SUDAN UNIVERSITY OF SCIENCE AND TECHNOLOGY

COLLEGE OF POSTGRADUATE STUDEIS AND SCIENTIFIC RESAERCH

CHARACTERIZATION OF THE HEART IN HYPERTENSIVE PATIENTS USING ULTRASOUND

توصيف القلب للمصابين بإرتفاعُ ضّغْط الدّمْ بإسْتخْدَام المُوجَاتْ فَوْقْ الصَّوْتيّة

ATHESIS SUBMITTED FOR PARTIAL FULFILMENT OF MASTER DEGREE IN MEDICAL ULTRASOUND

BY: ASHRAF MUSTAFA MOHAMMED OSMAN

SUPERVISOR: Associated Professor ELSAFI AHMED ABDALLAH

(RD, Msc, PHD-L, PHD-E)

2013-2014

DEDICATION

To my parents **MUSTAFA ABO HARAZ&ZAHRAA BIT ATTEBEIR,** who encouraged me to enjoy the intellectual challenge of medicine and the love of making a difference in patients' lives.

To my wife, **ZUBAIDAH**, for all the unsparing help, support and joy you bring to my life.

Acknowledgements

My deepest appreciation and sincerest gratitude:

To all the staff in the Ribat university hospital-echocardiography department, personally to **Dr SAAD ABDERRAHIM SOBAHI** the cardiologist & the director of the department for his permission & unsparing help provided by him, also to **MAJOR BILAL MAGZOB** (sonographer & echo technician), to **CAPTAIN NEMAT OMER** (sister & echo technician), and to **FIRST LIEUTENANT RABAB EL SHEIKH** (sister & echo technician), for their extensive & continuous teaching. Their help and never-ending support made this work possible .I cannot thank them enough for their efforts on this project.

LIST OF CONTENTS

ABSTRACT …… …..…..8

CHAPTER ONE

1-1 INTRODUCTION……..…9
1-2 STATEMENT OF THE PROBLEM …….....12
1-3 RESEARCH OBJECTIVE……...13
1-4 THESES OVERVIEW…….....14

CHAPTER TWO

2-1 LITRATURE REVEW …….…15

-ANATOMY……15

-PHYSIOLOGY……...…16

-PATHOLOGY…….…….. 18

2-2 NORMAL SONOGRAOHIC APPEARANCE OF THE HEART……..……..24

-NORMAL CARDIAC MEASUREMENT…….…….28

2-3 PREVIOUS STUDIES……...…….29

CHAPTER THREE (MATERIALS AND METHODS)

3-1 MATERIALS & TOOLS……..32

3-2 METHODS……32

3-3 INCLUSION CRITERIA……..32

3-4 EXCLUSION CRITERIA…….32

3-5 ULTRASOUND TECHNIQUE OF THE HEART…….…….….33

3-6 STATISTICS……..…….....33

CHAPTER FOUR

DISCRIPTIVE STATISTIC……34

RESULTS & ANALYSIS……...……43

CHATER FIVE

5-1 DISCUSSION……...48

5-2 RECOMMENDATION…….…51

5-3 CONCLUSION…….…52

5-4 REFERENCES……..…52

APPENDIX 1 MACHINE.……....55

NORMAL ECHO TEMLATE……56

DATA COLLECTION SHEET……..57

IMPORTANT ABREVIATIONS……...58

APPENDIX 2 IMAGES…….……..59

APPENDIX 3 THE QUESTIONNAIRE……...…….……...76

PATIENT AGREEMENT…….……..77

ملخّص البحث

هذا البحثِ تم إجراؤه لِكي نتعْرفَ على أهمية تصوير القلب بواسطة الموجات فوق الصوتية (echocardiography) في السيطرةِ على ومتابعة وتشخيص الأنواعِ المختلفةِ للمضاعفات القلبيةِ لإرتفاع ضغط الدمِ (معرفة تأثير إرتفاع ضغط الدم على عضلاتِ القلبَ والصماماتَ وأوعية الدَمّ القلبية بإستخدام الموجات فوق الصوتية- الرمادية والملونة).

البيانات المستخدمة في هذا البحث جُمِعتْ مِنْ مستشفى الرباط الوطني التعليمي بأَستُعمال ماكينةَ تصوير القلب بالموجات (echocardiographic machine) تسمى سيمينز (SIEMENS-ACUSON CV70 Ultrasound imaging system), وهذا يَتضمّنُ الأُذين الأيسر و القطر البطيني الأيسر كلاهما في حالتي إنقباض وإنبساط القلب و يدخلُ بالإضافة إلى ذلك حساب الأقطارِ الصماميةِ وأقطار تجاويف الأذين والبطين وكذا سرعة تدفق الدم من وإلى القلب.

هذه الدراسةِ التى هي حول تأثيرِ إرتفاع ضغط الدم على تشريح ووظيفةِ القلبَ كتبت فى خمسة فصول . الفصلَ الأولَ كان عبارةُ عن تمهيد حول الكلمات الدليليةِ في العنوانِ بالإضافة إلى الأهدافِ العامّةِ والمعيّنةِ للدراسةِ لتَعديد متغيّراتْ وخطواتْ الدراسةِ؛ هذا بعد عرضنا لقصّةِ المشكلةِ والتبريرِ العلمي والأكاديمي لإجْراء البحث ومختصرها ضرورة إدخال الموجات في متابعة مرضى الضغط وذلك لأن بعض التعقيدات الدقيقة المميتة والتي يمكن تلافيها لا تُشخّص إلا عبر هذه الطريقة, ووضعت نظرة عامّة على الدراسةِ كلمحة إضافيةُ في هذا الفصلِ. الفصل الثاني كتبت فيه المادة العلمية التي وضعت من قبل العلماء في هذا الشأن والبحوثَ العلميةَ السابقَة ذات الصلة وهذ يَتضمّنُ عِلْمَ التشريح وكذا عِلْم وظائف الأعضاء وعِلْم الأمراض و الظهور الطبيعي للقلبِ والمقاييسِ القلبيةِ الطبيعيةِ التي تظهر في صورة الموجات. في الفصلِ الثالثِ أُدرجتُ الطرق والأدوات التقنية بالإضافة إلى الموادِ والإجراءاتِ المهمِة لجَمْع البياناتِ والمعلومات المَذْكُورة في البحث، أيضاً التقنية والطرق الإحصائية التي أُستُعملت لتحليل وجُدولتْ البيانات وتوضيح إستخدام الإختبارِ المُمَثَّل أوالمُحَلَّل كاي Chi 2 وذلك كله كان بإستخدام الحزمة الإحصائية المشهورة بإسم ((الحزمة الإحصائية للدراسات الإجتماعية والعلمية النسخة رقم 16)).

((SPSS version 16- Statistical Package for social studies.))

الفصلَ الرابعَ كان حول تحليلِ البياناتَ والنَتائِجَ و يَصِفُ الفصل الأخير المُناقشةُ والتوصياتُ والخاتمةُ. في المُناقشةِ وَجدنَا بأنّ هناك علاقة بين ضغط الدمّ العالي والتغييرات في حجم و chiوظيفةِ القلبِ يَتراوحانِ ما بين علاقة ضعيفة وأخري قويةِ والذي عُكسَ مِن قِبل إختبار كما هو موضح في الجداول (11 و12). توصيتنا القوية إلى كُلّ العُمّال والموظفين المعنيين بصحةِ القلب والشرايين هي إستعمال تصوير القلب بالموجات فوق echocardiography الصوتية في تشخيصِهم ومتابعتهم لأمراضِ القلب وإرتفاع ضغط الدمِّ ويكون ذلك إلزاميُ بتوجيه من أصحاب المسؤلية وإتخاذ القرار وذلك بوضع بروتوكولات لتطبيق هذه السياسة للسيطرة على التعقيدات الخطرة.

يَتضمّنُ الفصلِ الخامس أيضاً المراجع الببليوغرافيةَ كتباً كانت أو من المواقع الألكترونية بالإضافة إلى الملاحقِ (1-2-3).

ABSTRACT

This research is conducted so as to know the importance of ultrasonography (Echocardiography) in the control, follow-up & the diagnosis of the different types of cardiac complications of hypertension (the impact of HTN on the heart muscles, valves and blood supply). The data was collected from Ribat teaching hospital, using an echocardiographic machine. This includes the left atrium, the left ventricular diameter in both systolic and diastolic phases as well as the valvular diameters, ejection fraction and SPSA. This study is about the effect of HTN on the heart anatomy and function. The first chapter is an introductory phrase about the key words of the title as well as the general and specific objectives of the study to enumerate the steps and the variables of the study; this is after showing us the story of the problem and the justification to conduct it and why about the heart effects in HTN. An overview of the study is added in this chapter. The second chapter gives us the previous scientific literature and researches, also includes anatomy, physiology, pathology, normal sonographic appearance of the heart and the normal cardiac measurements using M-mode. In the third chapter the technical methods and tools as well as the materials and procedures important to collect the data are mentioned, also the technique and the statistical methods used, SPSS version 16 by which the data has been tabulated represented and analyzed using Chi^2 test. The fourth chapter is about the results and data analysis. The last chapter describes the discussion, recommendations and conclusion. In the discussion we found that there is a relationship between a high blood pressure or HTN and the changes in the anatomy and function of the heart ranging from week to strong one which reflected by a significant chi square test (tables 11 & 12).Our strong recommendation to all health workers that, the use of echocardiography in their diagnosis and follow up of hypertensive heart diseases is mandatory. Also there is strong relationship between age & LVH, seen in the crosstab tables.

This chapter also includes the bibliographic references as well as the appendixes (1-2-3).

CHAPTER ONE

1-1INTRODUCTION

The heart is the most important vital organ in the body located within the chest in the left side posterior to the sternum in the middle mediastinum, relatively small conical approximately the size of a person's clenched fist (Chummy S. Sinnatamby, 2004).It weighs about 250 to 350 grams, rotated such that its right side or border {Rt atrium and ventricle} located more anteriorly, while its left side or border {Lt atrium and ventricle} is located more posteriorly (1). It pumps the deoxygenated blood to the lungs through the pulmonary trunk via the pulmonary valve to be oxygenated. This right ventricular systole is done by the myocardium including the interventricular septum. On the other hand the left atrial filling {diastole} is with oxygenated blood which is send by both passive and active flow to the left ventricle via the mitral valve {atrial systole} ,however, this is also the left ventricular diastole . Its systole is the ejection of blood to the aorta via the aortic valve by the strong and large myocardium of this chamber. This cardiac cycle is controlled by the conduction system of the heart and the autonomic nervous system (Roderick NM Mac Sween& Keith Wholly, 1998).

These alternate cycles of the wall contraction and relaxation develop the so called blood pressure. Minimum blood pressure is essential to push the blood through the blood vessels to the body tissues for nutrient supply and waste exchange. High blood pressure more than (140/90) is considered in some types of classifications as HYPERTENTION.

Hypertension (HTN) or high blood pressure, sometimes called arterial hypertension, is a chronic medical condition in which the blood pressure in the arteries is elevated.(Chobanian AV, Bakris GL, Black HR et al. (December 2003). This requires the heart to work harder than normal to circulate blood through the blood vessels. Blood pressure is summarized by two measurements, systolic and diastolic, which depend on whether the heart muscle is contracting (systole) or relaxed between beats (diastole) and equate to a maximum and minimum pressure, respectively. Normal blood pressure at rest is within the range of 100-140mmHg systolic (top reading) and 60-90mmHg diastolic (bottom reading). High blood pressure is said to be present if it is persistently at or above 140/90 mmHg.

Hypertension is classified as either primary (essential) hypertension or secondary hypertension; about 90–95% of cases are categorized as "primary hypertension" which means high blood pressure with no obvious underlying medical cause.[Carretero OA, Oparil S (January 2000].The remaining 5–10% of cases (secondary hypertension) are caused by other conditions that affect the kidneys, arteries, heart or endocrine system.

Hypertension is a major risk factor for stroke, myocardial infarction (heart attacks), heart failure, aneurysms of the arteries (e.g. aortic aneurysm), peripheral arterial disease and is a cause of chronic kidney disease. Even moderate elevation of arterial blood pressure is associated with a shortened life expectancy. Dietary and lifestyle changes can improve blood pressure control and decrease the risk of associated health complications, although drug treatment is often necessary in people for whom lifestyle changes prove ineffective or insufficient.

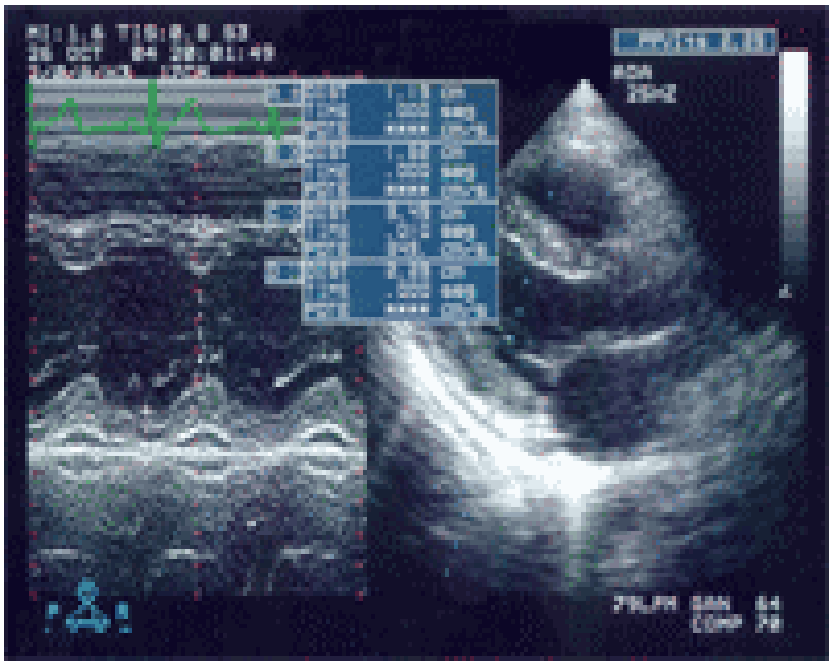


FIG (1-1) Normal echocardiogram – M mode

Echocardiogram and high blood pressure:

An echocardiogram is a scan which gives a detailed view of the structures of your heart, and which can show how well your heart is working. The scan uses a probe that sends out sound waves, which are reflected back by the muscles and tissues in your heart. These reflected waves are picked up by the probe and translated into images on a screen.

Echocardiograms can show if your heart is working as well as it should. They are particularly useful for revealing if you have an enlarged left side of the heart, or problems with your heart valves. They can also be used to investigate the causes and effects of heart murmurs and heart attacks.

How is an echocardiogram carried out?

An echocardiogram is a painless test that takes roughly 30-45 minutes. It can be carried out in one of two ways. If your doctor is looking for an enlarged heart muscle or heart valve problems, the probe is normally placed on your chest. Lubricating jelly will be put on your chest and a small probe will be moved around on your chest. Moving the probe around will give different views of your heart.

If your doctor is looking for more detailed information about how your heart is working, a small probe will be passed down your throat so that it lies behind your heart. This means that your doctor will have a much clearer view, as your heart and ribcage will not be in the way. You will receive a sedative and/or local anaesthetic for this procedure, but it should not require an overnight stay in hospital.

What do I need to do before an echocardiogram?

If the probe is going to be placed on the chest, no preparations needed. If the probe is going to be passed down the throat (transoesophageal), the patient will be asked not to eat anything for a few hours before the test because he will be given a sedative. Also, he will be asked not to drive for 24 hours after the test because he may still be slightly sleepy. For both tests the patient should continue to take his blood pressure medicines and any other medicines as normal.

Echocardiography; the use of ultrasound to examine the heart; is a safe, powerful, non-invasive and painless technique. Sound is a disturbance propagating in a material (air, water, body tissue or a solid substance).Echocardiography (echo or echocardiogram) is a type of

ultrasound test that uses high-pitched sound waves to produce an image of the heart. The sound waves are sent through a device called a transducer and are reflected off the various structures of the heart. These echoes are converted into pictures of the heart that can be seen on a video monitor. There is no special preparation for the test, only ultrasound gel is applied to the transducer to allow transmission of the sound waves from the transducer to the skin. The transducer transforms the echo (mechanical energy) into an electrical signal which is processed and displayed as an image on the screen. The conversion of sound to electrical energy is called the piezoelectric effect. (SAM KADDOURA, 2002).

Ultrasound shows in easy way without penetration or any harm to the patient the effect of hypertension on the heart. Six windows are used in echocardiography to image the heart, the left parasternal (short & long views) - apical –sternal – sub costal – right parasternal – transoesophageal (SAM KADDOURA, 2002).

1-2 STATEMENT OF THE BROBLEM

In long standing hypertension (HTN) and to some extent in newly discovered disease one or more of the following complications appear. Depending on the severity and the period as well as the control of HTN; ischemia in the heart and brain, cardiomegaly, valvular lesions, renal affection, eye problems and other system affection will appear.

The most common cardiac complications of HTN include myocardial thickening which may lead to myocardial infarction, left ventricular diameter abnormalities, septal thickening, valvular lesions, intra and extra cardiac masses, fluid collections and abnormal direction of blood flow. The problem here is that weather there is really a cardiac complication or not and to what extent? Is this has got any relation with the period? The severity and the degree of control or not? Are the social status, age, sex and nutrition will participate in the incidence and prevalence of the above mentioned complications?

High blood pressure is the most common chronic medical problem prompting visits to primary health care providers. The estimated direct and indirect costs of high blood pressure and its heart complications is

increasing with time due to bad life style, but only 48% of people aware that they have hypertension are adequately controlled.[Lloyd-Jones D, Adams RJ, Brown TM et al. (February 2010].Adequate management of hypertension can be hampered by inadequacies in the diagnosis, treatment, and/or control of high blood pressure. [Alcocer L, Cueto L (June 2008]Health care providers face many obstacles to achieving blood pressure control, including resistance to taking multiple medications to reach blood pressure goals. People also face the challenges of adhering to medicine schedules and making lifestyle changes. Nonetheless, the achievement of blood pressure goals is possible, and most importantly, lowering blood pressure significantly reduces the risk of death due to heart disease and stroke, the development of other debilitating conditions, and the cost associated with advanced medical care.

In order to diagnose as well as to participate in the control of these effects and to improve the health style of patients of HTN this study is conducted and it selects echocardiography to study the heart.

1-3 RESEARCH OBJECTIVES

1-2-1 General objectives :

To study the impact of hypertension on the heart muscles, cavities and valves including structure and function.

1-2-2 Specific objectives :

- To measure the left ventricular walls (left ventricular posterior wall – LVPW, interventricular septum - IVS) thickness in hypertensive patients.
- To measure the different left ventricular diameters {left ventricular end systolic diameter (LVESD) and the left ventricular end diastolic diameter (LVEDD)}.
- To characterize the valvular lesions that result from HTN (aortic, mitral, tricuspid and pulmonary stenosis and regurgitation).
- To identify the motion activity of the myocardium and the diastolic as well as the systolic dysfunction if ever in HTN.
- To assess the blood flow in HTN using Doppler capability.

In order to achieve all these objectives, proper methods and tools should be selected to pick up the variable and to collect the data.

1-4 THESIS OVERVIEW:

This study consists of five chapters:

- **Chapter one**: contains introduction and objectives (general and specific).
- **Chapter two**: literature review in two parts; part one is about anatomy, physiology and normal sonographic appearances of the heart and part two is about the pathology of the heart in hypertensive patients.
- **Chapter three**: contains the materials and methods.
- **Chapter four:** contains the results presentation as well as tabulation and graphs.
- **Chapter five:** contains the discussion, conclusion and recommendations as well as references used in this research.

CHAPTER TOW

Literature review

Firstly we will review the anatomy, physiology, ultrasound technique as well as the echocardiographic features of the heart and secondly the pathological features in this organ in hypertensive patient and previous studies.

2-1- Anatomy of the heart:

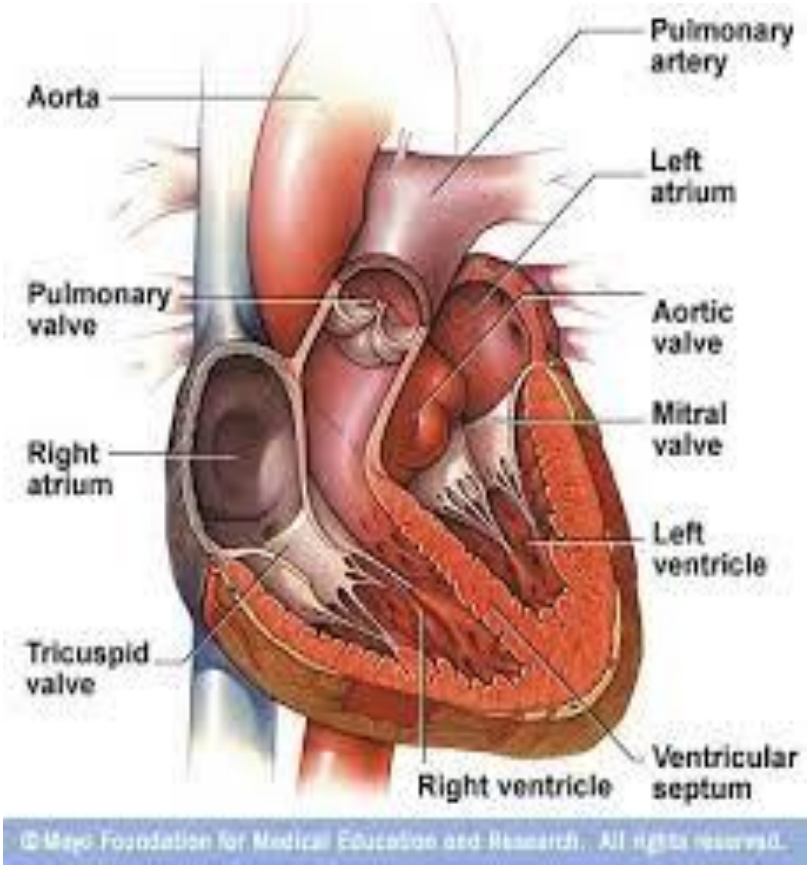


FIG (2-1) coronal section of the heart showing muscles and valves

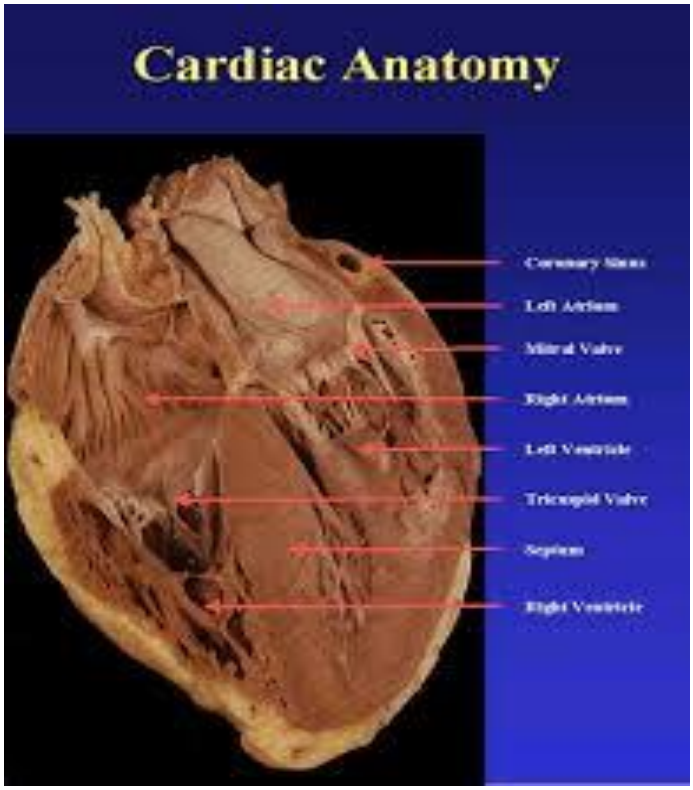


FIG (2-2) Cadaveric heart sectioned coronally

2-2- Physiology of the heart:

The Heart

The heart itself is made up of 4 chambers, 2 atria and 2 ventricles. De-oxygenated blood returns to the right side of the heart via the venous circulation. It is pumped into the right ventricle and then to the lungs where carbon dioxide is released and oxygen is absorbed. The oxygenated blood then travels back to the left side of the heart into the left atria, then into the left ventricle from where it is pumped into the aorta and arterial circulation.

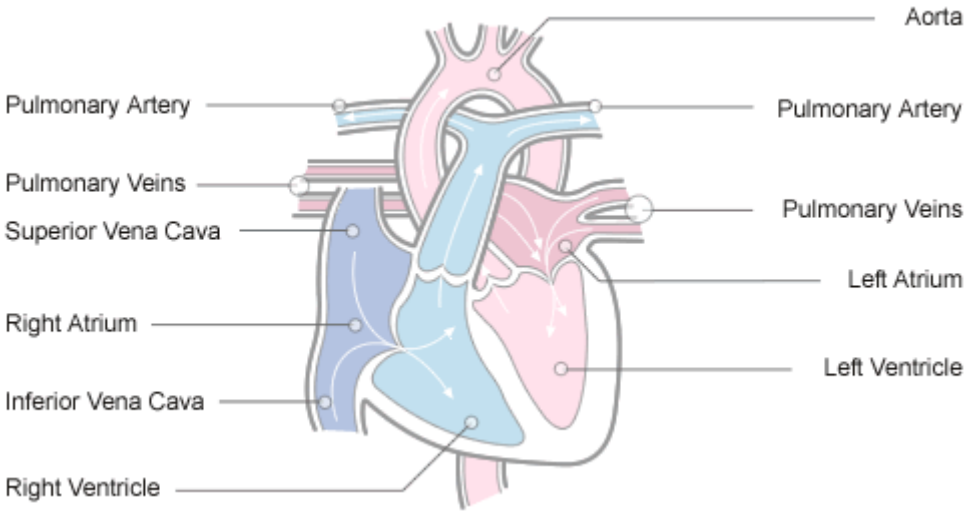


FIG (2-3) Image: The passage of blood through the heart

The pressure created in the arteries by the contraction of the left ventricle is the systolic blood pressure. Once the left ventricle has fully contracted it begins to relax and refill with blood from the left atria. The pressure in the arteries falls whilst the ventricle refills. **This is the diastolic blood pressure.** The atrio-ventricular septum completely separates the 2 sides of the heart. Unless there is a septal defect, the 2 sides of the heart never directly communicate. Blood travels from right side to left side via the lungs only. However **the chambers themselves work together**. The two atria contract simultaneously, and the two ventricles contract simultaneously. Ginners Guide to Normal Heart Function, Sinus Rhythm & Common Cardiac Arrhythmias.

Normal Function of the Heart:

The simplest way to describe the heart is as a "pump". Quite often doctors and nurses take the analogy further and talk to patients about their "plumbing". This analogy is reasonably accurate. The role the heart plays in the cardiovascular system is similar to the role played by the pump in your central heating system. The heart pumps blood through the arteries and veins to organs, muscles and tissues, just as the central heating pump forces hot water through the pipes to the radiators. But that is where the analogy ends.

The cardiovascular system is made up of: heart; lungs; arteries and veins, and all are under the control of the autonomic nervous system

(sympathetic and parasympathetic). In a healthy individual with a healthy heart, heart rate is dictated by the body's needs. If an individual is resting then organs, muscles and tissues require a reduced amount of blood and oxygen. The result of this is a reduction in blood pressure and a slowing down of heart rate and respirations.

When the individual becomes active then the organs, muscles and tissues require an increasing amount of blood and oxygen, resulting in raised blood pressure and an increase in heart rate and respirations. These responses are all involuntary, under the direct control of the autonomic nervous system. If the individual remains reasonably healthy with no cardiac complications then the cardiovascular system will continue to work just like this for life.

2-3- Pathology of the heart:

The etiology of hypertensive heart disease is a complex interplay of various hemodynamic, structural, neuroendocrine, cellular, and molecular factors. These factors play integral roles in the development of hypertension and its complications; however, elevated BP itself can modulate these factors.

Obesity has been linked to hypertension and LVH in various epidemiologic studies, with as many as 50% of obese patients having some degree of hypertension and as many as 60-70% of patients with hypertension being obese.

Elevated BP leads to adverse changes in cardiac structure and function in 2 ways: directly, by increased after load, and indirectly, by associated neurohormonal and vascular changes. Elevated 24-hour ambulatory BP and nocturnal BP have been demonstrated to be more closely related to various cardiac pathologies, especially in black persons. The pathophysiologies of the various cardiac effects of hypertension differ and are described in this section.

Left ventricular hypertrophy:

Of patients with hypertension, 15-20% develops LVH. The risk of LVH is increased 2-fold by associated obesity. The prevalence of LVH based on electrocardiogram (ECG) findings, which are not a sensitive marker at the time of diagnosis of hypertension, is variable. Studies have shown a direct relationship between the level and duration of elevated BP and LVH.

LVH, defined as an increase in the mass of the left ventricle, is caused by the response of myocytes to various stimuli accompanying elevated BP. Myocyte hypertrophy can occur as a compensatory response to increased after load. Mechanical and neurohormonal stimuli accompanying hypertension can lead to activation of myocardial cell growth, gene expression (of which some occurs primarily in fetal cardiomyocytes), and, thus, to LVH. In addition, activation of the renin-angiotensin system, through the action of angiotensin II on angiotensin I receptors, leads to growth of interstitial and cell matrix components. In summary, the development of LVH is characterized by myocyte hypertrophy and by an imbalance between the myocytes and the interstitium of the myocardial skeletal structure.

Various patterns of LVH have been described, including concentric remodeling, concentric LVH, and eccentric LVH. Concentric LVH is an increase in LV thickness and LV mass with increased LV diastolic pressure and volume, commonly observed in persons with hypertension; this is a marker of poor prognosis in these patients. Compare concentric LVH with eccentric LVH, in which LV thickness is increased not uniformly but at certain sites, such as the septum.

Although the development of LVH initially plays a protective role in response to increased wall stress to maintain adequate cardiac output, it later leads to the development of diastolic and, ultimately, systolic myocardial dysfunction. Interestingly, findings from a prospective study (The Multiethnic Study of Atherosclerosis [MESA] trial) also indicate a higher risk of developing systemic hypertension among patients in the higher quartiles of the LV mass at baseline.

Left atrial abnormalities :

Frequently underappreciated, structural and functional changes of the left atrium are very common in patients with hypertension. The increased afterload imposed on the LA by the elevated LV end-diastolic pressure secondary to increased BP leads to impairment of the left atrium and left atrial (LA) appendage function, plus increased LA size and thickness. Increased LA size accompanying hypertension in the absence of valvular heart disease or systolic dysfunction usually implies chronicity of hypertension and may correlate with the severity of LV diastolic dysfunction. In addition to LA structural changes, these patients are predisposed to atrial fibrillation. Atrial fibrillation, with loss of atrial contribution in the presence of diastolic dysfunction, may precipitate overt heart failure.

Valvular disease :

Although valvular disease does not cause hypertensive heart disease, chronic and severe hypertension can cause aortic root dilatation, leading to significant aortic insufficiency. Some degree of hemodynamically insignificant aortic insufficiency is often found in patients with uncontrolled hypertension. An acute rise in BP may accentuate the degree of aortic insufficiency, with return to baseline when the BP is better controlled. In addition to causing aortic regurgitation, hypertension is also thought to accelerate the process of aortic sclerosis and cause mitral regurgitation.

Heart failure :

Heart failure is a common complication of chronically elevated BP. Patients with hypertension fall into 1 of the following categories:

- Asymptomatic but at risk of developing of heart failure - Stage A or B, per the American College of Cardiology (ACC)/American Heart Association (AHA) classification, depending on whether or not they have developed structural heart disease as a consequence of hypertension
- Suffering from symptomatic heart failure - Stage C or D, per the ACC/AHA classification

Hypertension as a cause of CHF is frequently under recognized, partly because at the time heart failure develops, the dysfunctioning left

ventricle is unable to generate the high BP, thus obscuring the heart failure's etiology. The prevalence of asymptomatic diastolic dysfunction in patients with hypertension and without LVH may be as high as 33%. Chronically elevated afterload and the resulting LVH can adversely affect the active early relaxation phase and the late compliance phase of ventricular diastole.

Diastolic dysfunction:

Diastolic dysfunction is common in persons with hypertension. It is often, but not invariably, accompanied by LVH. In addition to elevated afterload, other factors that may contribute to the development of diastolic dysfunction include coexistent coronary artery disease, aging, systolic dysfunction, and structural abnormalities such as fibrosis and LVH. Asymptomatic systolic dysfunction usually follows.

Systolic dysfunction:

Later in the course of disease, the LVH fails to compensate by increasing cardiac output in the face of elevated BP, and the LV cavity begins to dilate to maintain cardiac output. As the disease enters the end stage, LV systolic function decreases further. This leads to further increases in activation of the neurohormonal and renin-angiotensin systems, leading to increases in salt and water retention and increased peripheral vasoconstriction. Eventually, the already compromised LV is overwhelmed, and the patient progresses to the stage of symptomatic systolic dysfunction.

Decompensation:

Apoptosis, or programmed cell death, stimulated by myocyte hypertrophy and the imbalance between its stimulants and inhibitors, is considered to play an important part in the transition from compensated to decompensated stage. The patient may become symptomatic during the asymptomatic stages of the LV systolic or diastolic dysfunction, owing to changes in afterload conditions or to the presence of other insults to the myocardium (e.g., ischemia, infarction). A sudden increase in BP can lead to acute pulmonary edema without necessarily changing the LV ejection fraction.

Generally, development of asymptomatic or symptomatic LV dilatation or dysfunction heralds rapid deterioration in clinical status and a markedly increased risk of death. In addition to LV dysfunction, right ventricular (RV) thickening and diastolic dysfunction also develop as results of septal thickening and LV dysfunction.

Myocardial ischemia:

Patients with angina have a high prevalence of hypertension. Hypertension is an established risk factor for the development of coronary artery disease, almost doubling the risk. The development of ischemia in patients with hypertension is multifactorial.

Importantly, in patients with hypertension, angina can occur in the absence of epicardial coronary artery disease. The reason for this is 2-fold. Increased afterload secondary to hypertension leads to an increase in LV wall tension and transmural pressure, compromising coronary blood flow during diastole. In addition, the microvasculature beyond the epicardial coronary arteries has been shown to be dysfunctional in patients with hypertension, and it may be unable to compensate for increased metabolic and oxygen demand.

The development and progression of arteriosclerosis, the hallmark of coronary artery disease, is exacerbated in arteries subjected to chronically elevated BP. Shear stress associated with hypertension and the resulting endothelial dysfunction cause impairment in the synthesis and release of the potent vasodilator nitric oxide. A decreased nitric oxide level promotes the development and acceleration of arteriosclerosis and plaque formation. Morphologic features of the plaque are identical to those observed in patients without hypertension.

Cardiac arrhythmias:

Cardiac arrhythmias commonly observed in patients with hypertension include atrial fibrillation, premature ventricular contractions (PVCs), and ventricular tachycardia (VT).The risk of sudden cardiac death is increased. Various mechanisms thought to play a part in the pathogenesis of arrhythmias include altered cellular structure and metabolism, inhomogeneity of the myocardium, poor perfusion, myocardial fibrosis, and fluctuation in afterload. All of these may lead to an increased risk of ventricular tachyarrhythmias.

Atrial fibrillation (paroxysmal, chronic recurrent, or chronic persistent) is observed frequently in patients with hypertension. In fact, elevated BP is the most common cause of atrial fibrillation in the Western hemisphere. In one study, nearly 50% of patients with atrial fibrillation had hypertension. Although the exact etiology is not known, LA structural abnormalities, associated coronary artery disease, and LVH have been suggested as possible contributing factors. The development of atrial fibrillation can cause decompensation of systolic and, more importantly, diastolic dysfunction, owing to loss of atrial kick, and it also increases the risk of thromboembolic complications, most notably stroke.

Premature ventricular contractions, ventricular arrhythmias, and sudden cardiac death are observed more often in patients with LVH than in those without LVH. The etiology of these arrhythmias is thought to be concomitant coronary artery disease and myocardial fibrosis.

Cardiomyopathy:

Cardiomyopathy refers to diseases of the heart muscle. These diseases have many causes, signs and symptoms, and treatments. In cardiomyopathy, the heart muscle becomes enlarged, thick, or rigid. In rare cases, the muscle tissue in the heart is replaced with scar tissue. As cardiomyopathy worsens, the heart becomes weaker. It's less able to pump blood through the body and maintain a normal electrical rhythm. This can lead to heart failure or irregular heartbeats called arrhythmias . In turn, heart failure can cause fluid to build up in the lungs, ankles, feet, legs, or abdomen. The weakening of the heart also can cause other complications, such as heart valve problems.

The main types of cardiomyopathy are:

- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- Restrictive cardiomyopathy
- Arrhythmogenic right ventricular dysplasia

Other types of cardiomyopathy sometimes are referred to as "unclassified cardiomyopathy."Cardiomyopathy can be acquired or inherited. "Acquired" means you aren't born with the disease, but you develop it due to another disease, condition, or factor. "Inherited" means your

parents passed the gene for the disease on to you. Many times, the cause of cardiomyopathy isn't known. Cardiomyopathy can affect people of all ages. However, people in certain age groups are more likely to have certain types of cardiomyopathy. This article focuses on cardiomyopathy in adults.

Some people who have cardiomyopathy have no signs or symptoms and need no treatment. For other people, the disease develops quickly, symptoms are severe, and serious complications occur. Treatments for cardiomyopathy include lifestyle changes, medicines, surgery, implanted devices to correct arrhythmias, and a nonsurgical procedure. These treatments can control symptoms, reduce complications, and stop the disease from getting worse.

2-4- Normal sonographic (echocardiographic) appearance of the heart:

A- At rest, this is done by the following view:

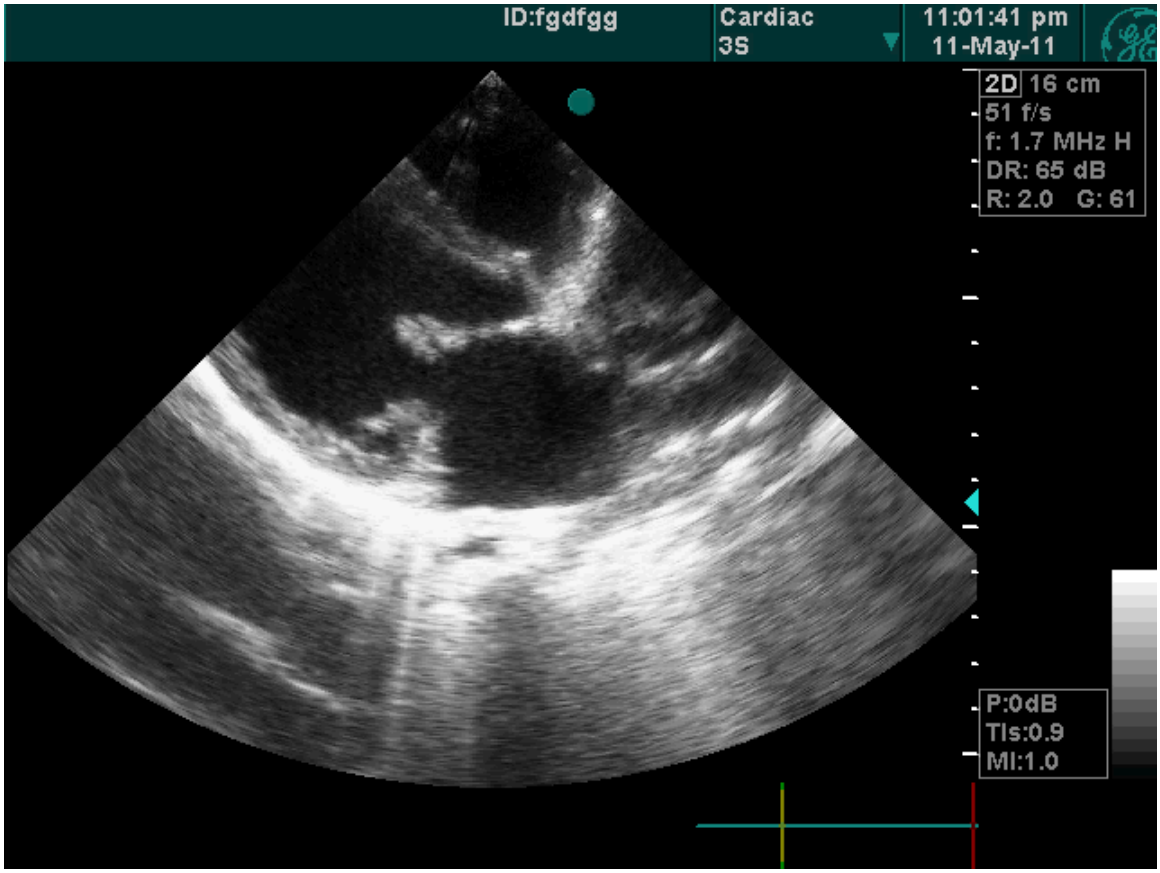
1- Parasternal views: the so called Lt & Rt

2- Apical view.

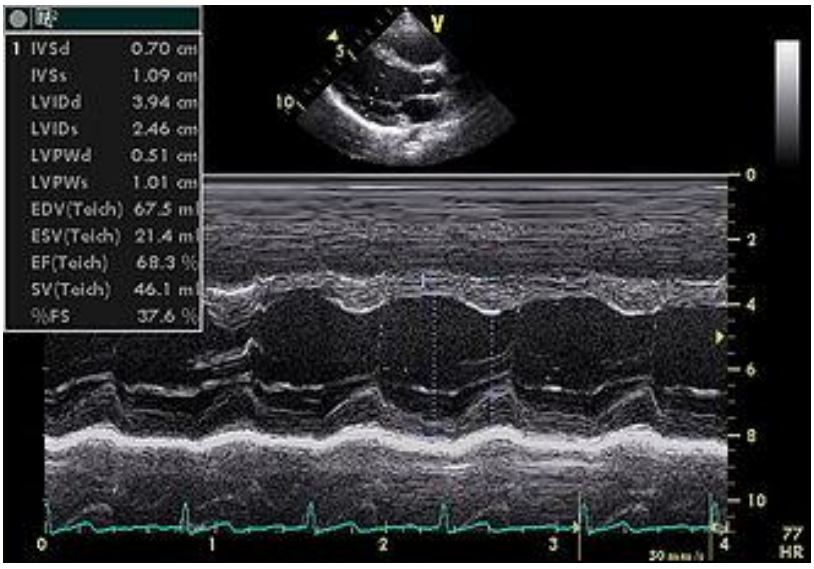
3- Sub costal view.

4- Suprasternal view.

5- Transoesophageal view.



a



b

FIG (2-4), a-Echocardiogram in the Lt Parasternal long-axis view, b- showing a measurement of the hearts left ventricle M-mode

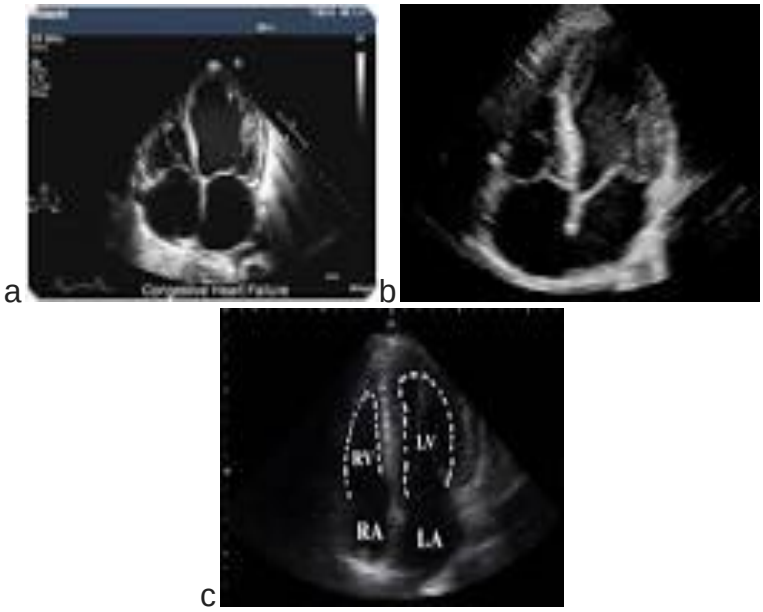


FIG (2-5) four chamber apical view (a-b-c)

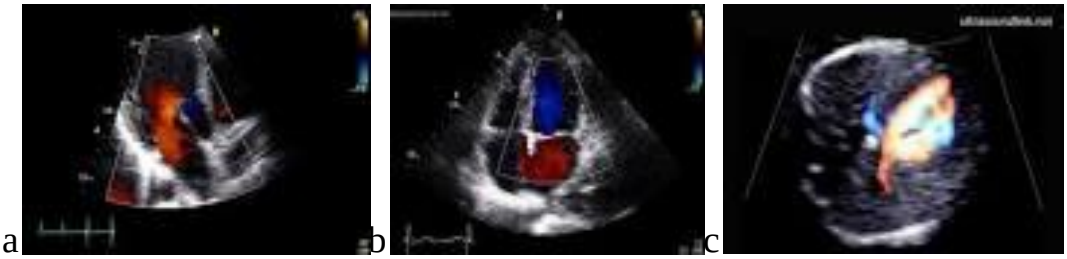


FIG (2-6) color Doppler four chamber apical view (a-b-c)

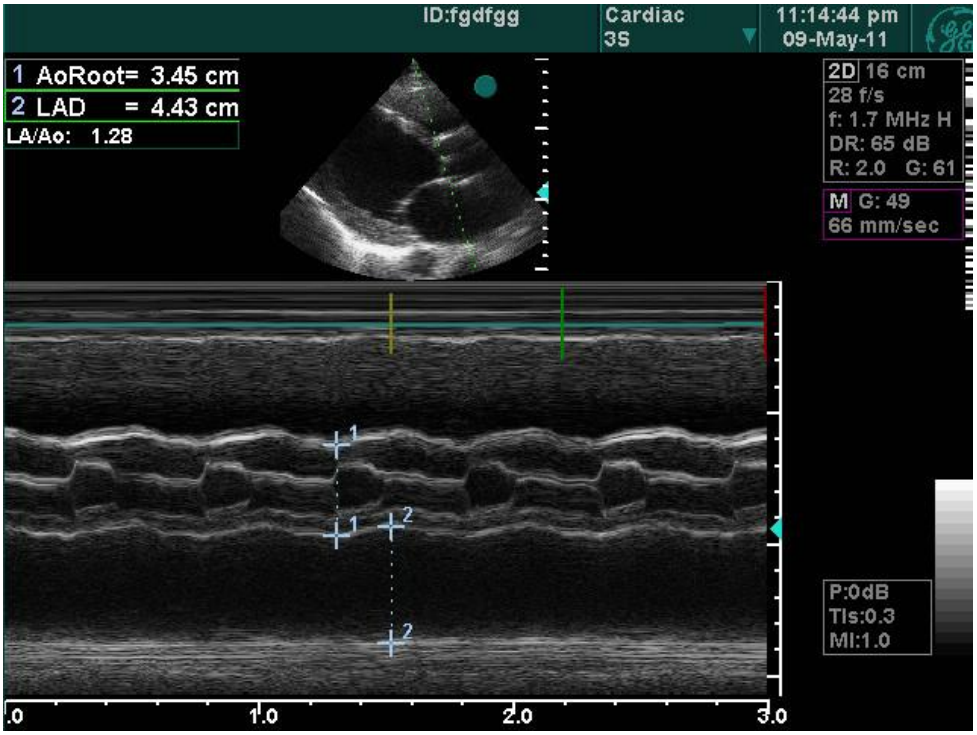


FIG (2-7) M- mode, used in measurements of diameters: +1+ LVSD, +2+ LVDD

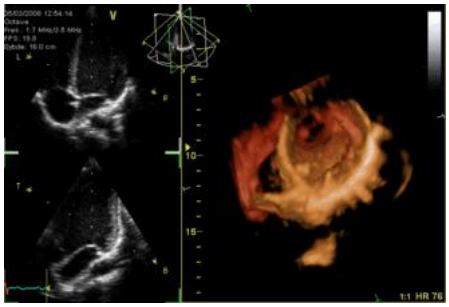


FIG (2-8) 3D echocardiogram of a heart viewed from the apex

2-5- Normal cardiac measurements: using M mode

PARAMETER	NORMAL RANGE
Aortic root	**(20-40 mm)**
Lt Atrium	**(19-40 mm)**
LVDD	**(35-56 mm)**
LVSD	**(22-40 mm)**
IVS	**(6-11 mm)**
LVPW	**(6-11 mm)**
FS	**(30-40 %)**
EF	**(55-75 %)**

Ejection Fraction (EF) is a key indicator of heart health and is frequently used to determine the pumping function of the heart. Simply stated, EF is the amount of blood pumped out of the heart during each beat or contraction. In a healthy heart, 55-75% of the blood is pumped out during each beat. This indicates that the heart is pumping well and able to deliver an adequate supply of blood to the body and brain. Many people with heart failure and heart disease pump out less than 50%. Heart failure (also known as congestive heart failure) is a condition in which the heart is not able to pump enough blood to meet the oxygen demands of the body. For heart patients, knowing your EF is key first step to determining your risk for Sudden Cardiac Arrest. A commonly used test to determine your EF is a heart ultrasound (echocardiogram). This is non-invasive and easy on the patient, and is often performed right in the doctor's office. By using ultrasound or sound waves, measurements are taken of the heart and with these measurements the pumping function the heart is calculated. If you have heart disease, it is important to have your EF measured regularly, the same way that you have your blood pressure and cholesterol checked regularly. Keep in mind that your EF number can

change, so it’s important that you talk to your doctor about tracking it over time.

B-Stress echocardiography:

Cardiac stress test

A stress echocardiogram, also known as a stress echo or SE, utilizes ultrasound imaging of the heart to assess the wall motion in response to physical stress. First, images of the heart are taken "at rest" to acquire a baseline of the patient's wall motion at a resting heart rate. The patient then walks on a treadmill or utilizes another exercise modality to increase the heart rate to his or her target heart rate, or 80% of the age predicted max heart rate (age predicted max heart rate = 220 − patient's age). Finally, images of the heart are taken "at stress" to assess wall motion at the peak heart rate. A stress echo assesses wall motion of the heart; it does not, however, image the coronary arteries directly. Ischemia of one or more coronary arteries could cause a wall motion abnormality which could indicate coronary artery disease (CAD). The gold standard test to directly image the coronary arteries and directly assess for stenosis or occlusion is a cardiac catheterization. A stress echo is a non-invasive test and is performed in the presence of a licensed medical professional, such as a cardiologist, and a cardiac sonographer.

2-7-Previous studies:

Study 1:

Articles: Effect of Hypertension and Cardiac Hypertrophy on Coronary Artery Morphology in Sudden Cardiac Death, done by

1. Allen P. Burke, MD;
2. Andrew Farb, MD;
3. You-hui Liang, MD;
4. John Smialek, MD;
5. RenuVirmani, MD

In the Department of Cardiovascular Pathology, Armed Forces Institute of Pathology, Washington, DC.

Methods and Results include Heart weight and coronary plaque morphology were prospectively compared in SCD in 36 hypertensive and 63 normotensives individuals. The frequency of CAD was similar in hypertensives (69%, n=25) and normotensives (73%, n=46). In 71 hearts with CAD, acute coronary thrombi were present in 76% of normotensives versus 36% of hypertensives (P=.002), LVH was present in 64% of hypertensives versus 33% of normotensives (P=.01) and in 72% of hypertensives with one-vessel disease versus 17% of normotensives with one-vessel disease (P=.0005), and a healed or acute infarct without acute thrombus was present in 36% of hypertensives versus 9% of normotensives (P=.007). Heart weight was higher in all cases of plaque rupture (519±109 g) than eroded plaque (381±92 g, P=.0002). In contrast to hypertensives, normotensives hearts with severe CAD showed a stepwise increase in heart weight with one-, two-, and three-vessel disease (P=.01).

Conclusions Severe CAD is present in most SCD in hypertensive and normotensives individuals, but acute thrombi are more common in normotensives. LVH is an important contributing mechanism of SCD in hypertensives, especially in cases of one-vessel disease. LVH is associated with plaque rupture and extent of disease in SCD in normotensives with severe CAD.

Study 2:

Articles:Effect of essential hypertension on cardiac autonomic function in type 2 diabetic patients, done by:

Naohiko Takahashi, MD; Mikiko Nakagawa, MD; TetsunoriSaikawa, MD; TatsuhikoOoie, MD; KunioYufu, MD; SakujiShigematsu, MD; Masahide Hara, MD; Hiroshi Sakino, MD; Isao Katsuragi, MD; ToshimitsuOkeda, MD; Hironobu Yoshimatsu, MD; Toshiie Sakata, MD .Using Echocardiography, M-mode two-dimensional echocardiography and cardiac Doppler recordings were obtained by means of a phase-array echo-Doppler system. Echocardiograms were obtained in a standard manner using standard parasternal, short-axis and apical views. Left ventricular (LV) mass was calculated according to a previous study:

where LVIDd = LV internal dimension at end-diastole; IVSTd = intraventricularseptal thickness at end-diastole and PWTd = posterior wall thickness at end-diastole. Left ventricular mass was divided by body surface area to calculate the LV mass index. Pulsed Doppler recordings were made from the standard apical four-chamber view. Mitral inflow velocity was recorded with the sample volume at the mitral annulus level; the average of ≥3 cardiac cycles was taken. The following measurements were made: peak velocity of early ventricular filling (E), peak velocity of late ventricular filling (A), their ratio (E/A) and deceleration time.

Measurement of BRS and plasma norepinephrine (NE) concentration

All subjects were studied while in supine position in a quiet room with dimmed lights between 9 and 11 AM. A catheter was inserted in the right cubital vein, and arterial BP was recorded noninvasively using tonometry (Jentow-7700, Nihon Colin, Komaki, Japan). The tonometric sensor was attached over the left radial artery. The accuracy of continuous BP monitoring using this system has been demonstrated previously. Arterial BP and the standard 12-lead ECG were monitored simultaneously, and data were stored in a PCM data recorder (RD-200T; TEAC, Tokyo, Japan). Three-lead precordial Holter ECG recordings (model-459, Del Mar Avionics, Irvine, California) were also obtained throughout the procedure for analysis of HRV.

After a waiting period of 30 min to allow cardiovascular baroreflex mechanisms to stabilize, patients were asked to breathe at a rate of 15 breaths/min using a metronome to maximize regularity between respiration and cardiovascular function. A blood sample was obtained from the venous catheter for measurement of plasma NE concentration. Baroreflex sensitivity was assessed by the phenylephrine method, as previously described. Phenylephrine (2 to 3 μg/kg) was injected over 15 s to obtain a 15 to 40 mm Hg systolic BP increase. Baroreflex sensitivity was calculated as the slope of the linear regression line relating systolic BP changes to RR interval changes. Regression lines with more than 20 data points and a correlation coefficient (r) >0.8 were accepted for analysis. The mean of the two slope values was taken as the BRS value.

CHAPTER THREE

Materials and methods

3-1- Materials & tools:

An echocardiographic machine (SIEMENS-ACUSON CV70 Ultrasound imaging system), with Doppler & M-mode capabilities is used. The probe is of a SECTER type .The transducer is a phased - array 3.5 MHZ, and ultrasound gel is applied to the transducer to prevent any attenuation or artifact. And thermal Paper Printer was used.

A questionnaire is used to collect the data and to number the patients.

3-2- Methods:

The study was conducted in Ribat university hospital, Khartoum city, from the first of May to the thirty first of August 2013; all attending hypertensive patients. From the study population, in a random way a total of FIFTY EIGHT patients (n=58) were selected to be the sample unit in this study.

The inclusion criterion is that any hypertensive patient attending the hospital in that period mentioned.

The exclusion criteria are that any child (less than 18 years) or a pregnant women with HTN. A written permission is issued and taken from the hospital director; also anyone in the study signed an agreement to be one of the study objects after had been told about what should be done for him.

To collect the suitable data for the study; personal information from any patient is written in the questionnaire paper as well as the result .This includes the following:

1- Name, sex, age, period of the disease, type of the drugs and the last three readings of BP (controlled or not).
2- Routine last investigations for chronic diseases (urine, renal & liver function tests, cardiac enzymes, lipid profile).

3-3-TECHNIQUE:

From the ultrasound technique, in which there are six windows the so called: (Rt&Lt parasternal view, apical view, sub costal view, suprasternal view and transoesophageal view); that use one or both of the two patient positions, either left lateral decubitus or supine, from it the following results is collected :

1- Left ventricular posterior wall (LVPW) size or thickness (using M – mode).

2- Interventricular septum (IVS) size (using M –mode).

3- Left ventricular end systolic diameter (LVESD) (using M-mode).
4- Left ventricular end diastolic diameter (LVEDD) (using M-mode).
5- Valvular sizes, including aortic root, mitral valve, tricuspid valve and the pulmonary valve (using M-mode).
6- Blood flow (using Doppler).
7- Ejection fraction (EF).

3-4- statistics:

Finally these data is tabulated, described, represented and analyzed using SPSS version 16,putting in mind that the p value is 0.05 using the chi square test to know the significance . The results of this analysis put in a scientific frames and facts from which the medical decision and recommendations is created in the discussion chapter.

CHAPTER FOUR

Results

4-1-Data Analysis:

The data was collected from the patients admitted in the Ribat University Hospital during the period from 1st April 2013 to 30th June 2013, and it is analyzed using the Statistical Package for Social Science – SPSS version 16.0. The following pages present and discuss the results of the analysis.

1/ Descriptive Statistics:

Table (1): Sex

	Frequency	Percent	Valid Percent	Cumulative Percent
Male	27	46.6	46.6	46.6
Female	31	53.4	53.4	100.0
Total	58	100.0	100.0	

Table (1) above shows that the gender of patients surveyed in this study is distributed 46.6% males and 53.4% females, as shown in the figure below.

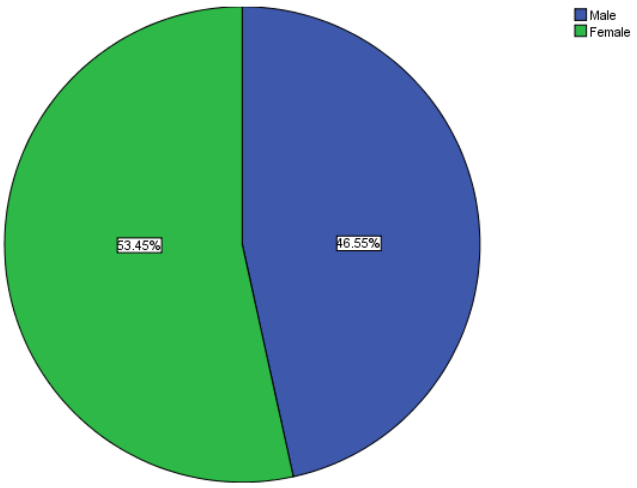


Figure (1): Patients Sex Distribution

Table (2): Age

	Frequency	Percent	Valid Percent	Cumulative Percent
30 Years and Less	2	3.4	3.4	3.4
31 - 40 Years	2	3.4	3.4	6.9
41 - 50 Years	2	3.4	3.4	10.3
51 - 60 Years	16	27.6	27.6	37.9
61 - 70 Years	24	41.4	41.4	79.3
71 Years and More	12	20.7	20.7	100.0
Total	58	100.0	100.0	

The table number (2) above indicates that the majority of the patients in this study aged between 61 – 70 years and 51 – 60 years with a percentage 41.4% and 26.6% respectively. The age class 71 years and more was 20.7%. Other age classes are 30 years and less, 31 – 40 years, 41 – 50 years were 3.4% for each, as the below figure illustrates.

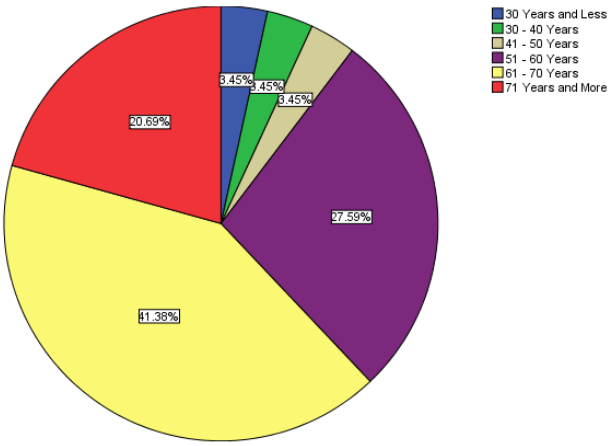


Figure (2): Patients Age Distribution

Table (3): Aortic Root

	Frequency	Percent	Valid Percent	Cumulative Percent
10 - 19 mm	1	1.7	1.7	1.7
20 - 29 mm	31	53.4	53.4	55.2
30 - 39 mm	21	36.2	36.2	91.4
40 mm and More	5	8.6	8.6	100.0
Total	58	100.0	100.0	

Table (3) above indicates that the majority of the patients in this study have an aortic root with a diameter in the range 20 – 29 mm with a percentage 53.4%, while those who have an aortic root with a diameter in the range 30 – 39 mm are 36.2%, and the other with an aortic root with a diameter in the range 10 – 19 mm, and 40 mm and more were 1.7% and 8.6% respectively, as depicted in the below figure.

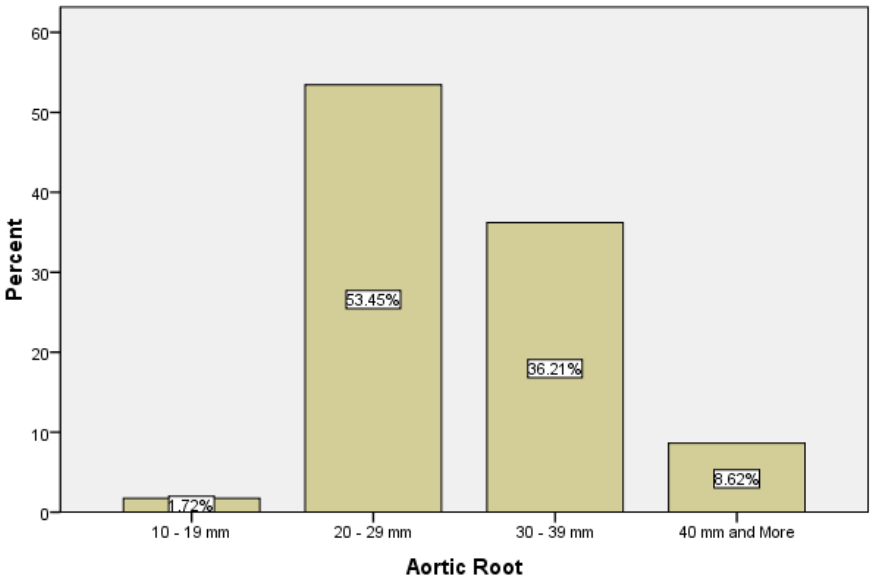


Figure (3): Aortic Root Thickness

Table (4): Left Atrium

	Frequency	Percent	Valid Percent	Cumulative Percent
20 - 29 mm	4	6.9	6.9	6.9
30 - 39 mm	30	51.7	51.7	58.6
40 - 49 mm	22	37.9	37.9	96.6
50 mm and More	2	3.4	3.4	100.0
Total	58	100.0	100.0	

The above table illustrates that the majority of the patients in this study are with left atrium thickness in the ranges between 30 – 39 mm and 40 – 49 mm with a percentage of 51.7% and 37.9%, respectively. The remaining patients are with 20 – 29 mm and 50 mm and more, left atrium thickness, with a percentage of 6.9% and 3.4%, respectively, as the below figure reveals.

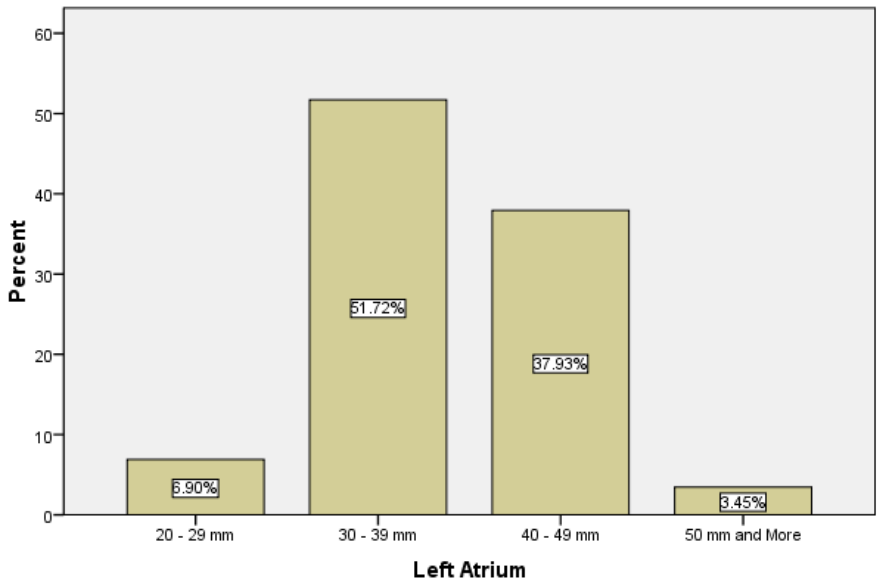


Figure (4): Left atrium Thickness

Table (5): L.V.D.D.

	Frequency	Percent	Valid Percent	Cumulative Percent
30 - 39 mm	4	6.9	6.9	6.9
40 - 49 mm	23	39.7	39.7	46.6
50 - 59 mm	23	39.7	39.7	86.2
60 - 69 mm	7	12.1	12.1	98.3
70 mm and More	1	1.7	1.7	100.0
Total	58	100.0	100.0	

The above table shows the distribution of the L.V.D.D. of the studied patients, it indicates that the majority of them are with an L.V.D.D. in the ranges 40 – 49 mm and 50 – 59 mm, each of them has a percentage of 39.7%, respectively. The below figure illustrates the distribution.

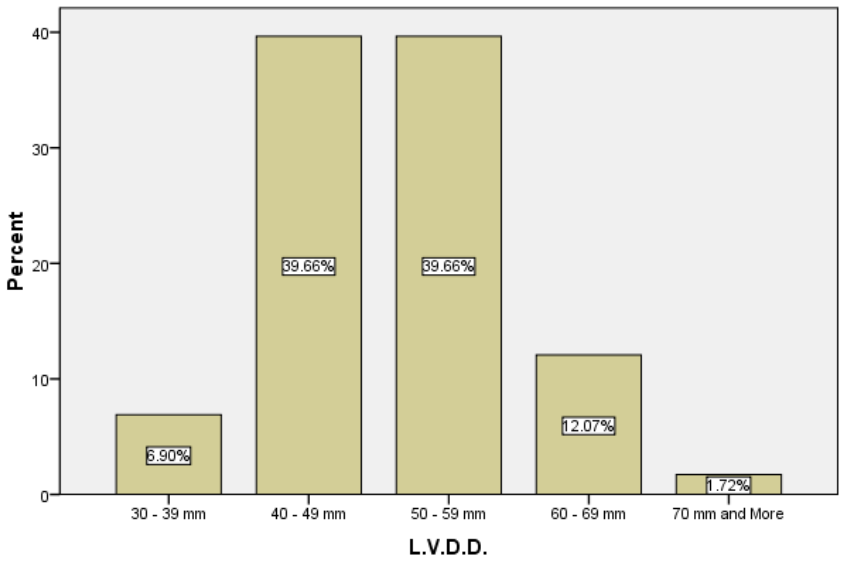


Figure (5): L.V.D.D. Thickness

Table (6): L.V.S.D.

	Frequency	Percent	Valid Percent	Cumulative Percent
20 - 29 mm	17	29.3	29.3	29.3
30 - 39 mm	20	34.5	34.5	63.8
40 - 49 mm	15	25.9	25.9	89.7
50 mm and More	6	10.3	10.3	100.0
Total	58	100.0	100.0	

The table above shows the distribution of L.V.S.D. of the patients studied, and is reveals that the highest percentage (34.5%) is of those whose L.V.S.D. in the range between 30 – 39 mm, while those with a L.V.S.D. ranges between 20 – 29 mm are (29.3%), and those with a L.V.S.D. ranges between 40 – 49 mm are (25.9%), and lastly those with a 50 mm and more L.V.S.D. are (10.3%) only. The figure below shows such a distribution.

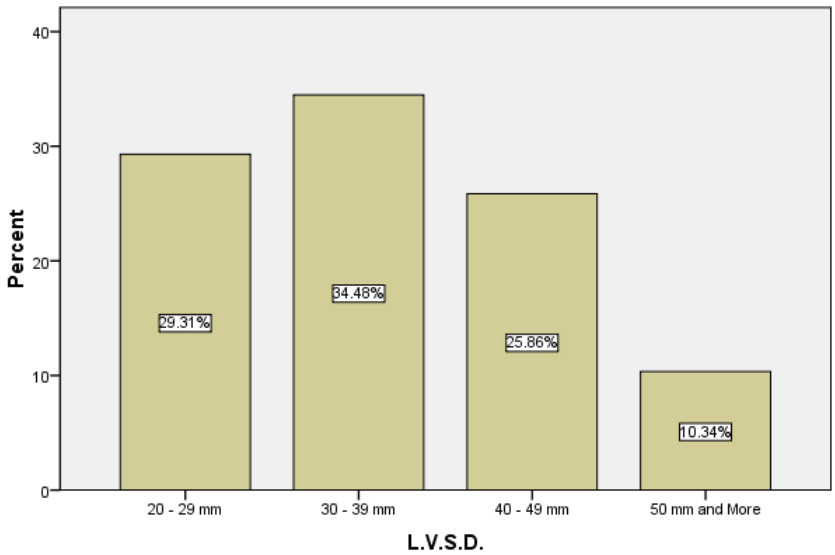


Figure (6): L.V.S.D. Thickness

Table (7): IVS

	Frequency	Percent	Valid Percent	Cumulative Percent
6 - 10 mm	12	20.7	20.7	20.7
11 - 15 mm	39	67.2	67.2	87.9
16 - 20 mm	6	10.3	10.3	98.3
26 - 30 mm	1	1.7	1.7	100.0
Total	58	100.0	100.0	

The table number (7) above represents the distribution of the IVS thickness in the studied patients, and it indicates that the majority (67.2%) of them have an IVS range between 11 – 15 mm, while those with an IVS thickness in the range between 6 – 10 mm, their percentage is (20.7%), and those IVS thickness in the range between 16 – 20 mm their percentage is (10.3%). Lastly, those is an IVS ranges between 26 – 30 mm, their percentage is only (1.7%), as revealed in the below figure.

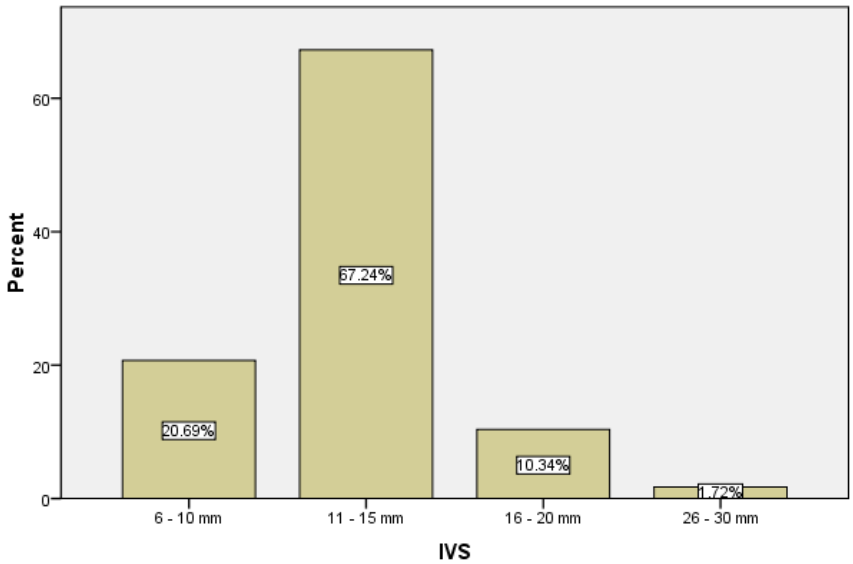


Figure (7): IVS Thickness

Table (8): L.V.P.W

Thickness	Frequency	Percent	Valid Percent	Cumulative Percent
6 - 10 mm	24	41.4	41.4	41.4
11 - 15 mm	32	55.2	55.2	96.6
16 - 20 mm	2	3.4	3.4	100.0
Total	58	100.0	100.0	

The data on the above table represents the distribution of thickness of L.V.P.W. in the studied patients. The majority of them have an L.V.P.W. in the ranges between 11 – 15 mm, and 6 – 10 mm with percentages (55.2%) and (41.4%), respectively. Only (3.4%) of them has a thickness of L.V.P.W. in the range between 16 – 20 mm. the below figure also represents this distribution.

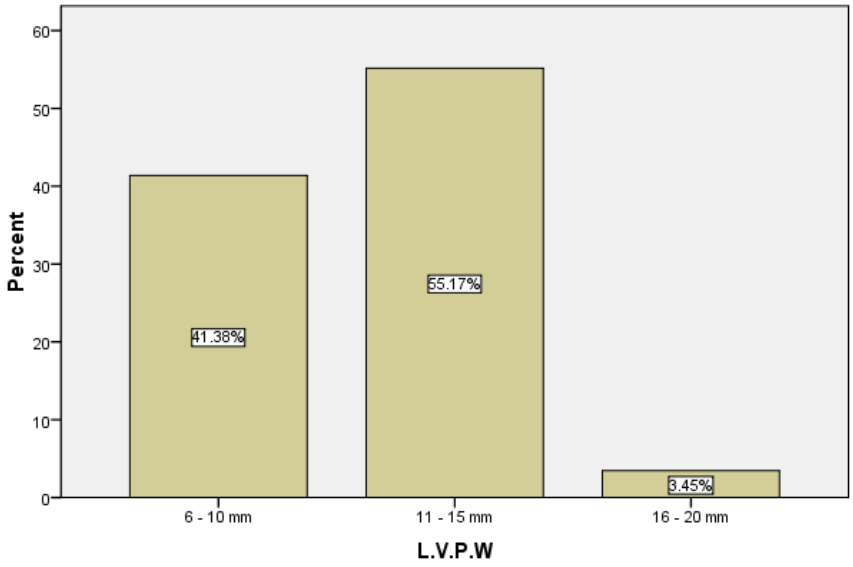


Figure (8): L.V.P.W. Thickness

Table (9): E.F.(ejection fraction)

Thickness	Frequency	Percent	Valid Percent	Cumulative Percent
20 - 29 %	2	3.4	3.4	3.4
30 - 39 %	10	17.2	17.2	20.7
40 - 49 %	14	24.1	24.1	44.8
50 - 59 %	6	10.3	10.3	55.2
60 % and More	26	44.8	44.8	100.0
Total	58	100.0	100.0	

The above table shows the distribution of effusion of blood of the hearts of the patients under study, it indicates that (44.8%) of the patients has an E.F. in the range 60% and more. (24.1%) of them are in the range between 40 – 49 % E.F., and (17.2%) are in the range between 30 – 39% E.F., and (10.3%) are in the range between 50 – 59% E.F. Only (3.4%) has an E.F. in the range between 20 – 29%, as the below figure indicates.

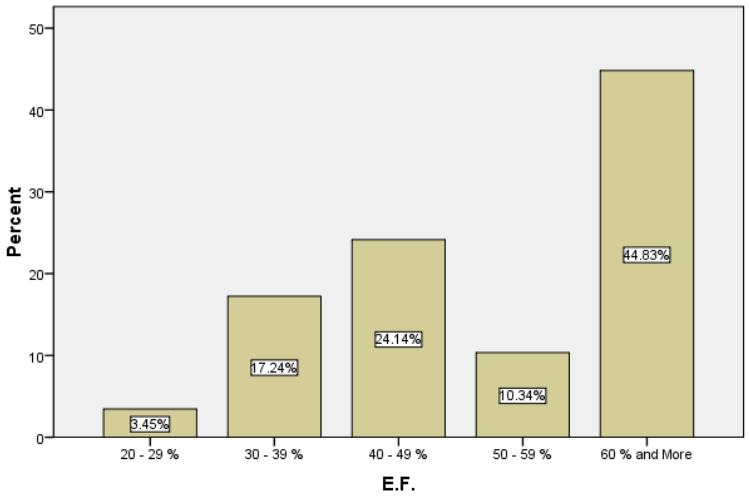


Figure (9): Distribution of E.F. percentage

Table (10): SPAP (Systolic Pulmonary Arterial pressure)

	Frequency	Percent	Valid Percent	Cumulative Percent
Normal	30	51.7	51.7	51.7
Abnormal	28	48.3	48.3	100.0
Total	58	100.0	100.0	

As presented in the above table, (51.7%) of the patients in this study has a normal SPAP, while (48.3%) has an abnormal SPAP. The figure below also reveals this fact.

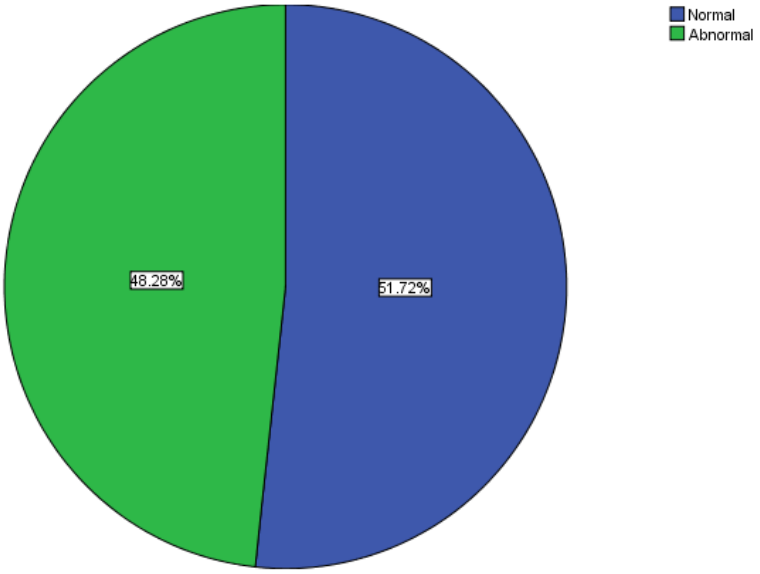


Figure (10): SPAP Normality

ANALYSIS: The Relationship between Hypertension and Heart Using Echo:

1/ SPAP and other Echocardiography Measurements:

Table (11): Calculated and Tabulated Chi-Squared Values of SPAP and other Echocardiography Measurements Cross-tabulation

Cross-Tabulation	Degree of Freedom	Calculated Chi^2	Tabulated Chi^2 at Significance Level (0.05)	Evaluation
SPAP * Aortic Root	3	7.421	7.815	Weak Relation
SPAP * Left atrium	3	4.797	7.815	Weak Relation
SPAP * L.V.D.D.	4	5.298	9.488	Weak Relation
SPAP * L.V.S.D.	3	4.599	7.815	Weak Relation
SPAP * IVS	3	4.167	7.815	Weak Relation
SPAP * L.V.P.W.	2	3.560	5.991	Weak Relation

As the above table shows, although there do exist relationships between SPAP and the diameters of Aortic Root, Left atrium, L.V.D.D., L.V.S.D., IVS, and L.V.W.P., but they are very weak according to the statistical principle which says that (*the greater the difference between the calculated Chi-Squared and the tabulated chi-squared, the stronger the relationship between the variables*). And also (*the larger the degree of freedom the less the possibility that the researcher makes mistake by*

rejecting the null hypothesis – i. e., rejecting the non-existence of relationships between the variables). Accordingly, despite the existence of the relationships between the variables, but these relationships are weak because the difference between the calculated chi-squared and the tabulated chi-squared at the level of significance (0.05) is very small. And accordingly the existence of relationship between SPAP and the Echo-Cardio-Graphy measurements can be neglected.

2/ Ejection Fraction (EF) and other Echo-Cardio-Graphy Measurements:

Table (12): Calculated and Tabulated Chi-Squared Values of EF and other Echocardiography Measurements Cross-tabulation

Cross-Tabulation	Degree of Freedom	Calculated Chi^2	Tabulated Chi^2 at Significance Level (0.05)	Evaluation
EF * Aortic Root	12	14.739	21.026	Moderate Relation
EF * Left atrium	12	24.864	21.026	Weak Relation
EF * L.V.D.D.	16	27.956	26.296	V. Weak Relation
EF * L.V.S.D.	12	61.728	21.026	V. Strong Relation
EF * IVS	12	13.082	21.026	Moderate Relation
EF * L.V.P.W.	8	9.586	15.507	Moderate Relation

As the table above indicates there is variation in the relationships between the EF and the Echo-Cardio-Graphy Measurements ranges from very strong relationship between EF * L.V.S.D. to moderate relationships between EF * Aortic Root, EF * IVS and EF * L.V.P.W, and weak and very weak relationships between EF * Left Atrium and EF * L.V.D.D., respectively, this is imputed to the variation in the difference between the calculated and tabulated chi-squared at (0.05) level of significance.

As a result, according to the above discussion the relationship which may shows significant interpretation of the effect of hypertension on the Heart using Echo-Cardio-Graphy measurements are those of EF * L.V.S.D., which shows very strong association between hypertension and heart size, and those which shows moderate association between EF and Aortic Root thickness, IVS, L.V.P.W.

Other measurements, although they show the existence of association between the SPAP and EF and the measurements made using Echo-Cardio-Graphy, but they are very weak and could not be used in interpreting the association of hypertension and the heart size.

CHAPTER FIVE

5-1 Discussion:

This study has been done in Echo Department of AlRibat university hospital, Khartoum city, from the first of May to the thirty first of August 2013for 58 hypertensive patients 46.6% male (27 of 58), 53.4%female (31 of 58) table (1)&Figure (1), was defined in six age groups .The table number (2) above indicates that the majority of the patients in this study aged between 61 – 70 years and 51 – 60 years with a percentage of 41.4% and 26.6% respectively. The age class 71 years and more was 20.7%. Other age classes are 30 years and less, 31 – 40 years, 41 – 50 years were 3.4% for each, as figure (2) illustrates.

The result of the study showed that the abnormalities are more prominent in the age groups between51to70 and that is as follows. Table (3) above indicates that the majority of the patients in this study have an aortic root with a diameter in the range of 20 – 29 mm with a percentage of 53.4%, while those who have an aortic root with a diameter in the range 30 – 39 mm are 36.2%, and the other with an aortic root with a diameter in the range 10 – 19 mm, and 40 mm and more were 1.7% and 8.6% respectively, as depicted in figure (3), put in mind that the normal range is (20-40) .Table (4) illustrates that the majority of the patients in this study are with left atrium thickness in the ranges between 30 – 39 mm and 40 – 49 mm with a percentage of 51.7% and 37.9%, respectively. The remaining patients are with 20 – 29 mm and 50 mm and more, left atrium thickness, with a percentage of 6.9% and 3.4%, respectively, as figure (4) reveals. The normal left atrium is between (19-40) mm, so 37.9% of the patients in the study are abnormal. The normal L.V.D.D is (35-56) mm,

table (5) shows the distribution of the L.V.D.D of the studied patients, it indicates that the majority of them are with a L.V.D.D in the ranges 40 – 49 mm and 50 – 59 mm, each of them has a percentage of 39.7%, respectively. Figure (5) illustrates the distribution. Table (6) shows the distribution of the L.V.S.D. of the patients studied, and is reveals that the highest percentage (34.5%) is of those whose L.V.S.D in the range between 30 – 39 mm, while those with a L.V.S.D. ranges between 20 – 29 mm are (29.3%), and those with a L.V.S.D. ranges between 40 – 49 mm are (25.9%), and lastly those with a 50 mm and more L.V.S.D. are (10.3%) only, as figure (6) above shows such a distribution. The table number (7) above represents the distribution of the IVS thickness in the studied patients, and it indicates that the majority (67.2%) of them have an IVS range between 11 – 15 mm, normal range (06-11) mm while those with an IVS thickness in the range between 6 – 10 mm, their percentage is (20.7%), and those IVS thickness in the range between 16 – 20 mm their percentage is (10.3%). Lastly, those is an IVS ranges between 26 – 30 mm, their percentage is only (1.7%), as revealed the figure No (7).The data on table (8) represents the distribution of thickness of L.V.P.W. in the studied patients. The majority of them have an L.V.P.W. in the ranges between 11 – 15 mm, normally between (06 –11 mm) and 6 – 10 mm with percentages (55.2%) and (41.4%), respectively. Only (3.4%) of them has a thickness of L.V.P.W. in the range between 16 – 20 mm. Figure (8) also represents this distribution.

The most important parameter of the heart function demonstrated in table (9) that shows the distribution of effusion of blood of the hearts of the patients under study, it indicates that (44.8%) of the patients has an ejection fraction (E.F.) in the range of 60% and more. (24.1%) of them are in the range between 40 – 49 % E.F., and (17.2%) are in the range

between 30 – 39% E.F., and (10.3%) are in the range between 50 – 59% E.F. Only (3.4%) has an E.F. in the range between 20 – 29%, as the figure indicates. Put in mind that the normal value is (55-75%).

As presented in table (10), 51.7% of the patients in this study have a normal SPAP, while (48.3%) has an abnormal SPAP. The figure also reveals this fact.

As the above discussion shows and in table (11), although there do exist relationships between SPAP and the diameters of Aortic Root, Left atrium, L.V.D.D., L.V.S.D., IVS, and L.V.W.P., but they are very weak according to the statistical principle which says that (*the greater the difference between the calculated Chi-Squared and the tabulated chi-squared, the stronger the relationship between the variables*). And also (*the larger the degree of freedom the less the possibility that the researcher makes mistake by rejecting the null hypothesis i.e., rejecting the non-existence of relationships between the variables*). Accordingly, despite the existence of the relationships between the variables, but these relationships are weak because the difference between the calculated chi-squared and the tabulated chi-squared at the level of significance (0.05) is very small. And accordingly the existence of relationship between SPAP and the Echo-Cardio-Graphy measurements can be neglected.

Statistically there is variation in the relationships between the EF and the echocardiography measurements ranges from very strong relationship between EF * L.V.S.D. to moderate relationships between EF * Aortic Root, EF * IVS and EF * L.V.P.W, and weak and very weak relationships between EF * Left Atrium and EF * L.V.D.D., respectively, this is imputed to the variation in the difference between the calculated and tabulated chi-squared at (0.05) level of significance.

As a result, according to the above discussion the relationship which may shows significant interpretation of the effect of hypertension on the Heart using Echo-Cardio-Graphy measurements are those of EF * L.V.S.D., which shows very strong association between hypertension and heart size, and those which shows moderate association between EF and Aortic Root thickness, IVS, L.V.P.W.

5-2 Recommendations:

After the enumeration of the results that related to the following thesis, there are some ideas which could help further in the field of research and better to be recommended as follow:

- Echocardiography should be used as a routine checkup and follow up to help treatment and control of hypertension.

- Echocardiography is very important to hypertensive patients to detect the complications as heart failure as well as myocardial infarction (MI) early, and could be avoided.
- More heart Echo studies will evaluate the correlations among Doppler sonographic findings in hypertensive patients and patient weight, laboratory investigations and ethnic group should be considered with application of Doppler to study the valvular lesions (turbulent jet).
- Echocardiography is very important to hypertensive patients want to do hajj, they should check before doing so because they will face more efforts there in **Makkkah**, **Madinah Munawara** and **Mena** as well as **Muzdalefa.** Therefore, fit healthy cardiac muscles are one of the list for **Muslims** to be able to do **hajj.**

5-3 Conclusion:

This study has been done in Echo Department of AlRibat university hospital, Khartoum city, from the first of May to the thirty first of August 2013 for 58 hypertensive patients 46.6% male, 53.4% female, the age from 18 years and more with exclusion of pregnant women. The goal of the study is to evaluate the heart size in patients with HTN by ECHO finding regarding to the left ventricle, E.F, the I.V.S, and the valves.

The results conclude that there is increase in heart size comparing with normal range that made it susceptible to heart failure, valvular lesions and myocardial infarction as well as other pathophysiological disturbances.

Echocardiography scanning is very important to detect any cardiac changes in hypertensive patients to avoid the complications.

5-4-REFERENCES

1- Chummy S. Sinnatamby, 2004, Last's Anatomy Regional & Applied, 10th edition, Churchill Livingstone, China, 191-200.
2- JHON JIRGIS JHODAH, 2012, JHON'S NOTES IN APPLIED ANATOMY, Khartoum, CVS.
3- Roderick NM Mac Sween& Keith Wholly, 1998, Mur's texts book of pathology, 13th edition, Arnold international edition, New York, 483-523.
4- SAM KADDOURA, 2002, Echo Made Easy, first edition, Churchill Livingstone, China, 165-168.
5- Thomas Boehmeke & Ralf Doliva, 2006, Atlas of Echocardiography, Thieme Stuttgart, New York, 4-8.
6- Chobanian AV, Bakris GL, Black HR et al. (December 2003). "Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure". *Hypertension* 42 (6): 1206–52. doi:10.1161/01.HYP.0000107251.49515.c2. PMID 14656957.

7- Carretero OA, Oparil S (January 2000)."Essential hypertension. Part I: definition and etiology". *Circulation* 101 (3): 329–35. doi:10.1161/01.CIR.101.3.329. PMID 10645931.

8- Lloyd-Jones D, Adams RJ, Brown TM et al. (February 2010). "Heart disease and stroke statistics--2010 update: a report from the American Heart Association". *Circulation* 121 (7): e46–e215.doi:10.1161/CIRCULATIONAHA.109.192667. PMID 20019324.

9- Alcocer L, Cueto L (June 2008). "Hypertension, a health economics perspective". *Therapeutic Advances in Cardiovascular Disease* 2 (3): 147–55. doi:10.1177/1753944708090572. PMID 19124418. Retrieved 2009-06-20.

10- Chobanian AV, Bakris GL, Black HR et al. (December 2003). "Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure". *Hypertension* 42 (6): 1206–52. doi:10.1161/01.HYP.0000107251.49515.c2. PMID 14656957.

11- Carretero OA, Oparil S (January 2000). "Essential hypertension. Part I: definition and etiology". *Circulation* 101 (3): 329–35. doi:10.1161/01.CIR.101.3.329. PMID 10645931.

12- Fisher ND, Williams GH (2005). "Hypertensive vascular disease". In Kasper DL, Braunwald E, Fauci AS, *et al.*.*Harrison's Principles of Internal Medicine* (16th ed.). New York, NY: McGraw-Hill. pp. 1463–81. ISBN 0-07-139140-1.

13- Marshall, IJ; Wolfe, CD; McKevitt, C (2012 Jul 9). "Lay perspectives on hypertension and drug adherence: systematic review of qualitative research.".*BMJ (Clinical research ed.)* 345: e3953. PMC 3392078. PMID 22777025.

14-O'Brien, Eoin; Beevers, D. G.; Lip, Gregory Y. H. (2007).*ABC of hypertension.* London: BMJ Books. ISBN 1-4051-3061-X.Zubcevic J, Waki H, Raizada MK, Paton JF (June 2011). "Autonomic-immune-vascular interaction: an emerging concept for neurogenic hypertension". *Hypertension* 57 (6): 1026–33. doi:10.1161/HYPERTENSIONAHA.111.169748. PMC 3105900. PMID 21536990.

15-Papadopoulos DP, Mourouzis I, Thomopoulos C, Makris T, Papademetriou V (December 2010). "Hypertension crisis".*Blood Press.* 19 (6): 328–36. doi:10.3109/08037051.2010.488052. PMID 20504242.

16-Marik PE, Varon J (June 2007). "Hypertensive crises: challenges and management". *Chest* 131 (6): 1949–62. doi:10.1378/chest.06-2490. PMID 17565029.

17-Rodriguez-Cruz, Edwin; Ettinger, Leigh M (April 6, 2010). "Hypertension". *eMedicine Pediatrics: Cardiac Disease and Critical Care Medicine*. Medscape. Retrieved 2009-06-16.

18 -Ehret GB, Munroe PB, Rice KM et al. (October 2011). "Genetic variants in novel pathways influence blood pressure and cardiovascular disease risk". *Nature* 478 (7367): 103–9. doi:10.1038/nature10405. PMC 3340926.PMID 21909115.

19-Lifton, RP; Gharavi, AG, Geller, DS (2001-02-23). "Molecular mechanisms of human hypertension". *Cell* 104 (4): 545–56. doi:10.1016/S0092-8674(01)00241-0. PMID 11239411.

20-He, FJ; MacGregor, GA (2009 Jun). "A comprehensive review on salt and health and current experience of worldwide salt reduction programmes". *Journal of Human Hypertension* 23 (6): 363–84. doi:10.1038/jhh.2008.144. PMID 19110538.

21-Dickinson HO, Mason JM, Nicolson DJ et al. (February 2006). "Lifestyle interventions to reduce raised blood pressure: a systematic review of randomized controlled trials". *J. Hypertens.* 24 (2): 215–33. doi:10.1097/01.hjh.0000199800.72563.26. PMID 16508562.

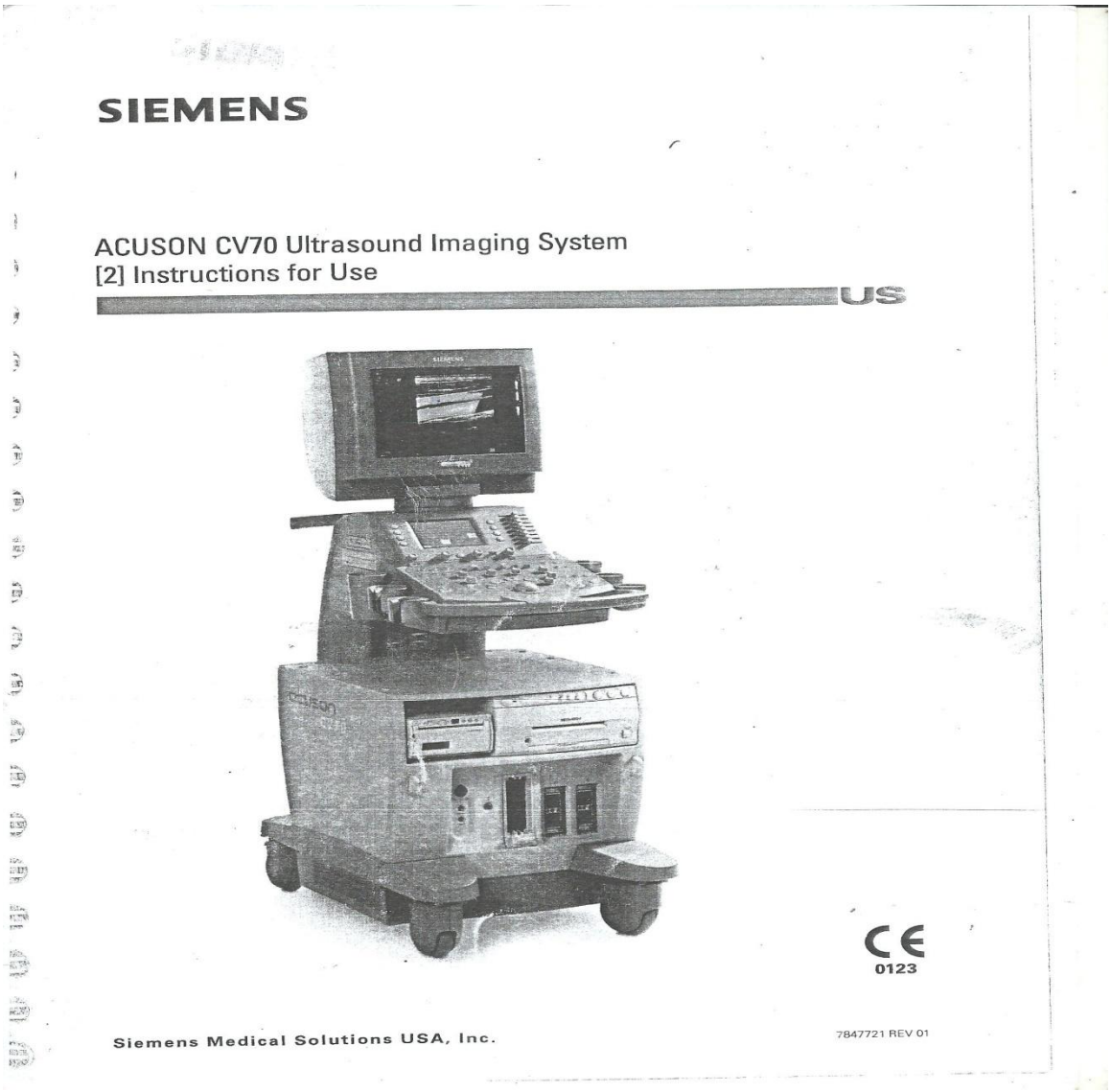
22-Haslam DW, James WP (2005). "Obesity". *Lancet* 366 (9492): 1197–209. doi:10.1016/S0140-6736(05)67483-1. PMID 16198769.

23-. *Canadian Journal of Cardiology* 24 (6): 441–4. doi:10.1016/S0828-282X(08)70617-2. PMC 2643187. PMID 18548140."Hypertension, a health economics perspective"10.1177/1753944708090572PMID191244182009-06-20.

24-William J. Elliott (October 2003). "The Economic Impact of Hypertension". *The Journal of Clinical Hypertension* 5 (4): 3–13. doi:10.1111/j.1524-6175.2003.02463.x. PMID 12826765.

APPENDIX 1

1- **The machine used in the Research:** An echocardiographic machine (SIEMENS-ACUSON CV70 Ultrasound imaging system), with Doppler & M-mode capabilities**.**


SIEMENS

ACUSON CV70 Ultrasound Imaging System
[2] Instructions for Use

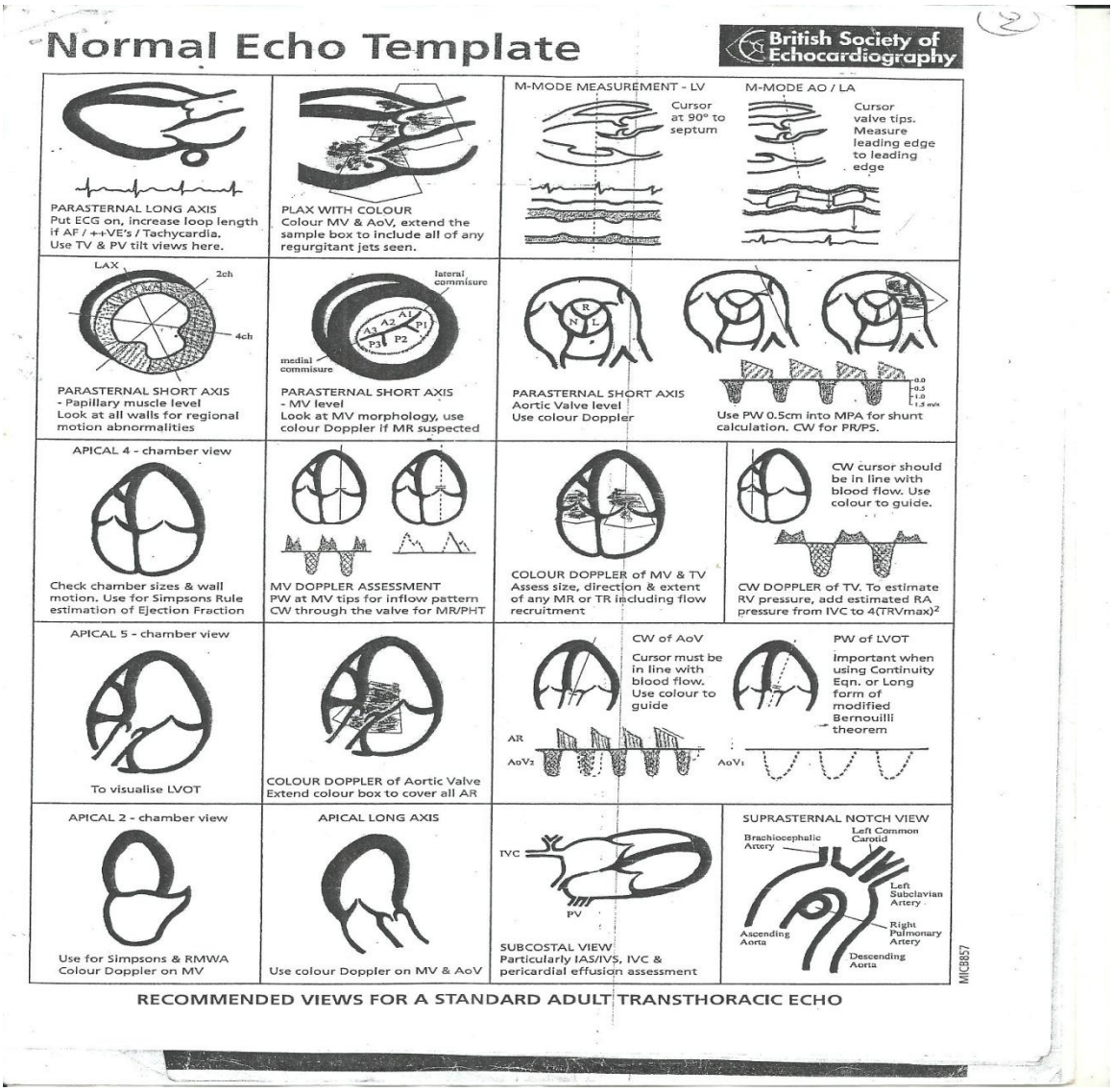
US

CE
0123

Siemens Medical Solutions USA, Inc.

7847721 REV 01

2- Normal Echo Template



3- Data collection sheet :

NATIONAL RIBAT UNIVERSITY

RIBAT UNIVERSITY HOSPITAL

ECHOCARDIOGRAPHY REPORT

Name:		Hospital NO. :	Age:	Sex:
Weight:	Height:	Word/Clinic :	P :police	C:
Consultant :		Department :MED	Date :28/1/2014	

Measurement:-

Parameter	Normal	Record
• Aortic root :	(20 -40) mm	
• Left atrium :	(19 -40) mm	
• L .V .D .D :	(35 -56) mm	
• L .V .S .D :	(22 _40) mm	
• IVS :	(06 -11) mm	
• L .V .P .W :	(06 -11) mm	
• F .S :	(30 -40 %)	
• E .F :	(55 -75 %)	
• R .V .D :	(7 -23) mm	

Report:-

- Normal **LV** internal dimensions, with preserved **LV** systolic function.
- No regional wall motion abnormalities at rest.
- Mild **LVH.**
- Mild **MR.**
- Normal **RV** systolic function.
- Normal **SPAP.**
- **No pericardial effusion.**

Technologist sig: Nemat Omer **Dr sig: Hyder Satti**

4- Important Abbreviations:

EF	**Ejection fraction**
IVS	**Interventricular septum**
LVPW	**Left ventricular posterior wall**
LVESD	**Left ventricular end systolic diameter**
LVEDD	**Left ventricular end diastolic diameter**
SPSS	**Statistical package for social studies**

APPENDIX 2

Echo images of some patients:

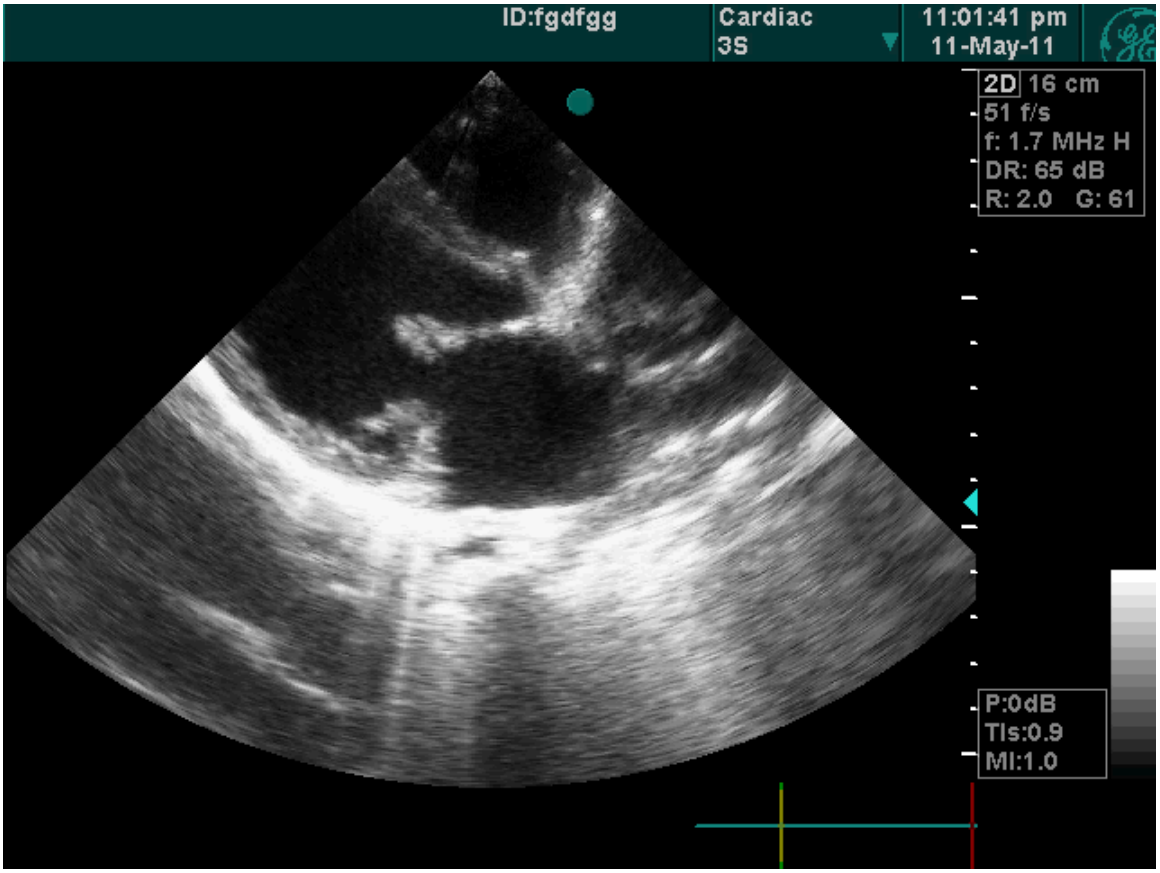


IMAGE NO (1) Long axis left parasternal view showing mitral regurgitation with vegetations

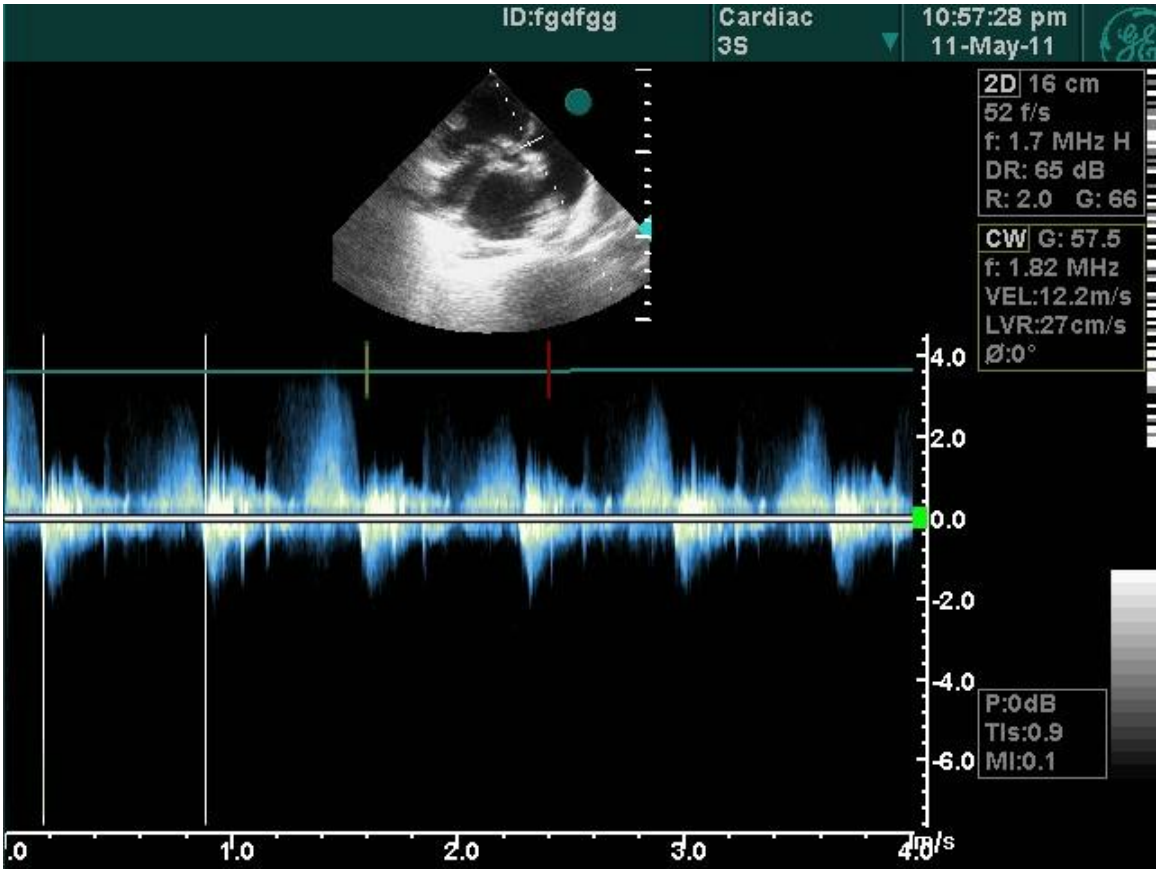


IMAGE NO (2) CW Doppler at the aorta

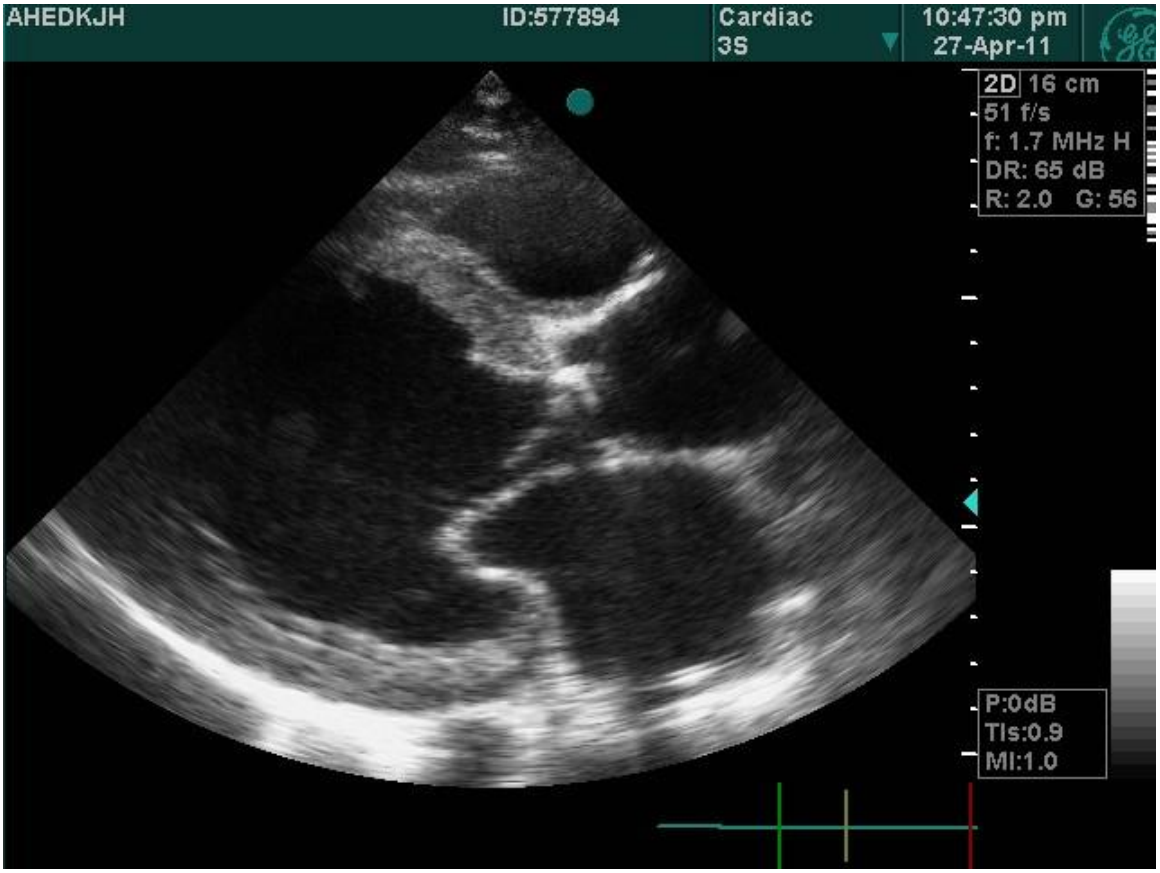


IMAGE NO (3) left parasternal long axis view showing LVH and aortic stenosis

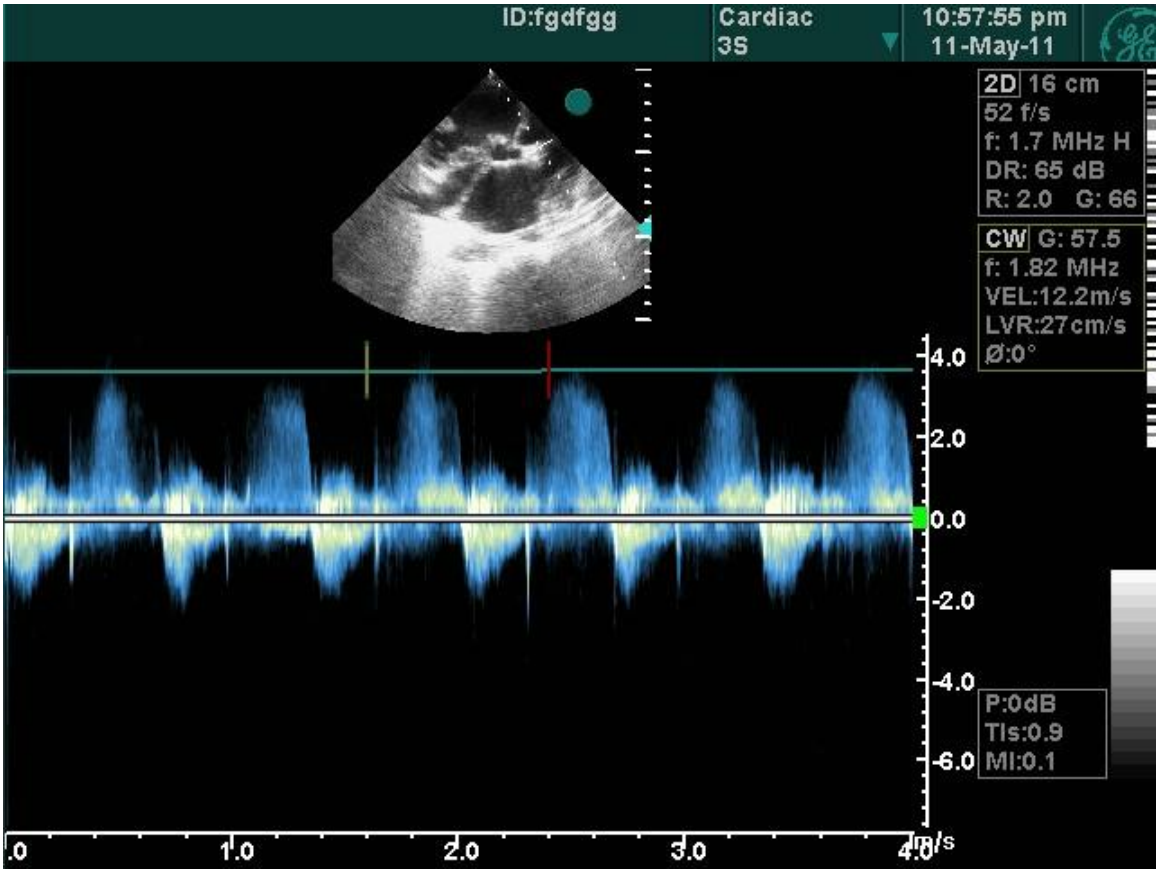


IMAGE NO (4) CW Doppler in a patient with HTN

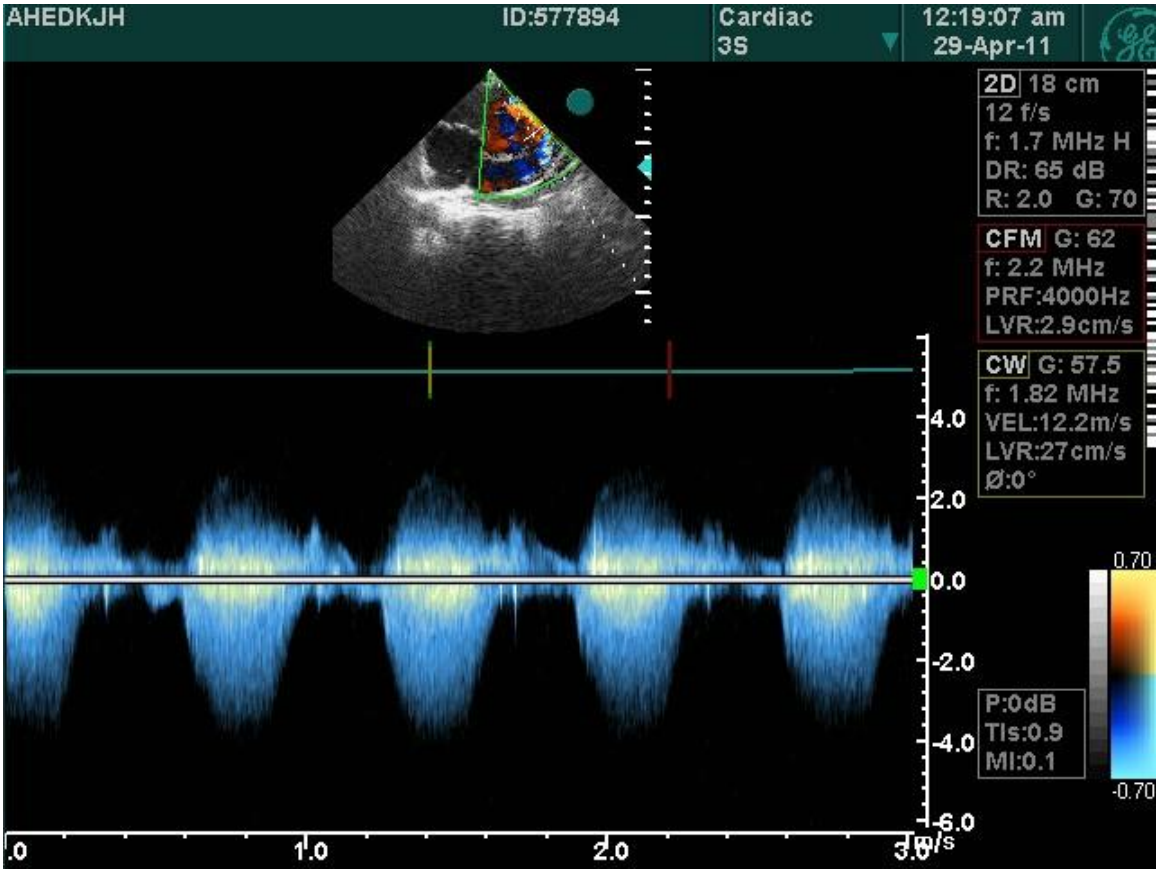


IMAGE NO (5) CW color Doppler in a patient with HTN

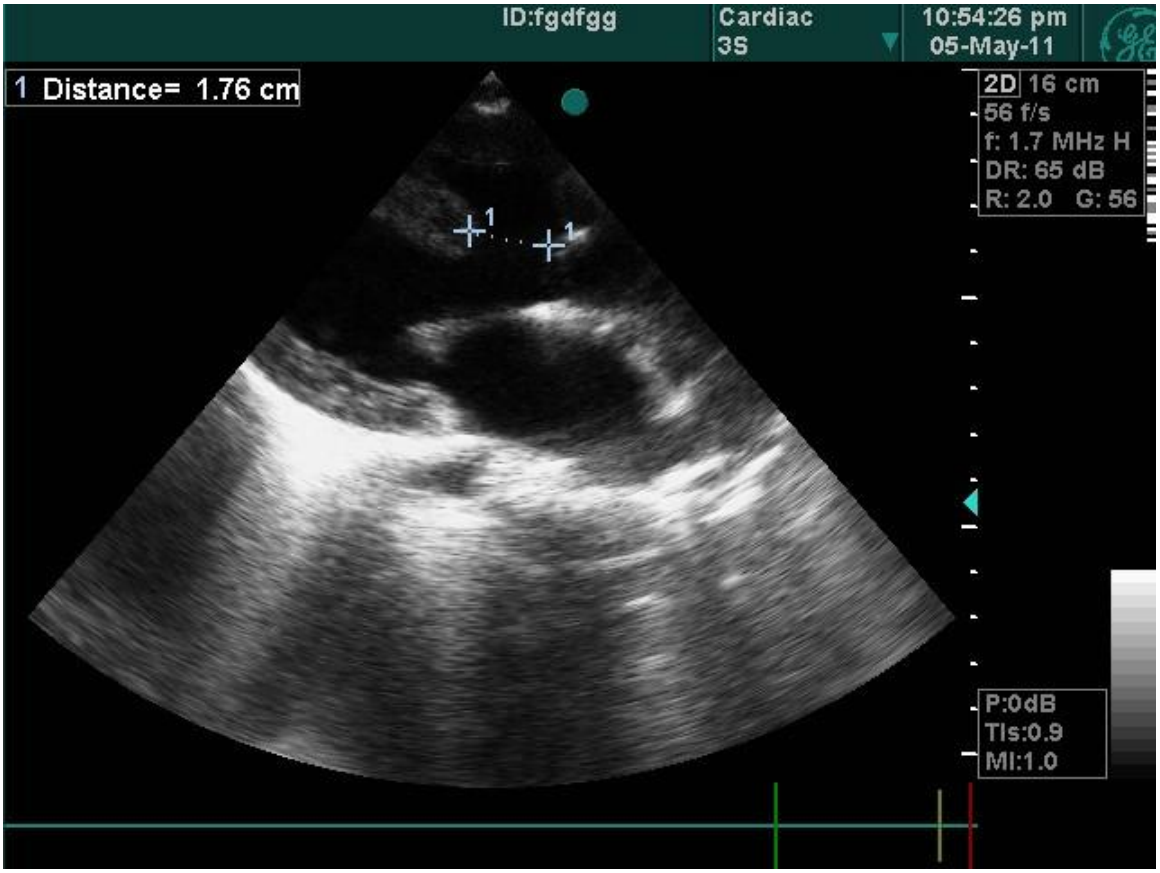


IMAGE NO (6) VSD, in along parasternal view measuring 1.76 cm

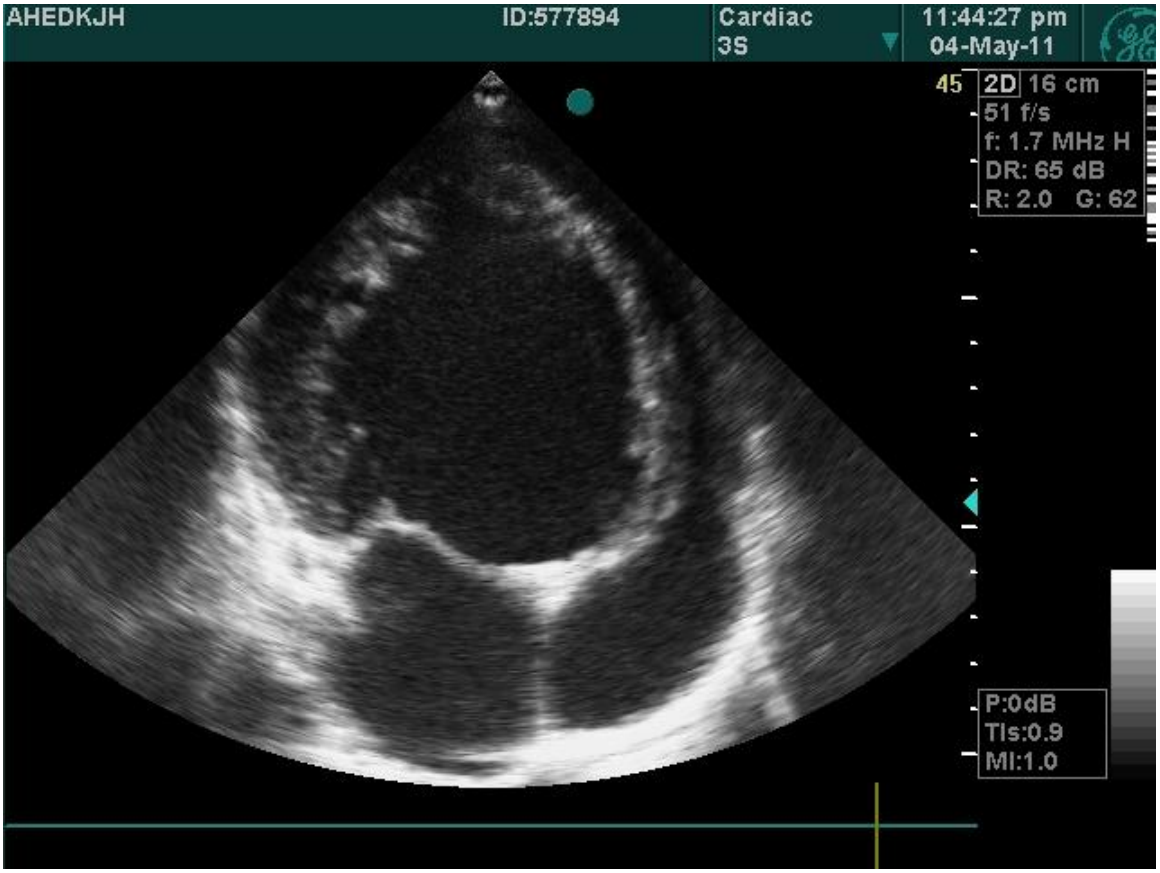


IMAGE NO (7) APICAL 4 camper view in a hypertensive patient showing LVH and increased IVS

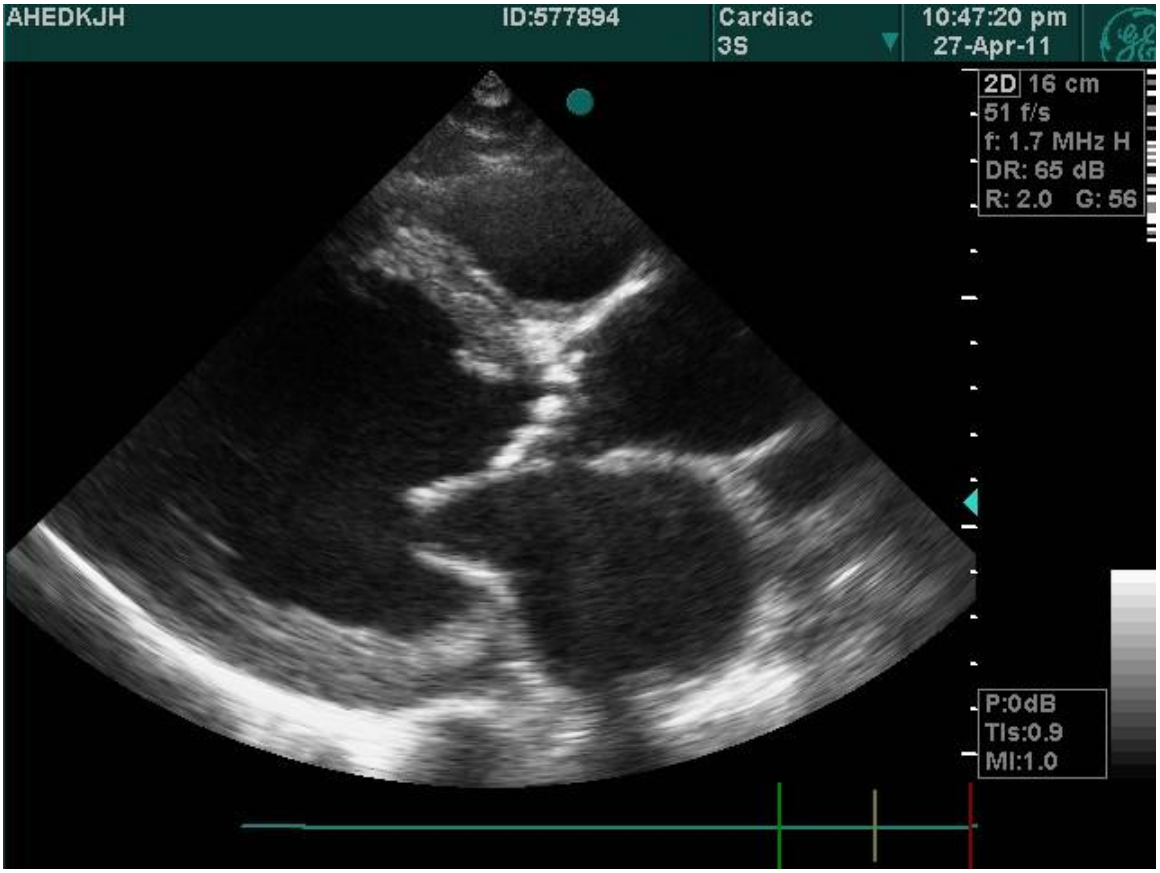


IMAGE NO (8) a parasternal long axis view in apatient with increased LVPW and left ventricular hypertrophy

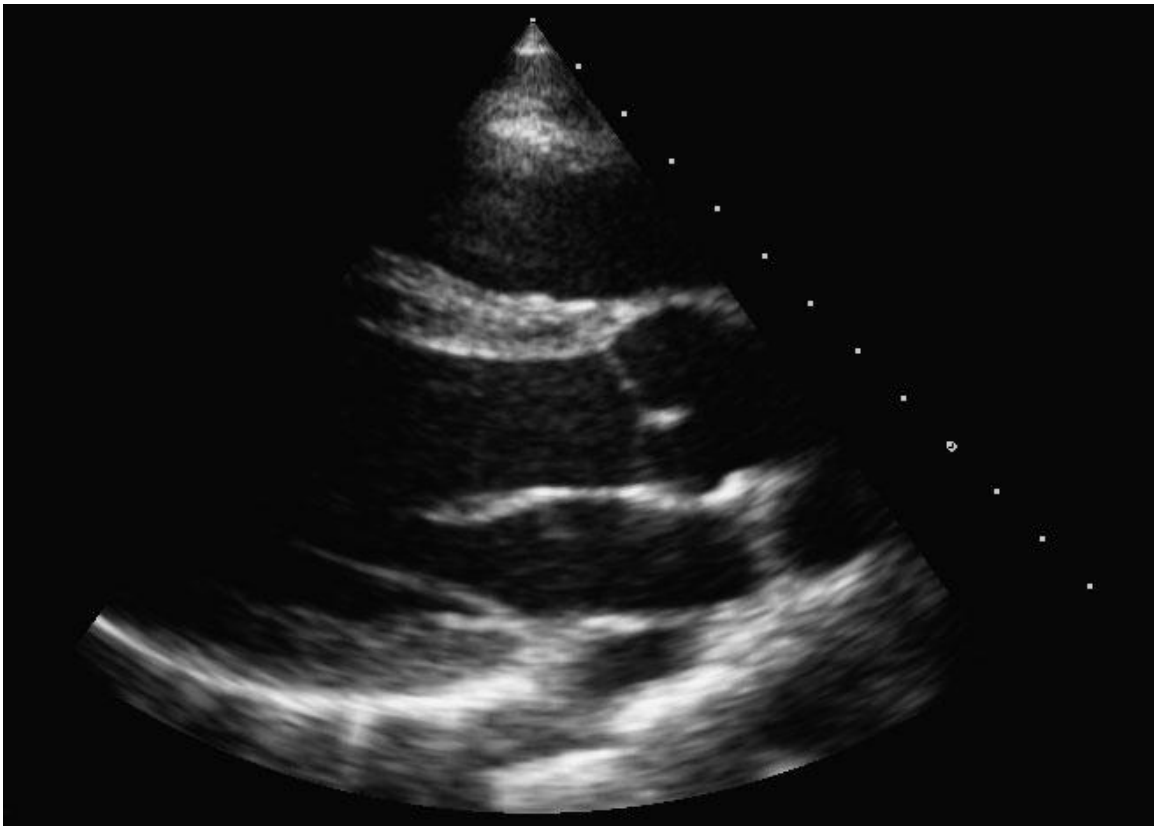


IMAGE NO (9) Parasternal long axis view showing the left ventricle, the left atrium, the right ventricle, and the aortic valve

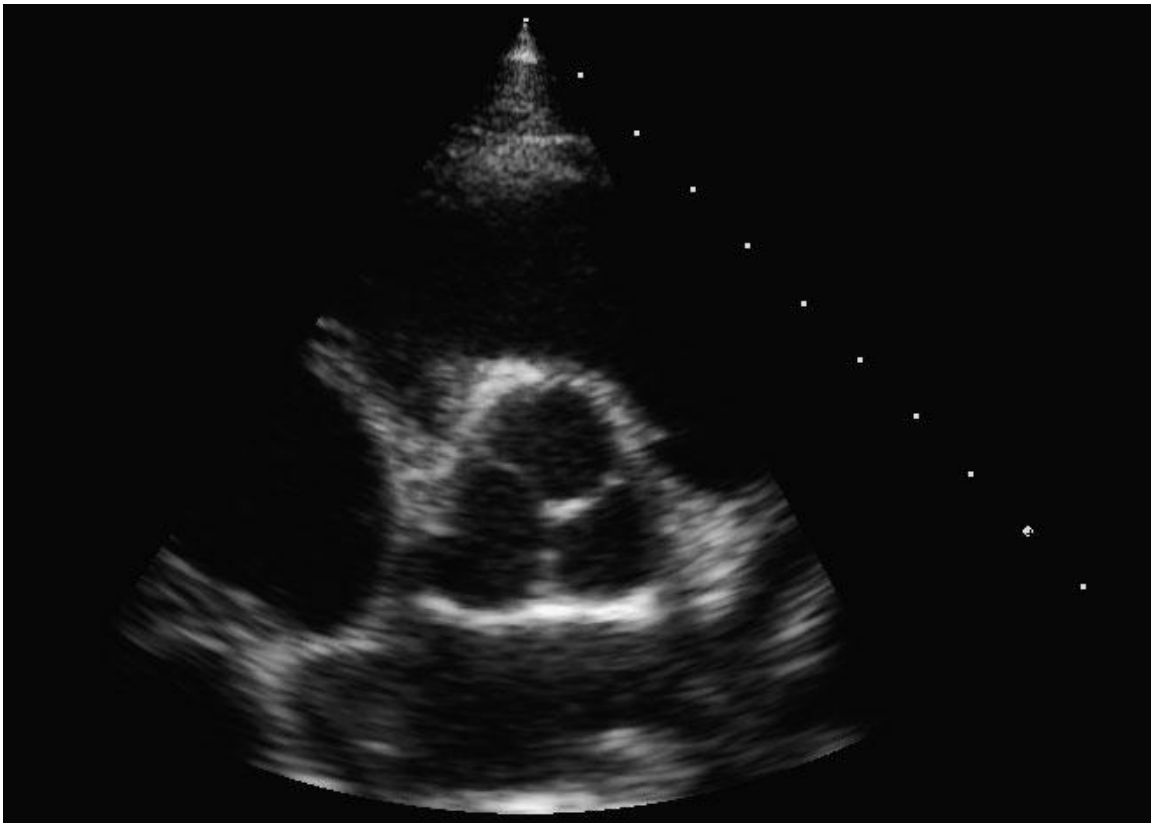


IMAGE NO (10) short axis- Left parasternal view at the level of the aorta showing the Mercedes Benz sign

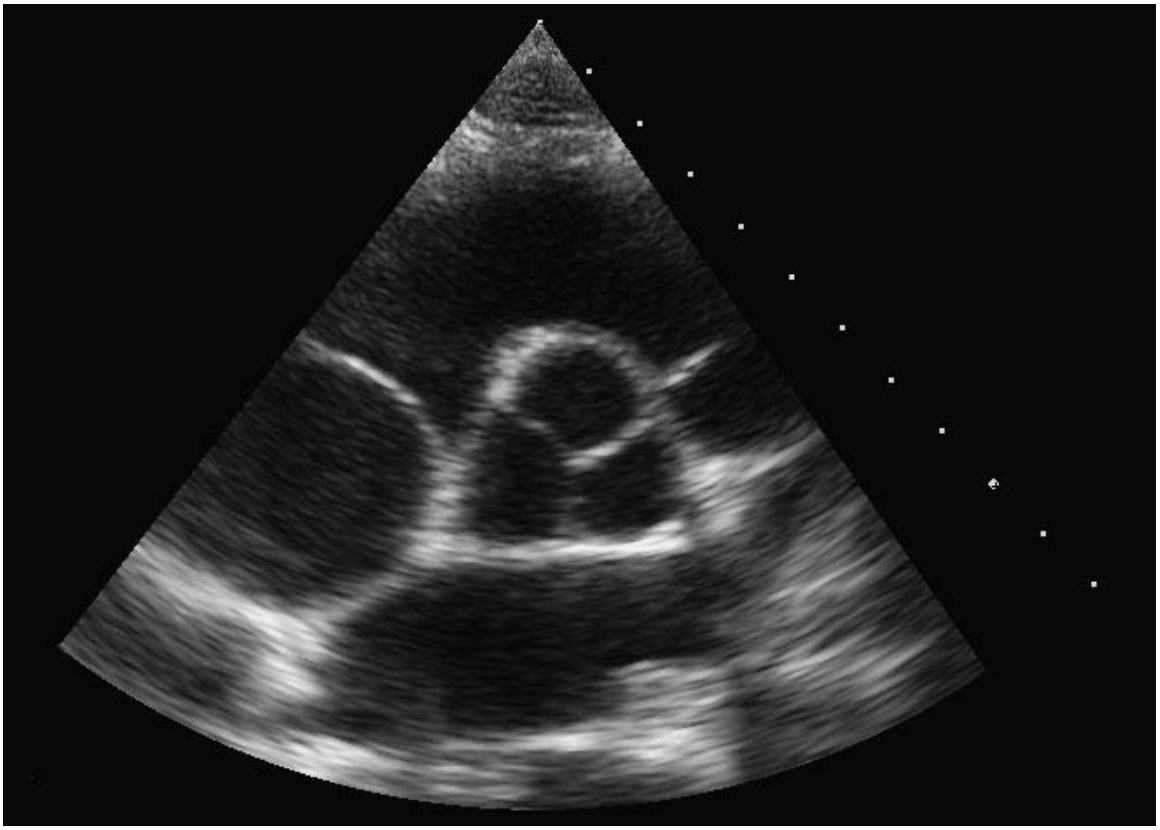


IMAGE NO (11) another short axis- Left parasternal view at the level of the aorta showing the Mercedes Benz sign

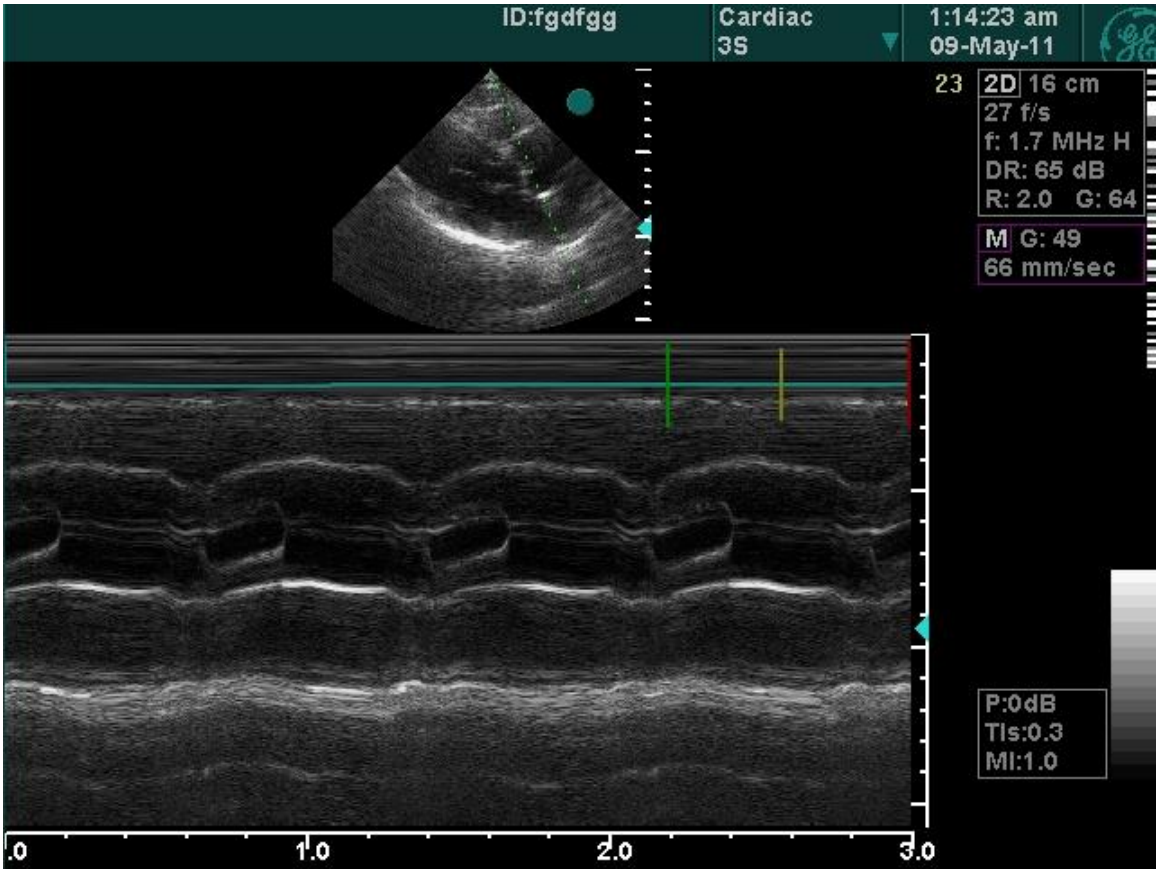


IMAGE NO (12) a hypertensive patient to measure the LVPW

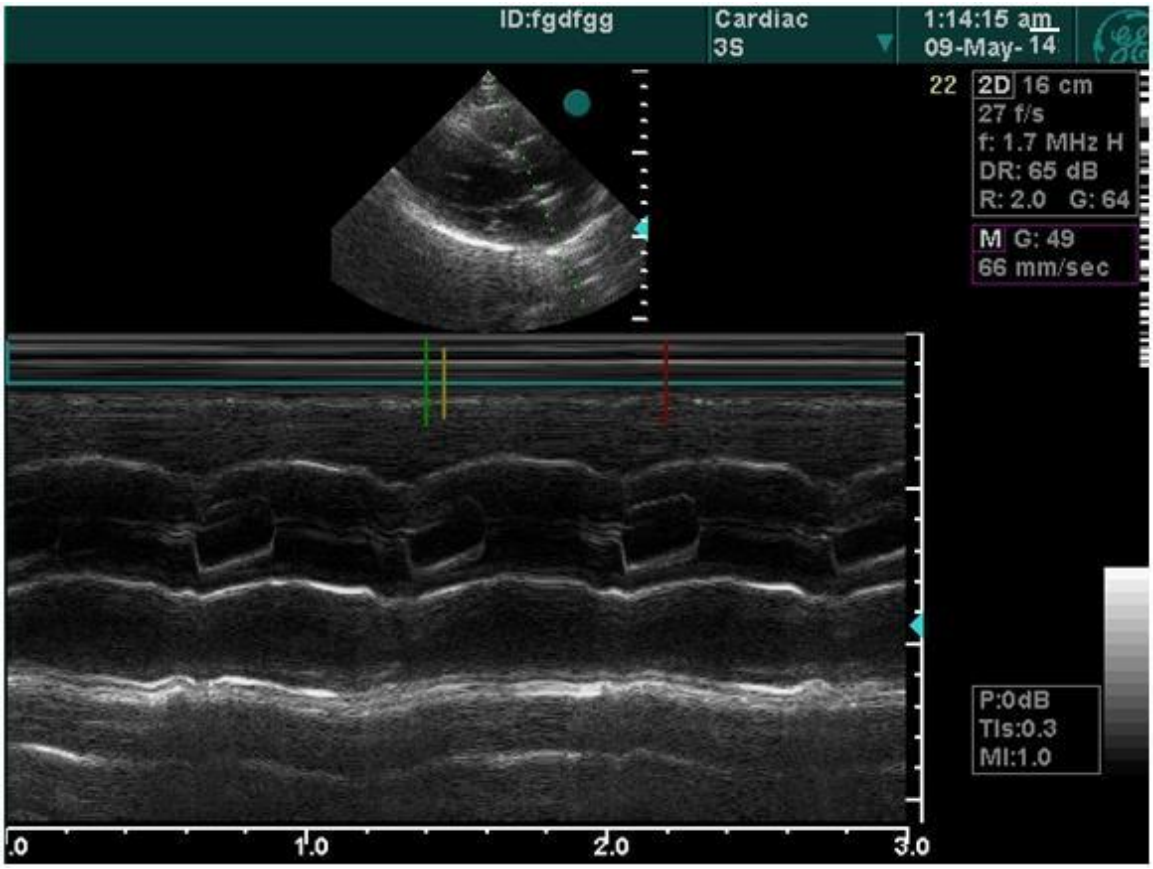


IMAGE NO (13) M- mode measurements of a patient having LVH

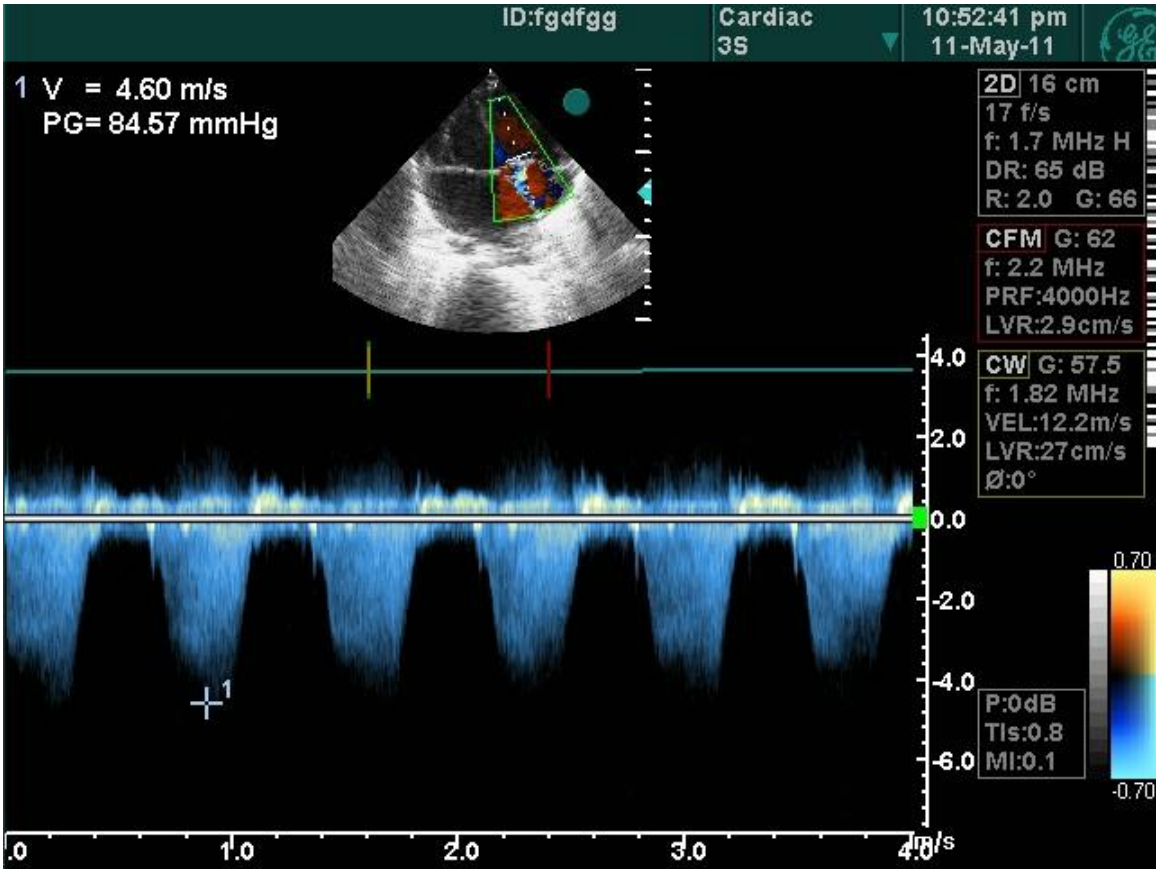


IMAGE NO (14) a CW Doppler of a patient with HTN

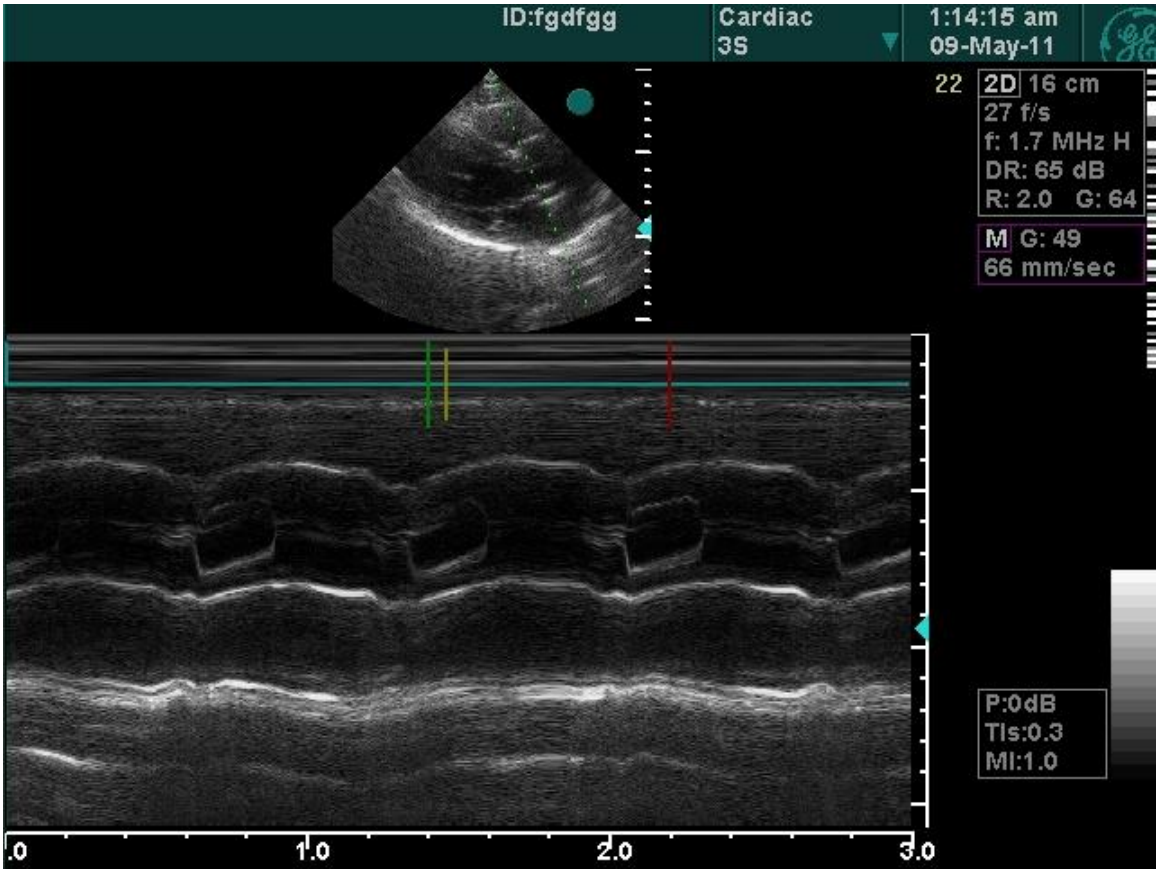


IMAGE NO (15) M–mode in the left parasternal view long axis to measure the ventricular walls

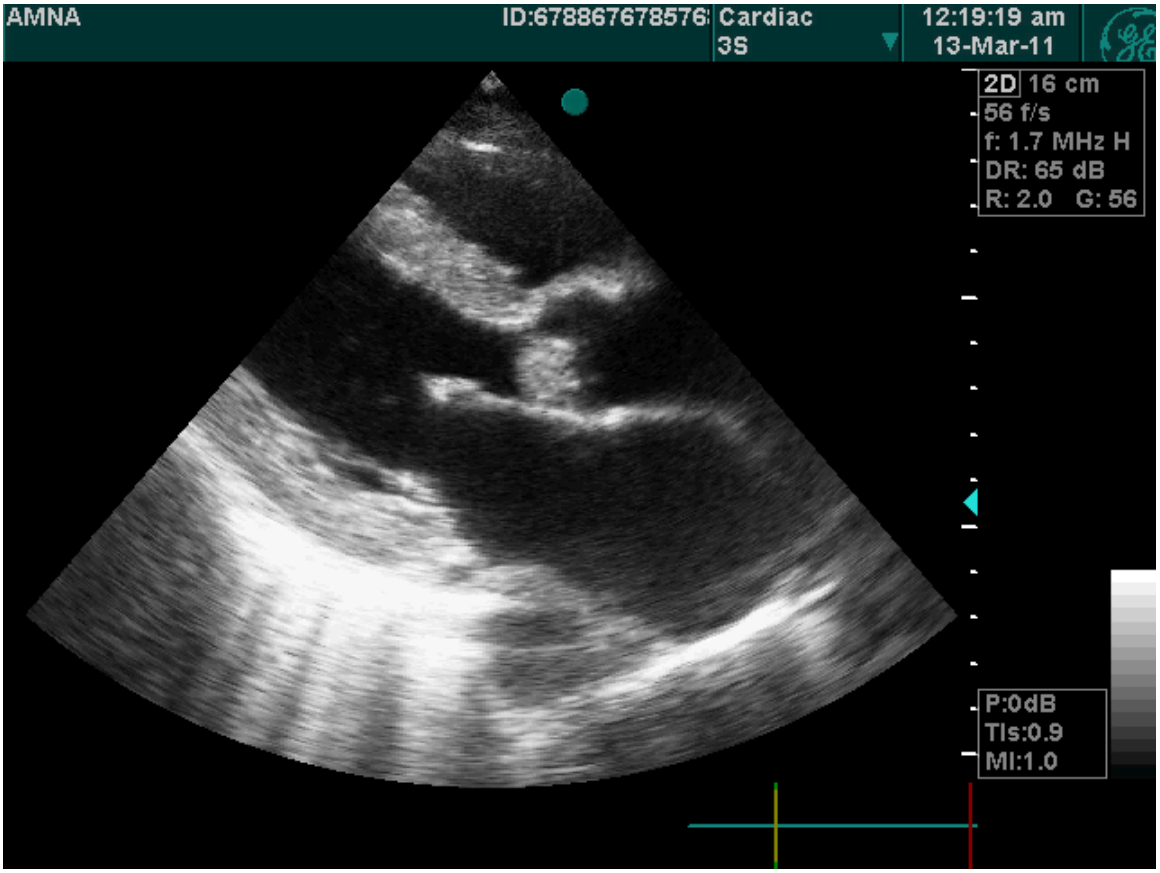


IMAGE NO (16), Left parasternal long axis view showing an aortic mass

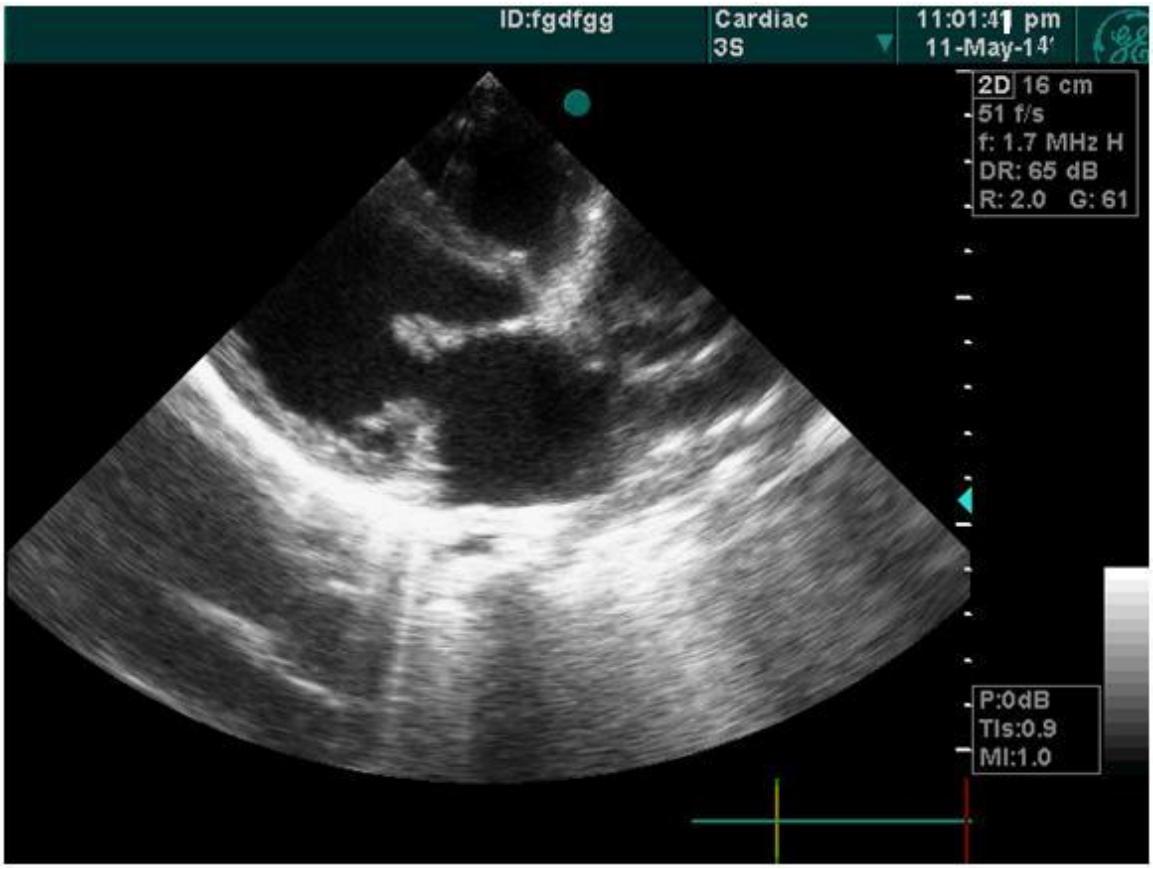


IMAGE NO (17) Long axis left parasternal view showing mitral stenosis

APPENDIX 3

(1)The questionnaire:

NAME: ……AGE…SEX…RESIDENCE: ……

FAMILY HISTORY OF HTN & DM: ……

PERIOD OF THE DISEASE: …….

WEIGHT OF THE PATIENT: (……KG), SMMOKING: ……..

LAST THREE READINGS: 1-BP ------, 2- BP ------, 3- BP------

THE RESULTS OF LAST INVESTIGATIONS DONE:

a- URINE……
b- BLOOD UREA & CREATININE……
c- LIVER FUNCTION TEST……
d- LIPID PROFILE……
e- CARDIAC ENZYMES ……
f- SODIUM & POTASIUM …….

COMPLICATIONS OTHER THAN CARDIAC:

1-……..……2-……..…3-……..…..…4-……

THE ECHO RESULTS:

a- THE WALLS.
b- THE VAVES.
c- THE PERICARDIUM.
d- SYSTOLIC FUNCTION.
e- DIASTOLIC FUNTION.
f- VENTRICULAR DIAMETER.
g- EJECTION FRACTION.
h- MASSES.
i- FLUID COLLECTIONS.

(2) Patient agreement:

موافقة المبحوث

انا .....

بعد أن تم شرح لى ما سوف يقوم به الباحث أوافق أن أكون من ضمن الأفراد المشاركين فى هذا البحث خدمة للعلم والوطن من بعد الله سبحانه وتعالى موافقة تامة وأعطى الباحث الوقت الكافى لعمل الكشف الطبي بالموجات فوق الصوتية بعد ان علمت من المختصين في هذا الشأن بأنه وسيلة آمنة لتشخيص الأمراض الخاصة بأمراض القلب .

الأسم/ .....

التوقيع على ذلك .....

البصمة فى حالة الأمي .....

التاريخ / /