Sudan University of Science and Technology

College of Graduated Studies

Evaluation of Left Ventricular Diastolic Dysfunction in Type 2 Diabetes Mellitus Using Echocardiography

تقييم اختلال وظيفة انبساط البطين الأيسر للمصابين بالنوع الثاني من مرض السكر باستخدام التصوير بالموجات فوق الصوتية للقلب

A thesis Submitted for Partial Fulfillment for the Requirement of the M.SC Degree in Medical Diagnostic Ultrasound

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بسم الله الرحمن الرحيم

الآية

{ يَرْفَعِ اللَّهُ الَّذِينَ آمَنُوا مِنكُمْ
وَالَّذِينَ أُوتُوا الْعِلْمَ دَرَجَاتٍ}

سورة المجادلة

الآية 11

صدق الله العظيم

Dedication

To My Lovely Parents & my husband Hisham…

To Soul of My Dear Friend (Asma)…

To All of My Family…

Closed Friends and Colleagues…

Sahar..

Acknowledgement

First full of thanks to Allah. And a lot of thanks and great fullness to my supervisor Dr. Ahmed Mostafa Abukonna for his valuable and continuous help and guidance.

I owe my most sincere gratitude to Dr. Adil Abdallah and the working team who gave me the opportunity to work with them in the department of ultrasound in Omdurman military hospital during the process of collection of data.

Abstract

Diabetes mellitus is one of the most common diseases in the world. Its prevalence is growing in both developed and developing countries.

Left ventricular diastolic dysfunction represents the first stage of diabetic cardiomyopathy preceding changes in systolic function, without any clinical manifestations.

The main objective of this study was to assess the diastolic dysfunction in diabetic patients. The study was conducted in Omdurman military hospital. 50 patients (26 males and 24 females) with known history of diabetes mellitus were enrolled in the study. All patients were scanned with ultrasound machine using linear high frequency transducer (7.5-10 MHz).

The results of this study showed that diastolic dysfunction was present in 45 (90 %) of the patients. Diastolic dysfunction was more common among female (95.8%) compared to male (84.6%). The prevalence of diastolic dysfunction increased with longer duration of diabetes, age and presence of Left Ventricular Hypertrophy (LVH).

The findings of this study indicate that myocardial damage in patients with diabetes affects diastolic dysfunction before systolic function.

Doppler echocardiography is one of the most useful clinical tools for the assessment of left ventricular diastolic function.

الملخص

داء السكر هو أحد الأمراض الشائعة فى العالم ومتزايد الإنتشار في الدول النامية وغير النامية.إختلال وظيفة ابساط البطين الايسر يمثل المرحلة الأولى لاعتلال عضلة القلب ويسبق إختلال وظيفة الإنقباض ويحدث بدون أي تغيرات سريرية. نظرا" للحقائق أعلاه كان الهدف الأساسي من هذه الدراسة هو تقييم اختلال وظيفة ابساط البطين الأيسر ا لمرضى داء السكر.

بيانات هذه الدراسة جمعت بمستشفى السلاح الطبى لعدد ٥٠ مريض لديهم تاريخ مرضي لداء السكر. كل المرضى تم تصويرهم بجهاز الموجات فوق الصوتية (Mylab40 U/S machine) باستخدام مسبار عالى التردد.

نتائج الدراسة أوضحت أن اختلال وظيفة ابساط البطين الأيسر وجد عند ٤٥ مريضا" (٩٠%) وهو أكثر شيوعا" عند النساء (٩٥،٨%) مقارنة بالرجال (٨٤،٦%). يتزايد اختلال وظيفة ابساط البطين الأيسر مع طول فترة الاصابة بداء السكر وتضخم عضلة البطين والزيادة فى العمر. كما أثبتت الدراسة أن تلف عضلة القلب فى مرضى داء السكر يؤثر فى اختلال وظيفة الانبساط قبل الانقباض.

الموجات فوق الصوتية بالدوبلر للقلب يعد من اهم الوسائل التشخيصية لتقييم اختلال وظيفة انبساط البطين الايسر .

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List of Abbreviations

A: peak A wave velocity;

Am: atrial contraction;

adur: duration of atrial reversal

CHD: Coronary heart disease

DHD: Diabetic heart disease

DM: Diabetes millets

DT: deceleration time

DT: deceleration time

E: peak E wave velocity

Em: early myocardial velocity

IVR: isovolumic relaxation time

LA: Left atrium

LVEDP: left ventricular end-diastolic pressure

LVDDF: Left ventricular diastolic dysfunction

LVSF: Left ventricular systole function

PW: pulsed wave

PVa = peak atrial reversal (a wave) velocity;

PVD = peak diastolic (Dwave) velocity

PVS = peak systolic (S wave) velocity

RCM: Restrictive cardiomyopathy

TDI: tissue Doppler imaging

Chapter one

1-1 introduction:

Diabetes mellitus is a chronic illness that requires continuing medical care and ongoing patient self-management education and support to prevent acute complications and to reduce the risk of long-term complications (Madhumathi et al 2014).

The existence of cardiomyopathy was first proposed by Ruber et al in 1972 .In 1974; Framingham study showed that heart failure was more common in diabetes due to diabetic cardiomyopathy (Kannel WB et al 1974).The Framingham heart study revealed a marked increase in congestive heart failure, coronary artery disease and myocardial Infarction in diabetic patients (Garcia et al 1976).

Patients with signs and symptoms of heart failure with preserved left ventricular systolic function i.e., ejection fraction of 60% are said to have diastolic heart failure. Diastolic heart failure (DHF) is observed in 40% of patients with other heart failure. Diabetes mellitus is one of the major risk factors for DHF. The mortality rates among the patients with diastolic heart failure ranges from 5-8% annually as compared with 10-15% among patients with systolic heart failure (Kleinman et al 1988).

Left ventricular diastolic dysfunction thus represent the first stage of diabetic cardiomyopathy preceding changes in systolic function, reinforcing the importance of early examination of ventricular function in individual with diabetes (Zarich , Nesto 2001).

The diastolic abnormalities are present in diabetic patients without diabetic complications of cardiovascular system (Sujino et al 2005), it is the earliest and specific functional abnormality in diabetic cardiomyopathy and can affect patients who are free of macro vascular complications and newly diagnosed

diabetes mellitus or even in those with a disease duration of less than 1 year (Vanninen et al 1992).

Diastole encompasses the time period during which the myocardium loses its ability to generate force and shorten and returns to an unstressed length and force. By extension, diastolic dysfunction occurs when these processes are prolonged, slowed, or incomplete. Moreover, if diastolic function is truly normal, it must remain normal both at rest and during the stress of a variable heart rate, stroke volume, end-diastolic volume, and blood pressure (Seneviratne 1977) .

Diastolic dysfunction refers to a condition in which abnormalities in mechanical function are present during diastole. The causes of diastolic dysfunction may be subdivided into a decrease in passive myocardial diastolic compliance, and an impairment in active LV relaxation. Abnormalities in diastolic function may occur in the presence or absence of a clinical syndrome of heart failure and with normal or abnormal systolic function. Therefore, whereas diastolic dysfunction describes an abnormal mechanical property, diastolic heart failure describes a clinical syndrome (Zarich , Nesto 2001).

Tissue Doppler imaging has markedly improved the echocardiographic detection of diastolic dysfunction in asymptomatic patients with Type 2 diabetes mellitus (Vanninen et al 1992).

Therefore, this study was conducted to determine the effect of glycemic status onleft ventricular diastolic function in normotensive Type 2 diabetic patients. This study highlights the problem of left ventricular diastolic dysfunction to be taken in consideration while dealing patients with Type 2 diabetes mellitus who were free from symptoms of heart failure.

1.2. Problem of study:

We do not have a definite data regarding the echocardiographic findings in asymptomatic type 2 diabetics in our population. Sudanese are genetically more susceptible to diabetes compared to other races cardiovascular complications are known to be the main cause of morbidity and death in diabetic patients.

1.3. Objective of study:

1.3.1. General objective:

To evaluate the left ventricular diastolic dysfunction in type 2 diabetes mellitus using echocardiography

1.3.2 ***Specific objective:***

To evaluate the left ventricular diastolic dysfunction in type 2 diabetes mellitus

- To study the correlation of duration of diabetes mellitus with the left ventricular diastolic dysfunction.
- To study the correlation between age and sex with left ventricular diastolic dysfunction .
- To study the correlation between present of left ventricular hypertrophy and left ventricular diastolic dysfunction .
- To study the correlation left ventricular systole function and left ventricular diastolic dysfunction.
- To study the correlation left atrial size and left ventricular diastolic dysfunction.

1.5 Over view of study:

The research contains Five Chapters:

Chapter One: Include introduction, statement of problem, objectives, and over view.

Chapter Two: Include literature review, heart anatomy and physiology,

Diabatic heart diseases,physic and technique of ulteasound and echo , echo assessment of left ventricular diastolic dysfunction, trans-thoracic echocardiography and previous studies.

Chapter Three: Include Material and Method

Chapter Four: Include the Results of the study

Chapter Five: Include Discussion, Conclusion and Recommendation, References and appendix showing a practical work

Chapter Two

Theoretical background and literature review

2.1 Heart Anatomy and physiology:

2.1.1. Anatomy of the Heart

The heart is a muscular organ located in the thoracic cavity medial to the lungs and posterior to the sternum, protected by the rib cage. About the size of a closed fist that functions as the body's circulatory pump. It takes in deoxygenated blood through the veins and delivers it to the lungs for oxygenation before pumping it into the various arteries (which provide oxygen and nutrients to body tissues by transporting the blood throughout the body).

On its superior end, the base of the heart is attached to the aorta, pulmonary arteries and veins, and the vena cava. The inferior tip of the heart, known as the apex. The base of the heart is located along the body's midline with the apex pointing toward the left side. Because the heart points to the left, about 2/3 of the heart's mass is found on the left side of the body and the other 1/3 is on the right.(Marieb , Hoehn2013)

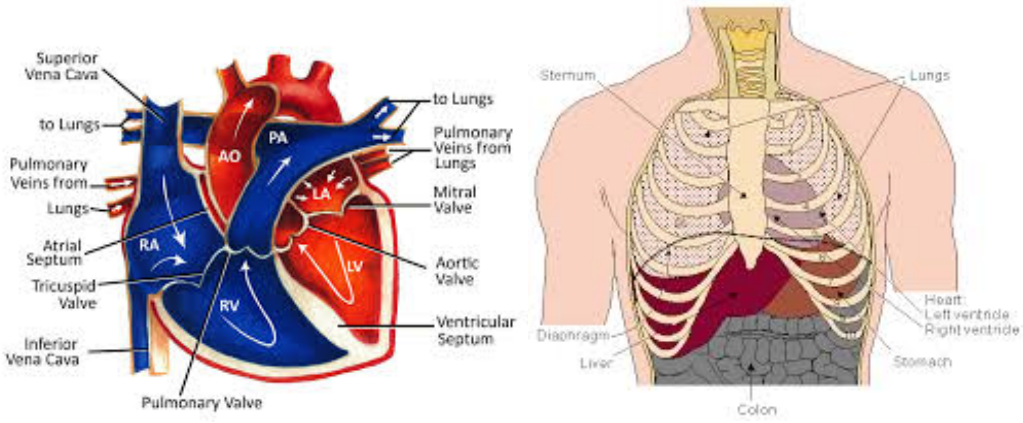


Figure 2.1: shows heart anatomy and location (Tony & Gill 2011)

2.1.2. Pericardium:

The heart sits within a fluid-filled cavity called the pericardial cavity. The walls and lining of the pericardial cavity are a special membrane known as the pericardium. Pericardium is a type of serous membrane that produces serous fluid to lubricate the heart and prevent friction between the ever beating heart and its surrounding organs. Besides lubrication, the pericardium serves to hold the heart in position and maintain a hollow space for the heart to expand into when it is full. The pericardium has 2 layers—a visceral layer that covers the outside of the heart and a parietal layer that forms a sac around the outside of the pericardial cavity .(Marieb , Hoehn2013)

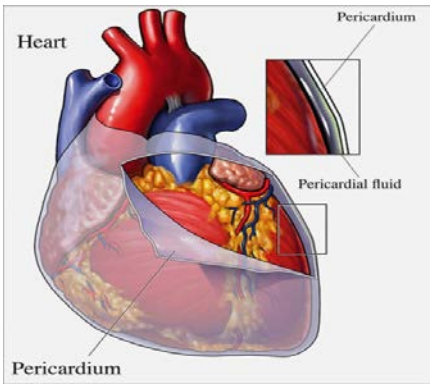


Figure 2.2: shows pericardium layer (Tony & Gill 2011)

2.1.3. Structure of the Heart Wall:

The heart wall is made of 3 layers: Epicardium, myocardium and endocardium.

2.1.3.1. Epicardium:

The epicardium is the outermost layer of the heart wall and is just another name for the visceral layer of the pericardium. Thus, the epicardium is a thin layer of serous membrane that helps to lubricate and protect the outside of the heart.

Below the epicardium is the second, thicker layer of the heart wall: the myocardium .(Marieb , Hoehn2013)

2.1.3.2. Myocardium:

The myocardium is the muscular middle layer of the heart wall that contains the cardiac muscle tissue. Myocardium makes up the majority of the thickness and mass of the heart wall and is the part of the heart responsible for pumping blood. Below the myocardium is the thin endocardium layer.(Marieb , Hoehn2013)

2.1.3.3. Endocardium:

Endocardium is the simple squamous endothelium layer that lines the inside of the heart. The endocardium is very smooth and is responsible for keeping blood from sticking to the inside of the heart and forming potentially deadly blood clots. .(Marieb , Hoehn2013)

The thickness of the heart wall varies in different parts of the heart. The atria of the heart have a very thin myocardium because they do not need to pump blood very far—only to the nearby ventricles. The ventricles, on the other hand, have a very thick myocardium to pump blood to the lungs or throughout the entire body. The right side of the heart has less myocardium in its walls than the left side because the left side has to pump blood through the entire body while the right side only has to pump to the lungs .(Marieb , Hoehn2013)

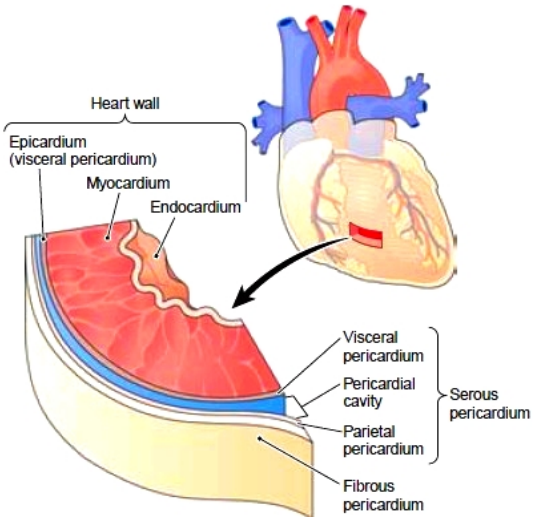


Figure 2.3: shows heart layers (Tony & Gill 2011)

2.1.4. Cardiac chambers and valves:

The heart consists of four main chambers (left and right atria, and left and right Ventricles) and four valves (aortic, mitral, pulmonary and tricuspid). Venous blood returns to the right atrium (RA) via the superior and inferior vena cava, and leaves the right ventricle (RV) for the lungs via the pulmonary artery. Oxygenated blood from the lungs returns to the left atrium (LA) via the four pulmonary veins, and leaves the left ventricle (LV) via the aorta (MAKING SENSE of Echocardiography, A hands-on guide, Second Edition, Andrew R Houghton).(Marieb , Hoehn2013)

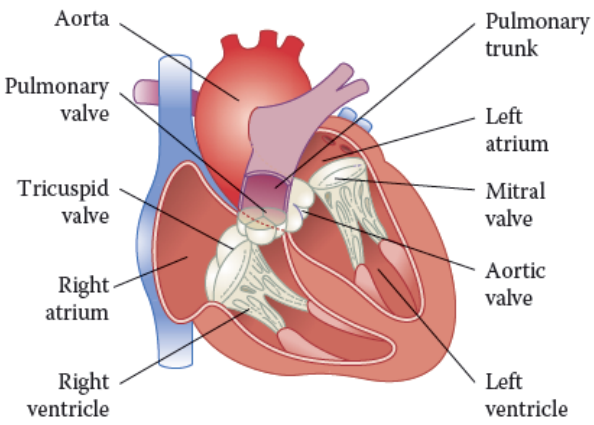


Figure 2.4: heart chambers and valve (Tony & Gill 2011)

2.1.4.1. The aortic valve:

The aortic valve lies between the left ventricular outflow tract (LVOT) and aortic root and has three cusps, which open widely during systole. In diastole, the valve closes .(Marieb , Hoehn2013)

2.1.4.2. The left ventricle:

It is the main pumping chamber of the heart and its wall is thicker (and myocardial mass greater), although less trabeculated, than that of the RV.(Marieb , Hoehn2013)

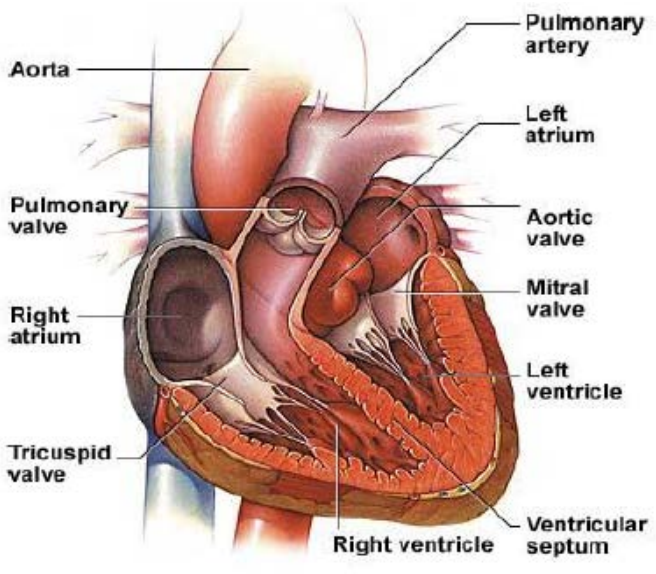


Figure 2.5: shows heart chambers and valves (Tony & Gill 2011)

2.1.4.3. The mitral valve:

The mitral valve lies between the left atrium and ventricle and has two leaflets that open during diastole and close in systole, to prevent regurgitation of blood from the LV back into the LA. The mitral valve needs to be thought of as more than just two leaflets, however, because the mitral annulus, papillary muscles and chordate tendineae are all essential to the valve's structure and function.

The mitral leaflets are termed anterior and posterior and attach around their base to the fibrous mitral annulus, an elliptical ring separating the LA and LV. The anterior mitral leaflet is longer (from base to tip) than the posterior leaflet, but the length of its attachment to the annulus is shorter and so the surface area of both leaflets is about equal. Each leaflet is divided into three segments, or scallops, which are named A1, A2 and A3 (anterior leaflet) and P1, P2 and P3 (posterior leaflet), There are two papillary muscles, named anterolateral and posteromedial (after the location of their attachment to the LV), and which are attached to the mitral leaflets via the chordae tendineae. Although there are two leaflets and two papillary muscles, each papillary muscle supplies chordae to both leaflets .(Marieb , Hoehn2013)

Chordae from the medial aspects of both leaflets attach to the posteromedial papillary muscle and from the lateral aspects to the anterolateral papillary muscle .(Marieb , Hoehn2013)

The chordae keep the mitral leaflets under tension during systole, preventing prolapse of the leaflets back into the LA. They are categorized into three groups:First order or marginal chordae, which attach to the free edges of the mitral leaflets ,Second order or strut chordae, which attach to the ventricular surface of the leaflets (away from the free edges) and Third order or basal chordae, which run directly from the ventricular wall (rather than the papillary muscles) to the ventricular surface of the posterior leaflet, usually near the annulus .(Marieb , Hoehn2013)

The mitral leaflets are normally thin and open widely during diastole, with the anterior leaflet almost touching the IVS .(Marieb , Hoehn2013)

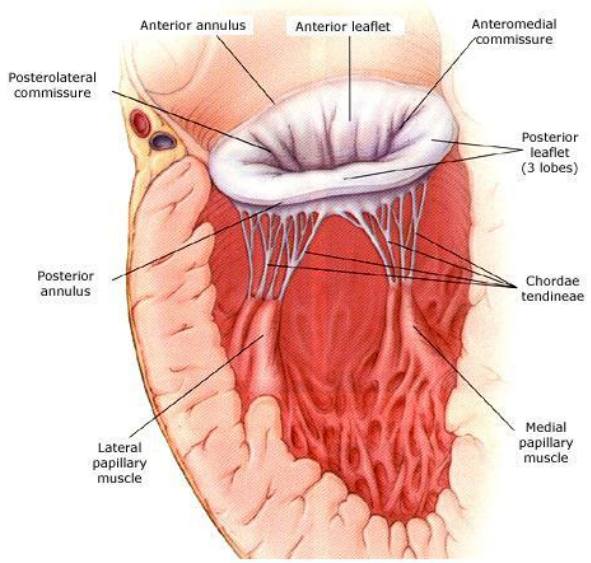


Figure 2.6: shows anatomy of mitral valve (Hatchett 2007)

2.1.4.4. The left atrium:

The LA is situated at the back of the heart, in front of the esophagus. The LA is a relatively smooth-walled structure. It is entered by four pulmonary veins carrying oxygenated blood from the lungs – two from the right lung and two from the left. (.(Marieb , Hoehn2013))

The LA is not just a passive conduit between the pulmonary veins and the LV, but contracts during atrial systole (immediately after the onset of the P wave) to provide additional diastolic filling of the LV (the 'atrial kick'). This is particularly important when diastolic filling is impaired.The LA is separated from the RA by the interatrial septum. (.(Marieb , Hoehn2013))

2.1.4.5. The pulmonary valve:

The pulmonary valve lies between the right ventricular outflow tract (RVOT) and pulmonary artery, opening during systole to allow blood to pass from the Ventricle into the pulmonary circulation, and closing in diastole to prevent regurgitation (asmall amount of 'physiological' pulmonary regurgitation is normal). The valve itself is structurally similar to the aortic valve, having three cusps (called anterior, left and right). (.(Marieb , Hoehn2013))

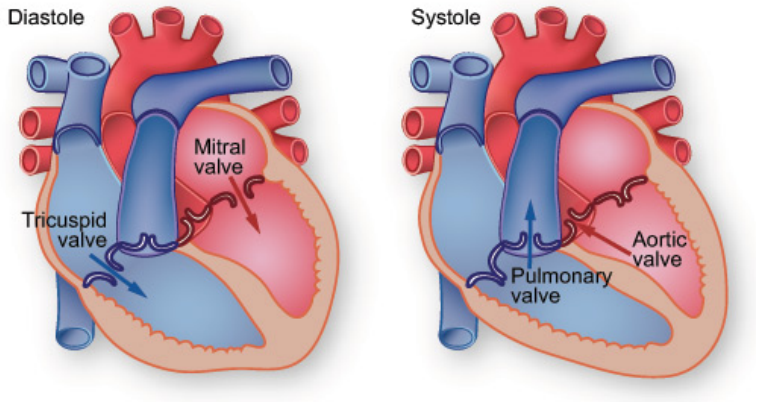


Figure 2.7: shows anatomy of pulmonary valve(Tony & Gill 2011)

2.1.4.6. The right ventricle:

The RV acts as the pumping chamber for deoxygenated blood returning from the body en route to the lungs. It is thinner walled than the LV. (.(Marieb , Hoehn2013))

2.1.4.7. The tricuspid valve:

The tricuspid valve lies between the RA and RV, opening during diastole to allow blood to pass from the atrium to the ventricle, and closing in systole to prevent regurgitation (although a small amount of 'physiological' tricuspid regurgitation is commonly seen in normal individuals).(Marieb , Hoehn2013)

As its name suggests, the tricuspid valve has three cusps – in order of decreasing size, these are called the anterior, posterior and septal cusps. There are also three papillary muscles, which, in a similar way to the mitral valve, are attached to the cusps via chordae tendineae. The orifice area of the tricuspid valve is greater than that of the mitral valve, normally >7.0 cm2. (.(Marieb , Hoehn2013))

2.1.4.8. The right atrium:

The RA receives blood returning to the heart via the superior and inferior vena cavae. It also receives blood draining from the myocardium via the coronary sinus,which enters the RA posteriorly, just superior to the tricuspid valve.

The Eustachian valve, an embryological remnant, may be seen in the RA near the junction with the inferior vena cava. (.(Marieb , Hoehn2013))

2.1.5. The coronary arteries:

The coronary circulation normally arises as two separate vessels from the sinuses of Valsalva – the LCA from the left coronary sinus, and the RCA from the right coronary sinus. The initial portion of the LCA is the left main stem,

which soon divides into the left anterior descending (LAD) and circumflex (Cx) arteries. The LAD artery runsdown the anterior interventricular groove giving rise to diagonal branches, which course towards the lateral wall of the LV, and septal perforators that supply the IVS. (.(Marieb , Hoehn2013))

The Cx artery runs in the left atrioventricular groove, giving rise to obtuse marginal branches which extend across the lateral surface of the LV.

The RCA runs in the right atrioventricular groove, and in most people gives rise to the posterior descending artery, which runs down the posterior interventricular groove. This defines ‘dominance’ – most people therefore have a ‘dominant’ RCA, but in some people the Cx gives rise to the posterior descending artery and they are said to have a ‘dominant’ Cx.(Marieb , Hoehn2013)

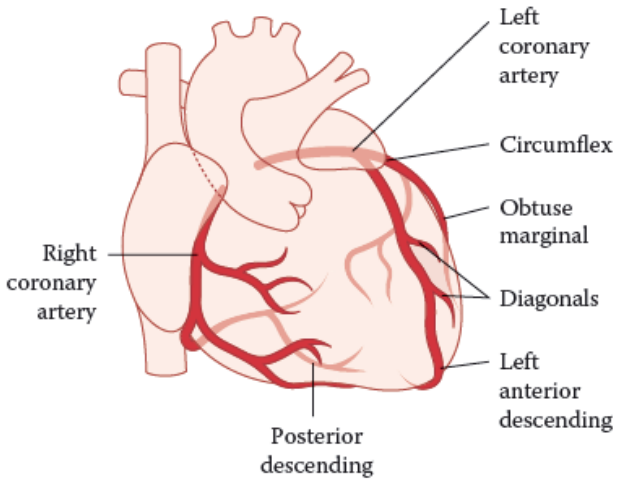


Figure 2.8: shows coronary artery(Tony & Gill 2011)

Physiology of the heart:

2.2.1. Coronary Systole and Diastole:

At any given time the chambers of the heart may found in one of two states:

2.2.1.1. Systole: During systole, cardiac muscle tissue is contracting to push blood out of the chamber. .(Marieb , Hoehn2013)

2.2.1.2. Diastole: During diastole, the cardiac muscle cells relax to allow the chamber to fill with blood. Blood pressure increases in the major arteries during ventricular systole and decreases during ventricular diastole. This leads to the 2 numbers associated with blood pressure—systolic blood pressure is the higher number and diastolic blood pressure is the lower number. For example, a blood pressure of 120/80 describes the systolic pressure (120) and the diastolic pressure (80). .(Marieb , Hoehn2013)

2.2.2. The Cardiac Cycle:

The cardiac cycle includes all of the events that take place during one heartbeat. There are 3 phases to the cardiac cycle: atrial systole, ventricular systole, and relaxation. .(Marieb , Hoehn2013)

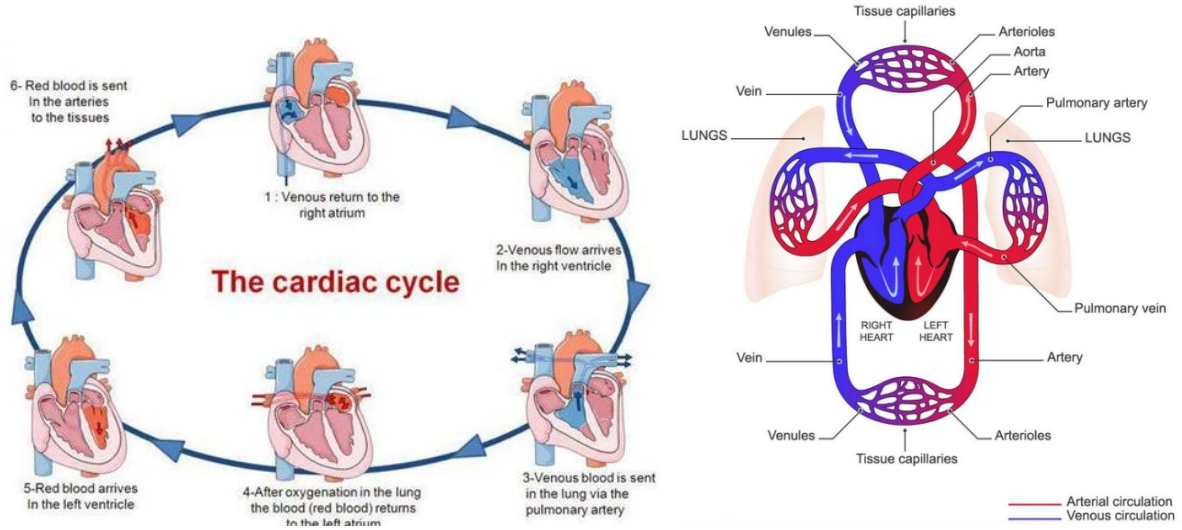


Figure 2.9: shows cardiac cycle(Tony & Gill 2011)

2.2.2.1. Atrial systole: During the atrial systole phase of the cardiac cycle, the atria contract and push blood into the ventricles. To facilitate this filling, the AV valves stay open and the semilunar valves stay closed to keep arterial blood from re-entering the heart. The atria are much smaller than the ventricles, so they only fill about 25% of the ventricles during this phase. The ventricles remain in diastole during this phase. .(Marieb , Hoehn2013)

2.2.2.2. Ventricular systole: During ventricular systole, the ventricles contract to push blood into the aorta and pulmonary trunk. The pressure of the ventricles forces the semilunar valves to open and the AV valves to close. This arrangement of valves allows for blood flow from the ventricles into the arteries. The cardiac muscles of the atria repolarize and enter the state of diastole during this phase. .(Marieb , Hoehn2013)

2.2.2.3. Relaxation phase: During the relaxation phase, all 4 chambers of the heart are in diastole as blood pours into the heart from the veins. The ventricles fill to about 75% capacity during this phase and will be completely filled only after the atria enter systole. The cardiac muscle cells of the ventricles repolarize during this phase to prepare for the next round of depolarization and contraction. During this phase, the AV valves open to allow blood to flow freely into the ventricles while the semilunar valves close to prevent the regurgitation of blood from the great arteries into the ventricles. .(Marieb , Hoehn2013)

2.1.4. Blood Flow through the Heart:

Deoxygenated blood returning from the body first enters the heart from the superior and inferior vena cava. The blood enters the right atrium and is pumped through the tricuspid valve into the right ventricle. From the right ventricle, the blood is pumped through the pulmonary semilunar valve into the pulmonary trunk. .(Marieb , Hoehn2013)

The pulmonary trunk carries blood to the lungs where it releases carbon dioxide and absorbs oxygen. The blood in the lungs returns to the heart through the pulmonary veins. From the pulmonary veins, blood enters the heart again in the left atrium. .(Marieb , Hoehn2013)

The left atrium contracts to pump blood through the bicuspid (mitral) valve into the left ventricle. The left ventricle pumps blood through the aortic semilunar valve into the aorta. From the aorta, blood enters into systemic circulation throughout the body tissues until it returns to the heart via the vena cava and thecycle repeats. .(Marieb , Hoehn2013)

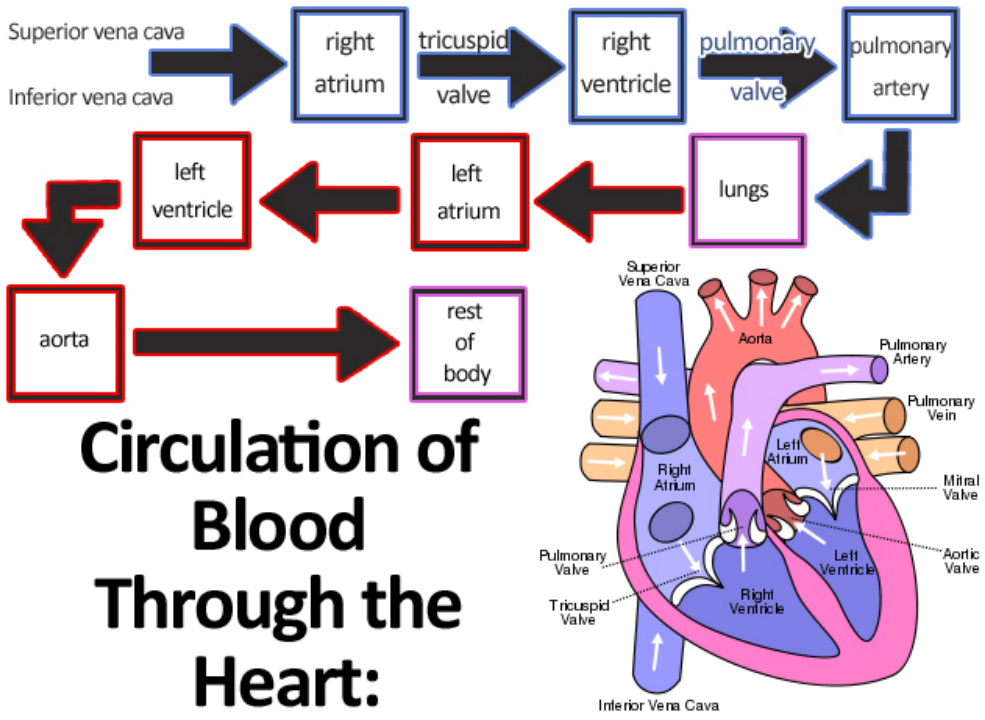


Figure 2.10: shows circulation of blood through the heart(Tony & Gill 2011)

2.2. Common diseases of the heart:

The main types of heart disease are listed here in order of seriousness:

2.3.1 Coronary heart disease:

This is the most common form of heart disease, so it is the one that you need to be most focused on avoiding. Coronary heart disease refers to the failure of your blood vessels to circulate blood to your heart and organs due to gunk build up on the walls of your circulatory system. (Nagueh 2009)

Figure 2.11: coronary heart disease(Boyer et al 2004)

Coronary heart disease is often confused with Coronary artery disease although coronary heart disease can be due to a few different causes, while coronary artery disease has only one cause. (Nagueh 2009)

2.3.2. Coronary artery disease (Ischemic heart disease):

Is a disease of the blood circulation system caused by junk within (Not on the walls like coronary heart disease) the walls of the arteries (The things that keep

our blood moving) that supply the rest of the body. Coronary artery disease is the build-up of plaque in the arteries. This can lead to such conditions as stroke or heart attack. (Cosson , Kevorkian 2003).

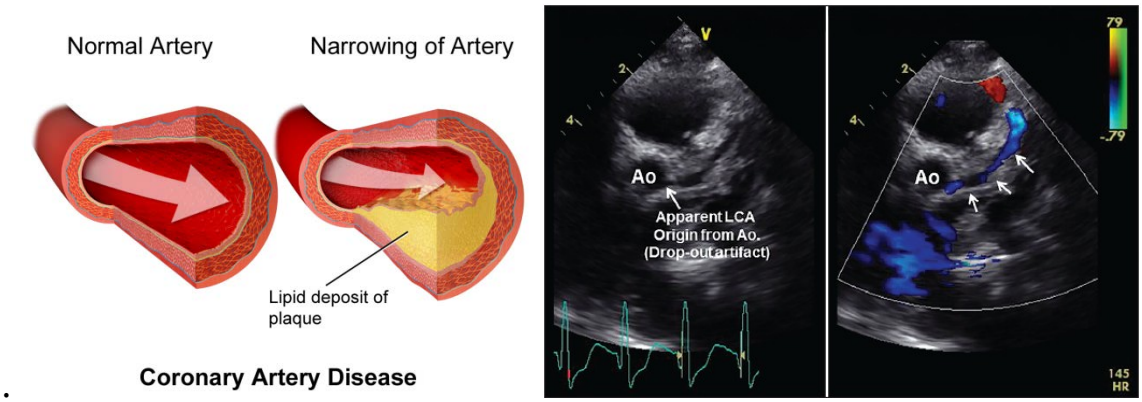


Figure 2.12: coronary artery disease(Boyer et al 2004)

2.2.3. Alcoholic heart disease:

This is pretty much self explanatory. But just to be clear this form of the disease is due to the overuse of alcohol. (Cosson , Kevorkian 2003).

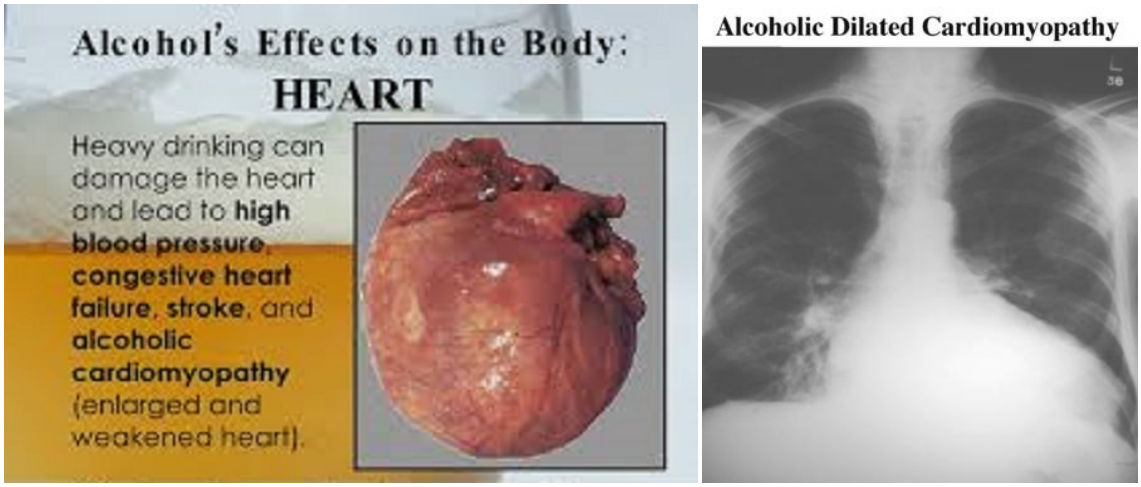


Figure 2.13: alcoholic heart disease (Boyer et al 2004)

2.2.4. Congenital heart disease:

This disease on our list of heart diseases is caused by birth defects that one way or other weaken the heart muscle and is one the primary causes of early childhood death in America. (Cosson , Kevorkian 2003).

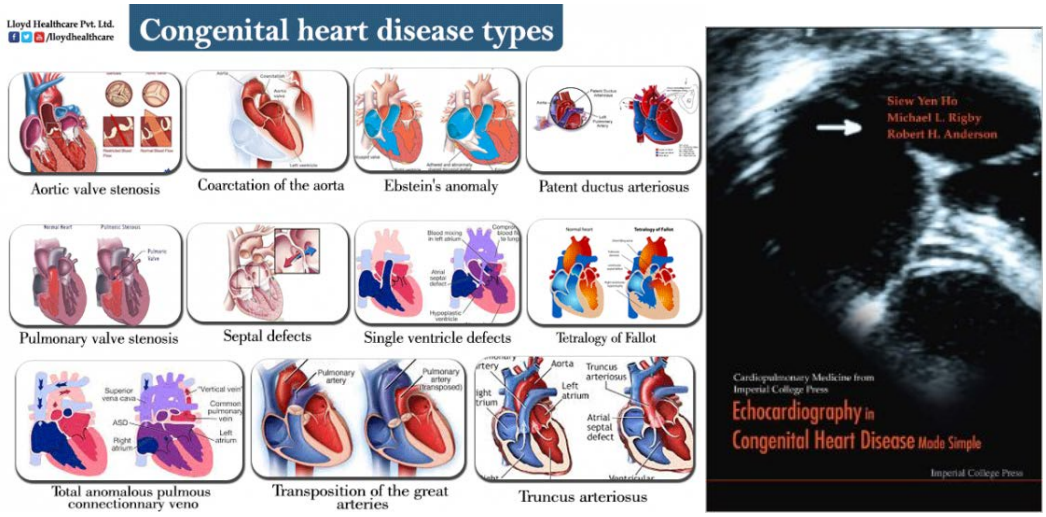


Figure 2.14: congenital heart disease types (Boyer et al 2004)

2.2.5. Hypertensive heart disease:

This is heart disease brought on by out of control, long term high blood pressure. It is just one of many diseases and conditions brought on by high blood pressure. High blood pressure also affects the liver very poorly among other organs(Cosson , Kevorkian 2003).

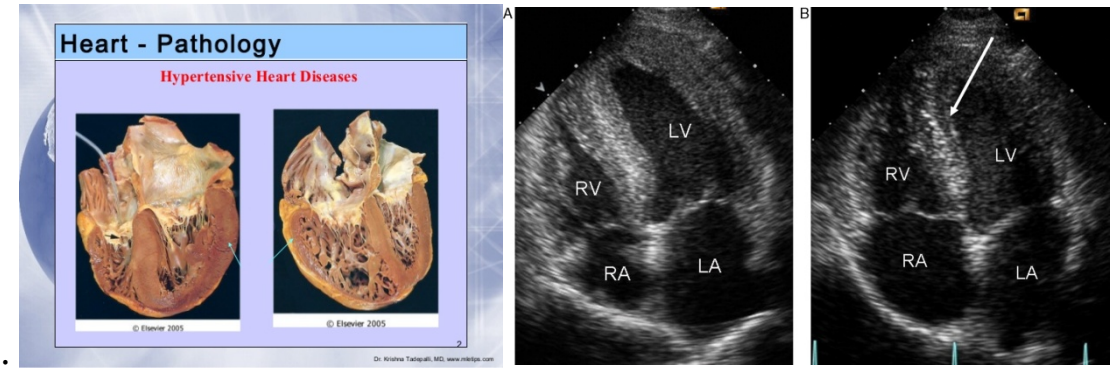


Figure 2.15: hypertensive heart disease (Boyer et al 2004)

2.2.6. Valvular heart disease:

Heart disease that affects the valves of the heart and not so much the heart itself, this can be congenital or can be developed over time (for example Rheumatic heart disease) . Heart murmurs may be benign or signs of a damaged heart valve. (Cosson , Kevorkian 2003).

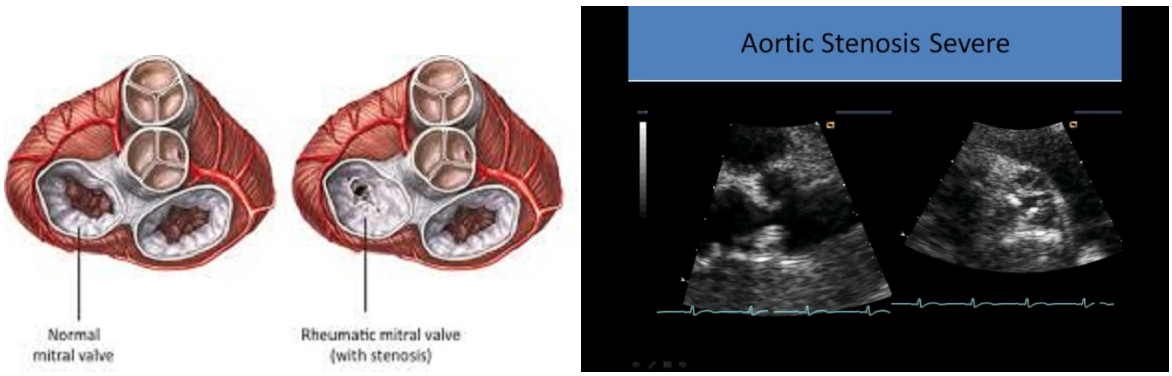


Figure 2.16: aortic stenosis severe (Boyer et al 2004)

2.2.7. Inflammatory heart disease:

Heart disease caused by inflammation (swelling) of the heart wall. This can be caused by bad bacteria getting past the immune system or stress. (For example: Pericarditis is an inflammation of the sac that encloses the heart). (Cosson , Kevorkian 2003).

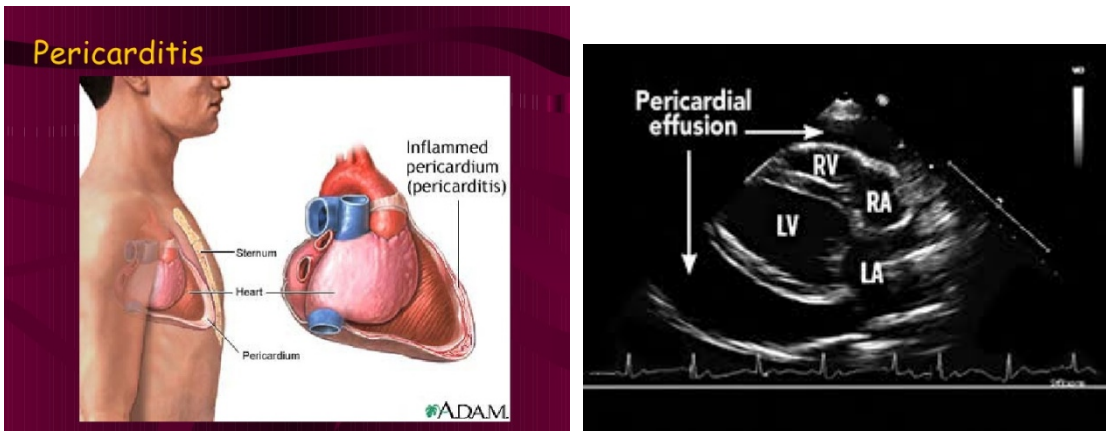


Figure 2.17: shows pericardial effusion(Boyer et al 2004)

2.2.8. Dilated Heart Disease:

In this disease the heart (especially the left ventricle) is enlarged and the pumping function limited causing a loss of blood to the rest of the body. (Cosson , Kevorkian 2003).

2.2.9. Hypertrophic Heart Disease:

This is a genetic disorder caused by various mutations in our genes. Here the heart muscle is thickened (Less elastic), which can obstruct blood flow and stop the heart from working right.(Cosson , Kevorkian 2003).

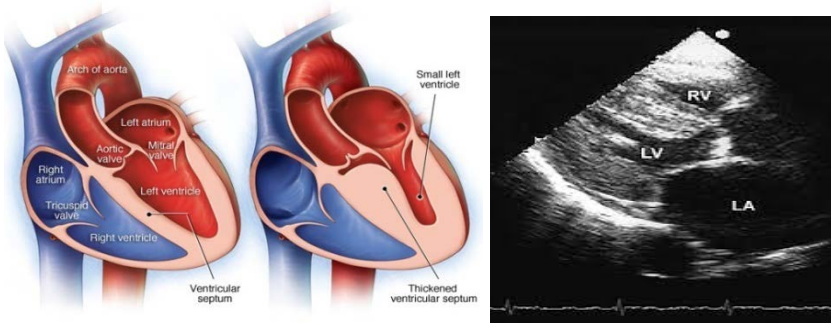


Figure 2.18: hypertrophic heart disease(Boyer et al 2004)

2.2.10. Arrhythmia:

It is when the heart beats in an irregular way. Heart valve disease happens when the valves that keep the blood flowing properly in and out of the heart malfunction. (Cosson , Kevorkian 2003).

Figure 2.19: Arrythmia(Boyer et al 2004)

2.2.11. Cardiomyopathy:

The heart is thickened or not as soft and supple as it should be. This makes it hard for the heart to pump blood efficiently. (Cosson , Kevorkian 2003).

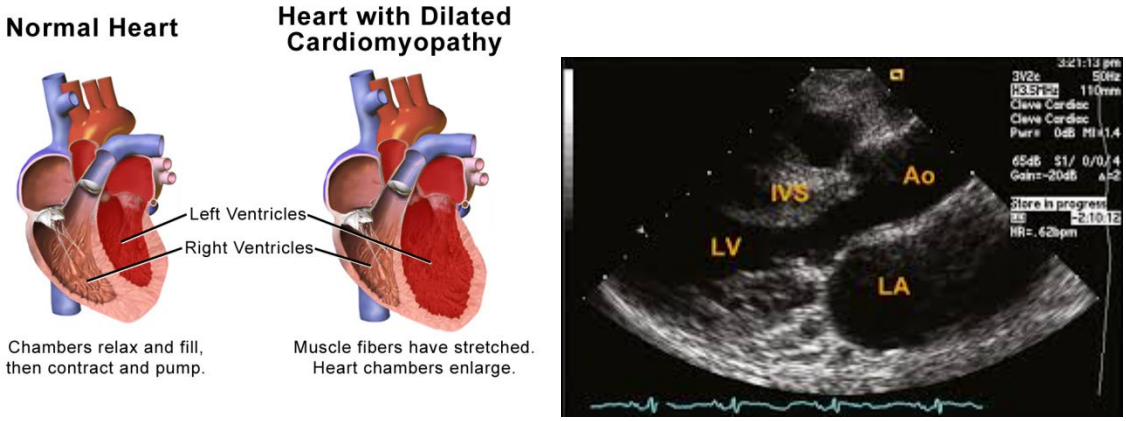


Figure 2.20: cardiomyopathy(Boyer et al 2004)

2.2.12. Heart failure:

Occurs when the heart is not able to pump blood through the body as well as it should. This means that other organs, which normally get blood from the heart, do not get enough blood. It does not mean that the heart stops. (Senior et al 2005)

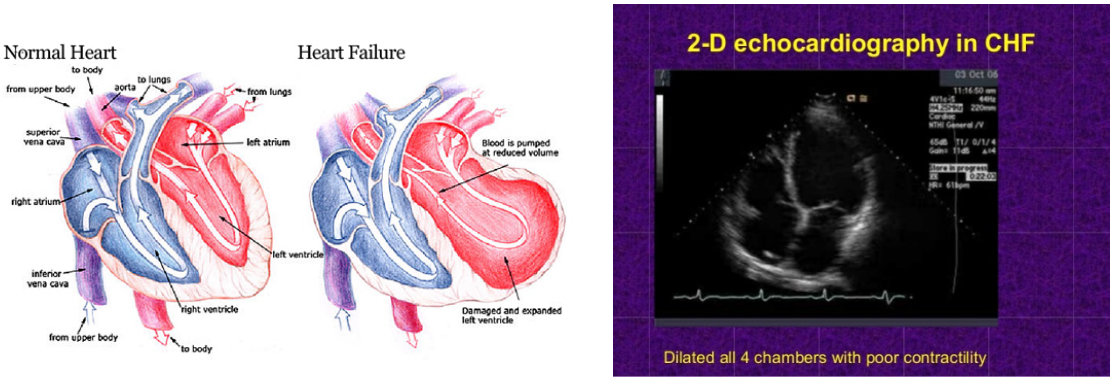


Figure 2.21: showed CHF(. (Nagueh 2009)

2.2.13. Restrictive heart disease:

This is the least common disease of the heart, the walls of the heart ventricles are stiff, but may not be thickened, and resists the normal filling of the heart with blood. (Senior et al 2005)

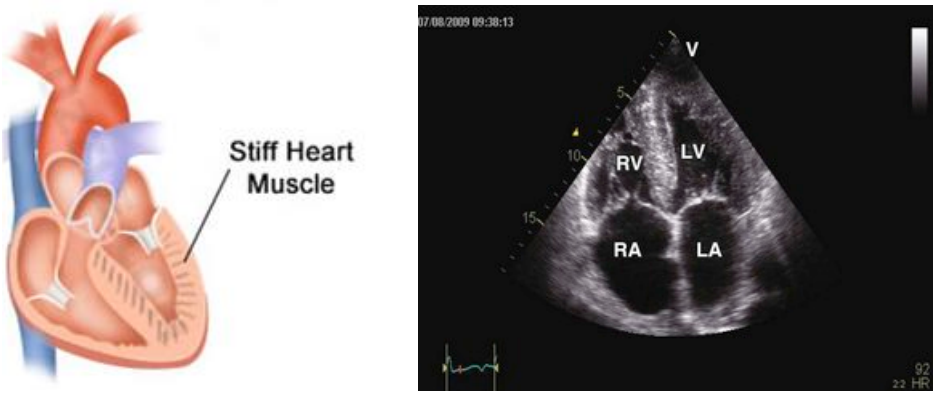


Figure 2.22: showed RCM(. (Nagueh 2009)

2.3. Specific diseases of the heart:

2.4.1. Diabetic heart disease (DHD):

The term "diabetic heart disease" (DHD) refers to heart disease that develops in people who have diabetes. Compared with people who don't have diabetes, people who have diabetes: are at higher risk for heart disease, have additional causes of heart disease and may develop heart disease at a younger age. (Senior et al 2005)

May have more severe heart disease Diabetes is a disease in which the body's blood glucose (sugar) level is too high. Normally, the body breaks down food into glucose and carries it to cells throughout the body. The cells use a hormone called insulin to turn the glucose into energy. (Senior et al 2005)

The two main types of diabetes are type 1 and type 2. In type 1 diabetes, the body doesn't make enough insulin. This causes the body's blood sugar level to rise. In type 2 diabetes, the body's cells don't use insulin properly (a condition called insulin resistance). At first, the body reacts by making more insulin. Over time, though, the body can't make enough insulin to control its blood sugar level (McMurray, 2012).

2.4.1.1. Type of Diabetic heart disease DHD:

2.4.1.1.1 Coronary Heart Disease:

In CHD, a waxy substance called plaque (plak) builds up inside the coronary arteries. These arteries supply your heart muscle with oxygen-rich blood.

Plaque is made up of fat, cholesterol, calcium, and other substances found in the blood. When plaque builds up in the arteries, the condition is called atherosclerosis. Plaque narrows the coronary arteries and reduces blood flow to your heart muscle. The buildup of plaque also makes it more likely that blood clots will form in your arteries. Blood clots can partially or completely block blood flow. (McMurray, 2012)

CHD can lead to chest pain or discomfort called angina, irregular heartbeats called arrhythmias, a heart attack, or even death (McMurray, 2012)

2.4.1.1.2 Heart Failure:

Heart failure is a condition in which your heart can't pump enough blood to meet your body's needs. The term “heart failure” doesn't mean that your heart has stopped or is about to stop working. However, heart failure is a serious condition that requires medical care. If you have heart failure, you may tire easily and have to limit your activities. CHD can lead to heart failure by weakening the heart muscle over time. (McMurray, 2012).

2.4.1.1.3 Diabetic Cardiomyopathy:

Diabetic cardiomyopathy is a disease that damages the structure and function of the heart. This disease can lead to heart failure and arrhythmias, even in people who have diabetes but don't have CHD.

2.4.1.2. Cardiovascular risk factors in people with diabetes:

Hypertension ,High blood cholesterol ,High triglycerides with low HDL ,Left ventricular hypertrophy ,Obesity and Smoking. (McMurray, 2012).

2.4.1.3. Reducing the Risks:

Lifestyle modification (including healthy eating habits, regular physical exercise, smoking cessation and sustained weight loss in the overweight) can be of major benefit in preventing non-communicable diseases such as diabetes and cardiovascular disease. (McMurray, 2012).

2.4.1.4. Complication of DHS:

Heart attack ,Stroke and Amputation of lower limps

2.4.1.5. Clinical manifestations of cardiovascular disease in diabetes:

Angina (including silent ischaemia),Heart attack (including • attack • claudication silent heart attack) ,Sudden death ,Heart failure ,Fainting attacks ,Stroke ,Transient ischaemic ,Dementia ,Gangrene ,Intermittent and Foot ulcers. (McMurray, 2012).

2.4.1.6. Treatment:

Lifestyle modifications Although and Drugs: oral hypoglycaemic agents, insulin. (Paulus et al 2007)

2.4.2. **LV diastolic dysfunction.**

Doppler pattern of impaired LV relaxation, characterized by reduced early and increased late diastolic flow, is an early sign of diastolic dysfunction (DD) (grade I). More advanced grades, manifested by predominant early diastolic filling and rapid velocity deceleration (restrictive filling patterns), are associated with the most severe LV decompensation (Nishimura RA, Tajik J 1997).

The causes of diastolic dysfunction may be subdivided into a decrease in passive myocardial diastolic compliance, and an impairment in active LV relaxation. (Cosson , Kevorkian 2003).

Abnormalities in diastolic function may occur in the presence or absence of a clinical syndrome of heart failure and with normal or abnormal systolic function. Therefore, whereas diastolic dysfunction describes an abnormal mechanical property, diastolic heart failure describes a clinical syndrome (Cosson , Kevorkian 2003).

2.4.2.1.**Causes of impaired LV diastolic function**

Diastolic dysfunction is thought to reflect ‘stiffness’ or impaired relaxation of the LV, and so occurs in conditions where the LV becomes less compliant:

Ageing ,hypertension ,LVH ,myocardial ischaemia ,aortic stenosis and infiltrative cardiomyopathies. (Paulus et al 2007)

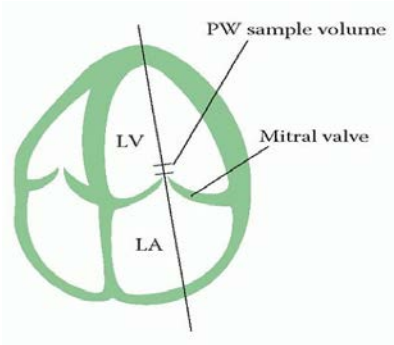
2.5. Echo assessment of LV diastolic function:

Many methods are available to characterize LV diastolic function on echo, but the most widely used are:

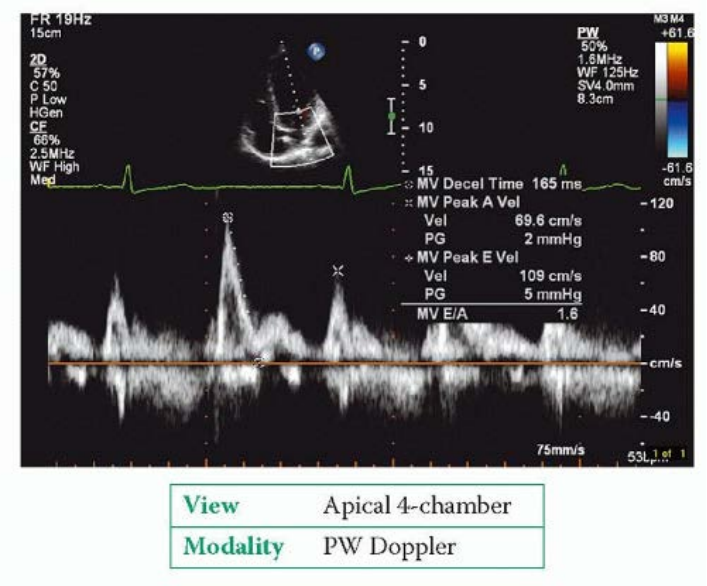
2.5.1. Inflow:

To assess LV inflow, perform PW Doppler in the apical 4-chamber view with a 1–3mm sample volume placed at the tips of the mitral valve leaflets (Fig. 2.23). Obtain a PW Doppler trace (Fig. 2.24) and measure: peak E wave velocity, peak A wave velocity, E:A ratio, E wave deceleration time (DT) and isovolumic relaxation time (IVRT). (Andrew, Houghton2002)

A sweep speed of 25 or 50 mm/s is used initially to look for respiratory variation in peak E and A wave velocities. The sweep speed is then increased to 100 mm/s before taking at least three sets of measurements with the patient's breath held at end-expiration. (Andrew, Houghton2002)



(Fig. 2.23): Positioning of sample volume for pulsed-wave (PW) Doppler of mitral valve inflow (Andrew, Houghton2002)



(Fig. 2.24): Pulsed-wave (PW) Doppler of mitral valve (MV) inflow. (Andrew, Houghton2002)

E:A ratio is simply the ratio between peak E and A wave velocities: E:Aratio= $\frac{\text{Peak E wave velocity}}{\text{Peak A wave velocity}}$.

The E wave is normally taller than the A wave, and the E:A ratio normally lies in the range 1–2. E wave deceleration time is the time period between the peak of the E wave and the end of the E wave (measured by extrapolating the E wave deceleration slope down to the baseline), and is normally 150–200 ms.

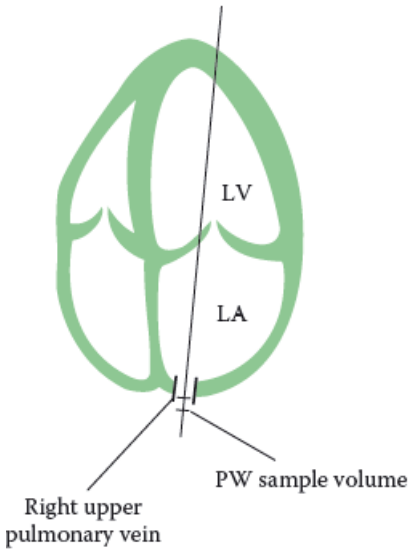
IVRT is the time period between aortic valve closure and mitral valve opening, during which LV pressure falls but there is no change in LV volume. There are various methods of measuring IVRT. The simplest is to tilt the probe, obtain a 5-chamber view and adjust the PW Doppler sample volume to lie between the mitral and aortic valves (so that both the mitral inflow and aortic outflow traces are seen on the same PW Doppler trace). Freeze the trace and measure the time period between the end of the aortic outflow trace and the start of the mitral inflow trace – this is the **IVRT**, and is normally 50–100 ms. (Andrew, Houghton2002)

2.5.2. Pulmonary venous flow:

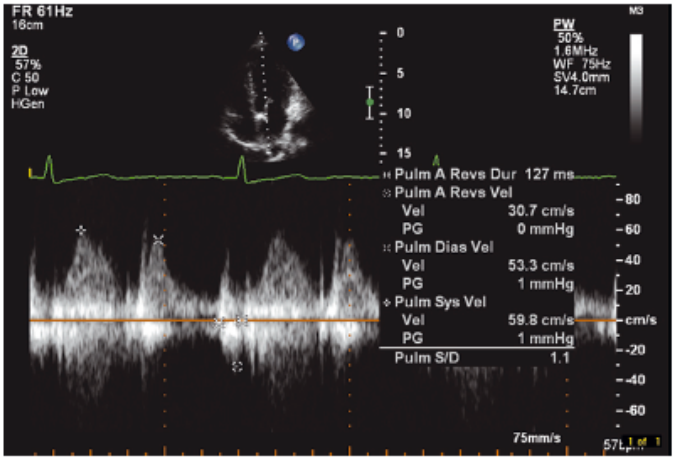
To assess pulmonary venous flow, perform PW Doppler in the apical 4-chamber view with a 2–3 mm sample volume placed 0.5 cm inside one of the pulmonary veins (the right upper pulmonary vein is usually easiest to locate, (Fig. 2.25).

Pulmonary vein flow normally consists of three components: the S wave represents forward flow into the left atrium during ventricular systole, and the smaller D wave represents forward flow during ventricular diastole. If the patient is in sinus rhythm, the S and D waves are followed by an ‘a’ wave, representing flow reversal in the pulmonary vein during atrial systole.

Obtain a PW Doppler trace (Fig. 2.26) and measure the peak systolic (S wave) velocity (PVS) and the peak diastolic (D wave) velocity (PVD) .(Senior R, Ashrafian H 2005)



(Fig. 2.25): Positioning of sample volume for pulsed-wave Doppler of pulmonary venous flow(Andrew, Houghton2002)



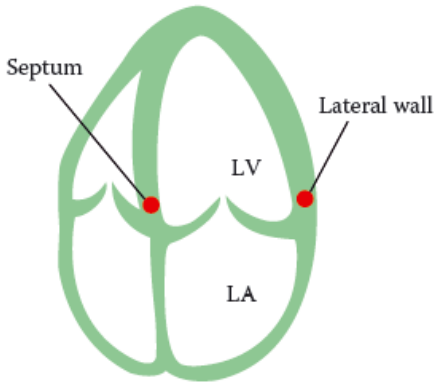
View	Apical 4-chamber
Modality	PW Doppler

(Fig. 2.26): Pulsed-wave (PW) Doppler of pulmonary venous flow. (Andrew, Houghton2002)

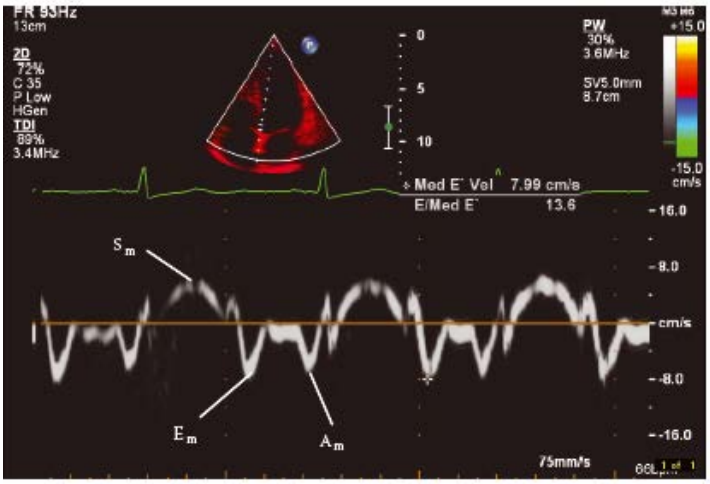
2.5.3. TDI of the mitral annulus:

TDI of the mitral annulus is undertaken in the apical 4-chamber view, placing the sample volume (which should be small, usually 2–3 mm) in the myocardium of the septum and then the lateral wall. The optimal location is 1 cm below the mitral annulus(Fig. 2.27).. In each location make a pulsed-wave tissue Doppler recording (Fig. 2.28). using a low gain setting and an aliasing velocity 15–20 cm/s. Set the sweep speed at 50–100 mm/s and take at least three sets of measurements with the patients breath held at end-expiration.

The mitral annular tissue Doppler recording shows an early myocardial velocity (Em or E') which corresponds to early diastolic relaxation, the myocardium moving away from the transducer. This is followed by a further movement away from the transducer, corresponding to atrial contraction (Am or A'). Normally Em > Am with a ratio between the two velocities in the range 1–2. If there is diastolic dysfunction, the Em:Am ratio reverses. The ratio between the peak LV inflow E wave velocity and Em should also be calculated; this ratio reflects LA pressure. Normal E/Em ratios are <8 at the septum and <10 at the lateral wall. .(Senior R, Ashrafian H 2005)



(Fig. 2.27): Positioning of sample volume for pulsed-wave tissue Doppler imaging (TDI) of the mitral annulus(Andrew, Houghton2002)



View	Apical 4-chamber
Modality	Pulsed-wave TDI

(Figure. 2.28): Pulsed-wave trace of medial mitral annulus (septal wall) obtained with tissue Doppler imaging (TDI). (Andrew, Houghton2002)

2.5.4. Interpretation of results:

The assessment of LV diastolic function combines each of the measures discussed above (Fig. 2.29). Using these measures, LV diastolic function can be

classified as normal, mildly impaired (abnormal relaxation), moderately impaired (pseudonormal) and severely impaired (restrictive filling) .(Senior , Ashrafian 2005)

(18) (19) (20) (21)

	Normal	Mild	Moderate	Severe
		↓ Relaxation	↓ Relaxation ↓ Compliance ↑ LVEDP	↓ Relaxation ↓ Compliance ↑↑ LVEDP
		Abnormal Relaxation	Pseudo-Normal	Restrictive Filling
LV Inflow Doppler				
E/A ratio	1–2	<1	1–2	>2
IVRT (ms)	50–100	>100	50–100	<50
DT (ms)	150–200	>200	150–200	<150
Pulmonary Venous Doppler				
PV_S/PV_D	$PV_S > PV_D$	$PV_S > PV_D$	$PV_S < PV_D$	$PV_S << PV_D$
PV_a (m/s)	<0.35	<0.35	≥0.35	≥0.35
$a_{dur} - A_{dur}$ (ms)	<20	<20	≥20	≥20
Mitral Annular Tissue Doppler				
E_m/A_m	1–2	<1	<1	<<1
E/E_m (septum)	<8	–	>15	–
E/E_m (lateral)	<10	–	>10	–

(Fig. 2.29): Classification of left ventricular (LV) diastolic dysfunction(Andrew, Houghton2002)

2.6. Trans-thoracic Echocardiography:

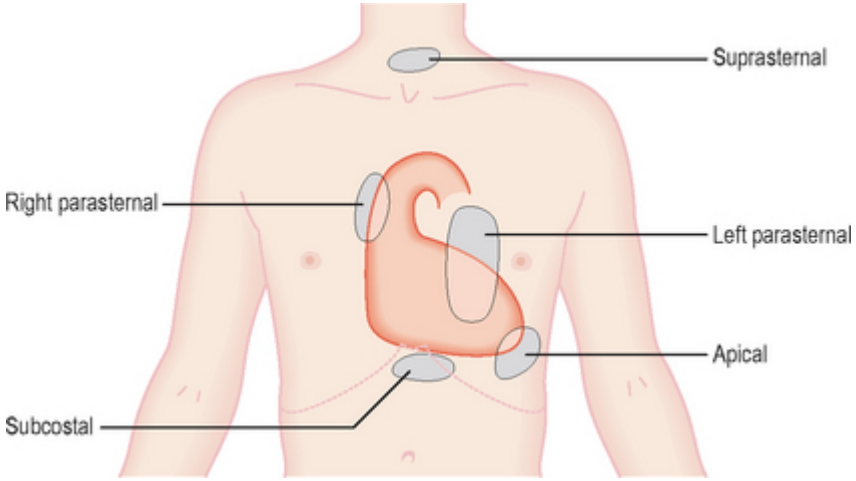


Figure 2.30: Transthoracic echo windows(Andrew, Houghton2002)

2.6.1. Patient preparation:

Patients attending for an echo study may feel anxious, not only about having the test itself but also about any abnormalities that it may reveal. To help reduce anxiety, describe the test to patients in clear and reassuring terms – explain to patients why they are having an echo, whether any special preparation is needed before they attend, what happens during the scan and how long it is likely to take.(Feigenbaum2010)

Reassure patients that having an echo is safe and painless. Patients can eat and drink normally before attending for a standard TTE, and they can take their medication as usual.

2.6.2. Standard windows and views:

There are five TTE windows (Fig. 2.30), each providing one or more views of the heart. The right parasternal window is optional and can be used when other views are suboptimal or when additional information is needed:

2.6.2.1. Left parasternal window:

The left parasternal window is located to the left of the sternum, usually in the third or fourth intercostal space, but in some patients you may need to adjust the position to optimize the image by moving the probe up/down a rib space or further towards/ away from the sternum. From the left parasternal window a number of views can be obtained. .(Feigenbaum2010)

2.6.2.1.1. Parasternal long axis view:

The parasternal long axis (LAX) view is shown in Fig. 2.31. To obtain the view with the probe in the left parasternal window, rotate the probe so that the probe's'reference point' (sometimes a 'dot') is pointing towards the patient's right shoulder. For an optimal view, aim to position the probe so that the view cuts through the centre of the mitral and aortic valves, without foreshortening the left ventricle (LV) or ascending aorta. .(Feigenbaum2010)

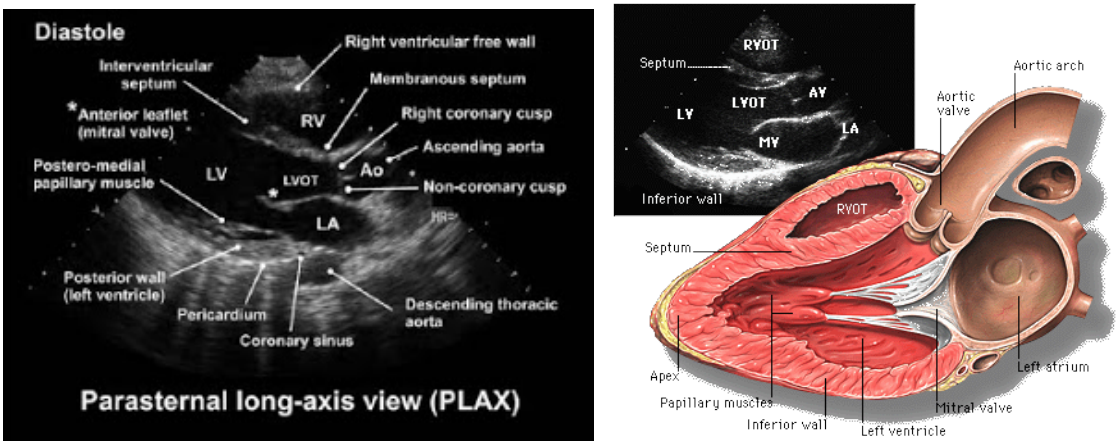
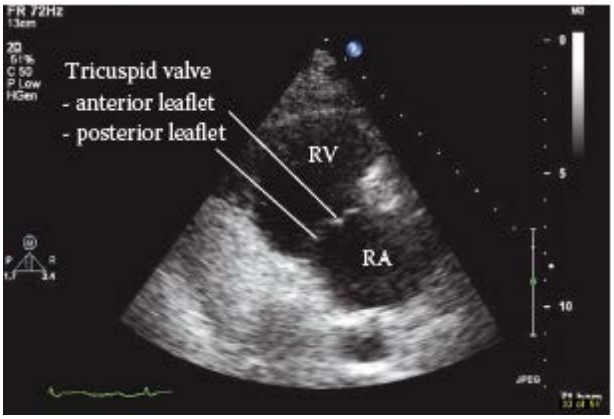


***Figure 2.31*:** Normal parasternal long axis view(.(Feigenbaum2010)).

2.6.2.1.2 Parasternal right ventricular (RV) inflow view:

This view is obtained from the left parasternal window by tilting the probe so that it points more medially and towards the patient's right hip, bringing the

right atrium (RA), tricuspid valve and RV .(Feigenbaum2010)



✓ ***Figure 2.32:*** Normal right ventricular inflow view(Andrew, Houghton2002).

2.6.2.1.3 Parasternal RV outflow view:

This view is obtained from the left parasternal window by tilting the probe so that it points more laterally and towards the patient's left shoulder, bringing the RVOT, pulmonary valve and pulmonary artery into view (Fig. 2.33). It may be possible to see the pulmonary artery bifurcation. .(Feigenbaum2010)

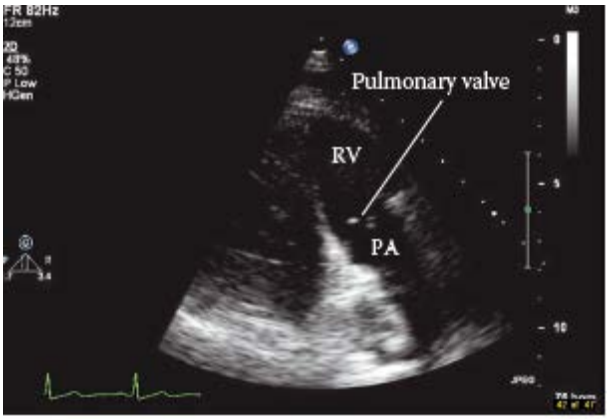


Figure 2.33: Normal right ventricular outflow view(Andrew, Houghton2002)

2.6.2.1.4 Parasternal short axis view (base, mid-cavity, apex):

To obtain the parasternal short axis (SAX) view, keep the probe in the left parasternal window and rotate it so that the 'dot' is pointing towards the patient's left shoulder. There are actually four SAX views, obtained by

sweeping the probe along the axis of the heart from the level of the aortic valve down to the apex. The standard SAX views are:

2.6.2.1.4.1 At the aortic valve level (Fig. 2.34):

Use 2D to assess the structure and function of the RVOT ,measure RVOT diameter at the aortic valve (AV) level (known as RVOT1) and at the pulmonary valve annulus level (known as RVOT2) ,assess the morphology of the main pulmonary artery up to its bifurcation and measure its diameter (known as PA1) ,assess the structure and mobility of the aortic valve; all three cusps should be visible ,inspect the LA and RA and interatrial septum, assess the structure and mobility of the tricuspid valve (the two leaflets seen are the septal and anterior leaflets) and toassess the structure and mobility of the pulmonary valve.(Feigenbaum2010)

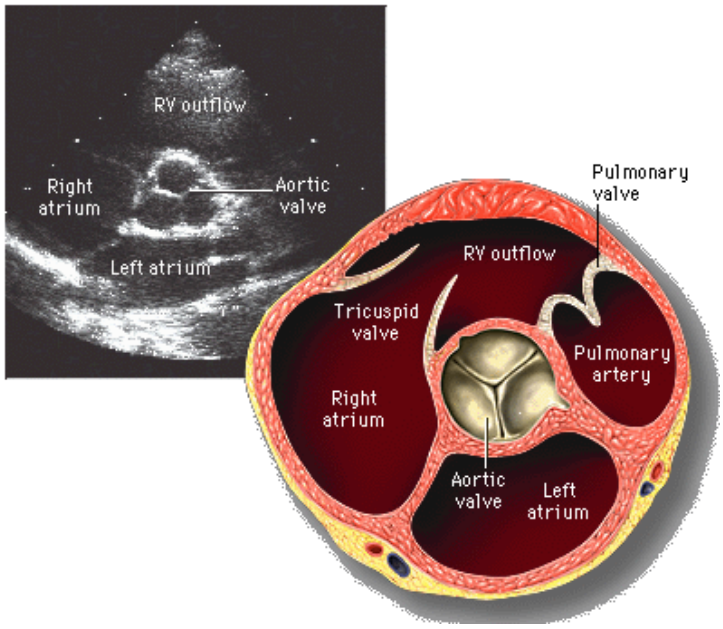


Figure 2.34: Normal parasternal short axis view .(Feigenbaum2010)

2.6.2.1.4.2 At the mitral valve level (Fig. 2.35):

Use 2D to inspect the MV leaflets, mitral annulus and subvalvular apparatus. The anterior and posterior leaflets are visible as is the classical mitral valve orifice, which can be planimetered to measure orifice area **,**assess the mobility of the mitral valve leaflets **,**assess LV radial function and look for any regional

wall motion abnormalities at the basal level **and to** assess RV size and function.(Feigenbaum2010)

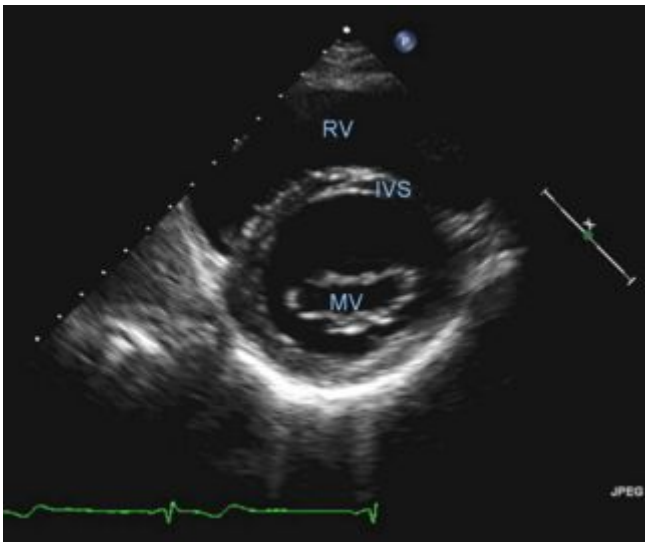


Figure 2.35: Normal parasternal short axis view (mitral valve level) (Andrew, Houghton2002)

2.6.2.1.4.3 At the papillary muscle level (Fig. 2.36):

Use 2D to assess the structure of the posteromedial and anterolateral papillary muscles ,measure LV wall thickness, assess LV radial function and look for any regional wall motion abnormalities at the mid-ventricle level and to assess RV size and function.(Feigenbaum2010)

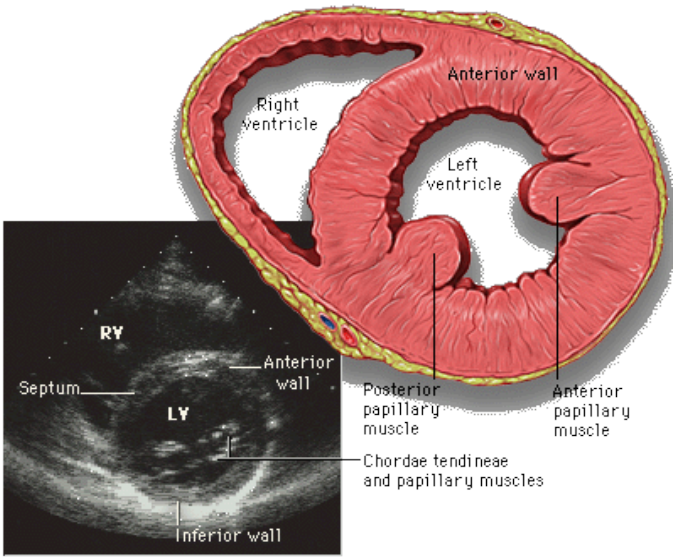


Figure 2.36: Normal parasternal short axis view (papillary muscle level) .(Feigenbaum2010).

2.6.2.2 Right parasternal window :

The right parasternal window is ‘optional’ but can be useful for assessing flow in the ascending aorta. With the patient lying on their right-hand side, place the probe to the right of the sternum in the third intercostal space (some adjustment may be required, as with the left parasternal window) and angle the probe downwards and pointing towards the heart. It is a challenging view, but it may be possible to visualize the ascending aorta and assess colour Doppler within it. This view is most useful for undertaking CW Doppler assessment of the aortic valve, particularly with a standalone pencil probe. .(Feigenbaum2010)

2.6.2.3. Apical window.

The apical window is located at the LV apex. This is normally in the mid-clavicular line and the fifth intercostal space, but may be displaced downwards and to the left if the heart is enlarged. From the apical window a number of views can be obtained. .(Feigenbaum2010)

2.6.2.3.1 Apical 4-chamber view:

To obtain this view, place the probe in the apical position with the ‘dot’ pointing towards the patient’s left. For an optimal view, aim to position the probe exactly at the apex to avoid distortion or foreshortening of the cardiac structures. The interatrial and interventricular septa should be in line with the probe and lie vertically on the screen (Fig. 2.37). .(Feigenbaum2010)

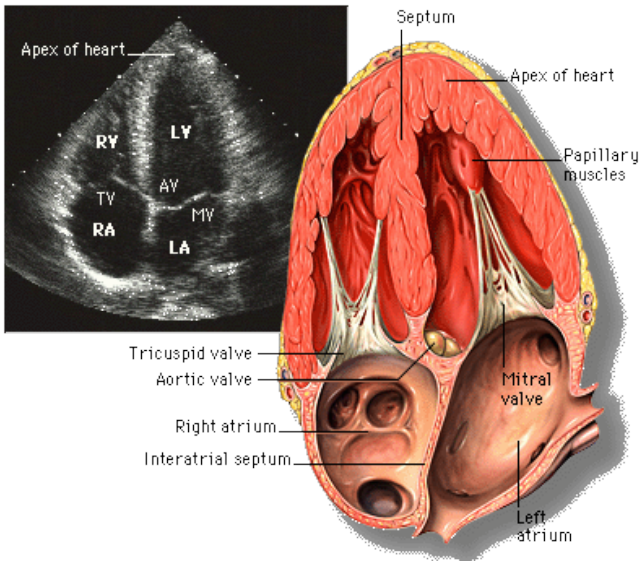


Figure 2.37: Normal apical 4-chamber view.(Feigenbaum2010)

2.6.2.3.2 Modified apical 4-chamber view:

To obtain an optimal view of the right heart, it is best to slightly adjust the standard apical 4-chamber view to centre the right heart on the screen and to ensure that there is no foreshortening. This is known as the ‘modified’ apical 4-chamber view.(Feigenbaum2010)

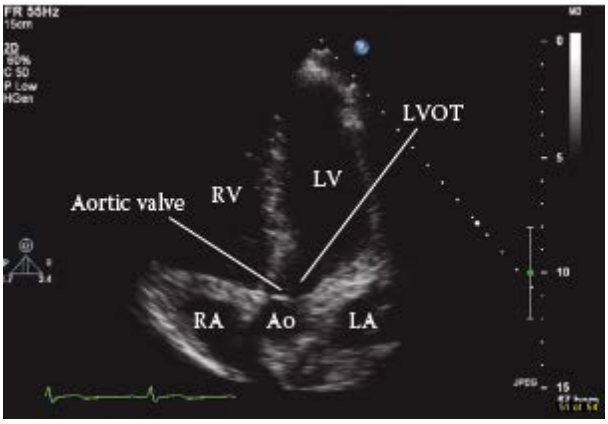


Figure 2.38: Normal apical 5-chamber view(Andrew, Houghton2002)

2.6.2.3.3. Apical 2-chamber view:

Return to the apical 4-chamber view and maintain the same window but rotate the probe about 60° anticlockwise so that the ‘dot’ points approximately towards the patient’s left shoulder. Stop rotating the probe before the LVOT

comes into view, and ensure that the mitral valve is centred in the image (Fig. 2.39). .(Feigenbaum2010)

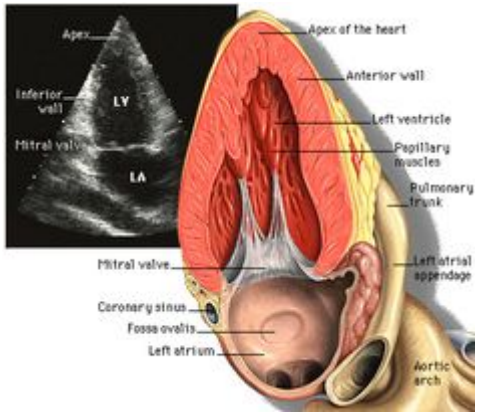


Figure 2.39: Normal apical 2-chamber view.(Feigenbaum2010)

2.6.2.3.4 Apical 3-chamber (long axis) view:

From the apical 2-chamber view, maintain the same window but rotate the probe a further 60° anticlockwise so that the 'dot' now points approximately towards the patient's right shoulder. Stop rotating the probe once the LVOT comes into view, and ensure that the mitral and aortic valves are centred and not foreshortened (Fig. 2.40). This view is the apical equivalent of the parasternal LAX view. .(Feigenbaum2010)

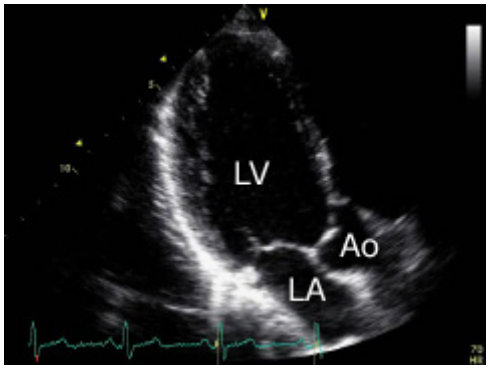


Figure 2.40: Normal apical 3-chamber view(Andrew, Houghton2002)

2.6.2.4. Subcostal window:

2.6.2.4.1 Subcostal long axis view:

The subcostal window is obtained with the patient lying supine with their arms by their sides. It is important that the abdominal wall is relaxed, and asking the patient to lie with their knees bent can help this. Place the probe just below the xiphisternum and angle it up towards the heart, with the ‘dot’ to the patient’s left. From the subcostal window a number of views can be obtained.

To optimize this view, ensure that the interatrial septum is perpendicular to the ultrasound beam (i.e. lies horizontally across the screen) with no foreshortening of the chambers (Fig. 2.41). .(Feigenbaum2010)

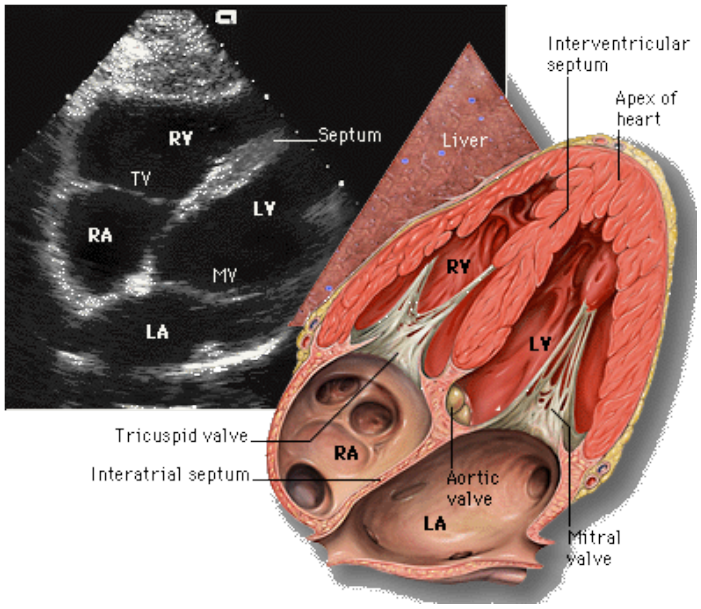


Figure 2.41: Normal subcostal long axis view.(Feigenbaum2010)

2.6.2.4.2 Subcostal short axis view:

Keeping the probe in the subcostal window rotate the probe 90° to obtain a SAX view (Fig. 2.42).

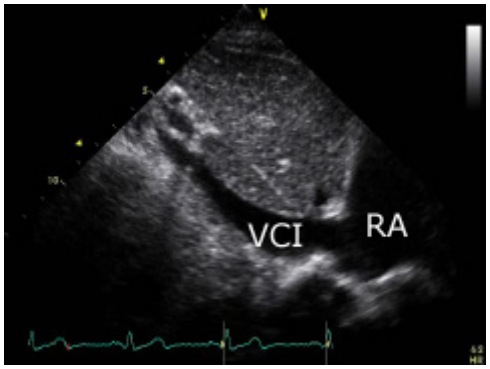


Figure 2.42: Normal subcostal short axis(Andrew, Houghton2002)

2.6.2.5 Suprasternal window:

The suprasternal window is located in the suprasternal notch. Ask the patient to lie supine and to raise their chin. Place the probe in the notch and angle it downwards into the chest. Be mindful that some patients find this uncomfortable. This view shows the aortic arch in LAX (Fig. 2.43). A similar view can, if needed, be obtained from the right supraclavicular position. .(Feigenbaum2010)

2.6.2.5.1 Aorta view:

Use 2D to assess the appearances and dimensions of the aortic arch.

Use colour Doppler to assess flow in the aorta, looking in particular for evidence of coarctation or persistent ductus arteriosus.

Use CW Doppler to: Assess flow in the descending aorta in the presence of a coarctation (it may be better to use a non-imaging ‘pencil’ probe if alignment is difficult using an imaging probe). .(Feigenbaum2010)

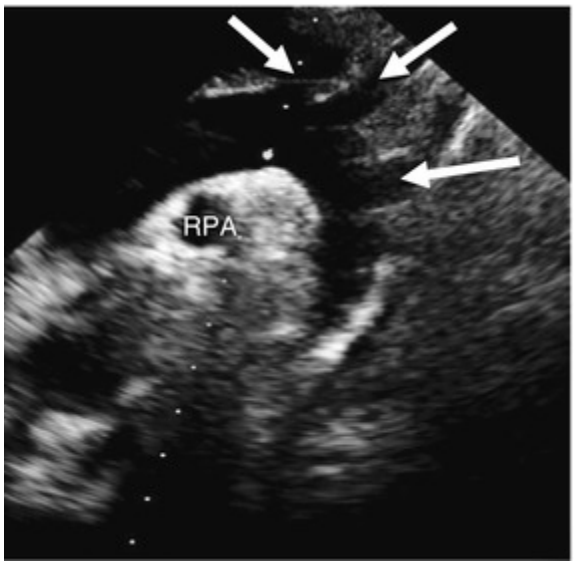


Figure 2.43: Normal suprasternal aorta view(Andrew, Houghton2002)

2.6.3 The transthoracic echo report:

Once you’ve completed the echo study, ensure that the report is written up on the same day. Structure your echo report clearly and systematically, ensuring it contains the Patient identifying and demographic information ,Detailed findings and Study summary. .(Feigenbaum2010)

Previous of studies:

A study done by Cosson S, Kevorkian JP in 2003 and other done by Zarich SW, Nesto RW in 2001was found that Left ventricular diastolic dysfunction represents the first stage of diabetic cardio myopathy preceding systolic function reinforcing the importance of early examination of ventricular function in individual with diabetes. This was in accordance with study done by Schannwell cm et al in 1999 who evaluated 92 type 2 diabetic patients without known cardiac disease and 50 controls with Doppler echocardiography.

He had found that diabetic patients with normal systolic ventricular function suffer a diastolic dysfunction which served as a marker of Diabetes.

Raev DC 1994 Soldatos *et al* in 2011 in their case control study of 55 individuals with type -2 DM found that Diastolic dysfunction, present in a significant proportion of population with Type 2 DM and Boyer et al in 2004 stated that the prevalence of LV diastolic dysfunction in asymptomatic, normotensive patients with type 2 diabetes disease is high. Diastolic dysfunction was found in 75% subjects.

In a study done by Madhumathi R, et al in 2014 it was observed that among the age group of 50 – 59years and 60 – 69years, diastolic dysfunction was almost comparable. There was a linear increase in the prevalence of diastolic dysfunction with the increasing age.

Schannwell et al. in 2002 in their study population of 87 subjects concluded that even young subjects with diabetes mellitus suffer from a diastolic dysfunction, while systolic ventricular function is normal. From the above discussion and comparison of present study findings with various studies, we found that there was high prevalence of diastolic dysfunction in subjects with asymptomatic type 2 DM, and it was correlated with age, duration of diabetes, and LV hypertrophy.

Chapter Three

3. Material and methods

3.1 Materials:

The study intended to evaluation the left ventricular diastolic dysfunction in diapatic patents . This study was done in Omdurman military hospital. The data has been collected from April to September 2016

3.1.1 Subjects:

Study cases were 50 patients (26 males and 24 females) with known history of diabetes mellitus.

3.1.2 Machine used:

All patents where scanned on Mylab40 U/S machine using sector low resolution probe (1.5-7MHZ) is essential when assessing the structures of the heart. Use 2D , PW Doppler and M mode to assess all structures of the heart with measurement.

Figure 3.1: Mylab 40 U/S machine

3.2 Method:

Technique used:

Patients attending for an echo study may feel anxious, not only about having the test itself but also about any abnormalities that it may reveal. To help reduce anxiety, describe the test to patients in clear and reassuring terms – explain to

patients why they are having an echo, whether any special preparation is needed before they attend, what happens during the scan and how long it is likely to take.

Reassure patients that having an echo is safe and painless. Patients can eat and drink normally before attending for a standard TTE, and they can take their medication as usual.

To assess left ventricle inflow, perform PW Doppler in the apical 4-chamber view with a 1–3mm sample volume placed at the tips of the mitral valve leaflets. and M mode to assess all structures of the heart with measurement. Once you've completed the echo study, ensure that the report is written up on the same day. Structure your echo report clearly and systematically, ensuring it contains the Patient identifying and demographic information ,Detailed findings and Study summary.

3.2.2 Data collection:

Data were collected using special designed data collection sheet to cover objective of the study.

3.2.3 Data analysis:

Data were analyzed in frequency distribution; mean and standard deviation were obtained. Crosstabulation between variables was performed. All statistical analysis was performed using SPSS version 19.

Chapter Four

Results

4.1 Results:

Table 4-1 Gender * LVDDF Crosstabulation

			LVDDF				
			Grade 1	Grade 2	Grade 3	Normal	Total
Gender	Female	Count	12	10	1	1	24
		% within Gender	50.0%	41.6%	4.2%	4.2%	100.0%
	Male	Count	7	10	5	4	26
		% within Gender	26.9%	38.5%	19.2%	15.4%	100.0%
Total		Count	19	19	6	6	50
		% within Gender	38.0%	38.0%	12.0%	12.0%	100.0%

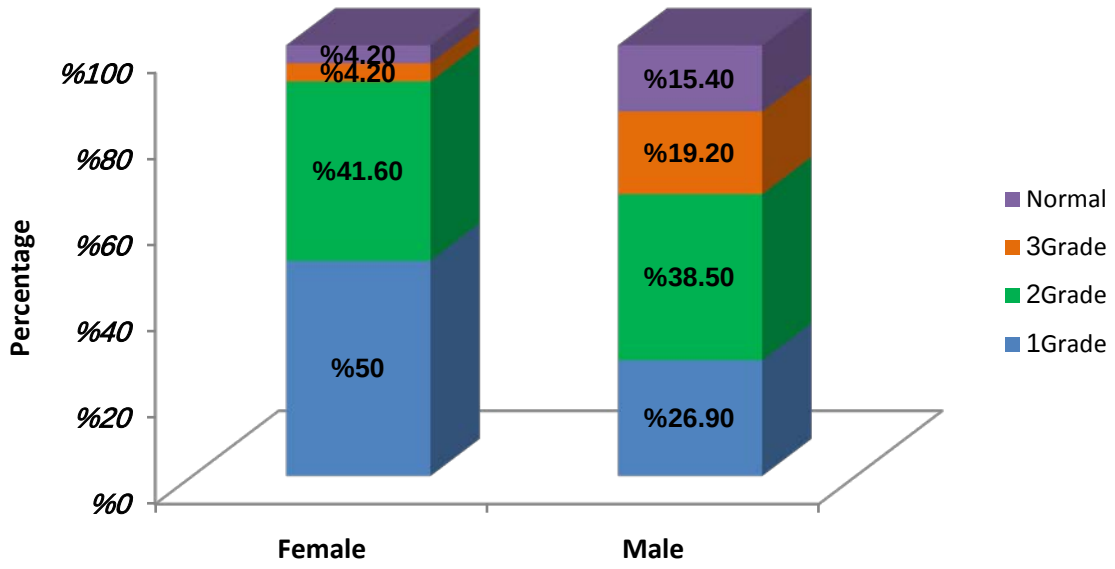


Figure 4.1 LVDDF distributions among gender

Tale 4-1 Age* LVDDF crosstabulation

Age	LVDDF		
	Present	***Absent***	***Total***
36-45	2 (50.0%)	2 (50.0%)	4 (100.0%)
46-55	11 (91.7%)	1 (8.3%)	12 (100.0%)
56-66	16 (88.9%)	2 (11.1%)	18 (100.0%)
67-above	16 (100.0%)	0 (0%)	16 (100.0%)

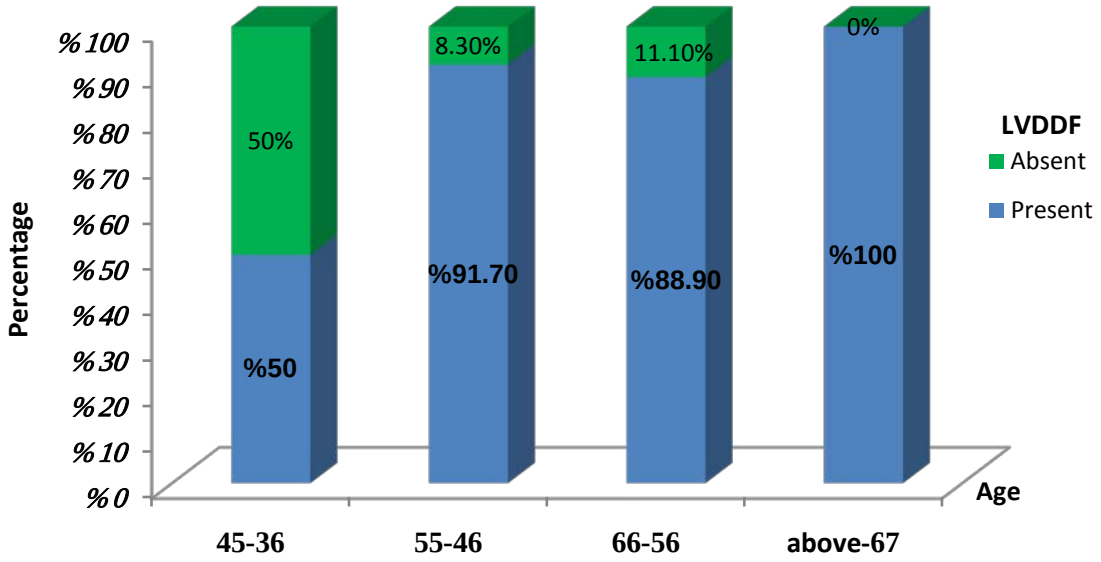


Figure 4.2 LVDDF distributions among age

Table 4.3. LVSF * LVDDF Crosstabulation

			LVDDF				
			Grade 1	***Grade 2***	***Grade 3***	***Normal***	***Total***
LVSF	Good	Count	17	12	6	5	40
		% within LVSF	42.5%	30.0%	15.0%	12.5%	100.0%
	Moderate impaired	Count	1	5	0	0	6
		% within LVSF	16.7%	83.3%	.0%	.0%	100.0%
	Severe impaired	Count	1	3	0	0	4
		% within LVSF	25.0%	75.0%	.0%	.0%	100.0%
Total		Count	19	19	6	6	50
		% within LVSF	38.0%	38.0%	12.0%	12.0%	100.0%

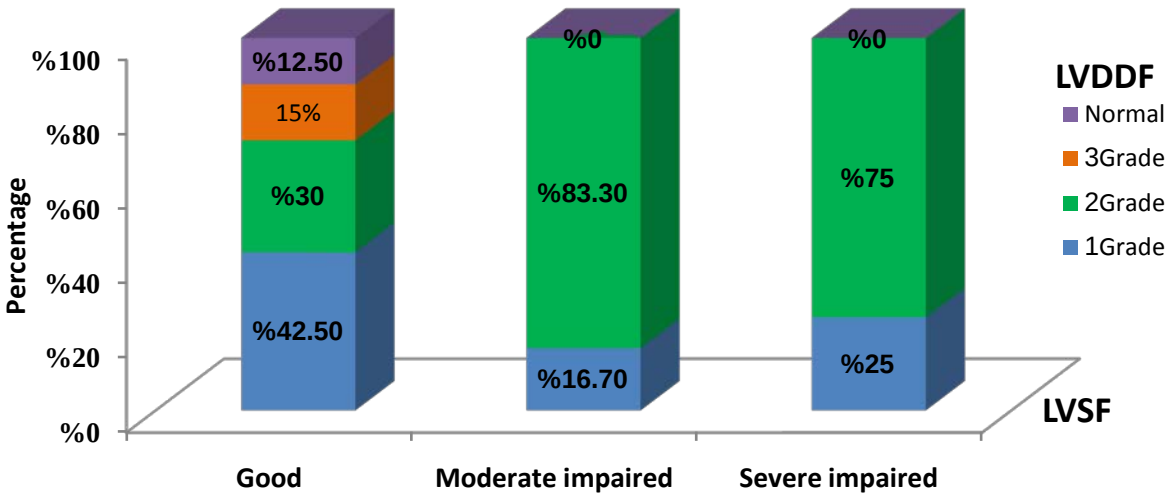


Figure 4.3 LVDDF distributions in relation to LVSF

Table 4.4 Duration of diabetes * LVDDF Crosstabulation

Duration	LVDDF		
	present	***Absent***	***Total***
1-5	12 85.7%	2 14.3	14 100.0%
6-10	16 94.1%	1 5.9%	17 100.0%
11-15	10 83.3%	2 16.7%	12 100.0%
16-above	7 100.0%	0 .0%	7 100.0%

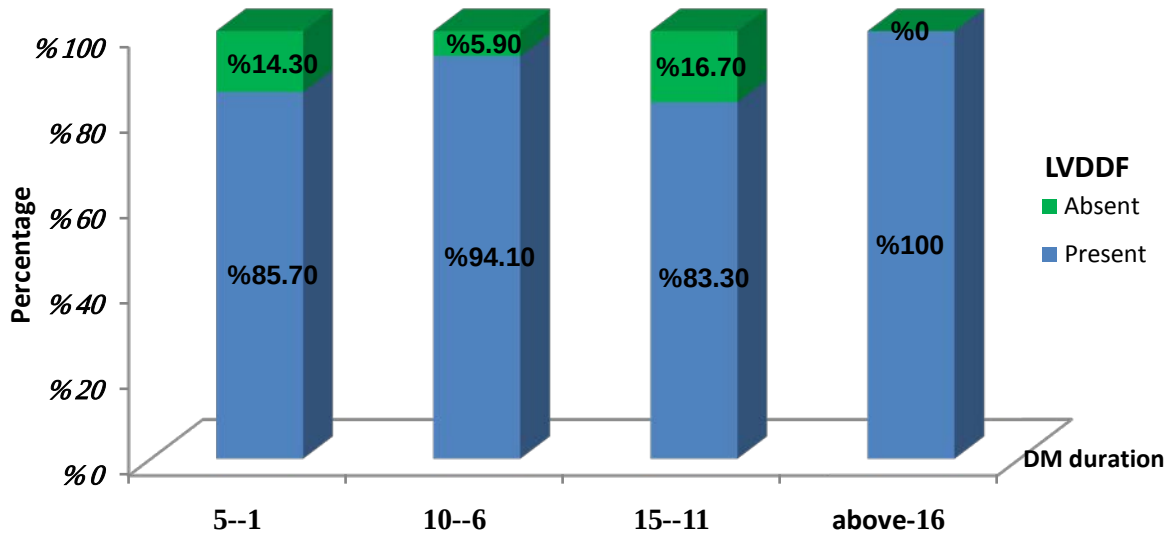


Figure 4.4 LVDDF distributions in relation to DM duration

Table 4.5 LA * LVDDF Crosstabulation

LVDDF / LA	Grade I	Grade II	Grade III	Normal	Total	Percentage
Normal:	18 40%%	16 35.6%	6 13.3%	5 11.1%	45 100.0%	90%
Dilated	1 20.0%	4 80.0%	0 .0%	0 .0%	5 100.0%	10%
Small	0 0%	0 .0%	0 .0%	0 .0%	0 0%	0%

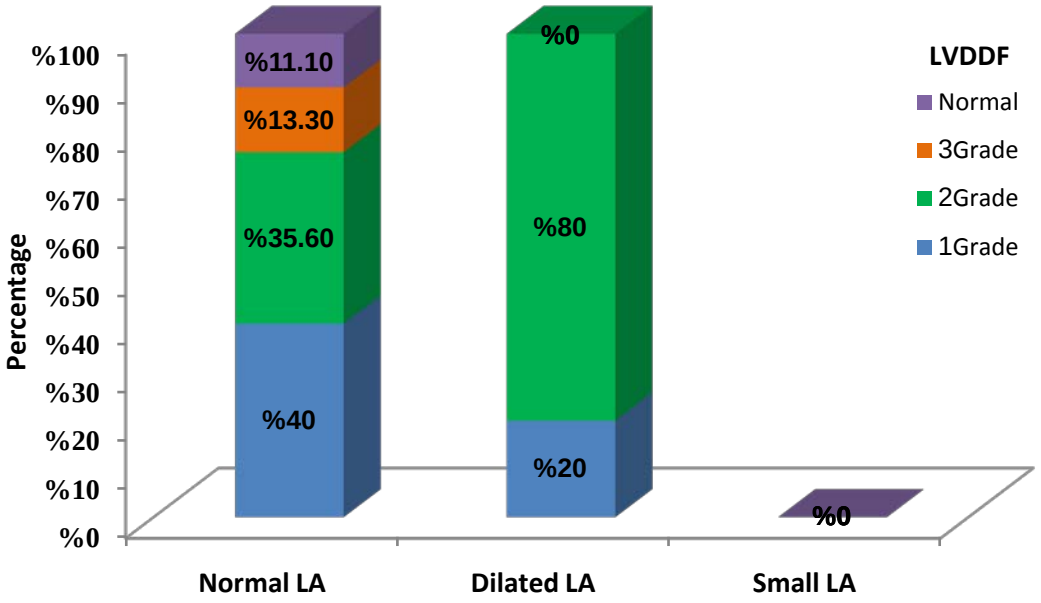


Figure 4.5 LVDDF distributions in relation to LA size

Table 4.6 Lt Ventricular Hypertrophy * LVDDF Crosstabulation

			LVDDF				Total
			Grade 1	Grade 2	Grade 3	Normal	
Lt Ventricular Hypertrophy	Present	Count	12	15	6	3	36
		% within Lt Ventricular Hypertrophy	33.3%	41.7%	16.7%	8.3%	100.0%
	Absent	Count	7	5	0	2	14
		% within Lt Ventricular Hypertrophy	50.0%	28.6%	.0%	21.4%	100.0%
	Total	Count	19	19	6	6	50
		% within Lt Ventricular Hypertrophy	38.0%	38.0%	12.0%	12.0%	100.0%

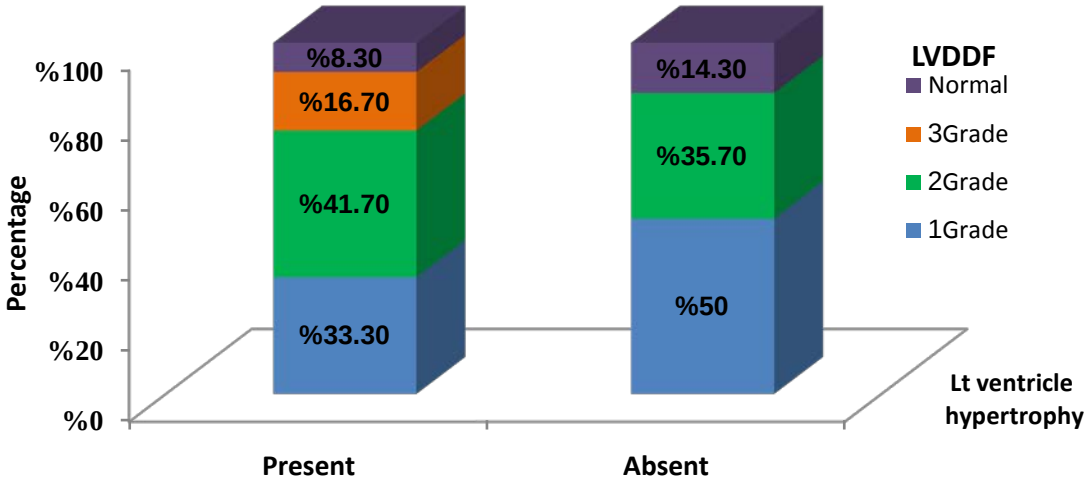


Figure 4.6 LVDDF distributions in relation to Lt Ventricular Hypertrophy

Chapter 5

Discussion, Conclusion and Recommendation

5.1 Discussion:

Left ventricular diastolic dysfunction represents the first stage of diabetic cardio myopathy preceding systolic function reinforcing the importance of early examination of ventricular function in individual with diabetes (Cosson S, Kevorkian JP 2003) (Zarich SW, Nesto RW 2001).

This study consists of 50 patients with type 2 DM, among whom 26 males and 24 females, most of the subjects were between 36 – 93 years of age (Table 1) . Diastolic dysfunction was present in 45 (90%) of the cases amongthem, 23 (95.8%) were fema,les 22 (84.6%) were males. Though statistically not significant, diastolic dysfunction was more prevalent in females. This was accordance with study done by Soldatos et al in 2011 in their case control study of 55 individuals with type -2 DM found that Diastolic dysfunction, present in a significant proportion of population with Type 2 DM and Boyer et al in 2004 stated that the prevalence of LV diastolic dysfunction in asymptomatic, normotensive patients with type 2 diabetes disease is high Diastolic dysfunction was found in 75% subjects.

In this study out of 45(90%) with Diastolic dysfunction maximum prevalence was found in 67- ab0ve years age group. 16(100%) patients in this group had diastolic dysfunction. In patients in the age group 46-55years 11(91.7%) patients of 12 had diastolic dysfunction. Among the age group of 67 – above years , diastolic dysfunction was almost comparable. There was a linear increase in the prevalence of diastolic dysfunction with the increasing age (Table 2). This was agree with study done by Madhumathi R, et al in 2014 it was observed that Among the age group of 50 – 59years and 60 – 69years, diastolic dysfunction was almost comparable. There was a linear increase in the prevalence of diastolic dysfunction with the increasing age.

In this study from40(100%) patents who have good LVSF, the Diastolic dysfunction was present in 35 (87.5%) patents. 6 patients have moderate impaired LVSF with grade 1 and grade 2 diastolic dysfunction , and 4 patents have severe impaired LVSF with 1 patent in grade 1 and 3 patents in grade 2 LVDDF (Table 3). This was in accordance with study done by Schannwell cm et al in 1999 who evaluated 92 type 2 diabetic patients without known cardiac

disease and 50 controls with Doppler echocardiography. He had found that diabetic patients with normal systolic ventricular function suffer a diastolic dysfunction which served as a marker of a Diabetes .

The study comparing with duration of diabetes, we had 14(28%) patients with 1- 5 year duration of diabetes 12(24%) patents had LVDDF, and 17(34%) patients with 6-10 years duration of diabetes with 16(32%) patents whom have LVDDF . Statistically it was significant as we had higher percentage of patients with diastolic dysfunction as duration of diabetes increased (Table 4).this was agree with study done by Virendra C et al in 2011 in their cases a total 127 subjects with type-2 diabetes mellitus (cases) and 100 healthy age and sex matched controls Duration of diabetes mellitus of 11 to 15 years had more prevalence of diastolic dysfunction as compared to the 6 -10 years group.

In this study 90% of cases had normal LA size with the 10% of cases had dilated in LA size , LVDDF , CV disease(IHD ,CCF) and impaired to sever impaired in LVSF , just one patient with good LVSF but it have HHD(table 5). this was agree with study done by Matsuda M in 1996 demonstrated that LA maximal volume increased with increasing severity of DD as defined by invasive hemodynamic study. Thus, it has been hypothesized that CV disease leads to DD, which results in chronic diastolic atrial pressure overload and subsequent LA enlargement. The current findings are supportive of that concept as DD was independently associated with LAVi even when controlling for the presence of CV disease, LV systolic dysfunction, and LV hypertrophy.

In the present study Diastolic dysfunction was prevalent in 45 (90%) of the cases amongthem, 36 (72%) 0f cases had LT ventricular hypertrophy 12 in grade 1 , 15 in grade 2 and 6 in grade 3 . just 3 (8.3%) 0f cases had Lt ventricular hypertrophy without LVDDF14 (28%) of cases who did not have LT ventricular hypertrophy and it was 12 patients who had diastolic dysfunction most patetns in grade 1 LVDDF (7 patents) and 5 patents in grade 2 LVDDF (table 6). this was agree with study done by Sherif F et al in 2009 in

their study say although diastolic dysfunction is not uncommon in patients with normal wall thickness; LV hypertrophy is among the important reasons for it. This study demonstrates that the incidence of diastolic dysfunction is high in type 2 DM subjects. Furthermore, we found that there is a direct correlation between the duration of DM and diastolic dysfunction; and, that significant diastolic dysfunction occurs > 5 years after the onset of DM. Therefore, future studies should be conducted to test the hypothesis that screening and aggressive management of diabetic patients with pre-clinical diastolic dysfunction may delay the progression to heart failure.

5.2. Conclusion:

The prevalence of left ventricular diastolic dysfunction in patients with type 2 DM without significant coronary artery disease is much higher than previously suspected as evidenced by the results of this study and also of similar other studies. LV diastolic dysfunction correlated with duration of diabetes, age, and LVH. Echocardiography with measurements of diastolic functional parameters appears to be a sensitive noninvasive method for evaluating the manifestation and course of early diabetic cardiomyopathy. Early diagnosis and institution of treatment for LVDD in diabetic patients will reduce the morbidity and improve the outcomes by preventing future development of heart failure.

5.3 Recommendation:

The echocardiography is very effective and accurate, it must be used as the first tool.

Improve health through healthy food choices and physical activity.

Making lifestyle changes and taking prescribed medicines can help to prevent or control many risk factors 0f DHD.

Prevent and treat the chronic complications of diabetes. Modify nutrient intake and lifestyle as appropriate for the prevention and treatment of obesity, dyslipidemia, cardiovascular disease, hypertension, and nephropathy.

follow the treatment plan and the doctors advises may help to avoid or delay serious problems, such as a heart attack or heart failure.

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.**Ultrasound images:**

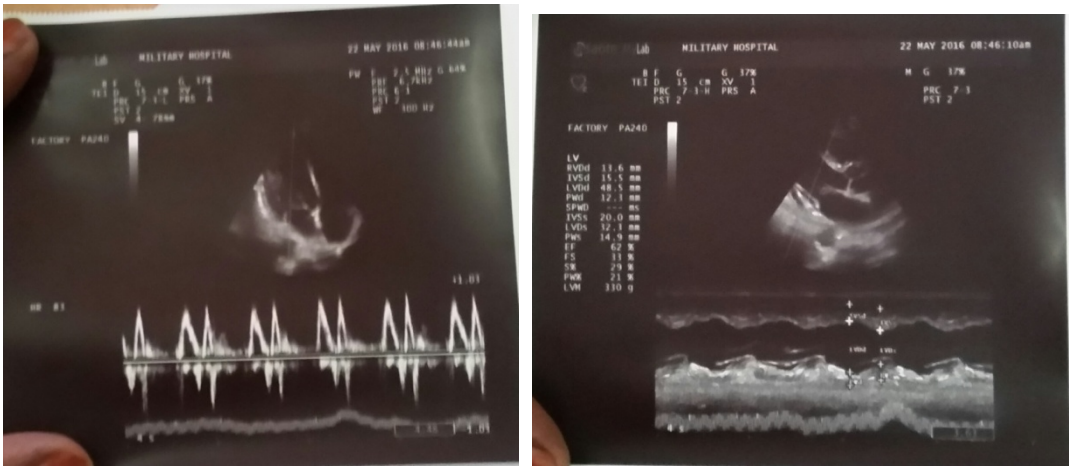

Image 1: female 58, LVDD, LVH

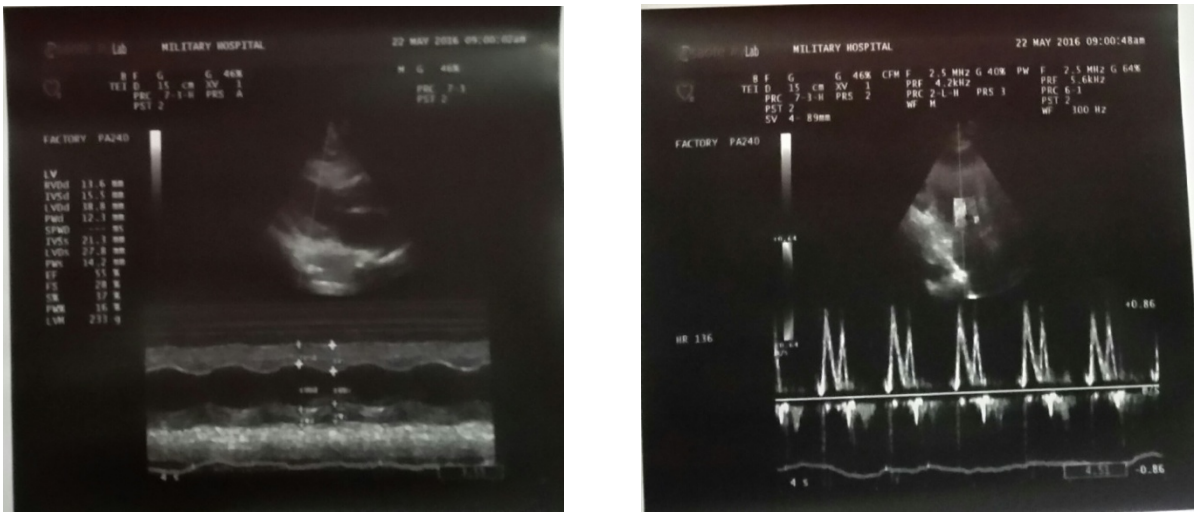

Image 2: female 85, LVDD, LVH

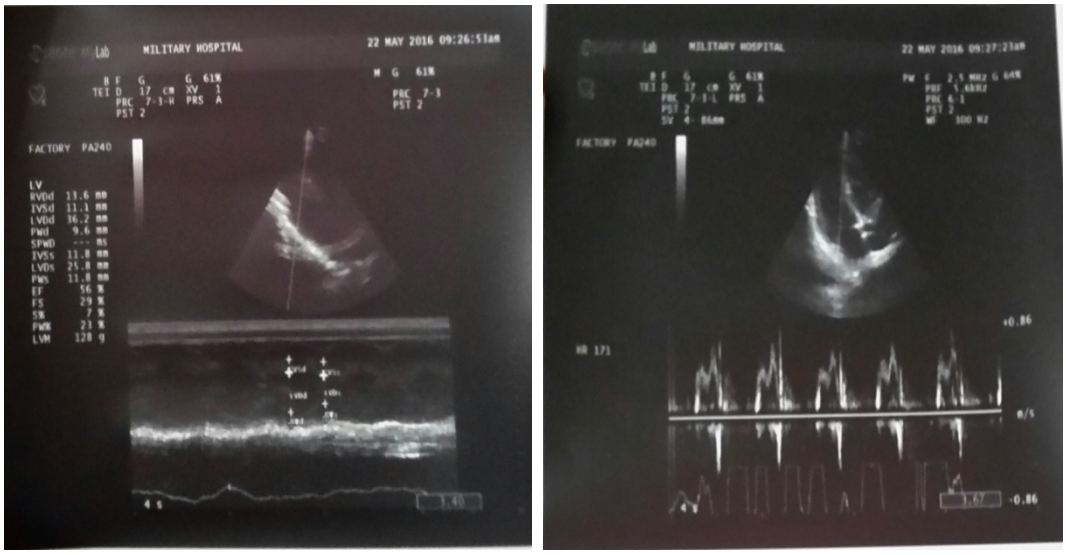

Image 3: male 65, LVDD, LVH

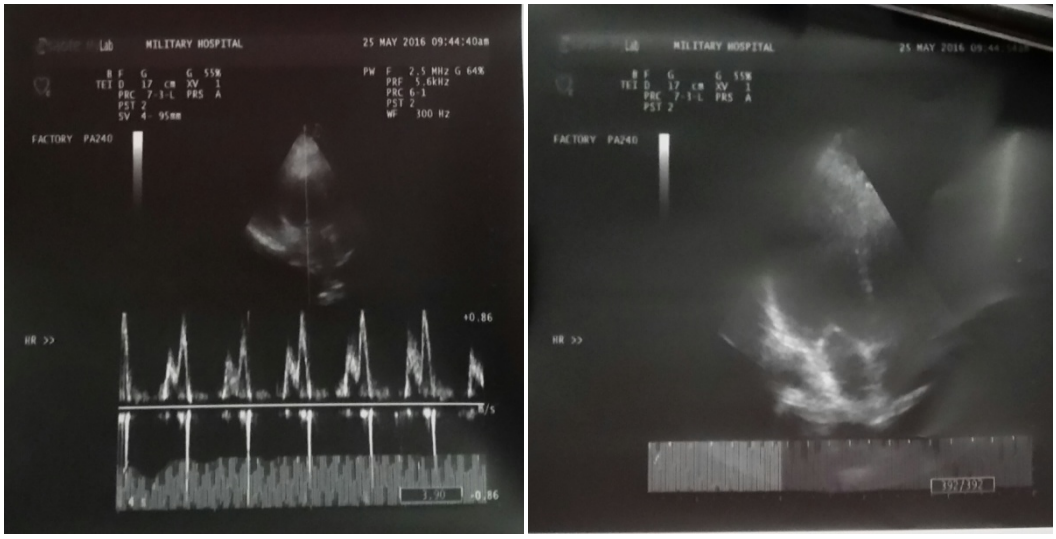

Image : female 55, LVDD

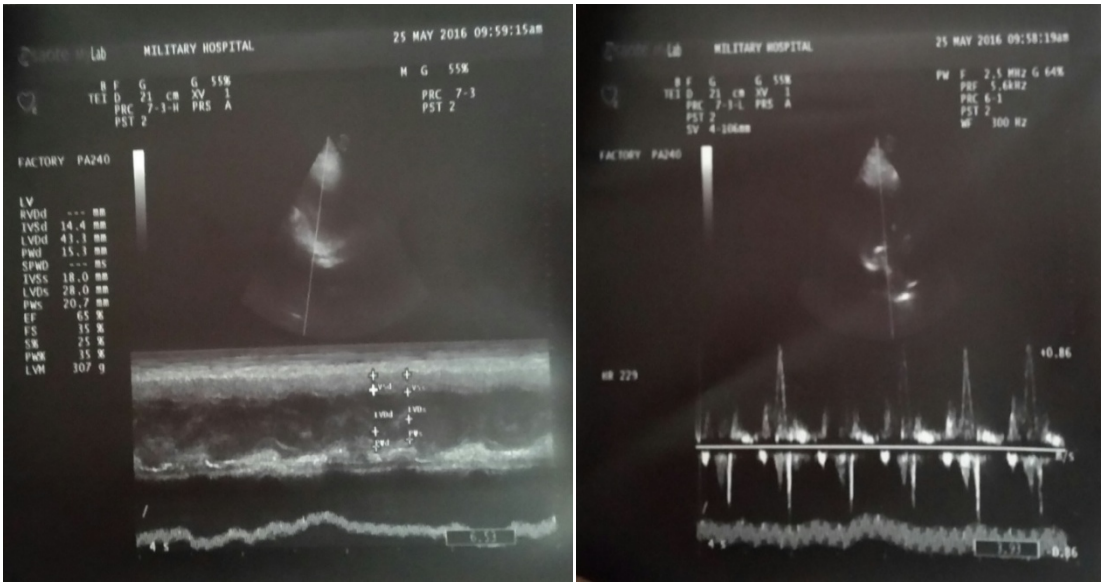

Image 5: male 86, LVDD, LVH

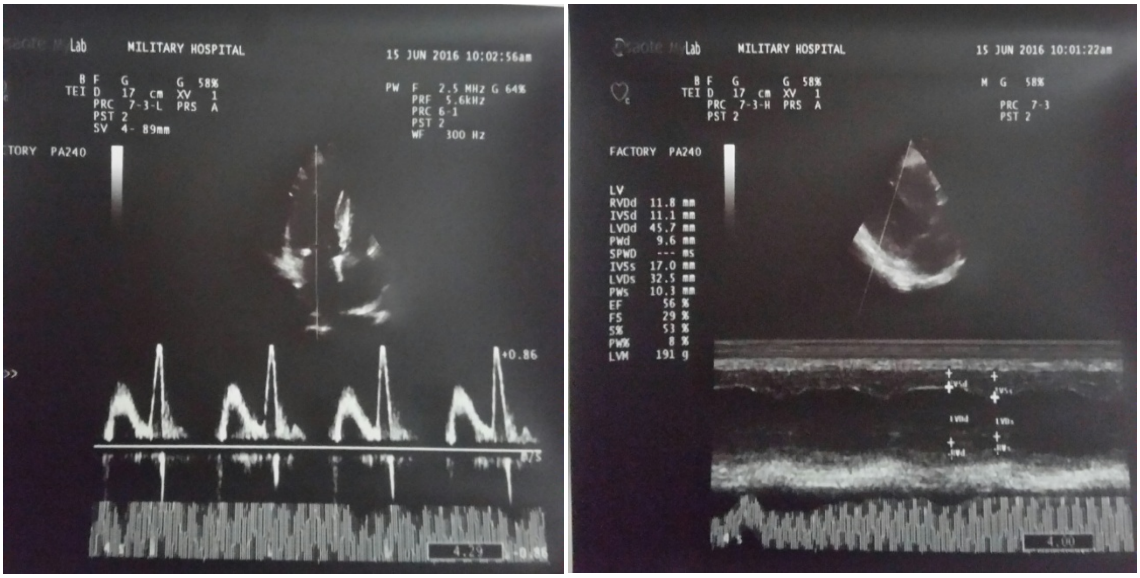

Image 6: male 75, LVDD

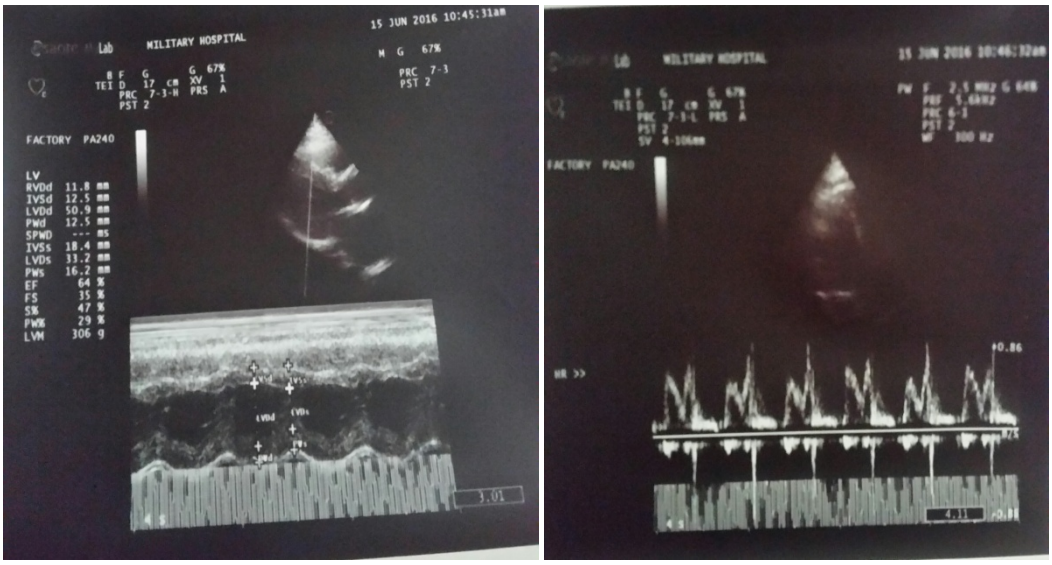

Image 7: male 60, LVDD, LVH

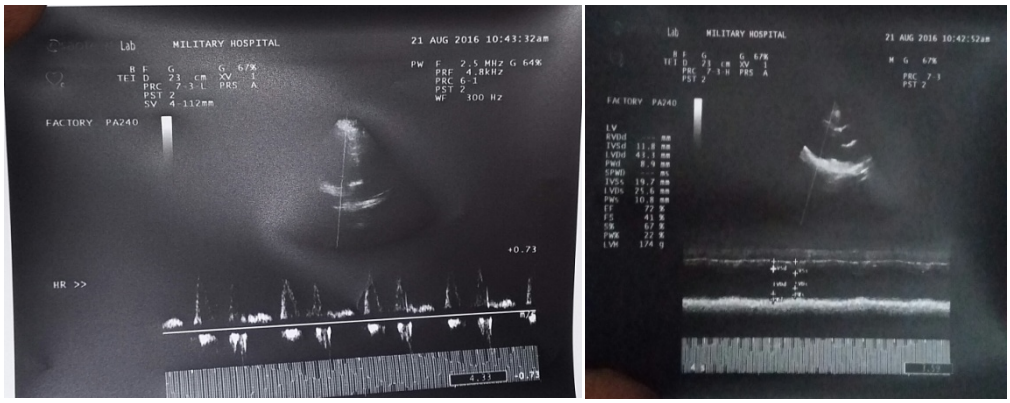

Image 8: male 58

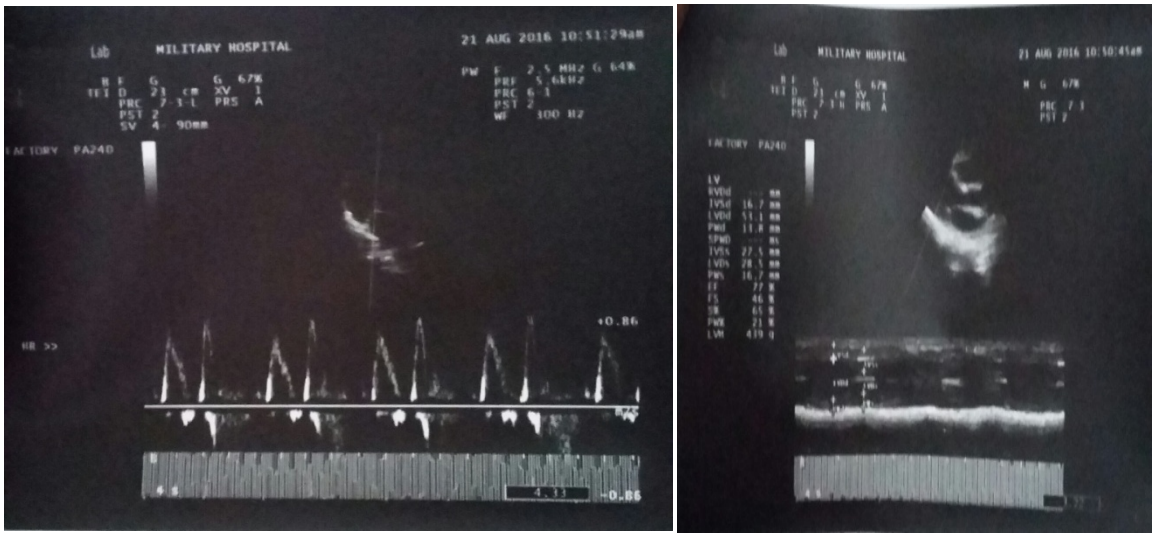

Image 9: male 65, LVDD, LVH

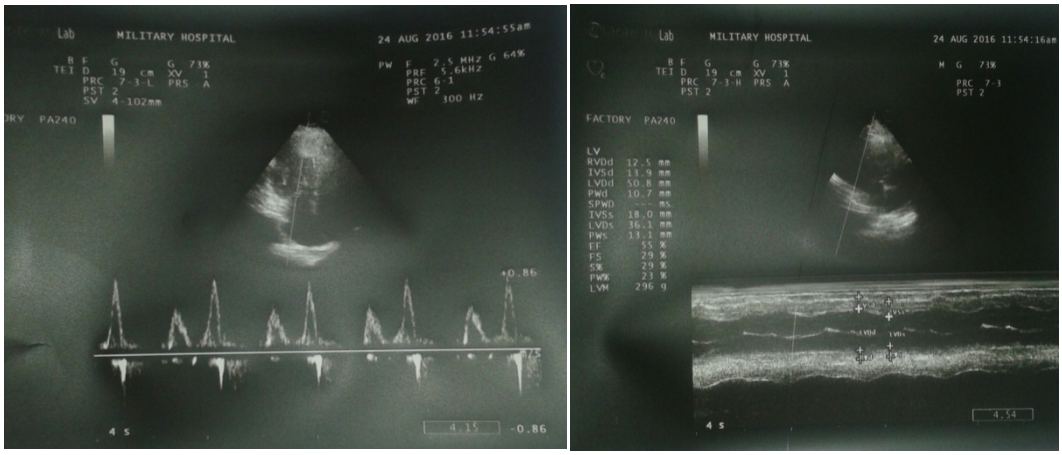

Image 10: female 65, LVDD, LVH