%), with *p* value < 0.05, and the change in ejection fraction (EF%) was (-1.49) with p value > 0.05. In conclusion, diabetic patients with hypertension had an increase in IMP and reduced E/A indicating deterioration in cardiac performance despite normal ejection fraction and insignificant change in LV dimensions.

Mottram 2013 To examine the relation of arterial compliance to diastolic dysfunction in hypertensive patients with suspected diastolic heart failure (HF). 70 medically treated hypertensive patients with exertional dyspnoea (40 women, mean (SD) age 58 (8) years) and 15 normotensive controls. Main outcome measures**:** Mitral annular early diastolic velocity with tissue Doppler

imaging and flow propagation velocity were used as linear measures of diastolic function. Arterial compliance was determined by the pulse pressure method. presents the characteristics of the study participants. Women slightly outnumbered men. Hypertensive patients were overweight compared with controls. In the group as a whole, diastolic blood pressure was better controlled than systolic pressure. The majority of patients were taking vasodilator medication, which consisted of a calcium channel blocker for 42 patients, an angiotensin converting enzyme inhibitor for six patients, and prazosin for two patients. Diuretic medication was either thiazide or loop diuretics, with no patients taking aldosterone antagonists. Relative wall thickness and LV mass indexed to height were increased in hypertensive patients, as was left atrial area. As a result of the increased body mass index in hypertensive patients, LV mass indexed to body surface area in this group was similar to that of controls. Among the 70 patients with hypertension, conventional Doppler echocardiography showed normal diastolic function in 33 patients and abnormal diastolic function in 37 patients (22 women). The majority of those with abnormal diastolic function had an impaired relaxation pattern (n = 28), whereas a pseudonormal LV filling profile was documented in nine patients. Table 22 presents the clinical and echocardiographic variables according to category of diastolic function. According to conventional Doppler echocardiography of transmitral and pulmonary venous flow, diastolic function was classified as normal in 33 patients and abnormal in 37 patients. Of those with diastolic dysfunction, 28 had mild (impaired relaxation) and nine had advanced (pseudonormal filling) dysfunction. Arterial compliance was highest in controls (mean (SD) 1.32 (0.58) ml/mm Hg) and became progressively lower in patients with hypertension and normal function (1.04 (0.37) ml/mm Hg), impaired relaxation (0.89 (0.42) ml/mm

Hg), and pseudonormal filling (0.80 (0.45) ml/mm Hg, $p = 0.011$). In patients with diastolic dysfunction, arterial compliance was inversely related to age ($p = 0.02$), blood pressure ($p < 0.001$), and estimated filling pressures ($p < 0.01$) and directly related to diastolic function ($p < 0.01$). After adjustment for age, sex, body size, blood pressure, and ventricular hypertrophy, arterial compliance was independently predictive of diastolic dysfunction. In hypertensive patients with exertional dyspnoea, progressively abnormal diastolic function is associated with reduced arterial compliance. Arterial compliance is an independent predictor of diastolic dysfunction in patients with hypertensive heart disease and should be considered a potential target for intervention in diastolic HF.

Radaideh 2010 the main objective to explore the pattern of left ventricular hypertrophy caused by hypertension and to compare it with idiopathic hypertrophic cardiomyopathy. The retrospective study was conducted at the echocardiography lab of Rashid Hospital, Dubai, from January 2009 to January 2010. Cases of 11 patients with significant left ventricular hypertrophy (septum >15mm) due to underlying hypertension were analysed and compared with 11 cases of idiopathic hypertrophic cardiography (septum >15mm) to assess the two groups with similar baseline echocardiographic features. Minitab software was used for statistical analysis. Although the pattern of hypertrophy in hypertensive patients was more concentric (n=5; 45%), there was also asymmetrical septal hypertrophy in 4 (36%) cases, particularly the elderly with sigmoid shape septum. There was evidence of resting mid-cavity gradient due to reduced left ventricular end-systolic diameter in 4 (36%) cases.
Although the equation between hypertension and left ventricular hypertrophy

is more concentric, but it can be associated with left ventricular outflow tract obstruction and significant mid-cavity gradients similar to that seen in idiopathic hypertrophic cardiomyopathy., Left ventricular hypertrophy, Left ventricular outflow tract. (JPMA 63: 16; 2013).

Olaniyi 2013 Left ventricular (LV) hypertrophy is an important predictor of morbidity and mortality in hypertensive patients, and its geometric pattern is a useful determinant of severity and prognosis of heart disease. Studies on LV geometric pattern involving large number of Nigerian hypertensive patients are limited. We examined the LV geometric pattern in hypertensive patients seen in our echocardiographic laboratory. A two-dimensional, pulsed, continuous and color flow Doppler echocardiographic evaluation of 1020 consecutive hypertensive patients aged between 18 and 91 years was conducted over an 8-year period. LV geometric patterns were determined using the relationship between the relative wall thickness and LV mass index. Four patterns of LV geometry were found: 237 (23.2%) patients had concentric hypertrophy, 109 (10.7%) had eccentric hypertrophy, 488 (47.8%) had concentric remodeling, and 186 (18.2%) had normal geometry. Patients with concentric hypertrophy were significantly older in age, and had significantly higher systolic blood pressure (BP), diastolic BP, and pulse pressure than those with normal geometry. Systolic function index in patients with eccentric hypertrophy was significantly lower than in other geometric patterns. Doppler echocardiographic parameters showed some diastolic dysfunction in hypertensive patients with abnormal LV geometry. Concentric remodeling was the most common LV geometric pattern observed in our hypertensive patients, followed by concentric hypertrophy and eccentric hypertrophy. Patients with concentric hypertrophy were older than those with

other geometric patterns. LV systolic function was significantly lower in patients with eccentric hypertrophy and some degree of diastolic dysfunction were present in patients with abnormal LV geometry. One of the most important individual risk factors for cardiovascular disease (CVD) is hypertension.1 Left ventricular hypertrophy (LVH), which is defined as an abnormal increase in left ventricular (LV) mass, is one of the organic processes resulting from hypertension.1 LVH has been widely documented to be an independent cardiac risk factor in hypertensive patients,2,3 and the structural classification of LV geometry also provides useful and additional prognostic information.4,5 Some authors6,7 have observed that age significantly affects LV structure and geometric patterns. The LV adaptation to hypertension takes four different geometric patterns using the combinations of left ventricular mass index (LVMI) and relative wall thickness (RWT). The heart may adapt to hypertension by developing concentric hypertrophy (CH) with increased LVMI and RWT, eccentric hypertrophy (EH) with increased LVMI and normal RWT, concentric remodeling (CR) with normal LVMI and increased RWT, or by retaining normal geometry (NG) with both normal LVMI and RWT. Each geometric pattern is associated with a distinct combination of pressure and volume stimuli, contractile efficiency (reduced in those with concentric hypertrophy and concentric remodeling), and prognosis (worst with concentric hypertrophy and best with normal geometry).

Chapter Three

Material and Method

3-1Materials:

3-1-1Patients:

60 patients aged between 35-85 years old, main affected age was 68-78, 25 of them are female percentage 41.7% and 35 are male percentage 58.3%, all patients have hypertension disease durations from 1-35 years. All congenital heart disease blood vessels and normal patients are excluded.

3-1-2 Machine:

The study done by Vivid General Electric (GE) USA, using **3s** probe

3-2 Method:

3-2-1 Echocardiography Technique:

All patient left lateral decubitus, transthoracic echocardiography using pulsed Doppler through apical 4 chamber at mitral valve to assess the flow pattern through the MV. Parasternal long-axis view of the left ventricular (LV) inflow and outflow tracts . Parasternal long-axis view of the right ventricular (RV) inflow tract, called the RV inflow view. Parasternal long-axis of the right ventricular (RV) outflow tract, RV outflow view . The parasternal short-axis views—at multiple short-axis levels, beginning with the PSAX view at the level of the aortic valve, at the level of the pulmonary artery bifurcation, the level of the mitral valve, the mid-LV level or papillary muscle level, and at the level of the LV apical segments (PSAX-apical level), including the apical cap of the LV. The PLAX view is where the transthoracic examination begins. Landmark cardiac structures—the right ventricle (RV), left ventricle (LV), aortic root (Ao), and left atrium (LA), and the mitral and aortic valves—can be readily aligned along the cardiac long axis in the PLAX view.

3-2-2 Image Interpretations:

The image done by qualified technologist and then presented by qualified and specialist cardiologist to confirm the result.

3-2-3 Statistical study

Frequency table and Statistical Package for Social Science (SPSS).

3-2-4 Ethical considerations:

Patients and hospital have permission to collect data.

Chapter Four

Results

Table (4-1) shows the gender distribution

Gender	Frequency	Percent
Female	25	41.7
Male	35	58.3
Total	60	100.0

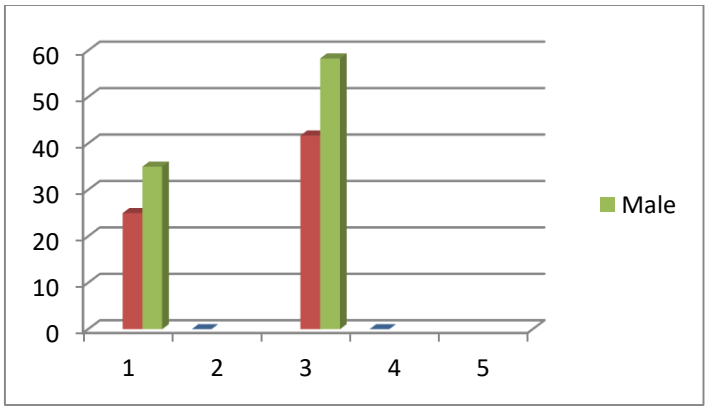

Figure (4-1) shows the gender distribution

Table (4-2) shows the age group

Age group	number	percentage
35-45	02	20
46-56	08	13.3
57-67	22	36.7
68-78	24	40
79-89	04	6.6
Total	60	100

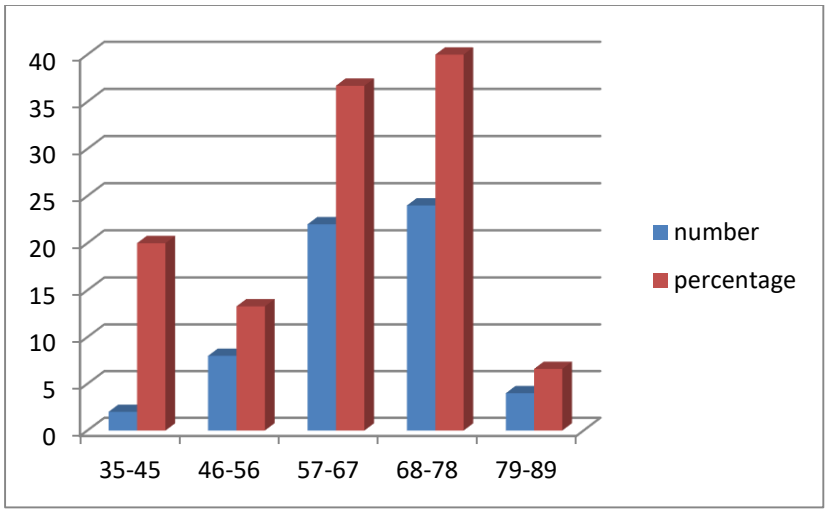

Figure (4-2) shows the age group

Table (4-3) shows the duration of hypertension

Duration	Frequency	Percent
1	2	3.3
3	4	6.7
4	3	5.0
5	6	10.0
6	3	5.0
7	2	3.3
8	4	6.7
9	2	3.3
10	5	8.3
11	1	1.7
12	2	3.3

13	3	5.0
15	3	5.0
17	2	3.3
18	2	3.3
20	5	8.3
21	1	1.7
22	2	3.3
23	2	3.3
25	3	5.0
28	1	1.7
30	1	1.7
35	1	1.7
Total	60	100.0

Table (4-4) shows control and uncontrolled hypertension

Control	Frequency	Percent
No	24	40.0
Yes	36	60.0
Total	60	100.0

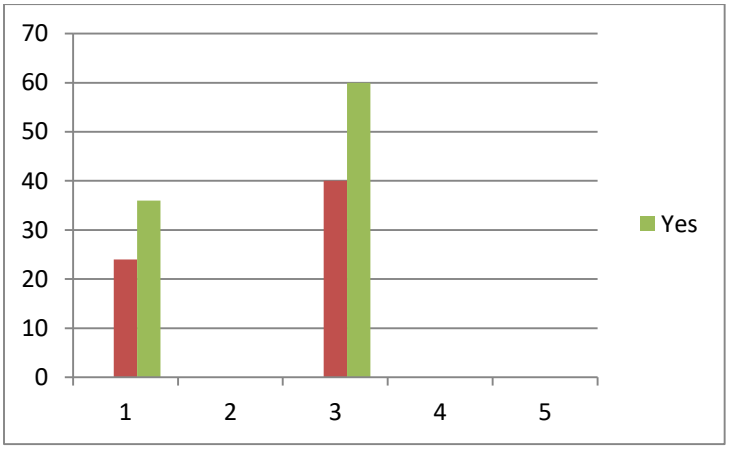

Figure (4-3) shows control and uncontrolled hypertension

Table (4-5) shows medication of hypertension

Treatment	Frequency	Percent
Regular	32	53.3
Irregular	5	8.3
None	23	38.3
Total	60	100.0

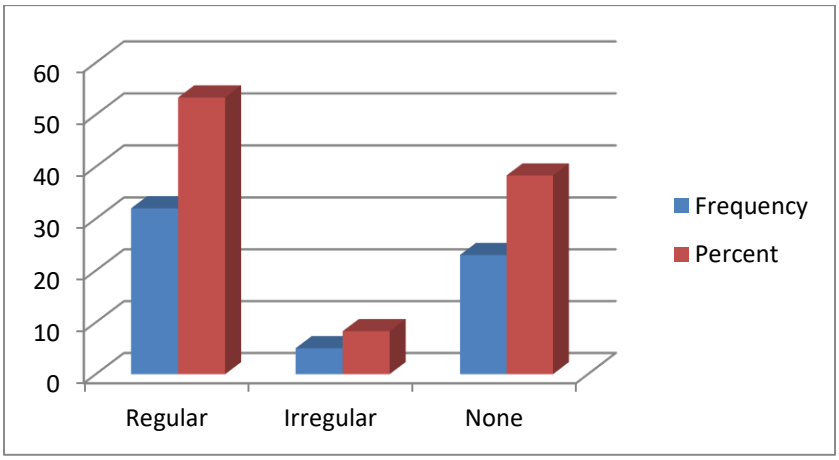

Figure (4-4) shows medication of hypertension

Table (4-6) shows the left ventricular Hypertrophy (LVH)

LVH	Frequency	Percent
Yes	34	56.7
NO	22	36.7
Mild	4	6.7
Total	60	100.0

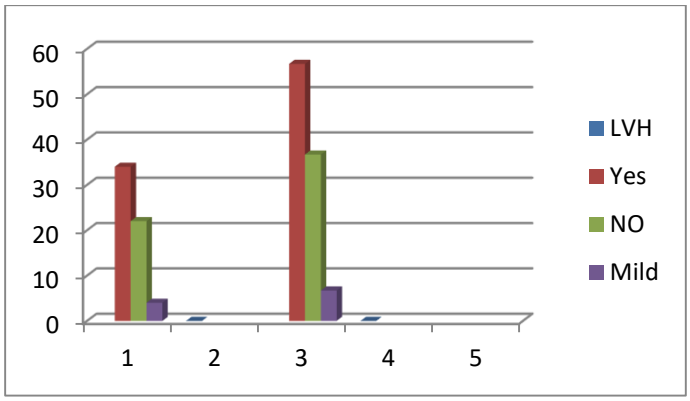

Figure (4-5) shows the left ventricular Hypertrophy (LVH)

Table (4-7) shows the heart chamber dilated according to hypertension

Chamber Dilated	Frequency	Percent
NO	44	73.3
Dilated LV	4	6.7
Dilated LV/RV	5	8.3
Dilated All	7	11.7
Total	60	100.0

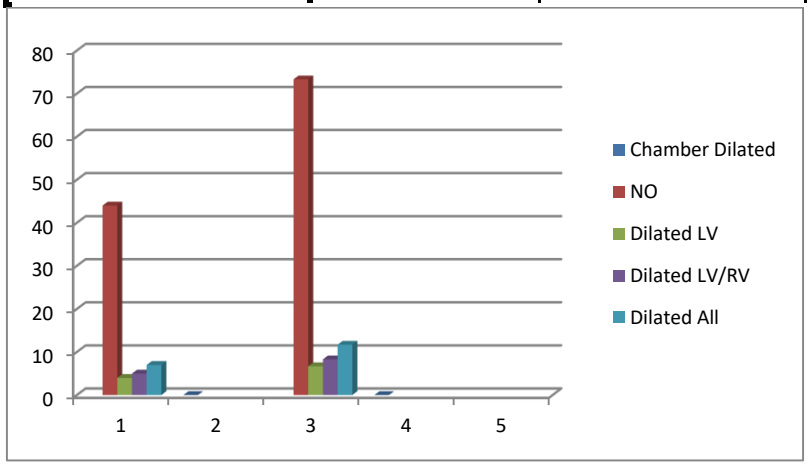

Figure (4-6) shows the heart chamber dilated according to hypertension

Table (4-8) shows the heart abnormalities according to hypertension

DD/Type	Frequency	Percent
1	30	50.0
2	21	35.0
3	9	15.0
Total	60	100.0

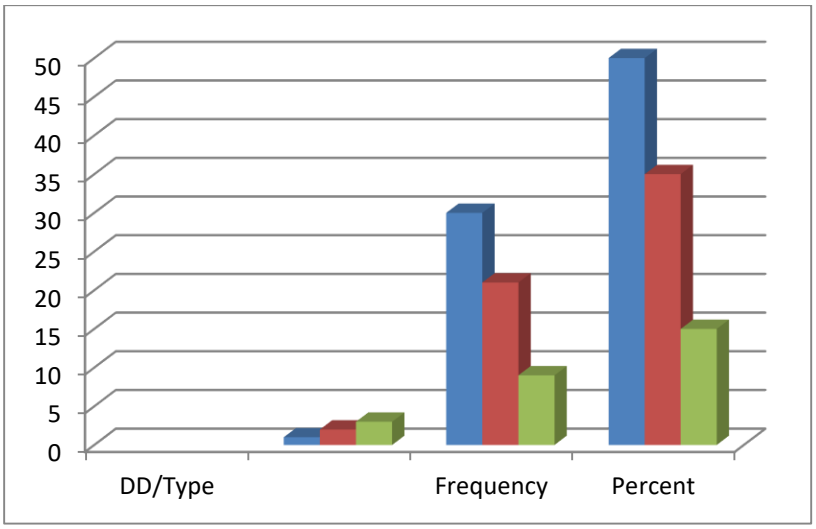

Figure (4-7) shows the heart abnormalities according to hypertension

Table (4-9) shows effect of hypertension in Aortic root

Aortic root	Frequency	Percent
Diltations	3	5.0
NO	55	91.7
Stenosis	2	3.3
Total	60	100.0

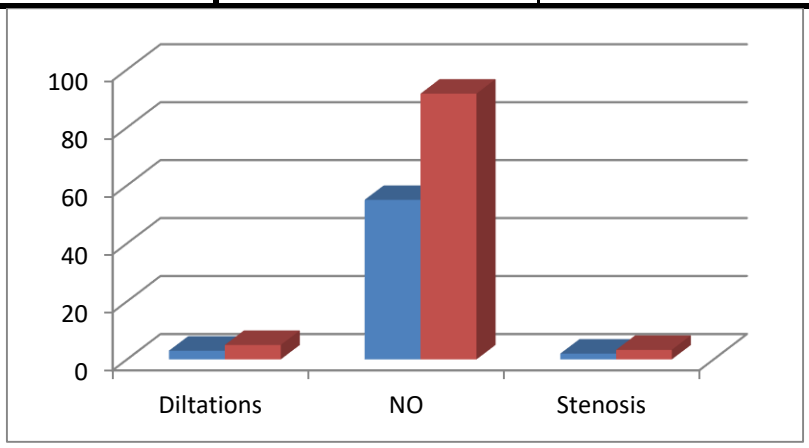

Figure (4-8) shows effect of hypertension in Aortic root

Table (4-10) shows effect of hypertension in Mitral Valve (MV)

MR	Frequency	Percent
MILD	12	20.0
Moderate	3	5.0
None	45	75.0
Total	60	100.0

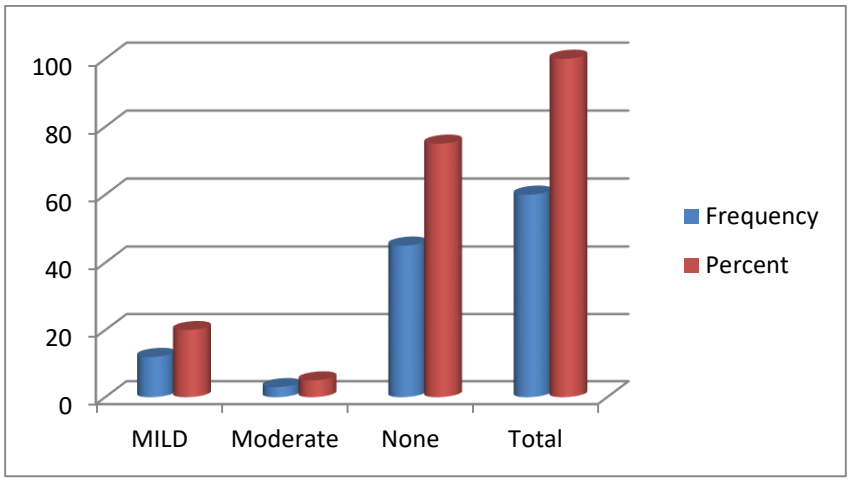

Table (4-9) shows effect of hypertension in Mitral Valve (MV)

Table (4-11) shows Aortic Relegations (AR) according to hypertensions

AR	Frequency	Percent
MILD	3	5.0
Moderate	1	1.7
Sever	2	3.3
No	54	90.0
Total	60	100.0

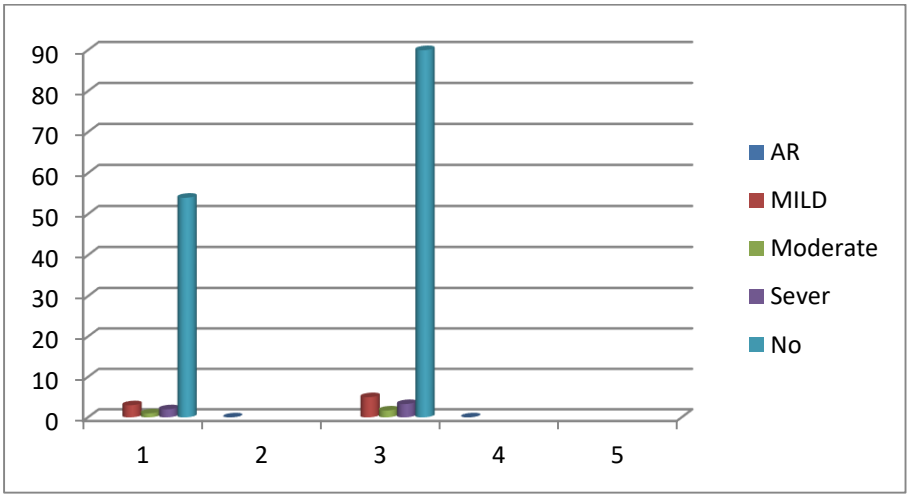

Figure (4-10) shows Aortic Relegations (AR) according to hypertensions

Table (4-12) shows Tricuspids Relegations (TR) according to hypertensions

TR	Frequency	Percent
Normal	52	86.7
MILD	6	10.0
Moderate	2	3.3
Total	60	100.0

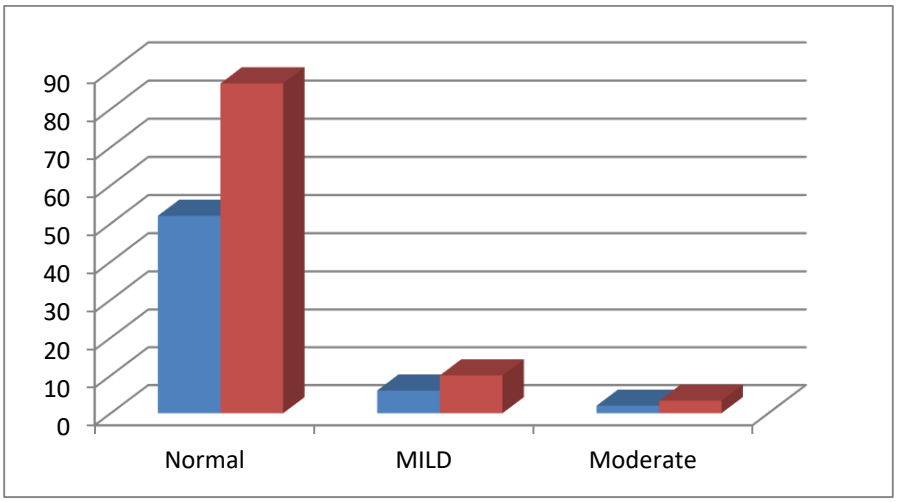

Figure (4-11) shows Tricuspids Relegations (TR) according to hypertensions

Table (4-13) shows Inter Ventricular Septal Diameter (IVSd) according to hypertensions

IVSd	Frequency	Percent
MORE	4	6.7
Few	8	13.3
Dilated	4	6.7
Normal	44	73.3
Total	60	100.0

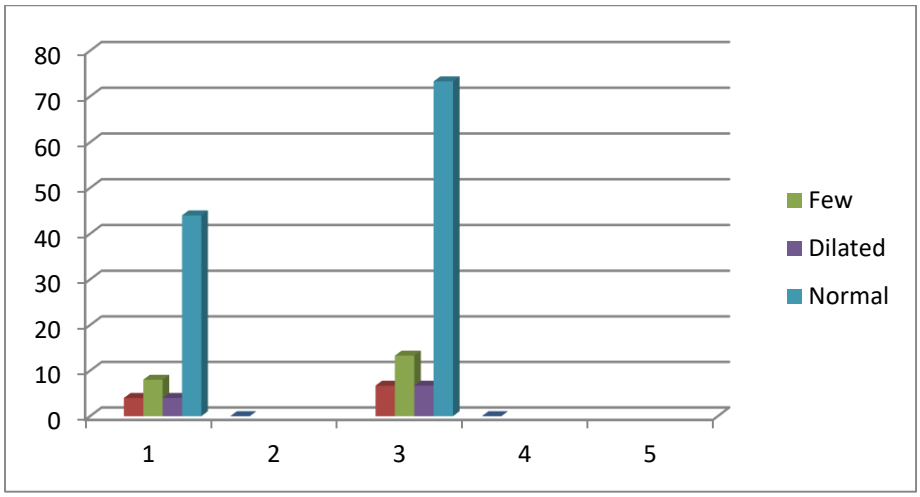

Figure (4-12) shows Inter Ventricular Septal Diameter (IVSd) according to hypertensions

Table (4-14) shows Posterior Wall Thickness diameter (PWTd) according to hypertensions

PWTd	Frequency	Percent
More	4	6.7
Few	8	13.3
Dilated	5	8.3
Normal	43	71.7
Total	60	100.0

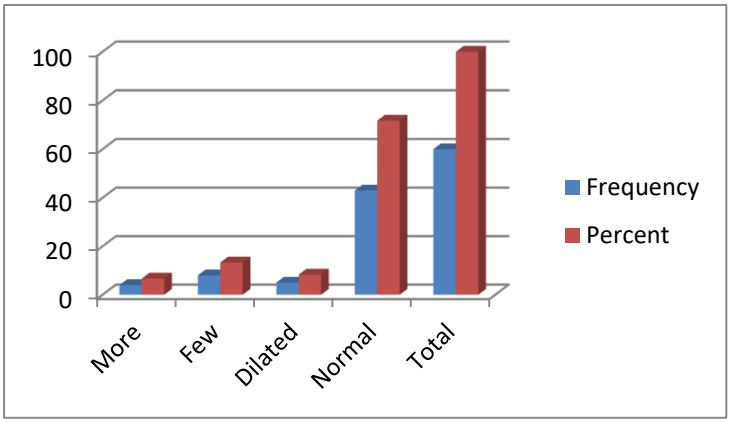

Figure (4-13) shows Posterior Wall Thickness diameter (PWTd) according to hypertensions

Table (4-15) shows last diagnosis of echocardiography in hypertensive patients:

Diagnosis	Frequency	Percent
Normal	5	8.3
Sclerotic MV/AV	36	60
Debakey	2	3.3
HDD	11	18.3
DD	1	1.7
IVH	4	6.7
PHT/HF	1	1.7
Total	60	100.0

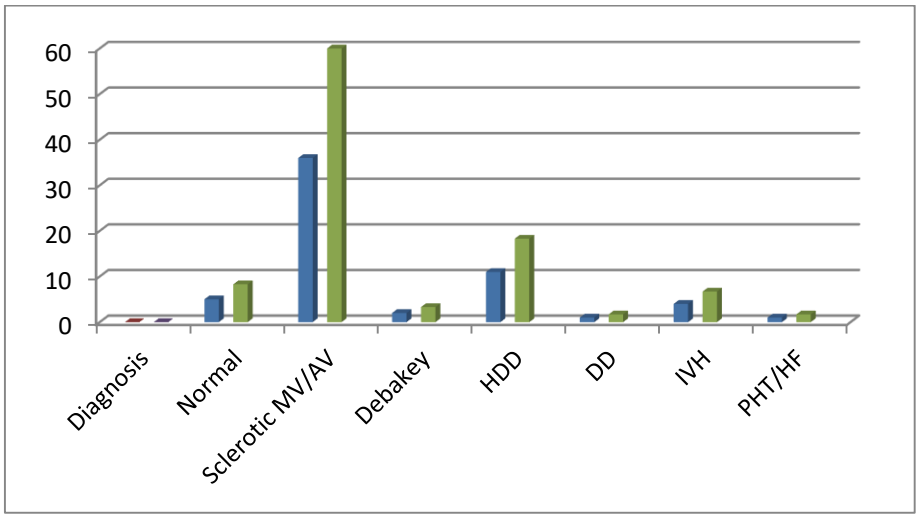

Figure (4-14) shows last diagnosis of echocardiography in hypertensive patients

Table (4-16) Cross tabulation between Diagnosis and Gender

Diagnosis	sex		Total
	Female	Male	
Normal	3	2	5
Sclertic MV/AV	14	22	36
Debakey	1	1	2
HDD	2	9	11
DD	1	0	1
IVH	3	1	4
PHT/HF	1	0	1
Total	25	35	60

Table (4-17) shows correlation between diagnosis and left ventricle hypertrophy

Diagnosis	LVH			Total
	Yes	NO	Mild	
Normal	2	3	0	5
Sclerotic MV/AV	18	16	2	36
Debakey	1	0	1	2
HDD	8	3	0	11
DD	1	0	0	1
IVH	3	0	1	4
PHT/HF	1	0	0	1
Total	34	22	4	60

Table (4-18) shows correlation between diagnosis and Aortic Root stenosis

Diagnosis	Aortic root			Total
	Yes	NO	Stenosis	
Normal	0	5	0	5
Sclertic MV/AV	2	33	1	36
Debakey	0	1	1	2
HDD	1	10	0	11
DD	0	1	0	1
IVH	0	4	0	4
PHT/HF	0	1	0	1
Total	3	55	2	60

Table (4-19) shows correlation between diagnosis and heart chamber dilatation

Diagnosis	Chamber Dilated				Total
	NO	Dilated LS	Dilated LV/RV	Dilated All	
Normal	5	0	0	0	5
Sclerotic MV/AV	28	2	2	4	36
Debakey	1	0	0	1	2
HDD	6	1	2	2	11
DD	1	0	0	0	1
IVH	3	1	0	0	4
PHT/HF	0	0	1	0	1
Total	44	4	5	7	60

Table (4-20) shows correlation between diagnosis and left ventricle posterior wall thickening

Diagnosis	PWTd				Total
	More	Few	Dilated	Normal	
Normal	0	1	0	4	5
Sclerotic MV/AV	3	3	1	29	36
Debakey	0	0	1	1	2
HDD	1	4	3	3	11
DD	0	0	0	1	1
IVH	0	0	0	4	4
PHT/HF	0	0	0	1	1
Total	4	8	5	43	60

Chapter Five

Discussion, Conclusion, and Recommendations

5-1 Discussion:

The study done in 60 hypertension patients aged between 35-85 years old, affected age between 68-78, agree with (Longwan 2014), all subject are examined in transthoracic echocardiography and pulsed Doppler through apical 4 chamber to assess the flow pattern through mitral valve. The study found that male 35 percentage 58.3% more affected than female 25 percentage 41.7% agree with (Ayoub 2013) table & figure (4-1), the affected age 68-78 years of 24 patients from all patients percentage 40% agree with (Olaniyi 2013) table (4-2). This study found that the duration of hypertension from 1-35 years agree with Ayoub 2013, table (4-3) and 36 patient had control this disease and 24 had uncontrolled, 32 percentage 53.3 regular in medications, 23 percentage 38.3 had no medications and 5 percentage 8.3 used medications but irregular. The important finding according to the effects of hypertensions that left ventricular hypertrophy in 38 patients percentage 62.7% (moderate 56.7% and mild 6.7%), these patients have Diastolic Dysfunctions (50% type1, 35% type 11, and 15% 111 DD) agree with (Mottran 2013). Aortic root affected in 5 patients percentage 8.3%, mitral regurgitations in 15 patients percentage 25%, Aortic regurgitations in 6 patients percentage 10%, Tricuspid regurgitations in 8 patients percentage 13.3%, Inter ventricular septal thickening in 16 patients 26.7%, left ventricular wall thickening in 17 patients percentage 28.3%. The study found that sclerotic mitral valve and aortic valve in 36 patients (14 female and 22 male) percentage 60%, hypertensive heart disease in 11 patients (2 female and 9 male) 18.3%, 8 of them have posterior wall thickening. Interventricular hypertrophy in 4 patients (3 female and 1 male) percentage

6.7% , the study found that 38 patients have different heart disease due to hypertension agree with (Merdts 2008).

5-2Conclusions:

The study conclude that male more affected than female, so that left ventricle Hypertrophy in all patients that all patients that have uncontrolled of hypertension, Heart chambers affected by hypertension. According to the long standing hypertension the heart had more affected and thickening of heart chamber and dilatation of left ventricle more than other chamber.

5-3Recommendations:

- Other study with large sample and many variations.
- Any patients have hypertension disease must control it.
- Echocardiography must used in follow up of hypertension patients.
- Comparative study between MRI & Echocardiography.
- Contrast echocardiography must be used as routine.

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Kyung Won Chung, Ph.D. Harold M. Chung, M.D 2012 Gross Anatomy Holly Reid McLaughlin Seventh Edition Copyright © 2012, Lippincott Williams & Wilkins, a Wolters Kluwer business. 351 West Camden Street Two Commerce Square Baltimore, MD 21201 2001 Market Street Philadelphia, PA 19103 Instruction. QS 18.2] QM31.C54 2012 611–dc22

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Appendix

zSData Collection Sheet of M.Sc Thesis in: Medical Diagnostic Ultrasound

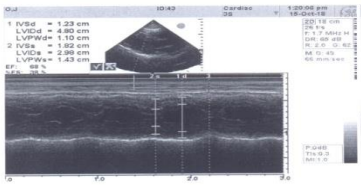
Impact of Hypertension in Heart using Echocardiography

Patients data					Echocardiography Finding					
No	age	gender	History of HB	Medications	PWdT	EF	IVSd	LVEDd	LV	LVESd
1										
2										
3										
4										
5										
6										
7										
8										
9										
10										

GE Medical Systems

4 Images Report

Name: o,j		
ID: 43	Sex: M	Exam Date: 11 Oct 2018
Birth Date:	Age:	Tape ID:
Height:	BSA: 0.00 m²	Tape counter: 0:00:00
Weight:	BP:	Referring M.D.:



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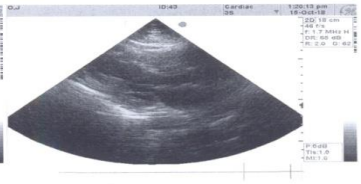

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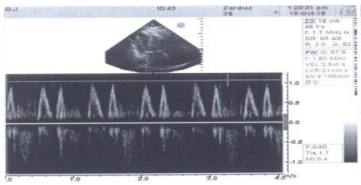

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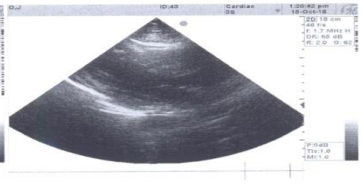
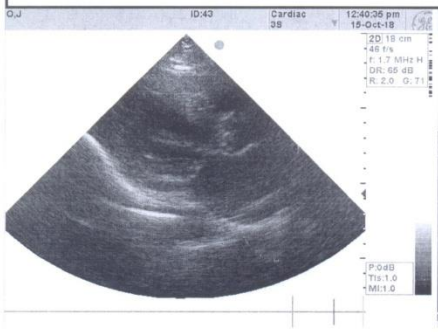


Image 4

65 years old female complain of hypertension for 13 years with control it the diagnosis grade 1 diastolic dysfunction with normal LV and normal LVEDd, LVESd , Ao, EF.

Patient Name o,j Patient ID 43



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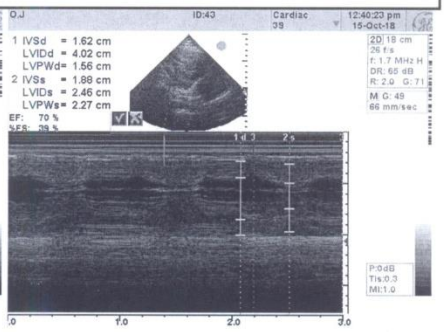

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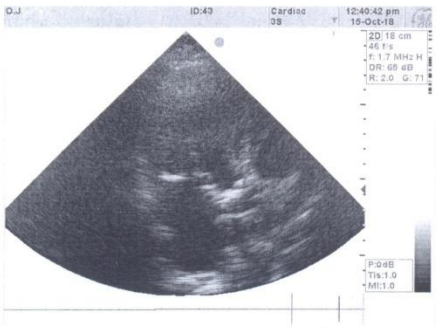

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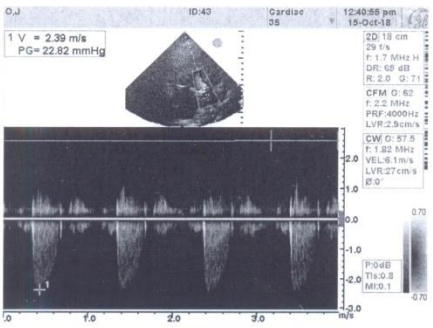

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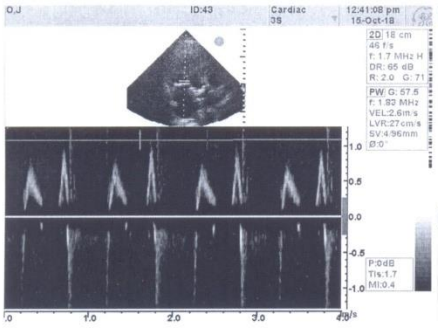

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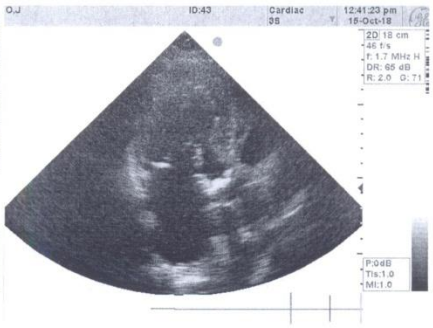

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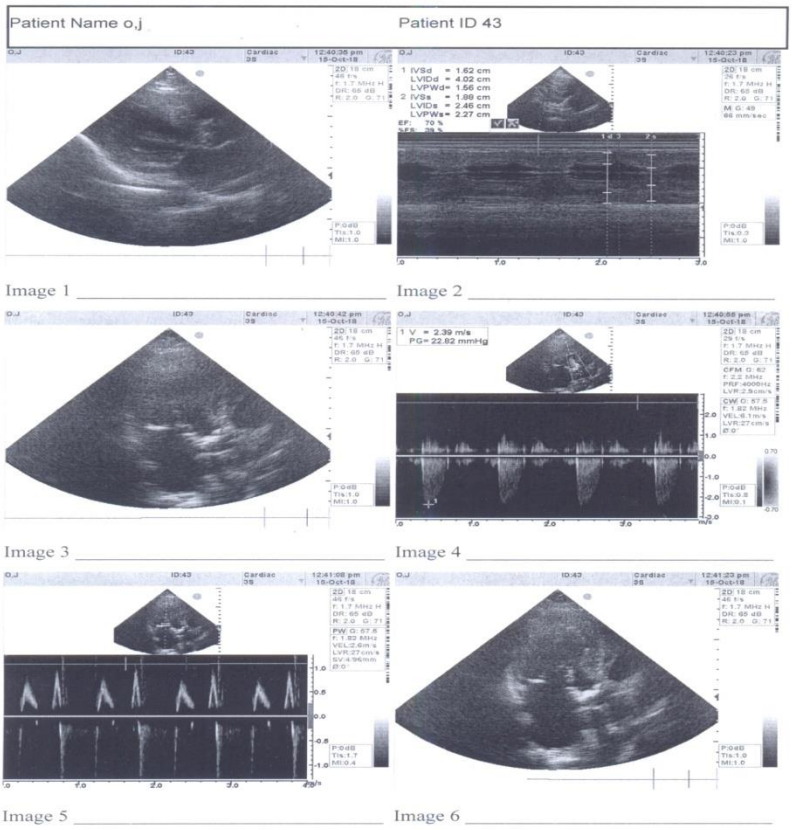
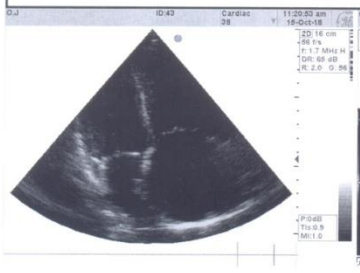


78 years old female complain of hypertension for 20 years with irregular medication the echocardiography shows, diastolic dysfunction type 1, LVH, Sclerotic AV, AS, travel AR, Sclerotic MV with mild MR

Patient Name o,j Patient ID 43



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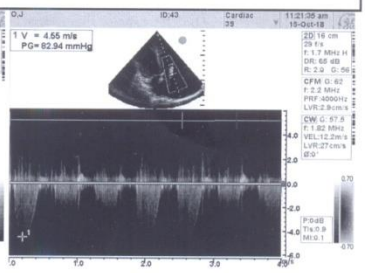

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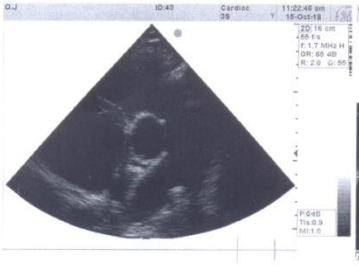

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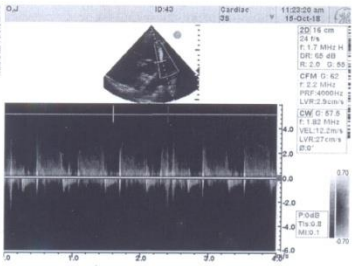

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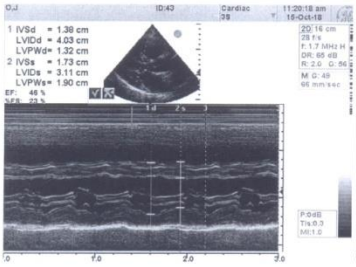

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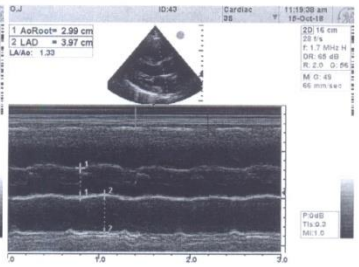
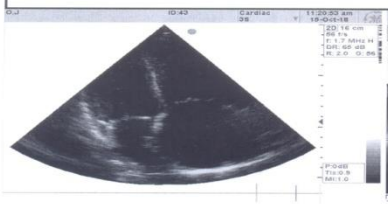


Image 6 _____

Page 1

35 years old female have hypertension for and uncontrolled it echocardiography report there was HHD complicated with Rt heart failure

Patient Name o.j Patient ID 43



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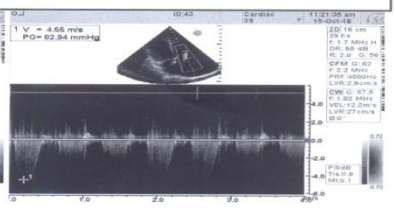

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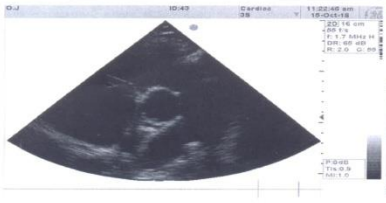

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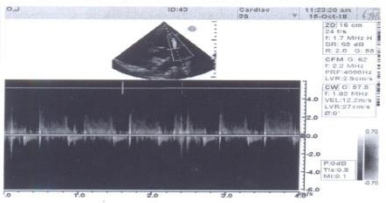

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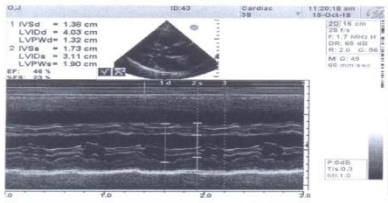

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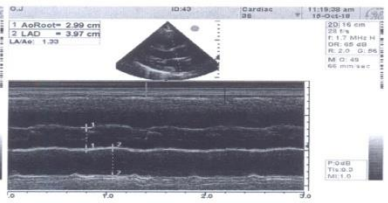

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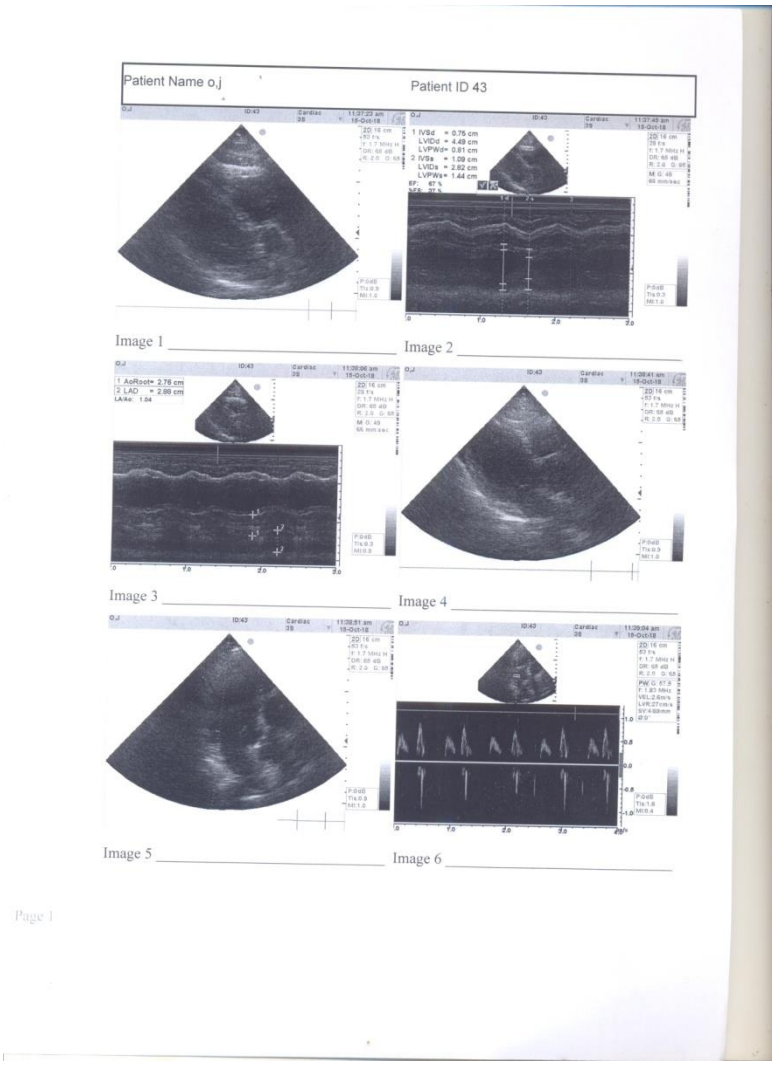
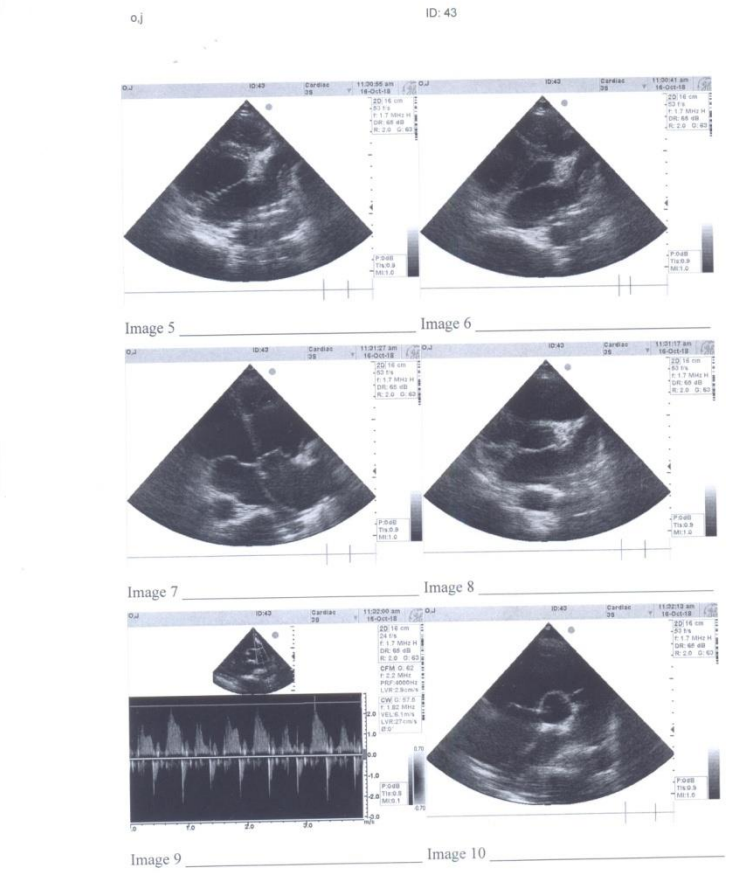
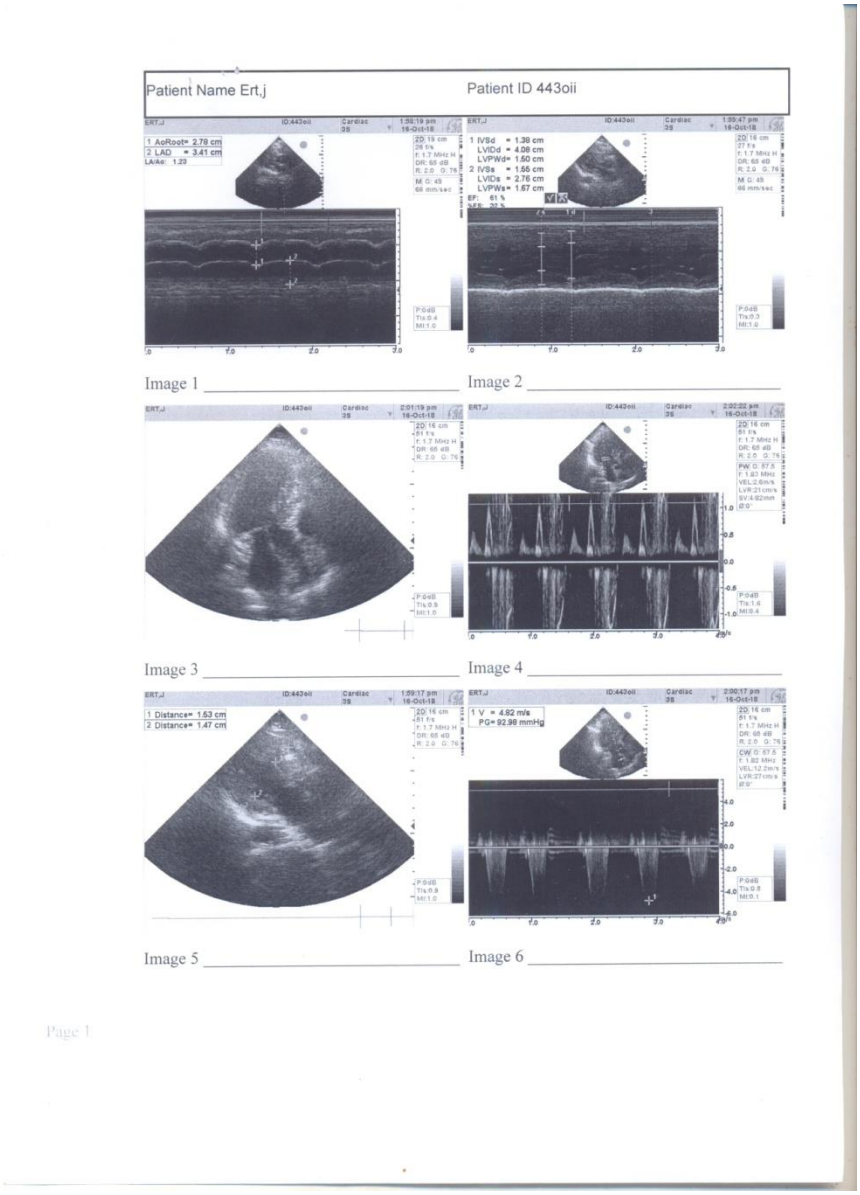


60 years old female, HBP for 20 years and control it echocardiography diagnosis is: grade 1 Diastolic Dysfunction with normal LV Systolic function and sclerotic AV.



43 years male HTN for 5 years uncontrolled the diagnosis are Dilated Cardiomyopathy (DCM)

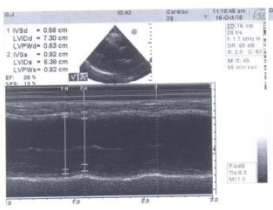


80 Year old female uncontrolled HTN for 20 y, echocardiography shows Hypertrophic Obstructive Cardiomyopathy (HOCM) (SAM&ASH)

GE Medical Systems

4 Images Report

Name: o,j		
ID: 43	Sex: M	Exam Date: 11 Oct 2018
Birth Date:	Age:	Tape ID:
Height:	BSA: 0.00 m²	Tape counter: 0:00:00
Weight:	BP:	Referring M.D.:



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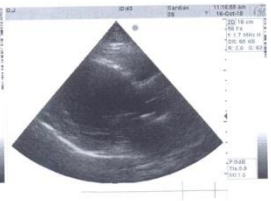

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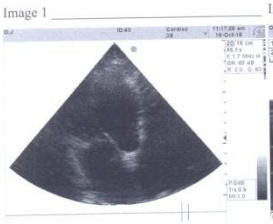

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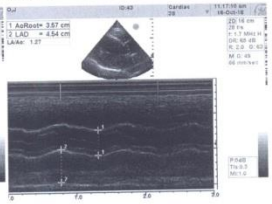

Image 4

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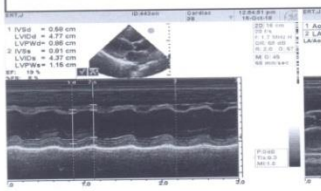
Patient Name Ert.j Patient ID 443oii



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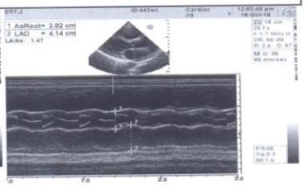

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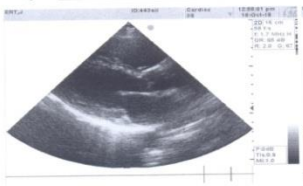

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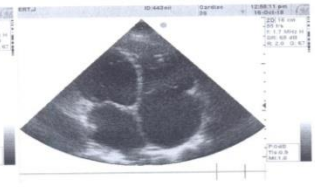

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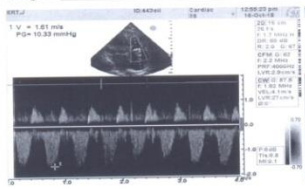

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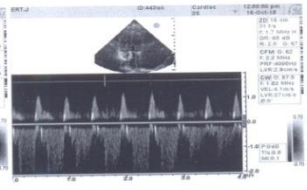
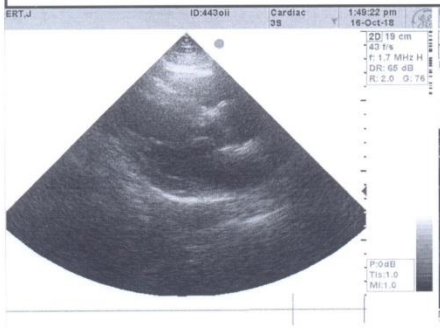


Image 6

Page 1

Patient Name Ert,j Patient ID 443oii



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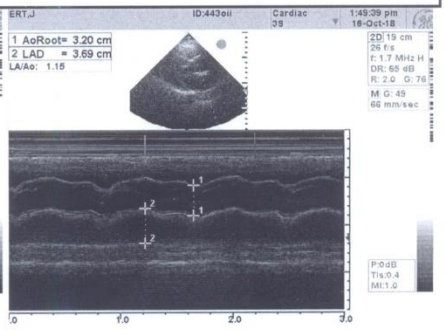

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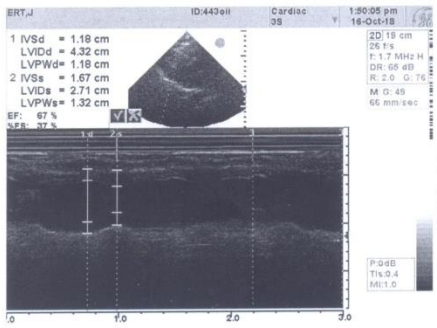

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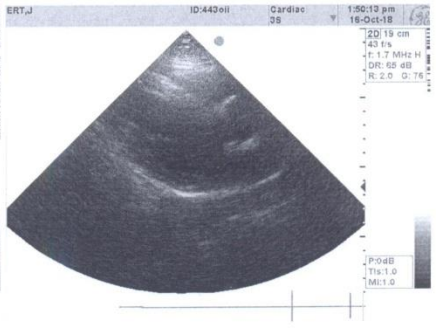

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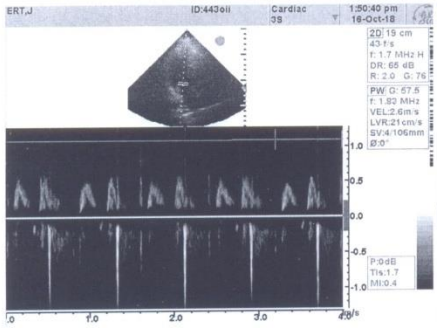

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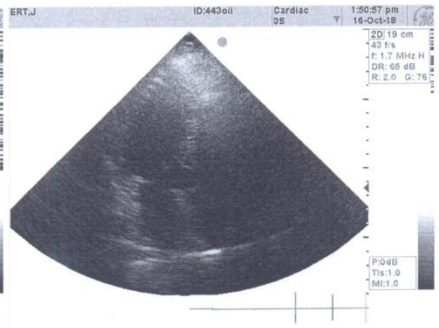

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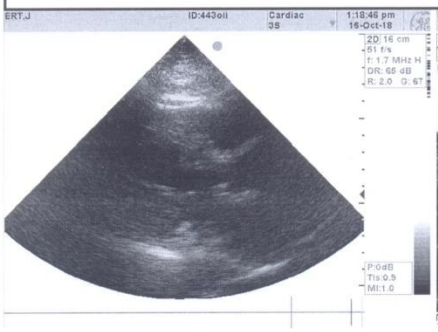
Patient Name Ert,j

Patient ID 443oii



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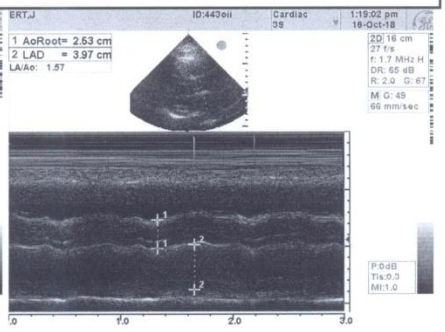

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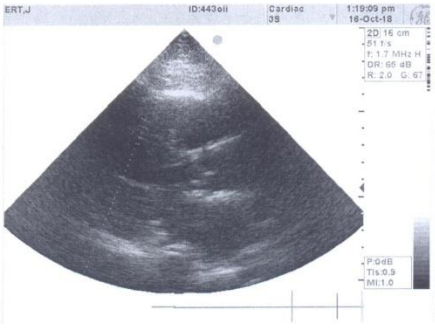

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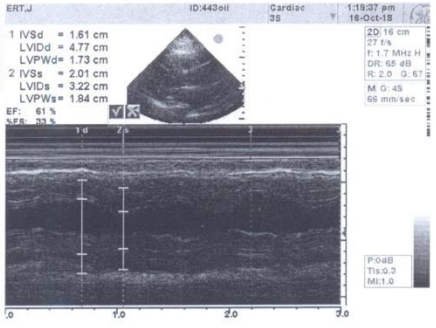

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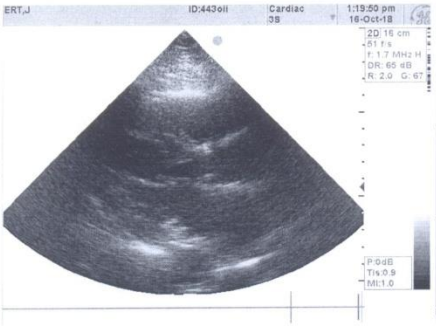

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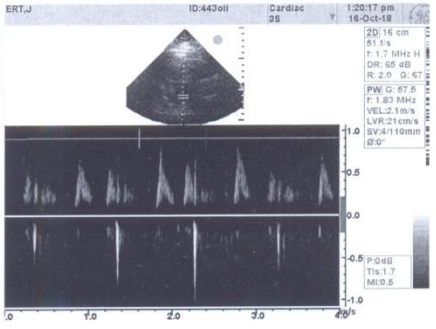

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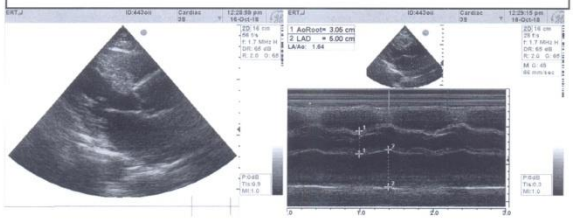
Patient Name Ert,j Patient ID 443oii



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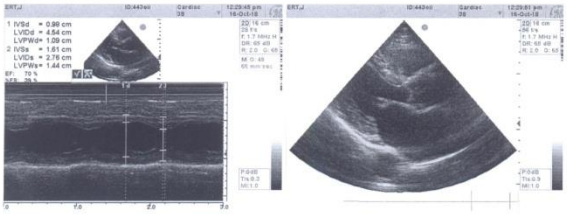

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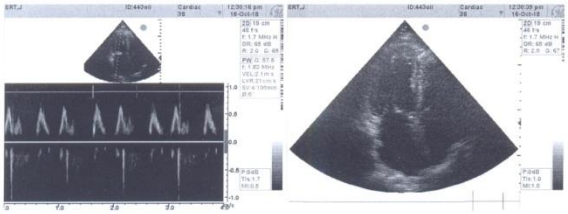

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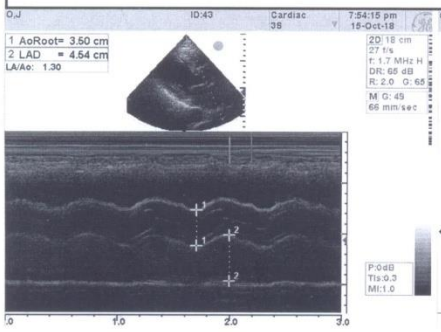
Patient Name o,j Patient ID 43



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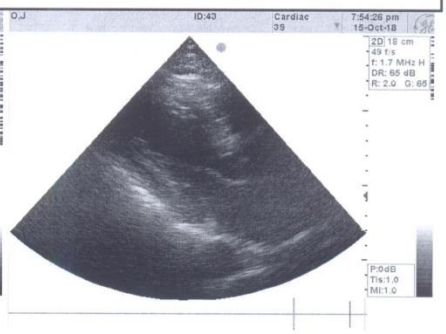

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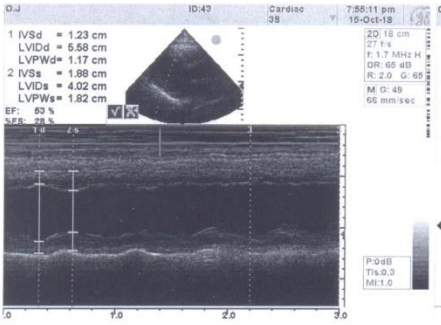

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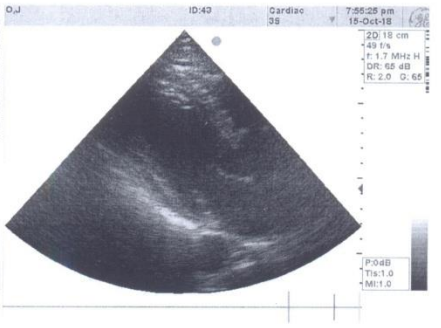

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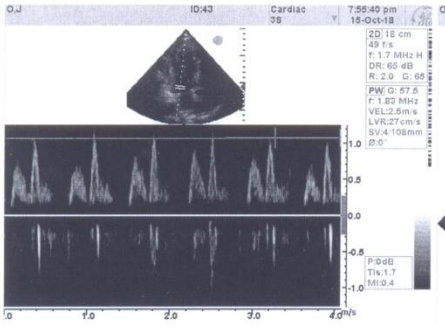

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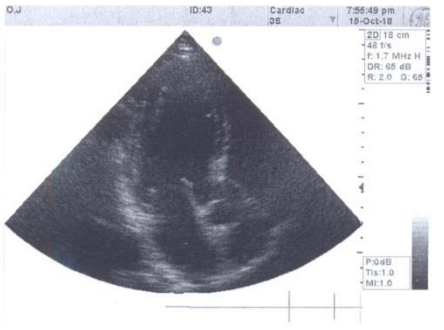

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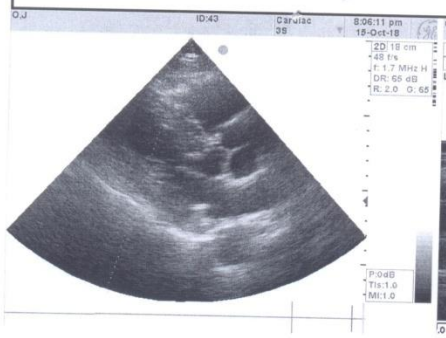
Patient Name o,j

Patient ID 43



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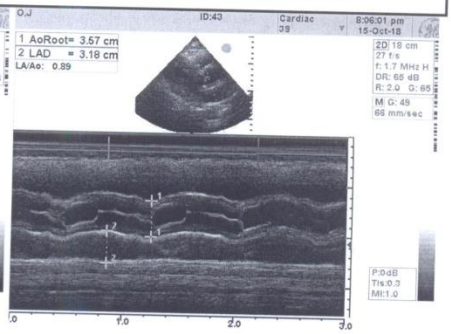

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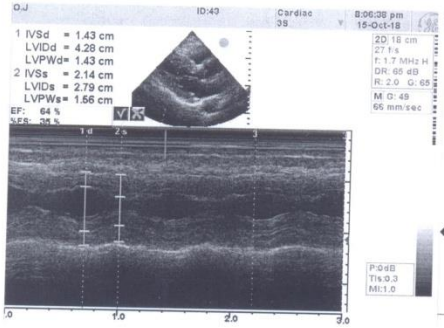

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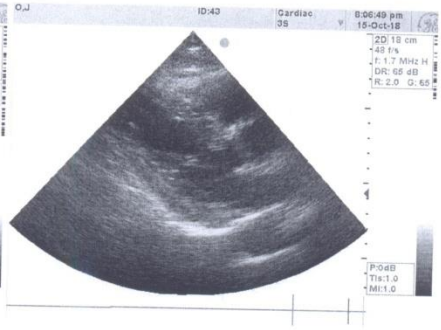

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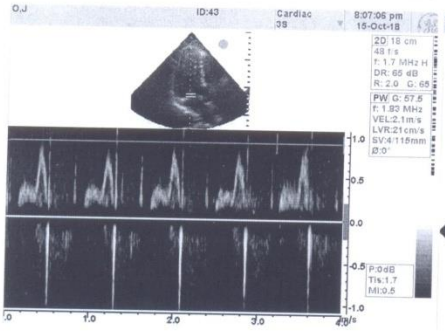

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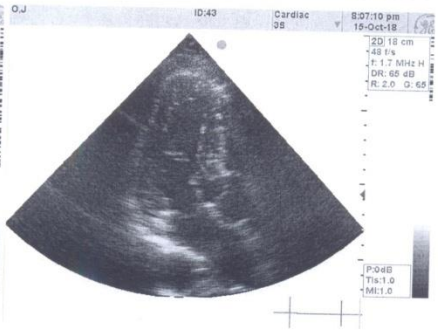


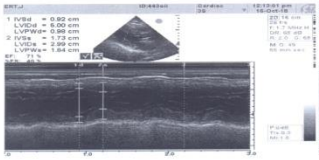
Image 6 _____

Page 1

GE Medical Systems

4 Images Report

Name: Ert,j		
ID: 443oii	Sex: M	Exam Date: 16 Oct 2018
Birth Date:	Age:	Tape ID:
Height:	BSA: 0.00 m²	Tape counter: 0:00:00
Weight:	BP:	Referring M.D.:



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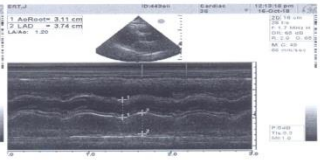

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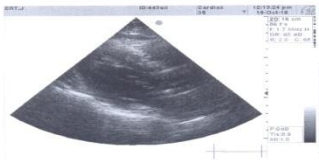

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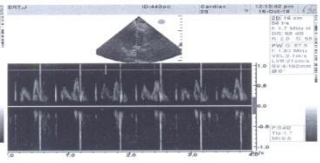
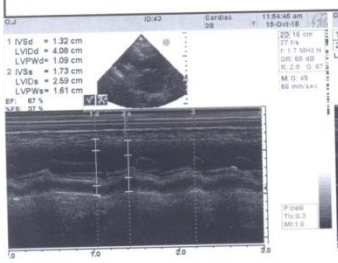


Image 4

Page 1

Patient Name o,j

Patient ID 43



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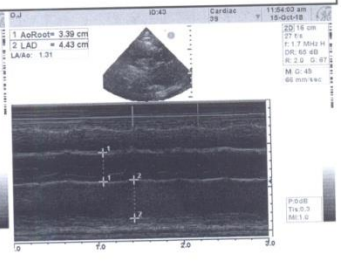

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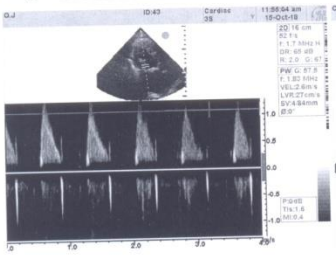

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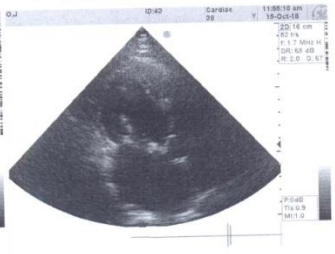

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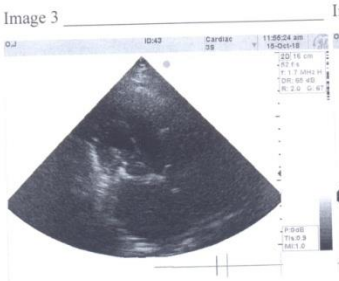

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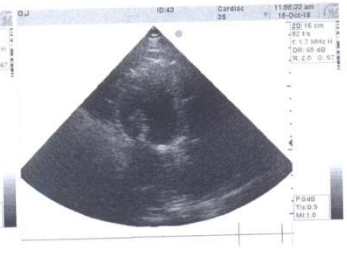

Image 6 _____

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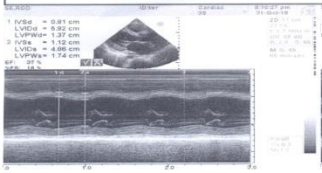
Patient Name Se,Rgd

Patient ID ter



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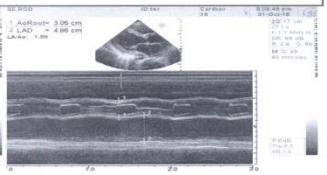

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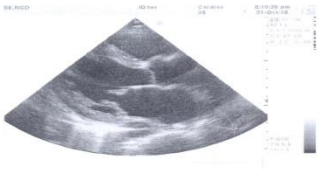

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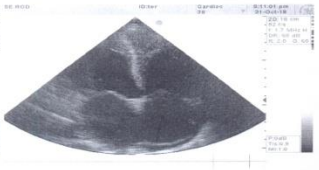

Image 4

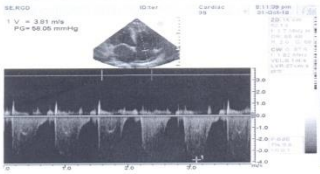

Image 5

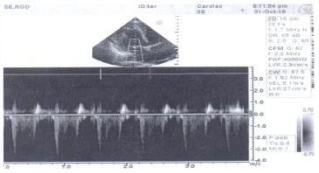

Image 6