Sudan University of science and technology

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Assessment of Left Ventricular Function in Hemodialysis Patients Using Tran's Thoracic Echocardiography

تقييم وظيفة البطين الأيسر لمرضي الاستصفاء الدموي باستخدام الموجات الصوتية علي القلب عبر الصدر

A thesis Submitted for Fulfillment of the Requirements of PhD Degree in Medical Diagnostic Ultrasonography

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الاية

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

أَوَمَنْ كَانَ مَيْتًا فَأَحْيَيْنَاهُ وَجَعَلْنَا لَهُ نُورًا يَمْشِي بِهِ فِي النَّاسِ كَمَنْ مَثَلُهُ فِي الظُّلُمَاتِ لَيْسَ بِخَارِجٍ مِنْهَا كَذَلِكَ زُيِّنَ لِلْكَافِرِينَ مَا كَانُوا يَعْمَلُونَ (122)

سورة الأنعام

Dedication

To my pain who taught me much.

To my father who did not know me, whose not share me this

Acknowledgement

I would like to express my sincere gratitude to Dr. Mohamed Mohamed Omer Mohamed Yousef and Dr.Mona Abushanab who has given me great advice and help in the whole process of my thesis for their fruitful day to day supervision, guidance, endless help and encouragement that built confidence in my work for their valuable and continuous help, their patience through all the years that made this work possible for giving this opportunity of study, and for endless encouragement and unlimited support. My special thanks to Ms. Islam Mohammed who help much in biostatistics. I extend my thanks to Sinner renal center and Dr. Islam merqany. Dr. Muaaz sidig my colleagues whose help me in data collection. And all staff of the College of Medical Radiological Sciences for their endless moral support and encouragement. I would like to thank everyone who assisted by one way or another to bring this study to the light.

Abstract

The aim of this study was to investigate the value of echocardiography in assessment of the left ventricular functions of long-standing hemodialysis patients. It was done in the renal center in Sinnar Teaching Hospital in echocardiography department using with sector probe of 2.5 mega hertiz. It included 200 cases under regular hemodialysis, using questioner included patient personal data, and echocardiography findings and the data was analyzed using computer programs for analysis (SPSS).The results of this study revealed that according to 99% of the patients had a thick septum, 81% thick posterior wall, 76% dilated cardiomyopathy, 37% impaired ejection fraction, 21% reduced stroke volume, 19% impaired cardiac output, 13% tachycardia, and 20% reduced wall motion. This study cleared that age had negative correlations with Septum, Posterior wall, COP, and a positive correlation with, LVD, EF, FS, and heart rate. In related Weight levels show negative correlations with heart rate, posterior wall, EF, and fractioning shorting, stroke volume and a positive correlation with LVD, and Septum thickness, COP, stroke volume, but the Duration showed negative correlation with LVD, COP, stroke volume, and FS, and positive correlation septum thickness, posterior wall thickness, Ejection fraction, and HR. This study concluded that echocardiography can be used for follow up hearts of these patients and adjust the suitable time for an intervention

الخلاصه

الهدف من هذه الدراسة تقيم وظيفة البطين الأيسر للمرضى المداومين على الاستصفاء الدموي باستخدام الموجات الصوتية للقلب. أجريت الدراسة في مركز الكلى بمستشفى سنار التعليمي بقسم الموجات الصوتية علي القلب وذلك باستخدام جهاز السونار به مسبار تردده الموجي 2.5 ميغاهيرتز. اشتملت الدراسة علي 200 حالة مداومين على الغسيل لفترات متفاوته باستخدام استبيان يحوى البيانات الشخصية للمرضى بالإضافة إلى نتائج الموجات الصوتية للقلب .

أظهرت نتائج الدراسة أن 99% من المرضي لديهم تضخم في الحاجز بين البطينين و 81% تضخم في الجدار الخلفي و 76% توسع في البطين الأيسر الانبساطي و63% قصور في الجزء المدفوع من الدم في القبضة الواحدة، 79% نقص في حجم الدم المدفوع في الضربة الواحدة، 81% نقص في حجم الدم المدفوع في دقيقه. و13% زيادة ي معدل ضربات القلب و 80% نقص في نسبة حركة عضلة القلب. كما أظهرت الدراسة بان عمر المريض له تأثير سلبي علي الحاجز بين البطينين والجدار الخلفي و حجم الدم المدفوع في دقيقه و اثر ايجابي علي عرض البطين الأيسر الانبساطي و نسبة الدم المدفوع في القبضة الواحدة، و معدل ضربات القلب و نسبة حركة عضلة القلب.وان وزن المريض له تأثير سلبي علي معدل ضربات القلب والجدار الخلفي و نسبة الدم المدفوع في دقيقه. و نسبة حركة عضلة القلب، و اثر ايجابي علي عرض البطين الأيسر الانبساطي و حجم المدفوع في القبضة الواحدة وحجم المدفوع في دقيقه و الحاجز بين البطينين. وكما أن عمر المريض في الغسيل الدموي له تأثير سلبي علي عرض البطين الأيسر الانبساطي و حجم الدم المدفوع في دقيقه وحجم المدفوع من الدم في القبضة الواحده و نسبة حركة عضلة القلب و اثر ايجابي علي الحاجز بين البطين والجدار الخلفي و نسبة الدم المدفوع في الضربة الواحدة و معدل ضربات القلب.

خلصت الدراسة أن الموجات الصوتية للقلب لها القدرة على متابعة هذه الحالات و المساعدة في اتخاذ القرارات المناسبة بشأنها

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List of abbreviations

2D	2-dimensional
3D	3-dimensional
ESRD	End-stage renal disease
HD	Hemodialysis
LVD	Left ventricular diameter at end diastole
EF	Ejection fraction
SV	Stroke volume
COP	Heart
HR	Heart rate
CKD	Chronic kidney disease.
CW	Continues waves
PW	Pulsed waves

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CHAPTER ONE

Introduction

1.1 Introduction

Chronic kidney disease is a progressive loss in renal function over a period of months or years. The symptoms of worsening kidney function are non-specific and might include feeling generally unwell and experiencing a reduced appetite *(K/DOQI 2008).*

Chronic kidney disease is identified by a blood test for creatinine, Higher levels of creatinine indicate a lower glomerular filtration rate and as a result a decreased capability of the kidneys to excrete waste products.

There is no specific treatment unequivocally shown to slow the worsening of chronic kidney disease. If there is an underlying cause of chronic kidney disease (CKD), such as vasculitis, this may be treated directly to slow the damage. In more advanced stages, treatments may be required for anemia and bone disease. In stage five CKD; renal replacement therapy is usually required, in the form of either dialysis or a transplant (Levin A et al. 2008). While renal replacement therapies can maintain patients indefinitely and prolong life, the quality of life is severely affected *(De Francisco et al., 2006).*

It is well recognized that dialyzed patients display hugely elevated rates of cardiac mortality (Ramizs et al, 2003). It is also becoming appreciated that this rate of cardiovascular attrition is not driven by the same variety of risk factors, or pathophysiological processes that are important in the general population (Jane et al., 2004). The classical complicated atherosclerotic disease appears not to be the predominant mode of death in hemodialysis (HD) patients. Records from the US Renal Data System have shown that HD is an independent risk factor for the development of both de novo and recurrent heart failure with a two-year mortality after a diagnosis of congestive heart failure as high as 51%*(Emanuel et al., 1999),*

making it one of the most common causes of cardiovascular mortality in this patient group. Also, a significant percentage of cardiac mortality is due to sudden death, and sudden death appears to be temporally related to the dialysis procedure *(K/DOQI 2008),* abnormal ventricular morphology and function appear to be major determinants of cardiac arrhythmias in this patient group.

It has long been suspected that myocardial ischemia may be precipitated by HD. Short intermittent HD treatments exert significant hemodynamic effects, and 20-30% of treatments are additionally complicated by episodes of significant intra-dialytic hypotension *(Bos WJ et al. 2000)*.In conjunction with this, HD patients are particularly susceptible to myocardial ischemia. In addition to the high prevalence of coronary artery atheroma, diabetic dialysis patients have been shown to have a reduced coronary flow reserve in the absence of coronary vessel lesions *(Ragosta et al, 2004)*.HD patients characteristically also exhibit left ventricular hypertrophy, reduced peripheral arterial compliance, impaired microcirculation *(Sigrist et al. 2008),* and ineffective vaso-regulation (in the face of HD coupled with ultrafiltration). All of these factors also predispose to demand cardiac ischemia. This article aims to review the evolving concepts surrounding HD-induced acute, recurrent myocardial ischemia and its contribution to the genesis of the observed constellation of cardiac abnormalities in patients undergoing maintenance HD and longer-term adverse cardiac outcomes.

Echocardiography has evolved into the highly specialized field of ultrasound .it originally began with M-mode techniques and developed into two -, three-, and even four-dimensional imaging combined with Doppler and color flow capabilities. Innovative technical advance, such as Tran's esophageal examination and contrast agent, added yet further diagnostic capability. Echo cardiology serves as an ideal noninvasive method to examine cardiac anatomy in the normal as well as abnormal states. The combinations of anatomical and functional information

provided by echocardiography makes it the diagnostic method of choice in a variety of clinical situation *(Krebs et al., 2004).* The heart is an extremely complex organ, and echocardiography provides a variety of techniques that can be applied to obtain comprehensive information about a very dynamic organ. When performing an echocardiography examination, It is important to consider not only the two – dimensional imaging information but also the Doppler and color flow finding .these techniques are performed as a complement one another *(Krebs et al.,2004* The evaluation of cardiac structures by echocardiography has many important parameters that must be fully understood and used in daily practice. Previously m-mode (time-motion mode) echocardiography was used, and it was regarded as an essential diagnostic tool for the practice of cardiology .The reason for its widespread use was its non-invasive, reproducible, and accurate assessment of cardiac structures in the evaluation of cardiac disease. The M-mode technique is limited, however, in that it provides only a one-dimensional or "icepick" view of the heart. The advent of tow – dimensional echocardiography has allowed cardiac structures to be visualized in a real time fashion. Thus, the echocardiography can now assess intra cardiac lesions , observe contractility , and studies provide an extremely accurate means of evaluating wall thickness, valvular orifice an chamber size, and contractility of the left ventricle (Sandra,2011). To perform a diagnostic echocardiogram examination the sonographer must be aware of anatomic and pathophysiologic parameters of the heart and understand the physical principles of sonography. These parameters are discussed relative to M-mode and two- dimensional technique the standard M-mode examination is presented first, followed by evaluation of the heart by combined two- dimensional and M-mode technique (Sandra, 2011*).*

1.2 Problem of the study:

The life span of ESRD patients become prolong because of hemodialysis and other supportive treatment, so they are reliable to many complications that for their chronic illness and the complication of hemodialysis. Long-term complications of hemodialysis include amyloidosis, neuropathy and various forms of heart disease. Cardiac ischemia, cardiac arrhythmias, and left ventricular systolic and diastolic dysfunction, *(controlled study) (Weinreich T et al. 2006).*This study tries to touch these complications and how to depend on echocardiography in the long term follow up to the hearts of these patients.

1.3. Objectives

1.3.1. General objective:

Evaluation the left ventricular function of long-standing hemodialysis patients by using trans-thoracic echocardiography.

1.3.2. Specific objectives:

- To measure septum and posterior wall diameters of all patients.
- To measure the diameter of the left ventricle during diastole and systole of all patients.
- To measure left ventricle ejection fracture of all patients.
- To measure the stroke volume of all patients.
- To calculate cardiac out- put of all patients.
- To compare these finding of the patients on hemodialysis for one year and two year and so up to five years with each other and with standard measurements.

1.4 Research outline:

The research included five chapters. Chapter one deal with the general introduction about the research, problem statement and the objectives of the study. Chapter two deals with literatures review cover the theoretical background

and previous studies. Chapter three deal with the methodology of the study, including materials, method and equipment. Chapter four will cover the results. And chapter five covers discussion, conclusion, recommendations and references.

Chapter two

Literature review

2.1. Cardiac anatomy:

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

2.1.1 Location and size of the heart:

The heart is located in the thoracic cavity between the lung .This area is called the mediastinum. The base of the cone-shaped heart is uppermost, behind the sternum, and the great vessels enter or leave here. The apex (tip) of the heart points downward and is just above the diaphragm to the left of the midline. This is why we may think of the heart as being on the left side, because the strongest beat can be heard or felt here. *(Scanlon & sanders, 2007).* For all its might, the heart is relatively small, roughly the same size (but not the same shape) as your closed fist. it is about 12 cm (5 in) long , 9 cm (3.5 in) wide at its broadest point , and 6 cm (2.5 inches)thick , with an average mass of 250 g (8 Oz) in adult females and 300 g (10 Oz) in adult males .*(Tortora&Derrickson , 2008)*

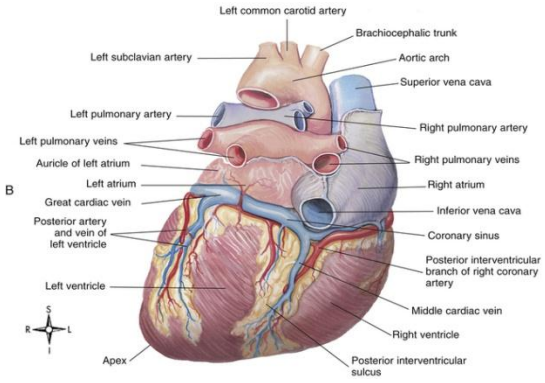

Figure 2.1 External anatomy and feature of the heart. An illustration shows the heart chambers and associated vessels, (*Https://clinicalgate.com*)

2.1.2 Surface of the heart:

The heart has three surfaces: stern o-costal (anterior), diaphragmatic (inferior), and a base (posterior). It also has an apex, which is directed downward, forward, and to the left. *(Snell, 2003).* The sterno-costal surface is formed mainly by the right atrium and the right ventricle, which are separated from each other by the vertical atrioventricular groove. The right border is formed by the right atrium; the left border, by the left ventricle and part of the left auricle. The right ventricle is separated from the left ventricle by the anterior interventricular groove. *(Snell, 2003*). The diaphragmatic surface of the heart is formed mainly by the right and left ventricles separated by the posterior interventricular groove. The inferior surface of the right atrium, into which the inferior vena cava opens, also forms part of this surface. (Snell, 2003), the base of the heart, or the posterior surface, is formed mainly by the left atrium. Into which open the four pulmonary veins. The base of the heart lies opposite the apex. *(Snell, 2003)*. The apex of the heart, formed by the left ventricle, is directed downward, forward, and to the left. It lies at the level of the fifth left intercostal space, 3.5 in (9cm) from the Medline. In the region of the apex, the apex beat can usually be seen and palpated in the living patient. *(Snell, 2003)*

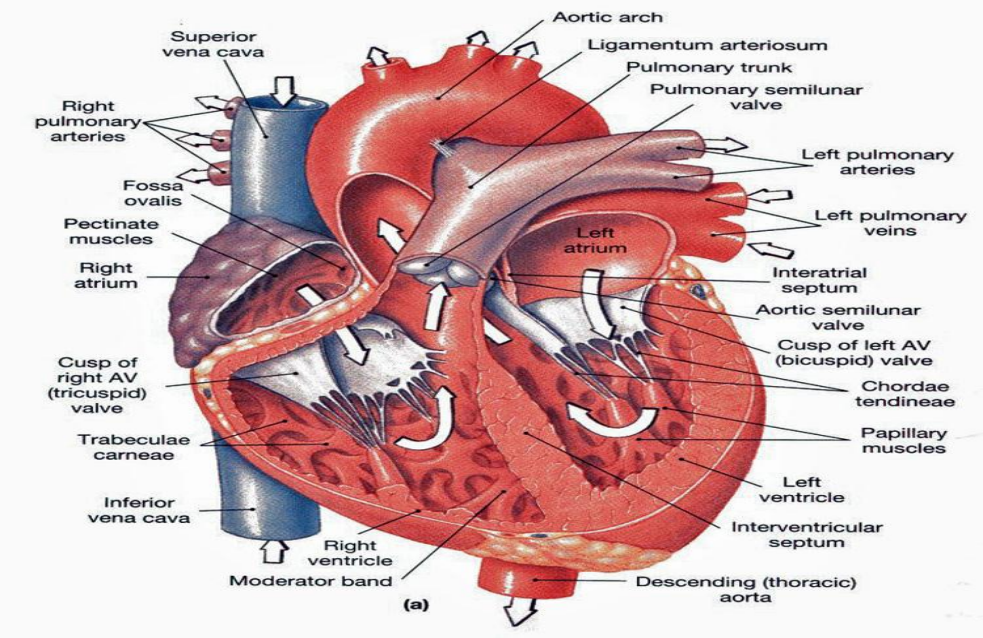

Figure 2.2 External anatomy and feature of the heart.an illustration shows the heart chambers and associated vessels and the base of the heart in a posterior view http://anatomyandphysiologyi.com/heart-anatomy-chambers-vessels-valves/

2.1.3 Border of the Heart:

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

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

The inferior border extend from the lower extremity of the right border to the apex of the heart .this lies in the fifth intercostal space immediately medial to a vertical line dropped through the midpoint of the clavicle (mid clavicular line). this border, formed mainly by the right ventricle, lies at the lower level in the erect than in recumbent posture and in inspiration than in expiration , it is normally slightly concave , but any condition leading to the hypertrophy of the right ventricle , e.g increased pulmonary arterial pressure , makes it convex , giving the heart a globular shape *(Romanes, 2008).* The left border marked by a convex line joining the left ends of the superior and inferior border. It is formed by the left ventricle except for small part formed by the left auricle superiorly. *(Romanes, 2008).* The coronary sulcus lies on a line joining the sternal ends of the third left and sixth right costal cartilage *(Romanes, 2008)* The great orifices of the heart and the valve which guard them lie on a line parallel and slightly inferior to the coronary sulcus. They will be seen when the heart is dissected. The pulmonary orifice lies is posterior to the sternal end of the third costal cartilage. The aortic orifice is posterior to the left margin of the sternum at the level of the third intercostal space. The left atrioventricular (mitral) orifice is posterior to the left half of the sternum at the level of fourth costal cartilage. The right atrioventricular (tricuspid) orifice is posterior to the middle of the sternum at the level of fourth intercostal space *(Romanes 2008)*

2.1.5 Heart chamber:

Right atrium

The right atrium receives the superior vena cava in its upper and posterior part, the inferior vena cava and coronary sinus in its lower part, and the anterior cardiac vein (draining much of the front of the heart) anteriorly Running more or less vertically downwards between the vena cava is a distinct muscular ridge, the crista terminalis (indicated in the outer surface of the atrium by a shallow groove _ the

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

2.1.6 Right ventricle

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

The right ventricle narrows as it passes upward toward the commencement of the pulmonary trunk. The interior of the cavity, whose walls are much thicker than those of the atrium, is thrown into a series of muscular ridges and bundles, the trabeculaecarneae. One of these ridges has broken free and lies in the cavity attached by its two ends to the interventricular septum and the anterior papillary muscle. This is the septo marginal trabecular (formerly the moderator band ; it contain part of the right branch of the conducting bundle . other bundle or bridges of muscle break free from the ventricular wall to form the papillary muscles which are attached to the cusps of the tricuspid valve *(McMinn , 2009)* .

The tricuspid valve guards the right atrioventricular orifice, it has three cusps and admits the tips of three fingers (the mitral valve has two cusps and admits two fingers). The three cusps, called anterior posterior and septal, are attached by their bases to the fibrous atrioventricular ring and are arranged to lie against the three walls of the ventricle _ anterior, inferior and septal. The edges and ventricular surface of the cusps receive the attachment of the chordae tendineae, inelastic cord which diverge from the papillary muscles and prevent the cusps from being everted when ventricle contracts. Usually the large anterior papillary muscle is connected by chordae to the anterior and posterior cusps a smaller posterior

papillary muscle is attached to the posterior and septal and several small papillary muscles join the septal and anterior cusps *(McMinn, 2009).*

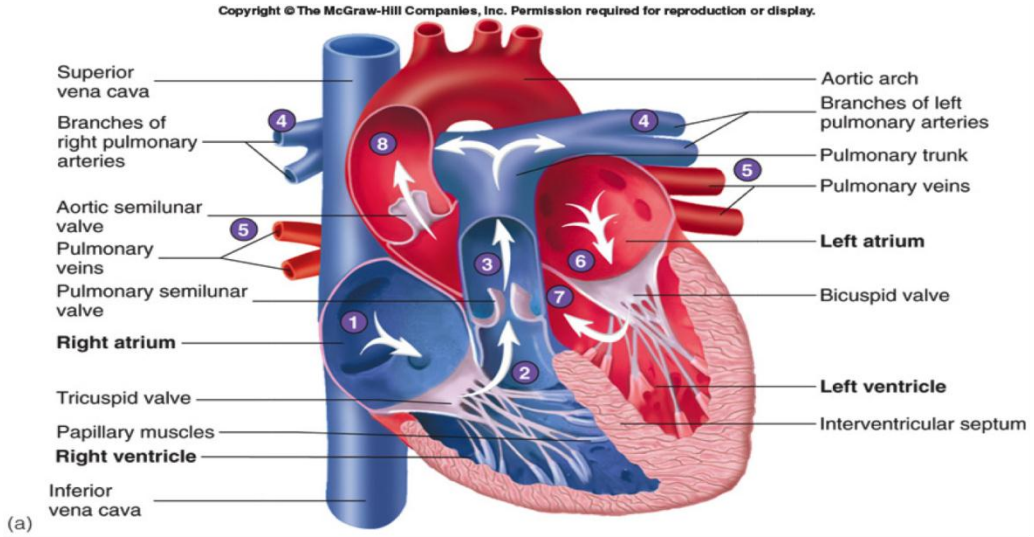

Figure 2.3 internal anatomy of the heart. And illustration reveals the internal structure of the heart including the valve and the musculature of the heart work https://www.pinterest.com/pin/11540542769552457/?lp=true

2.1.7 Left atrium:

Once gas exchange across in the lungs the oxygenated blood travel through the pulmonary veins to the left atrium the smooth posterior wall of the left atrium contains openings for approximately four pulmonary veins sometimes two of this vessels fuse prior to reaching the left atrium thus decreasing the number of openings through the atrial wall . Like the right atrium the left atrium also has pectinate muscle along its anterior wall as well as an auricle. *(Mck inly &o'loughlin , 2008).* Separating the left atrium from the left ventricle is the left atrioventricularopening .this opening is covered by the left atrioventricular (AV) valve (also called the bicuspid valve since it has to triangular cusps) .This valve is also sometimes called the mitral valve, because the two triangular cusps resemble a miter (the headpiece worn by a bishop) .Oxygenated blood, flows from the left atrium through the left atrioventricular opening when the valve is open into the

left ventricle. The left AV valve is forced closed when the left ventricle begins to concentrate preventing blood back flow into the left atrium (*Mck inly &o'loughlin , 2008).*

2.1.8 Left ventricle

The left ventricle is the thickest chamber of the heart, averaging 10 _15 mm (0.4 _ 0.6 in) and forms the apex of the like the right ventricle , the left ventricle contains trabeculaecarneae and has chordae tendineae that anchor the cusps of the bicuspid valve to papillary muscle. *(tortor&gerriickson , 2008).* The walls of this cavity are three times as thick as those of the right ventricle. The interventricular septum bulges into the cavity of the right ventricle, so that in cross section the left ventricle is circular, the right crescentic. Trabeculaecarneae are well developed. There are two papillary muscles, anterior and posterior, the anterior being the large. Both are connected by chordae tendineae to each valve cusp. The posterior cusp receives the chordae on both its margin and its ventricular surface, but since blood is squirted across both surface of the anterior cusp (down through the mitral orifice and up to the aortic) the chordae are attached to it only along its margins the upper and right end of the septal wall is smooth; between the smooth part and the anterior cusp of the mitral valve is the aortic vestibule, which lead up to the aortic orifice *(McMinn, 2009).* The left intraventricular blood pressure is six time higher than that inside the right ventricle *(Snell, 2003).* The interventricular septum lies vertically from side to side across the body: the cavity of the right ventricle lies in front of it and that of the left ventricle behind it. It is marked on the surface of the heart by the interventricular branches of right and left coronary arteries. Its muscle wall, equal in thickness to that of the left ventricle, bulges forward into the cavity of the right ventricle. At its attachment to the fibrous skeleton (conjoined atrioventricular rings) it is thinner and more fibrous. This is the membranous part of the septum, and the aortic vestibule lies between it and

the anterior cusp of the mitral valve *(McMinn, 2009).* The aortic orifice is guarded by the aortic valve, at the entrance to the ascending aorta. It lies at lower level than the pulmonary orifice, rather to its right side, and is more obliquely placed. Its three semilunar cusps are named right, left and posterior (in contrast to the anterior, right and left cusp of the pulmonary valve *(McMinn, 2009).* During fetal life, a temporary blood vessel, called the ductus arteriosus, shunts blood from the pulmonary trunk into the aorta. Hence, only a small amount of blood enters the nonfunctioning fetal lungs .the ductus arteriosus normally closes shortly after birth, leaving a remnant known as the ligamentum arteriosum, which connect the arch of the aorta and pulmonary trunk *(Tortor&Derrickson, 2008).*

2.1.9 Surface markings of valves:

As far as valves are concerned, they all lie behind sternum, marking a line with each other that is nearly vertical .the bases of tricuspid and mitral valves; attached to the atrioventricular ring; are indicated by vertical lines over the lower part of the sternum. The tricuspid valves lies behind the midline of the lower sternum, the mitral valve, over lapping it, lies higher and somewhat to the left. The aortic and pulmonary orifices lie behind the left border of the sternum at the third costal cartilage; the pulmonary is the higher of the two *(McMinn, 2009).*

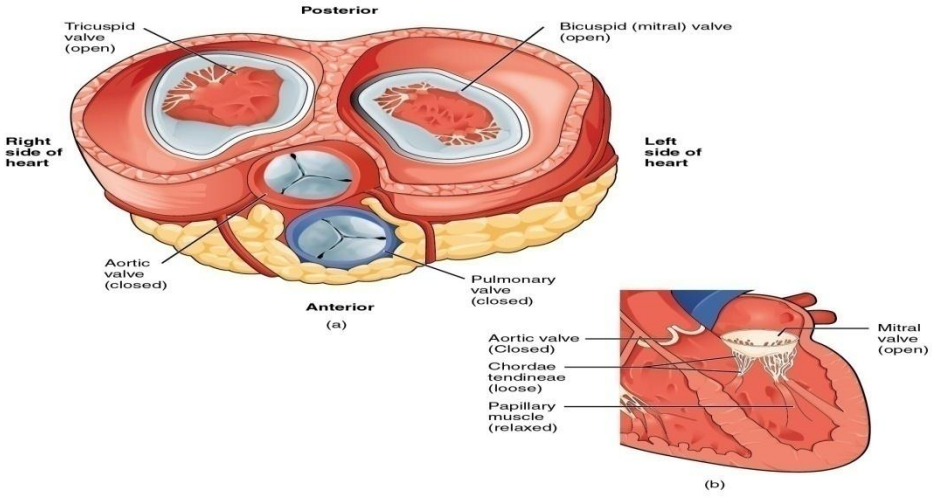

Figure 2.4 Heart valves superior view.

https://opentextbc.ca/anatomyandphysiology/chapter/19-1-heart-anatomy/

2.1.10 Heart wall structure

The heart wall consist of three distinctive layers: and external epicardium, a middle myocardium, and an internal endocardium. The epicardium is outermost heart layer and is also known as the visceral layer of the serous pericardium, the epicardium is composed of a serous membrane and areolar connective tissue. As we age, more fat is deposited in the epicardium, and so this layer becomes thicker and more fatty (McKinley &O'loughlin, 2008). The myocardium is the middle layer of the heart wall and is composed of cardiac muscle tissue. The myocardium is the thickest of the three heart wall layer. It lies deep to the epicardium superficial to the endocardium. The myocardial layer is where myocardial infractions (heart attacks) occur. The arrangement of cardiac muscle in the heart wall permits the compression necessary to pump large volumes of blood out of the heart ((McKinley &O'loughlin, 2008). The internal surface of the heart and the external surfaces of the heart valves are covered by endocardium. The endocardium is composed of a simple squamous epithelium, called an endothelium, and a layer of areolar connective tissue ((McKinley &O'loughlin, 2008).

2.1.11The development of the heart

Development of the heart commences in the third week, when the embryo becomes too large to receive its nutrients through diffusion alone .at this time, the embryo needs its own blood supply, heart, and blood vessels for transporting oxygen and nutrients through its growing body. The steps involved in heart development are complex, because the heart must being working before its development is complete (McKinley &O'loughlin, 2008) +.By day 19 (middle of week 3), two heart tubes (or endocardic tubes) form from mesoderm in the embryo. By day 21, these paired tubes fuse, forming a single primitive heart tube.

This tube develops the following named expansions that ultimately give rise to postnatal heart structures (McKinley &O'loughlin , 2008) . The primitive heart is a single tubes which soon shows grooves demarcating the sinus venosusatrium, ventricle and bulbuscordis from behind forwards. As this tube enlarges it kinks so that its caudal end, receiving venous blood, comes to lie behind its cephalic end with its emerging arteries (Harold, 2006). The sinus venosus later absorbs into the atrium and the bulbus becomes incorporated into the ventricle so that, in the fully developed heart, the atria and great veins come to lie posterior to the ventricles and the roots of the great arteries (Harold, 2006). The boundary tissue between the primitive single atrial cavity and single ventricle grows out as a dorsal and a ventral endocardial cushion which meet in the midline, thus dividing the common atrio-ventricular orifice into a right (tricuspid) and left (mitral) orifice. The division of the primitive atrium into two is a complicated process but an important one in the understanding of congenital septal defects. A partition, the septum Primmum, grows downwards from the posterior and superior walls of the primitive common atrium to fuse with the endocardial cushions. Before fusion is complete, however, a hole appears in the upper part of this septum which is termed the foramen secundum in the septum primum (Harold, 2006).

A second membrane , the septum secundum , then develops to the right of the Primmum but this is never complete ; it has a free lower edge which does , however ,extend low enough for this new septum to overlap the foramen secundum in the septum Primmum and hence to close it (Harold , 2006). The two overlapping defect in the septa form the valve _ like foramen oval which shunts blood from the right to left heart in the fetus. After birth, this foramen usually becomes completely fused leaving only the fossa oval's on the septal wall of the right atrium at its memorial. In about 10 % of adult subjects, however, a probe can still be insinuated through an anatomically patent, although functionally sealed

foramen (Harold, 2006). The primitive sinus venous absorbs into the right atrium so that the vena cava draining into the sinus come to open separately into this atrium. The smooth _walled part of the adult atrium represents the contribution of the sinus venosus, the pectinate part represents the portion derived from the primitive atrium (Harold, 2006). Rather similarly, the adult left atrium has a double origin. The original single pulmonary venous trunk entering the left atrium becomes absorbed into it, and donates the smooth _ walled part of this chamber with the pulmonary veins entering as four separate openings; the trabeculated part of the definitive left atrium is the remains of the original atrial wall (Harold, 2006).

2.1.12 Blood Supply

The arterial supply of the heart:

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

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

The following branches from the right coronary artery supply the right atrium and right ventricle and parts of the left atrium and left ventricle and the atrioventricular septum. (Snell, 2003).

Branches:

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

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

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

4. The posterior interventricular (descending) artery. It supplies branches to the posterior part of the ventricular septum but not to the apical part, which receives its supply from the anterior interventricular branch of the left coronary artery. A large septal branch supplies the atrioventricular node. In 10 % of individuals, the posterior interventricular artery is replaced by branch from the left coronary artery (Snell, 2003).

5. The atrial branches supply the anterior and lateral surfaces of the right atrium. One branch supply the posterior surface of the right and left atria .artery of the sinoatrial node supplies the node and the right and the left atria; in 35 % of individuals it arises from the left coronary artery (Snell, 2003). The left coronary artery, which is usually larger than the right coronary artery, supplies the major part of the heart, including the greater part of the left atrium, left ventricular septum. It arises from the left posterior aortic sinus of the ascending aorta and passes forward between the pulmonary trunk and the left auricle. it then enters the atrioventricular groove and divides into an anterior interventricular branch and circumflex branch (Snell , 2003).

Branches:

The anterior interventricular (descending) branch. Its branch supply the right and left ventricle with numerous branches that also supply the anterior part of the

ventricular septum. One of these ventricular branches (left diagonal artery) may arise directly from the trunk of the left coronary artery. A small left couns artery supply the pulmonary couns (Snell, 2003).

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

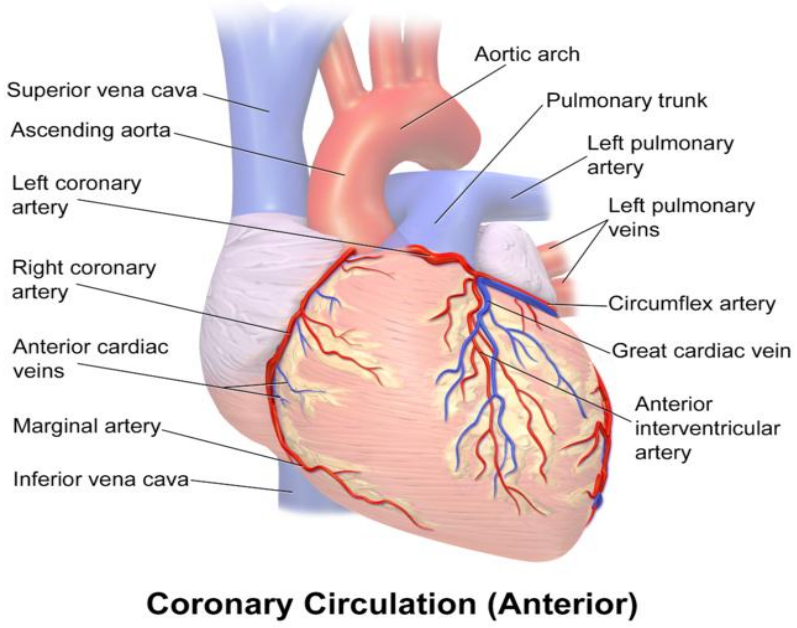

Figure 2.5 coronary circulation .anterior view of (a) coronary arteries

Venous Drainage:

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

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

It receives:

1. The great cardiac vein in the anterior interventricular groove.
2. The middle cardiac vein the inferior interventricular groove.
3. The small cardiac vein __ accompanying the marginal artery along the lower border of the heart.
4. The oblique vein _ descends obliquely on the posterior aspect of the left atrium.

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

Lymph drainage:

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

2.1.13 Nerves supply:

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

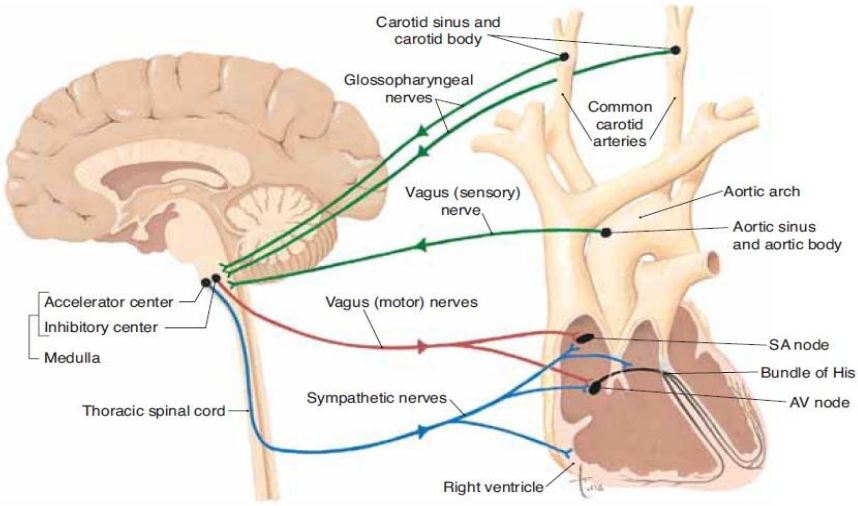

Figure 2.6nervous regulation of the heart. The brain and spinal cord are shown on the left. The heart and major blood vessels are shown on the right http://www.brainkart.com/article/Regulation-of-Heart-Rate_18759

2.2 physiology of the heart

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

AV node, through this node to the bundle of His, and through the branches of the bundle of His via purkinje system to the ventricular muscle (Ganong, 2005).

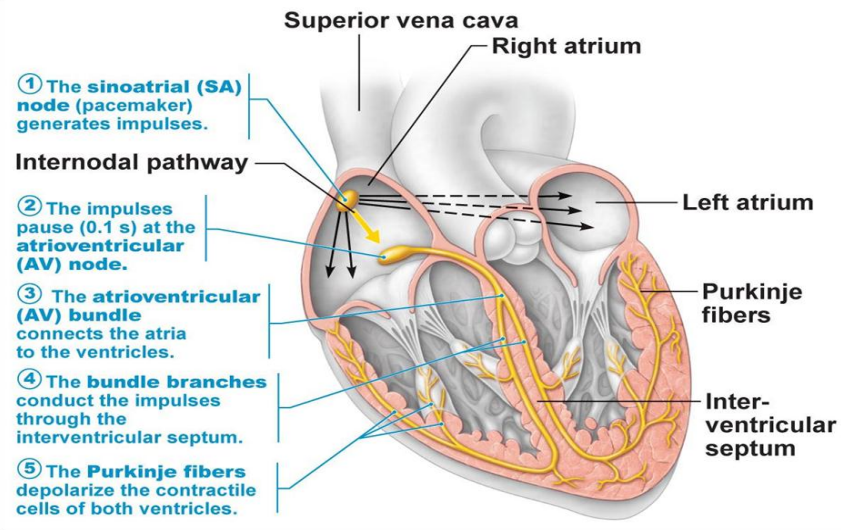

Figure 2.7conduction pathway of the heart. Anterior view of the interior of the heart.

http://anatomyandphysiologyi.com/heart-anatomy-chambers-vessels-valves/

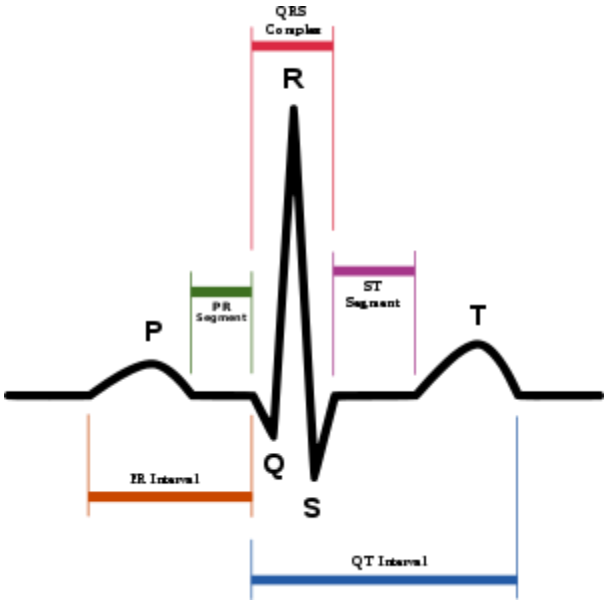

Figure 2.8 The electrocardiogram tracing is of one normal heart beat

2.2.1 Origin and spread of cardiac excitation

Anatomy consideration

In the human heart, the SA node is located at the junction of the superior vena cave with right atrium. The AD node is located in the right posterior portion of the interatrial septum. They are three bundles of atrial fibers that contain purkinje type fibers and connect SA node to the AV node: the anterialinternodal tract of packmen, the middle inter nodal tract of wenchebach , and the posterialinternodal tract of thoael . Conduction also occurs through atrial myocytes, but it's more rapid in this bundles. the AV node is normally is only conducting pathway between the atrium and ventricle , its continuous of the bundle of His , which gives of left bundle branch at the top of the enterventricular septum and continues at a right bundle branch . The left bundle branch divide into an anterial fascicle and posterior fascicle. The branches and fascicle run subindocardialy down either side of the septum and come in contact with purkinje system whose fibers spread to all part of the ventricular myocardium (Ganong, 2005). The conduction system is composed for the most part of modified cardiac muscle that has viewer striations and indistinct boundaries. The SA node and, two a lesser extent, the AV node, also contain small round cells with few organelles, which are connected by gap junctions. These are probably the actual pacemaker cell, and they fore they are called P cells, the atrial muscle fibers are separated from thus of the ventricle by fibrous tissue, a normally the only conducting tissue between the atrium and ventricle is the bundle of His (Ganong, 2005). The heart actually is composed of two syncytium: the atrial syncytium that constituted the walls of the two atriums, and the ventricular syncytium that constitutes the walls of the ventricle. Normally, potential are not conducted from the atrial syncytium into the ventricular syncytium directly through fibers tissue. Instead the conducted only by way of specialized conductive system called the A_V bundle (Guyton, Hall, 2006).

The action potential in cardiac muscle:

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

2.2.2 The cardiac cycle:

The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called the cardiac cycle. Each cycle is initiated by spontaneous generation of an action potential in the sinus node. This node is located in the superior lateral wall of the right atrium near the opening of the superior vena cava, and the action potential travels from here rabidly through both atrial and then through the AV node _ bundle into ventricle, because of this special arrangement of the conducting system from the atria into the ventricle, there is delay of more than 0.1 s during passage on the cardiac impulse from the atria into the ventricle. This allow the atria to contract a head of ventricular contraction they by pumping blood into the ventricle before the strong ventricular contraction begins. Thus, the atria at as primer pumps for the ventricle, and the ventricle in turn provide the major source of power for moving blood through the body's vascular system (Guyton, Hall, 2006).

2.2.3 Mechanical Events of the cardiac cycle

2.14.1 Diastole and systole:

The cardiac cycle consist of a period of relaxation called diastole, during which the heart fills with blood, followed by a period of contraction called systole. The top three curves show the pressure changes in the aorta, left ventricle, and left

atrium, respectively. The fourth curve depicts the changes in the left ventricular volume, the fifth the electrocardiogram, and the sixth a phonocardiogram, which is a recording of the sound produced by the heart __ mainly by the heart valve __ as it pumps (Guyton, Hall, 2006).

Events in the late diastole

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

Atrial systole and diastole, the p wave of the electrocardiogram (ECG) reflect atrial depolarization, which initiates atrial systole. Contraction of the atria "tops off" ventricular filling with a final, small volume of blood from the atria, producing the wave. Under resting conditions , atrial systole is not essential for ventricular filling and , In its absence , ventricular filling is only slightly reduced .However when increased cardiac output is required , as during exercise , the absence of atrial systole can limit ventricular filling and stroke volume . This happens in patients with atrial fibrillation, whose atria do not contract synchronously. The P wave is followed by and electrically quit period, during which atrioventricular (AV) node transmission occur (the PR segment) .During this electrical pause, the mechanical events of atrial systole and ventricular filling are concluded before excitation and contraction of the ventricle begin. Atrial diastole follows atrial systole and occurs during ventricular systole. As the left atrium relaxes, blood inter atrium from the pulmonary veins, simultaneously, blood enters the right atrium from the superior and inferior vena cava . The gradual rise in left atrial pressure during atrial diastole produces the V wave and

reflects its filling. The small pressure oscillation early in atrial diastole , called the C wave ,is caused by bulging of the mitral valve and movements of the heart associated with ventricular contraction(Rhades& Tanner , 2003).

Ventricular systole

At the start of ventricular systole, the mitral and tricuspid (AV) valves close. Ventricular muscle initially shortens relatively little, but intraventricular pressure rises sharply as the myocardium presses on the blood in the ventricle. This period of iso volumetric (iso volumetric, isometric) ventricular contraction lasts about 0.05 s ,until the pressure in the left and right ventricle exceed the pressure in the aorta (80 mm Hg , 10.6 Kpa) and pulmonary artery (10 mm Hg) and the aortic and pulmonary valves open , During iso volumetric contraction , the AV valves bulge into the atria , causing a small but sharp rise in atrial pressure (Ganong , 2005).

When the aortic and pulmonary valves open, the phase of ventricular ejection begins. Ejection is rapid at first, slowing down as systole progresses. The intraventricular pressure rise to a maximum and then declines somewhat before ventricular systole ends. Peak left ventricular pressure is about 120 mm Hg, and peak right ventricular pressure is 25 mm Hg or less. Late in systole, the aortic pressure actually exceeds the ventricular, but for a short period momentum keeps the blood moving forward. The Av valves are pulled down by the contraction of the ventricular muscle, and atrial pressure drops. The amount of blood ejected by each ventricle per stroke at rest is 70 _ 90 ml. The end diastolic ventricular volume is about 130 ml, thus about 50 mL of blood remains in each ventricle at the end of systole (end systolic ventricular volume), and the ejection fraction, the percent of the end diastolic ventricular volume that is ejected with each stroke, is about 65%. The ejection fraction is a valuable index of ventricular fraction , it can be measured by injecting radionuclide _ labeled red blood cells ,imaging the

cardiac blood pool at the end of diastole and the end of systole (equilibrium radionuclide angiocardiography), and then calculating the ejection fraction (Ganong, 2005).

Early Diastole

Once the ventricular muscle is fully contracted, the already falling ventricular pressure drop more rapidly. This is the period of proto diastole .it lasts about 0.04 s .it ends when the momentum of the ejected blood is overcome and the aortic and pulmonary valves close , setting up transient vibrations in the blood and blood vessel walls , After the valves are closed , pressure continues to drop rapidly during the period of iso volumetric relaxation . iso volumetric relaxation ends when the ventricular pressure falls below the atrial pressure and the AV valves open , permitting the ventricles to fill . Filling is rapid at first, then slows as the next cardiac contraction approaches. Atrial pressure continues to rises after the end of ventricular systole until the AV valves open, then drops and slowly rises again until the next atrial systole (Ganong, 2005).

2.2.4The electrocardiogram

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

Function of the Atria as primer pumps

Blood normally flows continually from the great veins into the atria; about 80 percent of the blood flows directly through the atria into the ventricles even before the atria contract. Then, atrial contraction usually causes an additional 20 percent filling of the ventricle. Therefore, atria simply function as primer pumps that increase the ventricular pumping effectiveness as much as 20 percent. However,

the heart can continue to operate under most condition even without this extra 20 percent effectiveness because it normally has the capability of pumping 300 to 400 percent more blood than is required by the resting body. Therefore, when the atria fail to function, the difference is unlikely to be noticed unless a person exercises; then acute signs of heart failure occasionally develop, especially shortness of breath (Guyton & Hall, 2006). The wave is caused by atrial contraction .Ordinarily, the right atrial pressure increases 4 to 6 mm Hg during atrial contraction, and the left atrial pressure increases about 7 to 8 mmHg. The C wave occurs when the ventricles begin to contract; it is caused partly by slight backflow of blood into the atria at the onset of ventricular contraction but mainly by bulging of the AV valves backward toward the atria because of increasing pressure in the ventricles (Guyton & Hall, 2006). The V wave occurs toward the end of ventricular contraction; it results from slow flow of blood into the atria from the veins while the AV valves are closed during ventricular contraction. Then, when ventricular contraction is over, the AV valves open, allowing this stored atrial blood to flow rapidly into the ventricles and causing the V wave to disappear (Guyton & Hall, 2006).

Function of the ventricles as pumps

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

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

(Guyton & Hall, 2006).

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

End _Diastolic volume, End _ Systolic volume, and stroke volume out put

During diastole, normal filling of the ventricles increases the volume of each ventricle to about 110 to 120 milliliters. This volume is called the end _diastolic volume. Then, as the ventricles empty during systole, the volume decreases about 70 milliliters, which is called the stroke volume output. The remaining volume in each ventricle, about 40 to 50 milliliters, is called the end _ systolic volume. The fraction of the end _diastolic volume that is ejected is called the ejection fraction __ usually equal to about 60 percent .when the heart contracts strongly , the end _

systolic volume can be decreased to as little as 10 to 20 milliliters . conversely , when large amount of blood flow into the ventricles during diastole , the ventricular end diastolic volumes can become as great as 150 to 180 milliliters in the healthy heart , by both increasing the end _diastolic volume and decreasing the end _ systolic volume , the stroke volume output can be increased to more than double normal (Guyton & Hall , 2006).

Also, because of the rapid closure and rapid ejection, the edges of the aortic and pulmonary valves are subjected to much greater mechanical abrasion than are the A_V valves .Finally, the A_V valves are supported by the chordae tineae, which is not true for the semilunar valves. It is obvious from the anatomy of the aortic and pulmonary valves that they must be constructed with an especially strong yet very pliable fibrous tissue base to withstand the extra physical stresses (Guyton & Hall, 2006).

2.2.5 Cardiac outputs.

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

Regulation of Heart pumping

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

Ventricular function curves

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

Control of the heart by the sympathetic and parasympathetic nerves

The pumping effectiveness of the heart also is controlled by the sympathetic and parasympathetic (vagus) nerves, which abundantly supply the heart. For given levels of input atrial pressure, the amount of blood pumped each minute (cardiac output) often can be increased more than 100 percent by sympathetic stimulation.

By contrast, the output can be decreased to as low as zero or almost zero by vagal (parasympathetic) stimulation (Guyton & Hall, 2006).

Parasympathetic (vagal) stimulation of the heart. Strong stimulation of the parasympathetic nerve fibers in the vagus nerves to the heart can stop the heartbeat for a few seconds, but then the heart usually "escapes" and beats at a rate of 20 to40 beats per minute as long as the parasympathetic stimulation continues. In addition, strong vagal stimulation and decrease the strength of the heart muscle contraction by 20 to 30 percent. The vagal fibers are distributed mainly to the atria and not much to the ventricle, where the power contraction of

the heart occurs. This explains the effect of vagal stimulation mainly to decrease heart rate rather than to decrease greatly the strength of the heart contraction. Never the less, the great decrease in the heart rate combined with slight decrease in the heart contraction strength can decrease ventricular pumping 50 percent or more (Guyton& Hall, 2006).

2.22 increasing the arterial pressure load (up to the limit doesn't decrease the cardiac output) Increasing the arterial pressure in the aorta doesn't decrease the cardiac output until the main arterial pressure rise above 160 mm Hg. in the other words, during normal function of the heart at normal systolic arterial pressure (80 to 140 mm Hg) , the cardiac output is determined almost entirely by the ease of blood flow through the body's tissue , which intern controls veins return of the blood to the heart (Guyton & Hall , 2006).

2.3 Pathology of the heart

Cardiovascular disease (CVD) is the leading cause of death in men and women in the United States. Because of economic advances, social structures, and demographics ,it is Predicted that CVD will become the leading cause of death worldwide by 2020,surpassing infectious diseases .it is estimated that the direct and indirect costs of CVD in the United States alone were $44805 billion for 2008 to reduce this increase in morbidity , mortality, and cost, strategies such as population-based public health measures ,preventative programs for high-risk subgroups, and the allocation of resources for treatments for CVD can be useful (Porth& mattfin,2009).

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

- Failure of the pump.in the most common circumstance, the cardia muscle contracts weakly or inadequately,and the chambers cannot empty properly.in

some conditions ,however, the muscle cannot relax sufficiently to permit ventricular filling (Kumar,etal,2005).

- An obstruction to flow, owing to a lesion preventing valve opening or otherwise causing increased ventricular chamber pressure (e.g., aortic valvar stenosis, systemic hypertension, or aortic coarctation). The increased pressure overworks the chamber that pumps against the obstruction (Kumar, et al, 2005).
- Regurgitant flow causes some of the output for each contraction to flow backward, adding a volume workload to each of the chambers, which must pump the extra blood (e.g., left ventricle in aortic regurgitation: left atrium and left ventricle in mitral regurgitation) (Kumar, etal, 2005).
- Disorders of cardiac conduction. heart block or arrhythmias owing to uncoordinated generation of impulses (e.g., atrial or ventricular fibrillation) lead to non-uniform and inefficient contractions of the muscular walls
- Disruption of the continuity of the circulatory system that permits blood to escape (e.g., gunshot wound through the thoracic aortic) (Kumar, et al,2005).

Renal system: introduction:

The urinary system consists of two kidneys, two ureters, the urinary bladder, and urethra (fig 2.1). The formation of urine is the function of the kidneys, and the rest of the system is responsible for eliminating the urine.

Body cells produce waste products such as urea, creatinine, and ammonia, which must be removed from the blood before they accumulate to toxic levels. As the kidneys form urine to excrete these waste products, they also accomplish several other important functions:-

1. Regulation of the volume of blood by excretion or conservation of minerals.
2. Regulation of the electrolyte content of the blood by the excretion of minerals.

3. Regulation of acid-base balance of the blood by excretion or conservation of ions such as H_ ions or HCO_ ions.

4. Regulation of all of the above in tissue fluid.

The process of urine formation, therefore. Helps maintain the normal composition, volume, and PH of both blood and tissue fluid by removing those substance that would upset the normal constancy and balance of these extracellular fluid (valeriec. Scanlon, 2007).

The gross anatomy of the kidneys:-

The two kidneys are located in the upper abdominal cavity on either side of the vertebral column, retroperitoneal, the upper portion of the kidneys rest on the lower surface of the diaphragm and are enclosed and protected by the lower ribs cage .

Each kidney has hilus on its medial side at which the renal artery enters the kidney, and the renal vein and ureter emerge.

The renal artery is a branch of abdominal aorta the renal vein returns blood to the inferior vena cava ,the ureter carries urine from the kidneys to the urinary bladder(valeriec. Scanlon, 2007).

Kidneys in situ

Anterior view

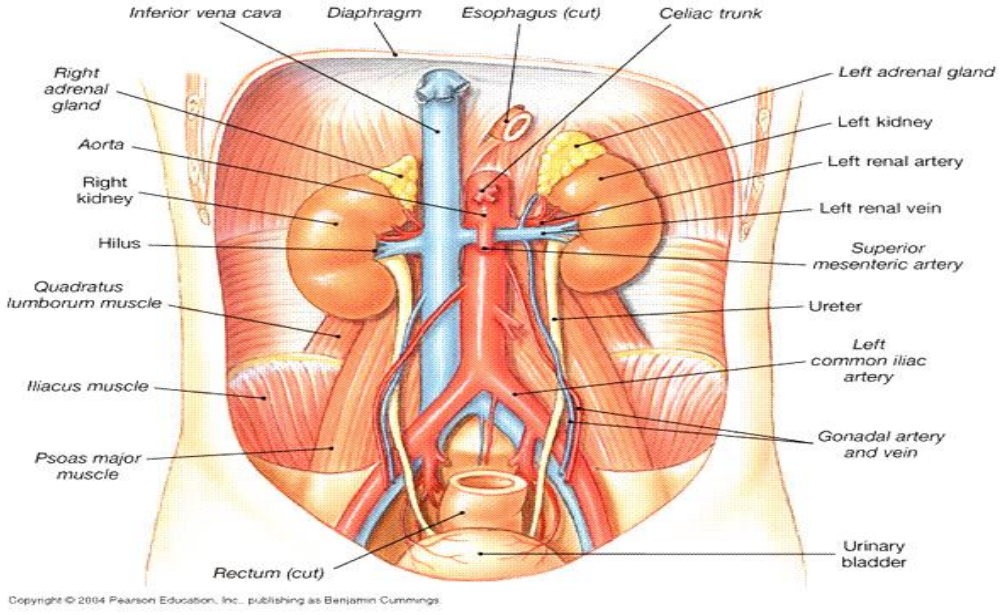

Fig 2 -9: the anterior view of the urinary.

(www.columiasurgary.org)

Internal structure of the kidney:-

In coronal or frontal section of the kidney three areas can be distinguished, the lateral and middle areas are tissue layers, and the medial area at the hilus is a cavity ,the outer tissue layer is called the renal cortex ,it's made of renal corpuscle and convoluted tubules, these are part of nephron.

The inner tissue layer is medulla which is made of loops of henle and collecting tubule also part of nephron.

The renal medulla consist of wedges shaped pieces called renal pyramids. The tip of each pyramid is its apex or papilla.

The third area called renal pelvis, it's a cavity formed by expansion of the ureter with in the kidney at hilus.

Urine flows from the renal pyramids in to the calyces which is funnel shaped extension of the renal pelvis then to the renal pelvis and out in to ureter (valeriec. Scanlon, 2007).

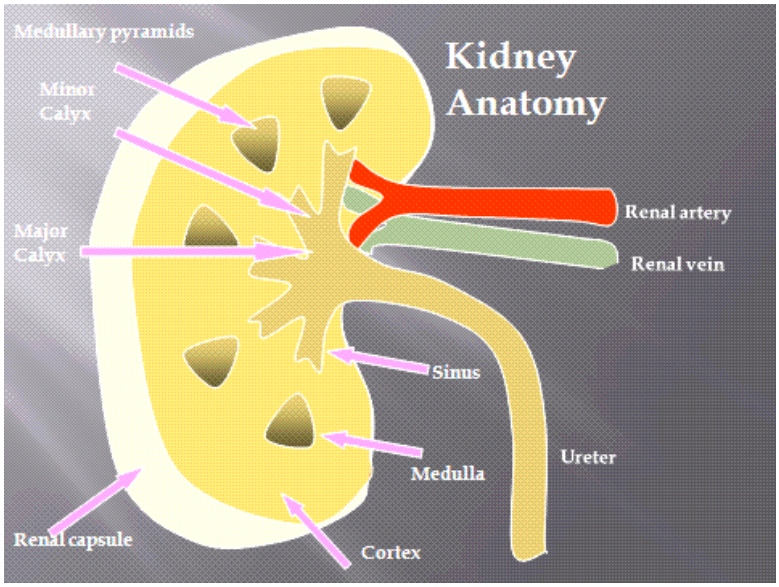

Fig 2-10 the internal structure of kidney.

(galacticonnection.com)

Normal Renal Measurements:-

The size of the kidneys is affected by age, sex (greater in men than in women), and body size; furthermore, the left kidney is slightly larger than the right in most individuals.

The normal renal length in females ranges from 9.5 to 12.1 cm and in males from 10.1 to 12.6 cm. Therefore, the normal adult kidney should measure 9-13 cm in length, 4 in thickness and 4 to 5 cm in width. These are good average measurements for exam purposes. Body habits and age should be considered since a single measurement could misrepresent the patient's condition. A 10cm long kidney is normal length: however, it is likely to be abnormal in a 20 years old male who is 6 feet tall and weighs 200 pounds (valeriec. Scanlon, 2007).

Parenchymal thickness is 11-18 mm in the male and 11-16 mm in the female.

Renal volume is a more sensitive indicator of size. .

Renal Pelvis Size:-

If the patient is non-hydrated, the renal pelvis is collapsed and

therefore not demonstrated on the scan. However, the renal pelvis with men larger than women and the left kidney larger than the right.The renal pelvis is influenced by many factors (bladder distention, diuretic drug effects) and in an estimated 5% of non-hydrated patients, there will be an anechoic separation of the central echo complex indicative of the renal pelvis(Hada, 2009).

Ultrasound appearance of the normal kidney:

The cortex of normal kidney is slightly hypo echoic when compared to adjacent liver parenchyma. The medullary pyramids are seen as regularly spaced, echo poor triangular structure between cortex and renal sinus. The renal sinus containing the pelvicalceal system is hyper echoic due to sinus fat which surround the vessels(Matthias Hofer, 1999).

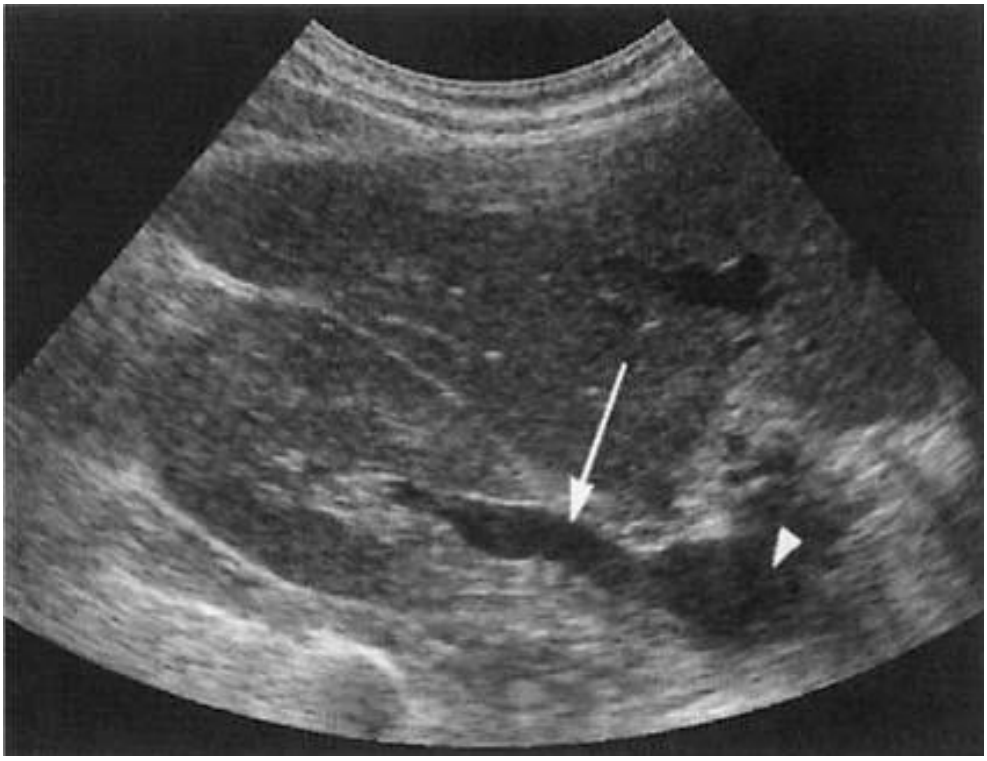

Fig2-11 transverse section through right kidney demonstrating right renal vein draining in to inferior vena cava (*Matthias Hofer, 1999*).

Sonographic measurements: The size of the kidneysis affected by age, sex(greater in the men than in women), and body size; furthermore, the left kidney is slightly larger than the right in most individuals, The normal renal length in females ranges from 9.5 to 12.1cm and in males from 10.1 to 12.6cm. therefore, the normal adult kidney should measure 9-13cm in length, 4cm in thickness and 4 to 5cm in width. These are good average(Matthias Hofer, 1999).

The main function of the kidneys are to regulate volume, composition, and PH of body fluids and to remove metabolic waste from the blood, and excrete them to the outside. Erythropoietin functions to regulate the production of red blood cells. Renin regulates blood pressure. Activation of vitamin D in active form that play

important role in calcium metabolism (valeriec.Scanlon, 2007). The main function of the nepheron is to control the composition of body fluid and remove waste from the blood.Urine is product produced by kidneys and contain wastes, excess water, and electrolytes. The three processes involved in urine formation are glomerular filtration, tubular reabsorption and tubular secretion. In glomerular filtration, blood plasma is filtered. The function of tubular reabsorption is to return most of the products filtered from plasma back to blood. The function of tubular secretion is to put waste product in to the filtrate tube excreted from the kidney(valeriec. Scanlon, 2007).

Pathology of the kidney: Disease of the kidneys are among the most important causes of death and disability in many countries throughout the world. Sever kidney diseases can be divided in to two main categories:

Acute renal failure, in which the kidneys abruptly stop working entirely or almost entirely but may eventually recover nearly normal function. The cause of acute renal failure can be divided in to three main categories: a. Pre renal acute renal failure, b. Intra renal acute renal failure, c. Post renal acute renal failure, and chronic renal failure, in which there is progressive loss of function of more and more nephrons that gradually decrease over all kidney function. Within these two general categories, they are many specific kidney diseases that can affect the kidney blood vessels, glomeruli, tubules, renal interstitium, and parts of urinary tract outside the kidney, including the ureters and bladder (Arthur. C. Guyton, 2006).

Pre-renal acute renal failure: Acute renal failure resulting from decreased blood supply to the kidneys; this condition is often referred to as prerenal acute renal failure to reflect the fact that the abnormality occurs in a system before the kidneys. This can be aconsequence of heart failure with reduced cardiac output

and low blood pressure or conditions associated blood volume and low blood pressure, such as sever hemorrhage.

Intra-renal acute renal failure: Resulting from abnormalities within the kidney itself, including those that affect the blood vessels, glomeruli, or tubules.like glomerulonephritis, tubular necrosis.

Post-renal acute renal failure: Multiple abnormalities in the lower urinary tract can block or partially block urine flow and therefore lead to acute renal failure even when the kidneys blood supply and other functions are initially normal. If the urine output of only one kidney is diminished, no major change in body fluid composition will occur because the contra lateral kidney can increase its urine output sufficiently to maintain relatively normal levels of extracellular electrolytes and solutes as well as normal extracellular fluid volume. With this type of renal failure, normal kidney function can be restored if the basic cause of the problem is corrected with in few hours. But chronic obstruction of the urinary tract, lasting or several days or weeks, can lead to irreversible kidney damage. Some of causes of post renal acute renal include: Bilateral obstruction of ureters or renal pelvises caused by large stones or blood clots, Bladder obstruction, and obstruction of the urethra.

Chronic renal failure: An irreversible decrease in the number of functioning nephrons. Chronic renal failure results from progressive and irreversible loss of the large numbers of functioning nephrons. Serious clinical symptoms often do not occur until the number of functioning nephrons fall to at least 70 to 75percent below normal. Chronic renal failure like acute renal failure, can occur because of disorder of blood vessels, glomeruli, tubules, renal interstitium, and lower urinary tract. Despite wide variety of disease that can lead to chronic renal failure, the end result is essentially the same- a decrease in the number of functioning nephrons, in many cases, an initial insult to the kidney lead to progressive deterioration of

kidney function and further loss of nephrons to the point where the person must be placed on dialysis treatment or transplanted with functioning kidney to survive. This condition is referred to as end- stage renal disease (Arthur. C. Guyton, 2006). The cause of chronic kidney disease is not always known. But any condition or disease that damage blood vessels or other structures in the kidneys can lead to kidney disease. The most common causes of chronic kidney disease are:

1. Diabetes: it causes about 35% of all chronic kidney disease. High blood sugar levels cause by diabetes damage blood vessels in the kidneys. If blood sugar level remains high this damage gradually reduce the function of the kidneys.
2. High blood pressure (hypertension): it causes another 30% of all kidney disease. Because blood pressure often rises with chronic kidney disease, high blood pressure may cause further damage kidney function even when another medical condition initially caused the disease.
3. Infection (pyelonephritis) primary or secondary disease of renal interstitium is referred to as interstitial nephritis. In general, this can result from vascular, glomerular, or tubular damage that destroys individual nephrons, or it can involve primary damage to the renal interstitium by poisons, drugs, and bacterial infections, renal interstitial injury caused by bacterial infection is called pyelonephritis. The infection can result from different types of bacteria but especially from Escherichia coli that originate from fecal contamination of the urinary tract. These bacteria reach the kidneys either by way of the ureters to the kidneys. Although the normal bladder is able to clear bacteria readily, there are two general clinical conditions that may interfere with the normal flushing of bacteria from bladder:

The inability of the bladder to empty completely, leaving residual urine in the bladder, and the existence of obstruction of urine out flow. With impaired ability to flush bacteria from the bladder, the bacteria multiply and the bladder becomes

inflamed, a condition termed cystitis. Once cystitis has occurred, it may remain localized without ascending to the kidney, or in some people, bacteria may reach the renal pelvis because of a pathological condition in which urine is propelled up one or both of ureters during micturition. This condition is called vesico-ureteric reflux and is due to failure of the bladder wall to occlude the ureter during micturition; as a result, some of the urine is propelled upward toward the kidney, carrying with it bacteria that can reach the renal pelvis and renal medulla, where they can initiate the infection and inflammation associated with pyelonephritis. Pyelonephritis begins n the renal medulla more than it affects the cortex, at least in the initial stages with long-standing pyelonephritis, invasion of the kidneys by bacteria not only causes damage to the renal medulla interstitium but also results in progressive damage of renal tubules, glomeruli, and other structures throughout the kidney. Consequently, large parts of functional renal tissue are lost, and chronic renal failure can develop (Arthur. C. Guyton, 2006).

Nephrotic syndrome (excretion of protein in the urine): Many patients with kidney disease develop the nephritic syndrome, which is characterized by loss of large quantities of plasma proteins in the urine. In some instance, this occurs without evidence of other major abnormalities of kidney function, but more often it is associated with some degree of renal failure (Arthur. C. Guyton, 2006).

Other conditions that can damage the kidneys and cause chronic kidney disease includes: bladder out let obstruction, neurgenic bladder, kidney stones in both ureters, obstruction of tubules, retroperitoneal fibrosis, vesico-ureteric reflux cystic kidney, such as alports syndrome (hereditary nephritis), nephrogenic fibrosis, polycystic kidney disease, congenital causes, having a narrowed or blocked renal.

Clinical picture Most people with chronic kidney disease have no symptoms because the body can tolerate even a large reduction in kidney function.

A change in kidney function is usually discovered through routine blood or urine test if the kidneys continue to lose function and there is progression towards kidney failure (established renal failure or ESRD), this will usually be tracked by blood tests and monitoring. If kidney failure does occur, the symptoms may include: Weight loss, poor appetite, swollen ankles, feet or hands (due to water retention) shortness of breath, blood or protein in urine, an increase need to urinate, particularly at night, itchy skin, muscle cramps, high blood pressure, nausea, and erectile dysfunction in men(Ayus et al, 2005).

Treatment of chronic renal failure (dialysis with an artificial kidney):-

Sever loss of kidney function, either acutely or chronically, is a threat to life or requires removal of toxic products and restoration of body fluid volume and composition toward normal. This can be accomplished by dialysis with an artificial kidney. In certain types of acute renal failure,, an artificial kidney may be used to tide the patient over until the kidneys resume their function. If the loss of kidney function is irreversible, it is necessary to to perfume dialysis chronically to maintain life. Because dialysis cannot maintain completely normal body fluid composition and cannot replace all the multiple functions performed by the kidneys, the health of patients maintained on artificial kidneys usually remains significantly impaired. A better treatment for permanent loss of kidney function is to restore functional kidney tissue by means of a kidney transplant.

Is no specific treatment unequivocally shown to slow the worsening of chronic kidney disease If there is an underlying cause to CKD, such as vasculitis, this may be treated directly to slow the damage. In more advanced stages, treatments may be required for anemia and bone disease. Severe CKD requires renal replacement therapy, which may involve a form of dialysis, but ideally constitutes a kidney transplant. Renal replacement therapy is a term used to encompass life-supporting

treatments for renal failure. It includes: hemodialysis, peritoneal dialysis, hemofiltration and renal transplantation.

Basic principles of dialysis:

The basic principle of the artificial kidney is to pass blood through minute blood channels bounded by a thin membrane. On the other side of the membrane is a dialyzing fluid into which unwanted substance in the blood pass by diffusion.

Kidney in which blood flows continually between two thin membranes of cellophane; outside the membrane is a dialyzing fluid. The cellophane is porous enough to allow the constituents of the plasma، except the plasma proteins, to diffuse in both directions-from plasma in to the dialyzing fluid or from the dialyzing fluid, back in to the plasma. If the concentration of a substance is greater in the plasma than in the dialyzing fluid, there will be a net transfer of substance from plasma in to the dialyzing fluid. The rate of movement of solute a cross the **dialyzing membrane depends on**:

1. The concentration gradient of solute between the two solutions.
2. The permeability of the membrane to the solute.
3. The surface area of the membrane.
4. The length of time that the blood and fluid remain in contact with membrane.

Thus, the maximum rate of solute transfer occurs initially when the concentration gradient is greatest (when dialysis is begun) and slows down as the concentration gradient is dissipated. In a flowing system, as in the case with hemodialysis in which blood and dialysate fluid flow through the artificial kidney, the dissipation of the concentration gradient can be reduced and diffusion of solute a cross the membrane can be optimized by increasing the flow rate of the blood, the dialyzing fluid, or both (Arthur. C. Guyton, 2006).

3.1 Complications during hemodialysis:-

Common complications: Patient complications, hypotension, muscle cramps, disequilibrium syndrome, nausea and vomiting, headache, chest pain, itching, fever and chills, pyrogenic reactio, hypertension.

Technical complications: Clotting, blood leak, power failure, hemolysis, air embolism, air in bloodlines, exsanguinations, dialyzer reactions.

Cardiac complication: Even though the safety of the hemodialytic procedure has improved greatly over the years, the procedure is not without risks. Common problems are listed below.

a. Hypotension:-

A decrease in blood pressure is the most frequent complication reported during hemodialysis. When fluid is removed during hemodialysis, the osmotic pressure is increased and this prompts refilling from the interstitial space. The interstitial space is then refilled by fluid from the intracellular space. Excessive ultrafiltration with inadequate vascular refilling plays a major role in dialysis induced hypotension. The immediate treatment to hypotension is to discontinue dialysis and place the patient in a trendelenburg position. This will increase cardiac filling and may increase the blood pressure promptly.

b. Cramps:-

In the majority of hemodialysis patients, cramps occur toward the end of the dialysis procedure after a significant volume of fluid has been removed by ultrafiltration. The immediate treatment for cramps is directed at restoring intravascular volume through the use of small boluses of isotonic saline. Prevention of cramps has been attempted with the prophylactic use of quinine sulfate at least 2 hours prior to dialysis.

c. Febrile reactions:-

Febrile episodes should be aggressively evaluated with appropriate wound and blood cultures. The suspicion of infection should be high. Treatment of endotoxin related fever is generally supportive with antipyretics. Temperatures should be recorded at the initiation and termination of dialysis treatment.

d. Arrhythmia:-

Patients on maintenance hemodialysis are at risk of cardiac arrhythmias. They occur predominately in association with hemodialysis or may occur in the interdialytic period. Both acute and chronic alterations in fluid, electrolyte, and acid-base homeostasis may be arrhythmogenic in these patients.

e. Hemolysis:-

Hemolysis may result from a number of biochemical and toxic insults during the dialysis procedure. The half-life of red blood cells in renal failure patients is approximately one half to one third of normal and the cells are particularly susceptible to membrane injury.

f. Hypoxemia:-

A fall in arterial PO2 is a frequent complication of hemodialysis that occurs in nearly 90% of patients. The drop ranges from 5 to 35 mm Hg, and reaches its peak between 30 - 60 minutes after beginning dialysis. This is obviously undesirable for patients with underlying cardiopulmonary disease. Also, patients on mechanical ventilators with constant minute volume and inspired oxygen concentration can still develop hypoxemia during hemodialysis. High cardiovascular mortality is major cause of reduced life expectancy of patients who are on hemodialysis. Classical risk factors cannot fully explain the magnitude of risk. This article addresses some nontraditional approaches to deal with expensive cardiovascular risk for patients who are on hemodialysis(*Eberhard Ritz*).

Cardiovascular disease in the haemodialysis population continues to contribute to mortality and morbidity. Disorders of left ventricular geometry and function are

highly prevalent and lead to increased mortality in this highly vulnerable population. Left ventricular often dysfunction as a result of hypertension, ischemic cardiac disease or dilated cardiomyopathy, has not been uniformly defined in the literature making diagnosis and therapy problem. Although routinely available, screening by echocardiography is critically volume dependent and prone to underestimation in left ventricular ejection fraction(*Manish et al 2008*). Prior studies of the effect of hemodialysis on left ventricular function have not distinguished between the removal of uremic toxins and the change in cardiac filling volume. To separate these effects, left ventricular function was examined by serial echocardiography in five stable hemodialysis patients before and after three different dialysis procedures: (a) hemodialysis with volume loss, (b) ultrafiltration (volume loss only), and (c) hemodialysis without volume loss. The patients were similarly studied under control conditions and after increased (50 of head down tilt for 90 min) and decreased (lower body negative pressure) cardiac filling volume (*J. V. NIXON 1982*). The existence of a uremic cardiomyopathy has remained an important, but unsettled issue in clinical medicine. While experimental studies have shown that high concentrations of uremic compounds are capable of depressing cardiac function in vitro, the demonstration of a uremic cardiomyopathy has been lacking(*Penpargkulet al 1972*)(*Uraoka et al 1975*), Such a demonstration is hampered by the plethora of complicating diseases that prohibit the dissociation of the independent effects of uremia(*Seldin et al 1971*). The commonly associated conditions of coronary artery disease, hypertension, and pericardial disease may individually or in combination affect left ventricular performance and make the influence of uremia per se difficult to evaluate(*Lewin et al 1971*)(*Lindner et al 1974)*.

2.4 Previous studies

Chen R et al in 2014 Their aim of the study was to assess the differences between the left ventricular (LV) myocardial function in hemodialysis and non-dialysis uremic patients based on three-dimensional speckle-tracking echocardiography,the study population consisted of 35 maintenance hemodialysis patients (the hemodialysis group), 30 uremic patients who were hospitalized for the creation of a primary arterio-venous fistula (the non-dialysis group), and 32 healthy volunteers. All of the patients had normal left ventricular ejection fractions (i.e., 55% or greater). Three-dimensional speckle tracking echocardiography was performed to assess the left ventricle's global three-dimensional strain, regional longitudinal strain, circumferential strain, and radial strain, te results of their study revealed that the left ventricular regional longitudinal strain, radial strain, circumferential strain, and global three-dimensional strain were significantly decreased in the non-dialysis patients compared with the other two groups (all, P,0.001). However, the three-dimensional strain and the regional longitudinal strain were lower in the hemodialysis patients than in the controls (P,0.01). In the hemodialysis patients and the control group, the longitudinal strain, circumferential strain, and radial strain were higher at the apical level than they were at the basal level and mid-levels. A multivariate linear regression analysis showed that the blood urea nitrogen and creatinine levels were independently associated with the values of the global three-dimensional strain (b =20.217, P = 0.000; b =20.243, P = 0.011, respectively) and the longitudinal strain (b =2 0.154, P = 0.032; b =20.188, P = 0.029, respectively).,it concluded that Three-dimensional speckle-tracking echocardiography may detect myocardial dysfunction in patients with uremia who have preserved LVEF. The global three-dimensional strain and the regional longitudinal strain appear to be superior in hemodialysis patients compared with non-dialysis patients

*Lissa Sugeng et al in 2006*they sought to compare both CCT and RT3DE measurements of left ventricular size and function with the standard reference technique, cardiac MR (CMR).In 31 patients, RT3DE data sets (Philips 7500) and long-axis CMR (Siemens, 1.5 T) and CCT (Toshiba, 16-slice MDCT) images were obtained on the same day without _-blockers. All images were analyzed to obtain end-systolic and end-diastolic volumes and ejection fractions using the same rotational analysis to eliminate possible analysis-related differences. Intertechnique agreement was tested through linear regression and Bland-Altman analyses. Repeated measurements were performed to determine intraobserver and interobserver variability. Both CCT and RT3DE measurements resulted in high correlation (*r*2_0.85) compared with CMR. However, CCT significantly overestimated end-diastolic and end-systolic volumes (26 and 19 mL; *P*_0.05), resulting in a small but significant bias in ejection fraction (_2.8%). RT3DE underestimated end-diastolic and end-systolic volumes only slightly (5 and 6 mL),with no significant bias in EF (0.3%; *P*_0.68). The limits of agreement with CMR were comparable for the 2 techniques. The variability in the CCT measurements was roughly half of that in either RT3DE or CMR values.

CCT provides highly reproducible measurements of left ventricular volumes, which are significantly larger than CMR values. RT3DE measurements compared more favorably with the CMR reference, albeit with higher variability (*LissaSugeng et al 2006).*

(*Darren Green et al 2012), (Eric et al 2006) and (Diana Yuan Yng Chiu) studied*

Cardiac Risk Assessment using 2D and 3D Transthoracic Echocardiography in Patients Undergoing Haemo-dialysis Diana Yuan Yng Chiu, For the degree of Doctor of Philosophy at the University of Manchester, March 2016

Haemodialysis (HD) patients have a high mortality risk and most have echocardiographic evidence of abnormal cardiac structure or function. Markers, such as left ventricular hypertrophy (LVH), show association with adverse outcome in the general population and can aid in clinical decision making. The aim of this research was to explore the prognostic utility of established and novel two-dimensional (2DE) and three-dimensional transthoracic echocardiographic (RT3DE) techniques in HD patients.

Adult maintenance HD patients from a single tertiary nephrology centre including satellite dialysis units were enrolled. Exclusion criteria were if patients were clinically unstable, unable to consent, or if required ambulance transportation for echocardiography visits. Consented patients underwent 2DE with speckle tracking (STE), RT3DE and Vic order Tm measurements of pulse wave velocity (PWV) on a non-dialysis day, after the short inter-dialytic break. Clinical phenotype data, 3-month averaged blood results and dialysis prescriptions were obtained from the hospital electronic patient records. All patients screened were followed-up until death, renal transplantation, moving out of the region, or 16th November 2015. Regression analysis was used to assess the cross-sectional relationship between echocardiographic parameters. Relationship of echocardiographic parameters with outcome was assessed by Cox regression analysis.

The first study explored whether patients recruited had similar characteristics and survival compared with patients who declined consent or who were excluded from the study. Patients who declined consent had an adjusted hazard ratio (HR) for all-cause mortality compared with recruited patients of 1.70, 95% confidence interval (CI) 1.10-2.52, and excluded patients had an adjusted HR of 1.30, 95% CI 0.75-2.25. Recruited patients may be a 'fitter' population and this needs to be considered when interpreting results.

The second study reports that when global longitudinal strain (GLS) is combined in a multivariable model with PWV; PWV is superior to GLS in its association with mortality (adjusted HR 1.23, 95% CI 1.03-1.47 *versus* HR 1.00, 95% CI 0.86-1.17). When this analysis was repeated in a sub-group of patients with LVH, neither GLS nor PWV were associated with mortality, whilst both were prognostic ally significant in a preserved LVEF sub-group (PWV: HR 1.23, 95% CI 1.04-1.4 and GLS: HR 1.16, 95% CI 1.01-1.33). Therefore GLS has different prognostic implications in different patient sub-groups.

The third study explored whether tissue motion mitral annular displacement (TMAD) measured by STE may be a more useful alternative to GLS as it measures strain but is quicker and less user-dependent. TMAD was closely correlated to GLS ($r=-0.614$, $p<0.001$), but had no prognostic power for mortality (adjusted HR 1.04,95% CI 0.91-1.19).

The correlation between 2DE and RT3DE determined LV mass and volume measurements and the prognostic significance of RT3DE measurements were assessed. Although there was good correlation between 2DE and RT3DE LV volume measurements, 2DE overestimated LV mass compared to RT3DE. RT3DE measures gave no added prognostic value, and there were added difficulties in obtaining adequate images for RT3DE (35% of patients who had adequate 2D images). Furthermore, although RT3DE determined LV mechanical dys-synchrony index was prolonged in HD patients compared with published general population controls, it failed to show any prognostic significance (HR 2.16, 9% CI 0.96-4.89) for mortality, but was associated with hospitalisation for heart failure (HR 1.03, 95% CI 1.00-1.06). These results indicate that novel measurements of sub-clinical cardiac dysfunction have the potential to aid prognostication in this high risk population. Follow-up studies exploring the longitudinal change in these

parameters is ongoing (*Darren Green et al 2012), (Eric et al 2006) and (Diana Yuan Yng Chiu).*

*Isabel et al 2012studied*The goal of their paper is to stratify dialyzed patients according to hydration status and to make an evaluation about the possible echocardiography alterations of the different groups.

a transversal study was carried out with 117 patients: 65 were on hemodialysis and 52 on peritoneal dialysis. We performed the following tests: multi frequency bio-impedance with the BCM-Body Composition Fresenius' Monitor system, transthoracic echocardiography, and blood tests. If ECW/TBW (extracellular water vs total body water) normalization ratio for age and gender was > 2.5% SD, the patient was considered overhydrated.

HD patients are significantly overhydrated before HD (67.1%) compared to DP patients (46.1%), and almost half of the overhydrated population presents arterial hypertension. However, after an HD session, a better control of the hydration status is reached (26.1%). DP patients frequently present high arterial pressure and/or are under antihypertensive treatment (DP 76.9% vs HD 49.2%). Left ventricular hypertrophy is much more common in HD overhydrated patients, eccentric LVH being more prevalent.

Overhydrated patients present significantly high values of LAVI, ILVM, OH/ECW. They concluded that: Bio-impedance technique allows for the detection of a large number of overhydrated patients.

Echocardiographic alterations in dialyzed patients show a high correlation between the hydration stage by ECW/TBW normalized ratio for age and gender and the LAVI and ILVM *(Isabel et al 2012).*

Terezie et al 2016 studied Arterial stiffness reflects the rigidity of the arterial wall. Studies of adult patients confirmed that arterial stiffness is a marker and risk factor for cardiovascular morbidity and mortality.

Non-invasive methods for arterial stiffness measurement, such as pulse wave velocity, have been increasingly used in both research and clinical practices to determine arterial stiffness in the pediatric population as well. With widespread use of these non-invasive techniques in children and adolescents, the knowledge of factors and conditions associated with arterial stiffening has expanded rapidly over the past years. These factors include traditional cardiovascular risk factors as well as other conditions, e.g. prenatal growth restriction, metabolic syndrome, obesity, diabetes mellitus, chronic kidney disease, vasculitides and vasculo-pathies associated with various syndromes, congenital heart disease, and several systemic diseases. Early identification of increased arterial stiffness in childhood may lead to early intervention. However, interventional longitudinal studies will be necessary for determining whether the improvement of arterial function in normal and at-risk pediatrics populations, will be associated with lowering cardiovascular risk in the early adulthood (*Terezie et al 2016*).

*Damir et al 2015studied.*Aims: Cardiovascular alterations contribute to a high mortality rate in patients with end-stage renal disease (ESRD). The aims of the present study are to evaluate left ventricular (LV) function and common carotid artery (CCA) parameters and to determine risk factors associated with these changes in patients undergoing peritoneal dialysis (PD). Methods: This longitudinal prospective study was conducted in 50 ESRD patients in whom PD had been initiated and who were observed for 18 months after the commencement of dialysis treatment, with echocardiography and CCA ultrasound parameter evaluation. Results: LV hypertrophy was observed in 78% of patients at baseline and in 60% after 18 months of PD treatment. LV systolic and diastolic function

was found to be significantly better after 18 months of PD treatment. Examining predictors of LV systolic function, it was found that total cholesterol was an independent positive predictor and endothelin-1 (ET-1) an independent negative predictor of LV systolic function after 18 months of treatment with PD ($p < 0.001$). Independent negative predictors of diastolic LV function were hemoglobin and type 2 diabetes mellitus, and daily collection of urine was an independent positive predictor ($p < 0.001$). Female gender was an independent negative predictor of CCA intima-media thickness, whereas body mass index,ET-1 and C-reactive protein were independent positive predictors ($p < 0.001$). Conclusions:The results suggest several novel modifiable mechanisms related to the short-term effects of dialysis that are potentially implicated in the development of uremic cardiomyopathy, *(Damir et al 2015).*

Silvio et al 2010studied Changes in cardiac structure and function detected by echocardiography are common in patients with chronic kidney disease undergoing hemodialysis, and have been recognized as key outcome predictors. This review attempts to summarize recent evidence pointing to the usefulness of the method in the detection of clinical and subclinical cardiac dysfunction, stratification of cardiovascular risk and assessment of intervention strategies (*Silvio et al 2010)*.

Jan et al , 2016*studied*Arterial hypertension is the leading cause of morbidity and mortality in adult population. In recent years, the prevalence of hypertension in children has been increasing, mainly due to increased prevalence of obesity. Similar to adult population, arterial hypertension can also manifest with target organ damage such as left ventricular hypertrophy (LVH), increased arterial stiffness, micro-albuminuria, brain damage or retinopathy in children and adolescents. Therefore, for diagnostic and therapeutic reasons, echocardiography is recommended as a primary tool for evaluating patients for target-organ abnormalities by assessing the presence or absence of LVH. The ESH hypertension

management guideline 2009 for children recommends evaluating the left ventricular mass and micro-albuminuria regularly in this case. Echocardiography is a non-invasive fundamental visualization method which has the ability to evaluate heart morphology and function in detail and to assess the progression or regression of hypertrophy. Thus, it is frequently used in children with hypertension. Even though it is easy to perform echocardiography, it is more complicated to assess left ventricular mass in children as this assessment depends on age. The objective of the publication is to review left ventricle parameters evaluation in children (left ventricular mass, LVM, left ventricular mass index, LVMI), particularly in diseases which may lead to higher cardiovascular risk as early as childhood or early adulthood(Jan et al , 2016).

Ahmed et al 2016studied To define the normal echocardiographic reference values for various cardiac measurements in young, healthy Egyptian adults.

Methods: This study was performed on 1,364 healthy adults aged 18–35 years. Standard trans-thoracic echocardiographic studies were performed to obtain end diastole measurements of left ventricular (LV) posterior wall thickness (PWd), interventricular septum thickness (IVSd), LV internal dimensions at end diastole (LVEDD) and end systole (LVESD), left atrial (LA) diameter, aortic root diameter and right ventricular outflow dimension (RVd). LV ejection fraction (LVEF), LV mass and relative wall thickness

were assessed. All measurements were then indexed to body surface area (BSA). Based on our results we proposed normal reference values for young Egyptian adults.

Results: On comparing subjects according to gender, absolute measurements showed males had significantly larger LVEDD, LVESD, IVSd, PWd, LV mass, LA anteroposterior diameter and aortic root diameter ($p<0.0001$ for each). LV ejection fraction was higher in females ($p=0.008$). There was no difference

regarding RVd (p=0.118). Females had larger indexed LVEDD (p=0.0003), indexed LA and aortic root diameters (p=0.0005 and 0.007 respectively) and indexed RVd (p<0.0001). However,

indexed LV mass was larger in males 83.56±20.37 versus 77.76±18.56 g/m2 (p<0.0001). We proposed normal values for absolute and indexed echocardiographic measurements based on our results.

Conclusions: Estimation of local reference values is important to define normalcy. The main difference

from international values was a higher upper reference limit for LV mass especially in females and relatively smaller LA dimensions in young Egyptians*(Ahmed et al 2016).*

Reid et al 2011studied Echocardiography is a widely used evaluation tool in cardiovascular research. Although rats are a common model in such research, normal echocardiographic values for young, developing rats have not been established. Furthermore, whether exercise during the developmental phase of the lifespan affects the structure or function of the heart is unclear. Male Sprague–Dawley rat pups (21 d) were assigned randomly to a non-exercise or voluntary exercise group for 12 wk. Echocardiograms were obtained before and at weekly intervals during the 12-wk observation period. Maturation resulted in changes in many echocardiographically derived variables, whereas voluntary exercise failed to alter the development of cardiac structure or function. This study provides normal echocardiographic variables for developing male rats and provides evidence that exercise during the developmental phase of the lifespan has little effect on cardiac morphology and function as assessed by echocardiography*(Reid et al 2011).*

CHAPTER THREE

MATERIALS AND METHODS

3.1study design and population:

A prospective, cohort study was carried out in Sinnar renal Center, between July 2014 to June 2018. The study population included 200 adult's patient under regular hemodialysis with ESRD and regular HD, but very ill patients or who are known cardiac disease patients are excluded.

3.2 Technical information identifies:

All patients subjected to echocardiography by two dimensions machine estate, my lab gold 30 cardiovascular machine(Italy), with phased array probe2.5MHz(serial number of 08900manufactured date of February 2012), convex face with a small footprint for "peaking" in between rib interspaces. Printing facility issued through the ultrasound-digital graphic-printer (serial number of UP-D897 made by sony corporation- Japan), 100V; 1.5A; and 50/60 Hz, this machine can perfectly match the latest technological innovations with ease of use and portability, with MYLAB 30 GOLD cardiovascular, any technological innovation and solution has been redesigned to be applied to the most advanced portable system, able to deliver unmatched performance, increased diagnostic confidence and extreme ease of use at the same time, moreover, the high-level platform ensures extended modularity and upgradeability as well as easy and fast services diagnosis and recovery procedures, also two probes simultaneously connected to the system allows fast selection and activation as well as an extended ranges of applications even in the portable configuration. The high-level of the system ensures probe compatibility with top-end systems. Not this only but also it contains high performance 15 inches LCD monitor ensure clear image visualization, low reflection, and eyestrain. It contains integrated lightweight battery enable it to

travel where it is are needed, this machine with high performance and high-density array transducers to always ensure optimal image quality, it has extended bandwidth to deliver A wide range of setting for increased application of use, including standard and harmonic imaging, high sensitivity for precise Doppler detection, reflected on color flow mapping (CFM), power and pulsed waves/continuous waves (PW/CW)signal, these transducer are light-weight and ergonomic approach for user comfort in daily routine, with flexible cable for easy maneuverability during scanning.

Because ultrasound cannot be transmitted through air, a dense coupling medium is needed between the transducer and the skin. (*Starkey, 1993 and Michlovitz, 1996).* To obtain a good image, a fluid medium is needed to provide a link between the transducer and surface of the patient. This fluid is called an acoustic coupling agent, often referred to as ultrasound gel (Palmer, 1995). Water is not good coupling agent because it evaporates rapidly owing to the heat of the body: it also runs away from the patient as the transducer is moved. It should only be used in an emergency when nothing else is available. Oil, either mineral or vegetable, is good coupling agent, but when used for a long time may dissolve the rubber or plastic shielding of the equipment. If oil gets on the operator's finger, as it inevitably will, it might damage the controls of the ultrasound the unit. (Palmer, 1995). The best acoustic coupling agent is the water-soluble gel. May are commercially available, but they are usually expensive and sometimes difficult to obtain. It is not necessary to use a particular coupling agent with specified equipment. Even though manufacturers often suggest that this is essential. Special coupling agents do not give a better image.(Palmer, 1995). The coupling agent is best applied using the squeeze bottle, from which it can be squirted on to the patient's skin. This avoids contamination. Any refillable plastic squeeze bottle is suitable, but it must be completely clean and dry before it is filled with a coupling

agent. If there is an open wound, skin rash, or any other risk of infection, cover the transducer (or the skin) with thin plastic; put coupling agent on both sides of the plastic. The transducer must be cleaned after every patient.(Palmer, 1995)

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

Ingredients

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

1. Carbomer. A synthetic high molecular weight polymer of acrylic acid cross linked with allyl sucrose and containing 56-68% of carboxylic acid groups. It's a white, fluffy, acidic, hygroscopic powder with a slight characteristic odour. Neutralized with alkali hydroxides or amines, it's very soluble in water, alcohol, and glycerol. 3 (Palmer, 1995). There three carbomers: the most suitable is carbomer 940, which forms a clear gel in aqueous and non-aqueous vehicles. If carbomer 940 is not available carbomer 934 or 941 can be used, however, they may not be quite so easy to mix (as described below) as carbomer 940. (Palmer, 1995).
2. EDTA (edetic acid). A white crystalline powder, very slightly soluble in water. Soluble in solutions of alklihyderoxides. (Palmer, 1995)
3. Propylene glycol. A colorless, odorless. Viscous, hygroscopic liquid with a slightly sweet taste. Density equal 1.035-1.037g/ml. (Palmer, 1995).

3-3 Study protocols methods (techniques)

Heart ultrasound scanning technique:

Tran's thoracic echocardiography was performed on all patients. The examination was performed in a supine, or 30 degrees left lateral decubitus position, with the left arm raised up above the head. This position brings the heart out toward the

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

Para sternal long-axis (PLA) view

The probe should be placed in the parasternal fourth or fifth intercostals space with transducer marker directed to the patient's right shoulder, this allows for typical identification of the right ventricle, left atrium, left ventricle, aortic valve, aortic root, aortic out flow tract, and surrounding pericardium.

Parasternal short axis view

From the parasternal long axis position rotate the probe clock wise 90 degrees such that the probe indicator is pointed toward the patient's left shoulder. This allows for identification of the left ventricle, right ventricle, and pericardium.

In this view, the right ventricle is closer to the surface and appears crescent-shaped. While the left ventricle is deep in the right ventricle and appears circular.

Apical four chamber view

If possible, have the patients raised the left arm

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

Subxiphoid 4-champer view

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

Septum thickness was measured using the parasternal long-axis view. M-mode measurements were made according to the recommendation of the American society of echocardiography. Thus, the line of measuring was perpendicular to the endocardium of the septum and posterior wall. With the patient in the recommended position. The transducer was placed in the fourth inter costal space at the left sterna border and directed posteriorly. Laterally and inferiorly to obtained a group of strong echoes from the posterior left ventricular wall.

3.4. Data collection:

Structured questionnaires were administered, and physical examinations were done in all cases. Information was obtained about demographic factors, socioeconomic status of the residential neighborhood, the drug described. Weight was measured with standardized protocols.

The son graphic data was obtained through direct ultrasound scanning of heart.

3.5. Statistical analysis:

Statistical techniques were used to analyze the data obtained from the field. Some of the computer software applications used for the survey were Statistical Package for the Social scientists (SPSS, Ins). The results were presented in both quantitative and qualitative terms. They were in the forms of frequency tables, histograms, line graphs, pie charts, Pearson correlation coefficient, two-independent samples *t*-test, Chi-square test applied for estimating the occurrence of categorical variables and Pearson's correlation coefficient. The p-value less than 0.05 were considered as significant. Inferences and calculations made from these measures and compared with the existing literature to conclude the study.

The data from the interview schedules were analyzed regarding percentages of various responses. This was done rate the percentages of a sample which responded in a particular way.

3.6. Ethical considerations:

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

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

CHAPTER FOUR

Results

The following tables and figures presented the results of the study

Table (4-1): Frequency percentage according to gender:

gender		
	Frequency	Percent
Male	86	43
Female	114	57
Total	200	100.0

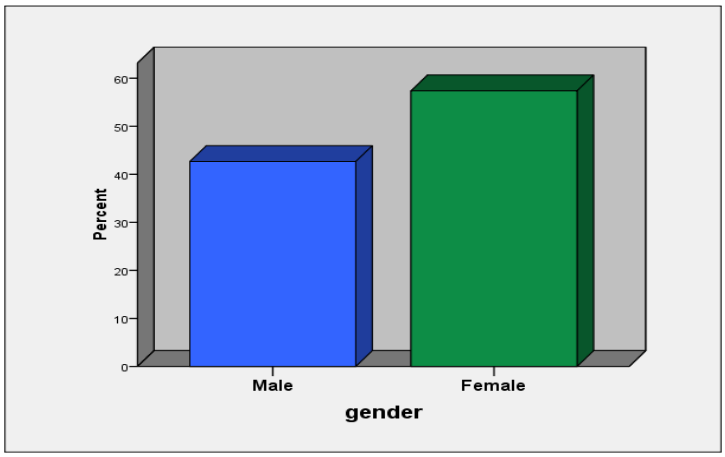

Figure 4-1:Frequency percentage according to gender

Table (4-2): Frequency percentage according to Age

Age		
	Frequency	Percent
20-30	54	27.0
30-40	44	22.0
40-50	24	12.0
50-60	36	18.0
More than 60	42	21.0
Total	200	100.0

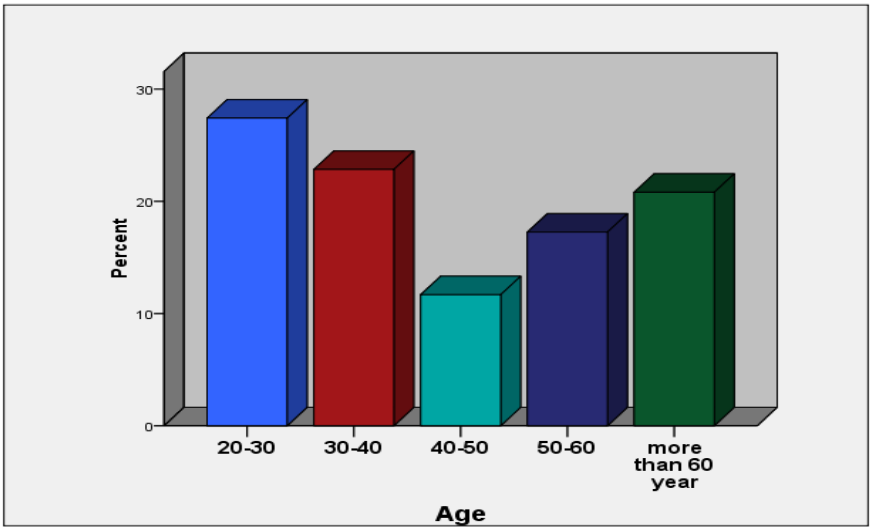

Figure4-2:Age

Table (4-3): Frequency percentage according to weight

weight		
	Frequency	Percent
20-40	28	14.0
40-60	110	55.0
More than 60	62	31.0
Total	200	100.0

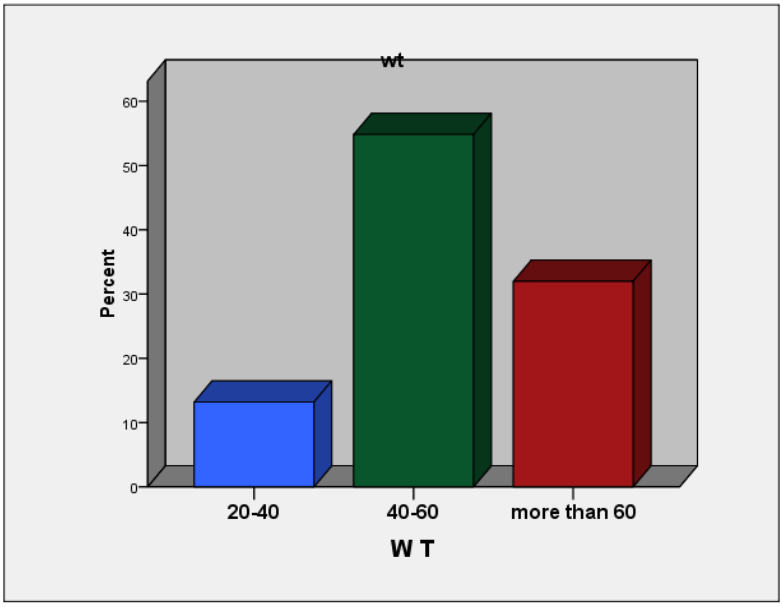

Figure 4-3: Frequency percentage of cystic change according to weight

Table (4-4): Frequency percentage according Duration

Duration		
	Frequency	Percent
1-24	66	33.0
25-48	76	38.0
49-72	36	18.0
More than 72	22	11.0
Total	200	100.0

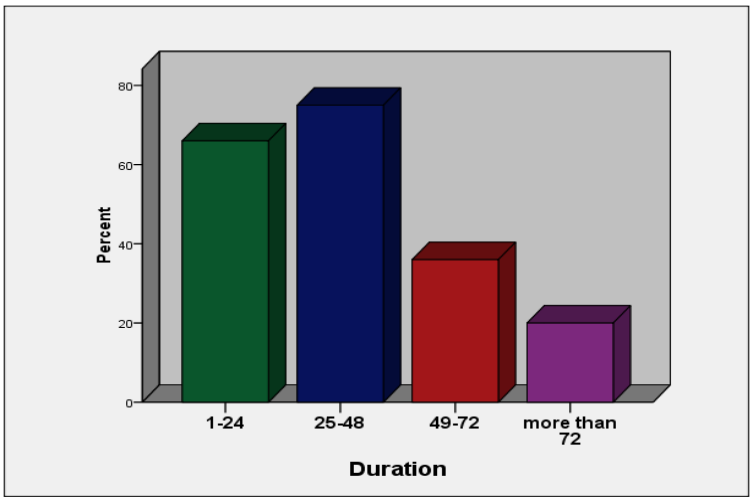

Figure 4-4: Frequency percentage accordingDuration

Table (4-5): Frequency percentage according Septum thickness

Septum		
	Frequency	Percent
Normal	2	1.0
Abnormal	198	99.0
Total	200	100.0

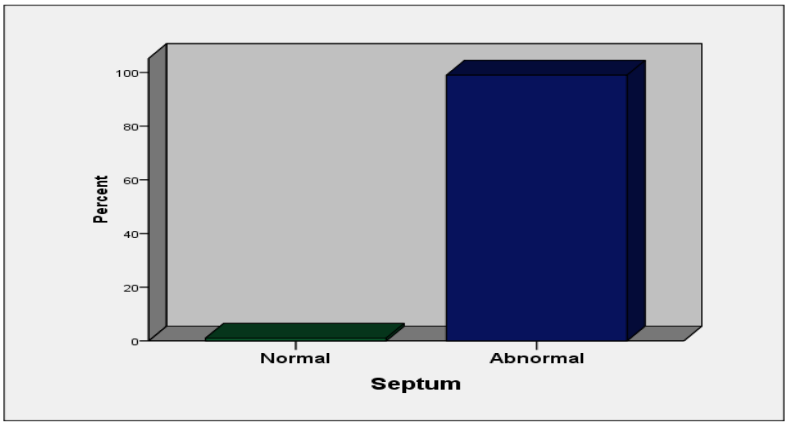

Figure4-5: Frequency percentage according Septum

Table (4-6): Frequency percentage according post wall thickness

post		
	Frequency	Percent
Normal	38	19.0
Abnormal	162	81.0
Total	200	100.0

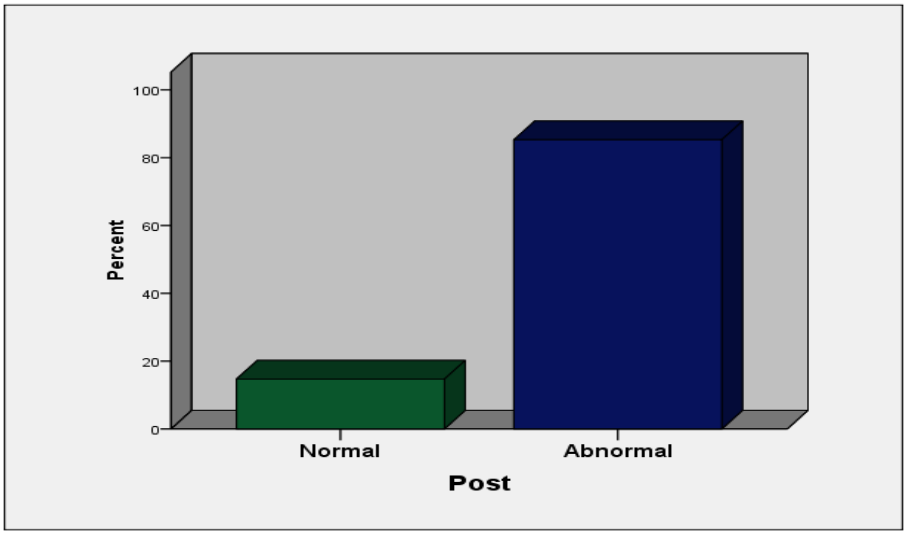

Figure4-6: Frequency percentage according Post wall thickness

Table (4-7): Frequencypercentage according LVD

LVD		
	Frequency	Percent
Normal	48	24.0
Abnormal	152	76.0
Total	200	100.0

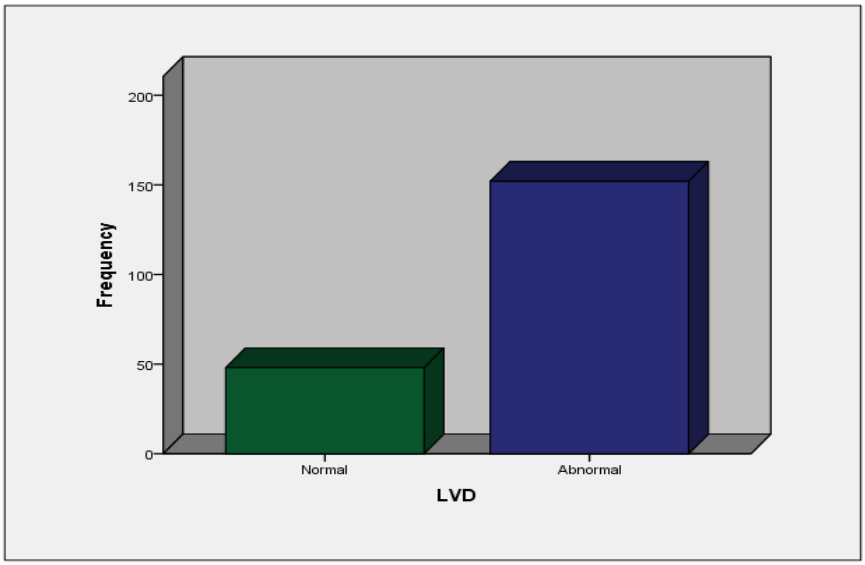

Figure4-7: Frequency percentage according LVD

Table (4-8): Frequency percentage according ejection fraction

E_F		
	Frequency	Percent
Normal	126	63.0
Abnormal	74	37.0
Total	200	100.0

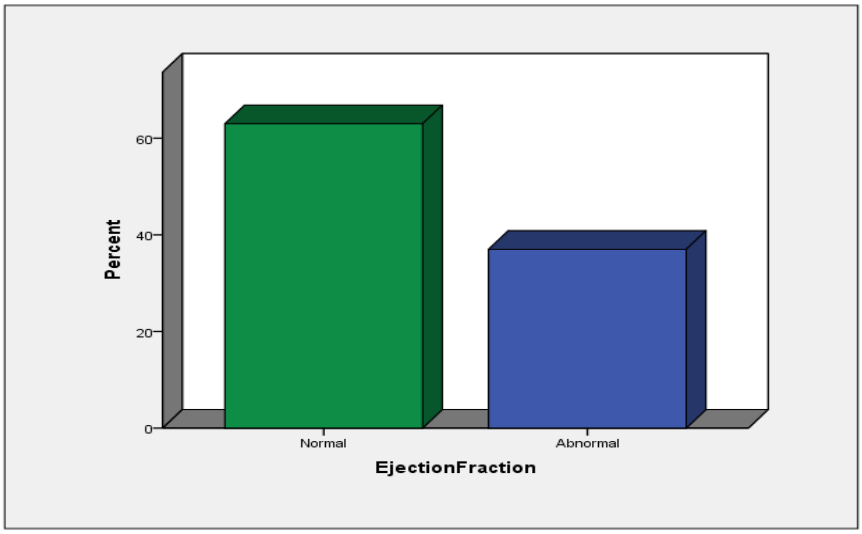

Figure 4-8: Frequency percentage according ejection fraction

Table (4-9): Frequency percentage according fraction shorting:

FS		
	Frequency	Percent
Normal	160	80.0
Abnormal	40	20.0
Total	200	100.0

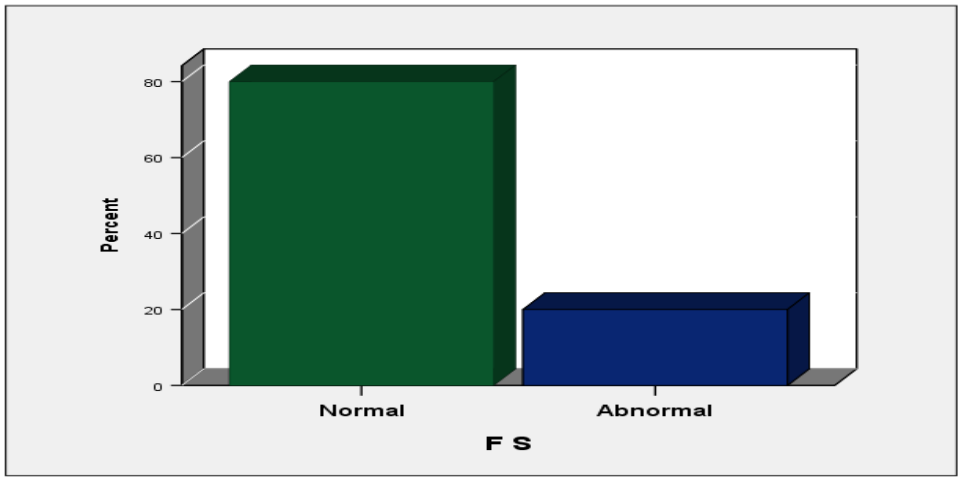

Figure4-9: Frequency percentage according fraction shorting

Table (4-10): Frequency percentage according cardiac out put

COP		
	Frequency	Percent
Normal	162	81.0
Abnormal	38	19.0
Total	200	100.0

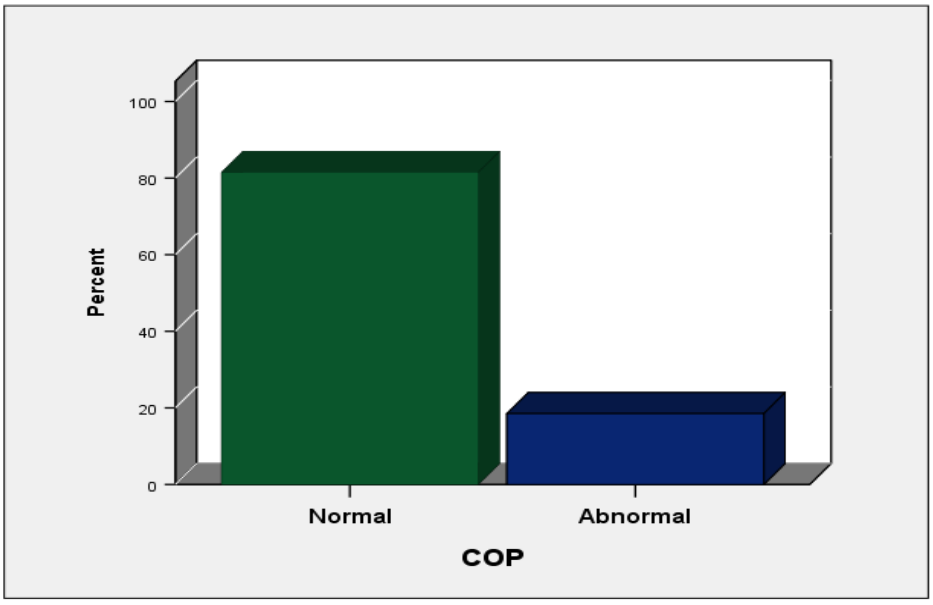

Figure4-10: Frequency percentage according cardiac out put

Table (4-11):Frequency percentage according stroke volume

SV		
	Frequency	Percent
Normal	158	79.0
Abnormal	42	21.0
Total	200	100.0

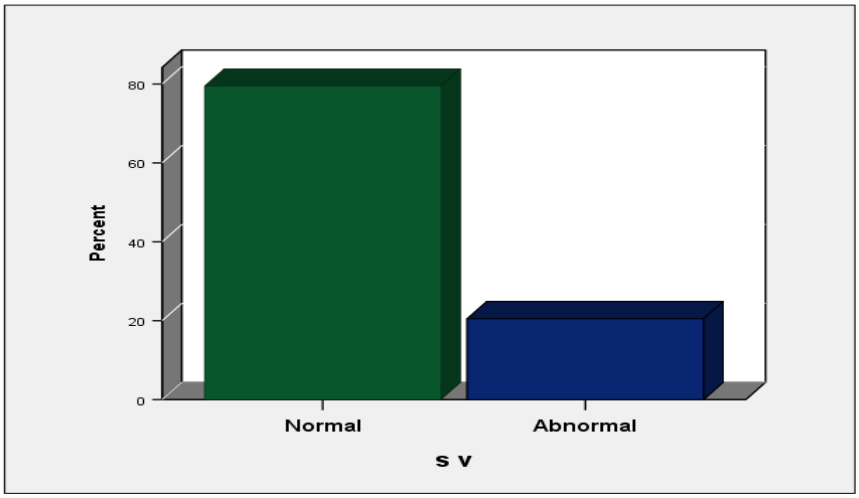

Figure4-11: Frequency percentage according stroke volume

Table (4-12):Frequency percentage according heart rate

HR		
	Frequency	Percent
Normal	174	87.0
Abnormal	26	13.0
Total	200	100.0

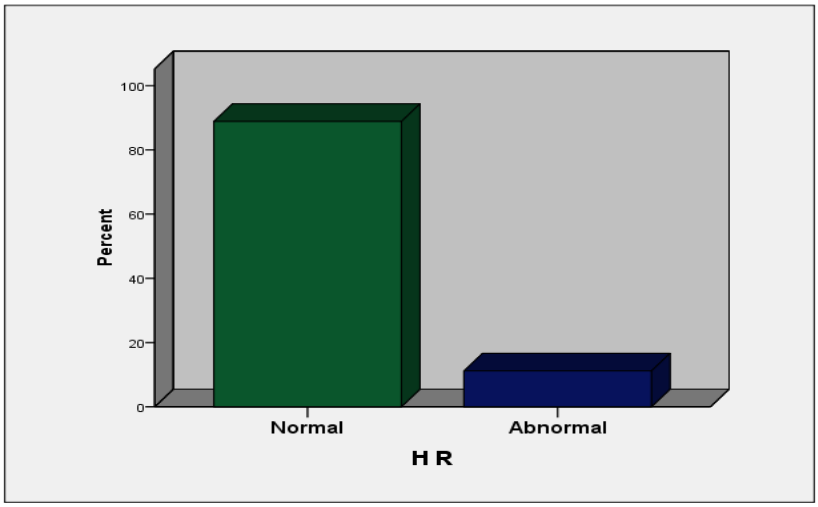

Figure4-12: Frequency percentage according heart rate

Table4-13: Chi-Square Tests Duration * septum

Duration		Septum		Total	Pearson Chi-Square	Sig (P-value)
		Normal	Abnormal			
1-24	Count	0	66	66	3.327[a]	.35
	Expected Count	.7	65.3	66.0		
25-48	Count	2	73	75		
	Expected Count	.8	74.2	75.0		
49-72	Count	0	36	36		
	Expected Count	.4	35.6	36.0		
more than 72	Count	0	20	20		
	Expected Count	.2	19.8	20.0		
Total	Count	2	195	197		
	Expected Count	2.0	195	197.0		

According this table p-value= 0.34 so there is no statistically significant association between duration and septum change.

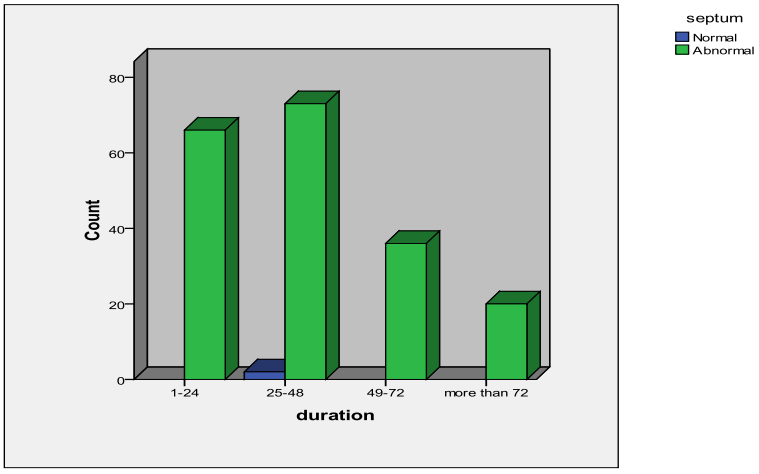

Figure4-13: Chi-Square of cystic change according Duration * Septum

Table4-14: Chi-Square Tests Duration * post

Duration		POST		Total	Pearson Chi-Square	Sig (p_value)
		Normal	Abnormal			
1-24	Count	18	48	66	6.156[a]	.10
	Expected Count	12.5	53.5	66.0		
25-48	Count	9	68	77		
	Expected Count	14.6	62.4	77.0		
49-72	Count	8	28	36		
	Expected Count	6.8	29.2	36.0		
more than 72	Count	3	18	21		
	Expected Count	4.0	17.0	21.0		
Total	Count	38	162	200		
	Expected Count	38.0	162.0	200.0		

According this table p-value= 0.10.There is no statistically significant association between duration and post change.

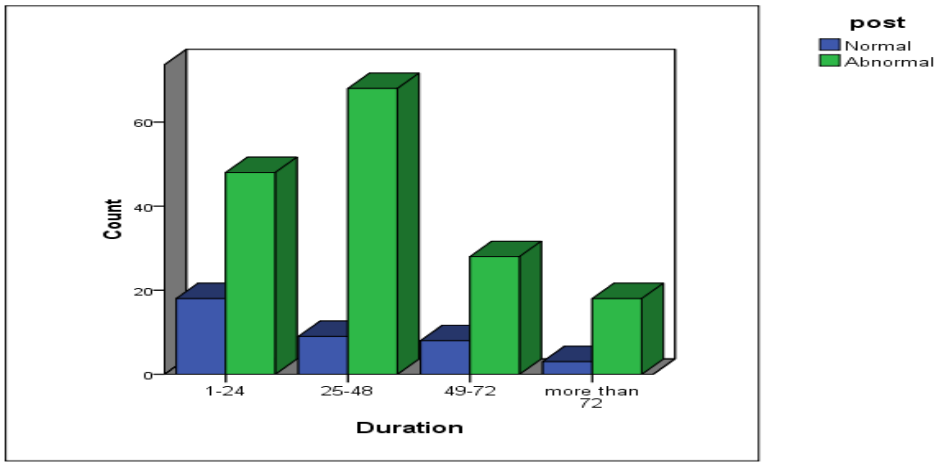

Figure4-14: Chi-Square of cystic change according Duration * post

Table4-15: Chi-Square Tests Duration * LVD

Duration		LVD		Total	Pearson Chi-Square	Sig (p_value)
		Normal	Abnormal			
1-24	Count	51	15	66	9.212[a]	.027
	Expected Count	50.2	15.8	66.0		
25-48	Count	51	26	77		
	Expected Count	58.5	18.5	77.0		
49-72	Count	33	3	36		
	Expected Count	27.4	8.6	36.0		
more than 72	Count	17	4	21		
	Expected Count	16.0	5.0	21.0		
Total	Count	152	48	200		
	Expected Count	152.0	48.0	200.0		

According this table p-value= 0.02. There is statistically significant association between duration and LVD change.

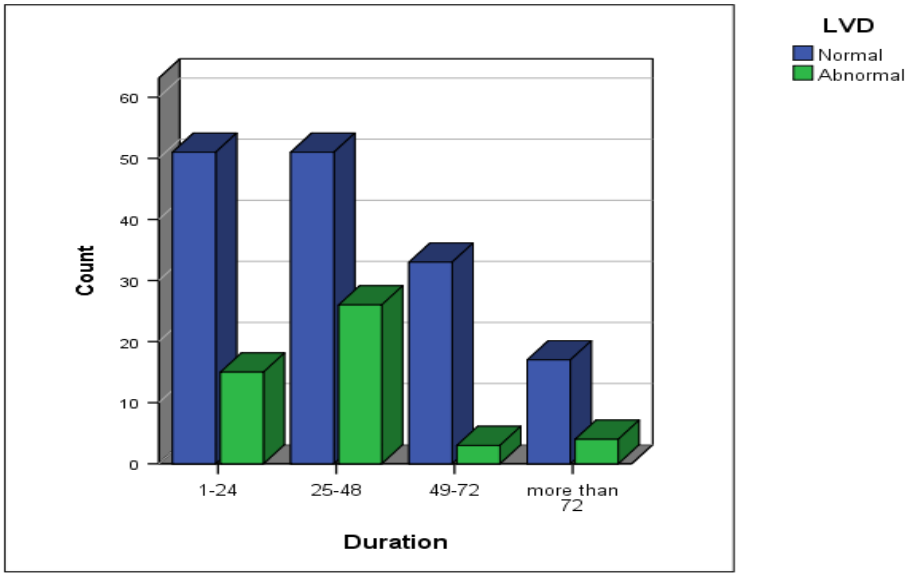

Figure4-15: Chi-Square of cystic change according Duration * LVD

Table4-16: Chi-Square Tests Duration * EF

Duration		EF		Total	Pearson Chi-Square	Sig (p_value)
		Normal	Abnormal			
1-24	Count	46	20	66	9.618[a]	.022
	Expected Count	41.6	24.4	66.0		
25-48	Count	47	30	77		
	Expected Count	48.5	28.5	77.0		
49-72	Count	16	20	36		
	Expected Count	22.7	13.3	36.0		
more than 72	Count	17	4	21		
	Expected Count	13.2	7.8	21.0		
Total	Count	126	74	200		
	Expected Count	126.0	74.0	200.0		

According this table p-value= 0.022. there is statistically significant association between duration and EF change.

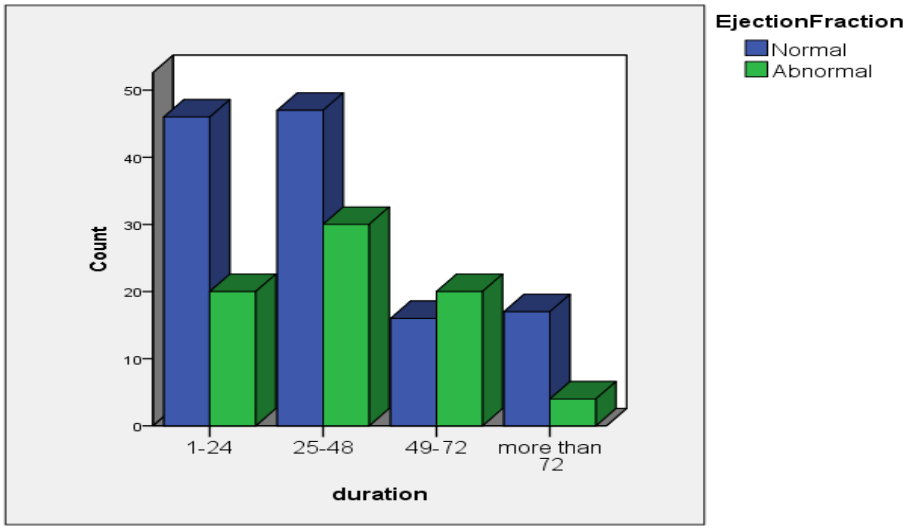

Figure 4-16: Chi-Square of cystic change according Duration * E_F

Table4-17: Chi-Square Tests Duration * COP

Duration		COP		Total	Pearson Chi-Square	Sig (p_value)
		Normal	Abnormal			
1-24	Count	52	14	66	3.287[a]	.35
	Expected Count	53.8	12.2	66.0		
25-48	Count	63	14	77		
	Expected Count	62.8	14.2	77.0		
49-72	Count	28	8	36		
	Expected Count	29.3	6.7	36.0		
more than 72	Count	20	1	21		
	Expected Count	17.1	3.9	21.0		
Total	Count	163	37	200		
	Expected Count	163.0	37.0	200.0		

According this table p-value= 0.35. There is no statistically significant association between duration and COP change.

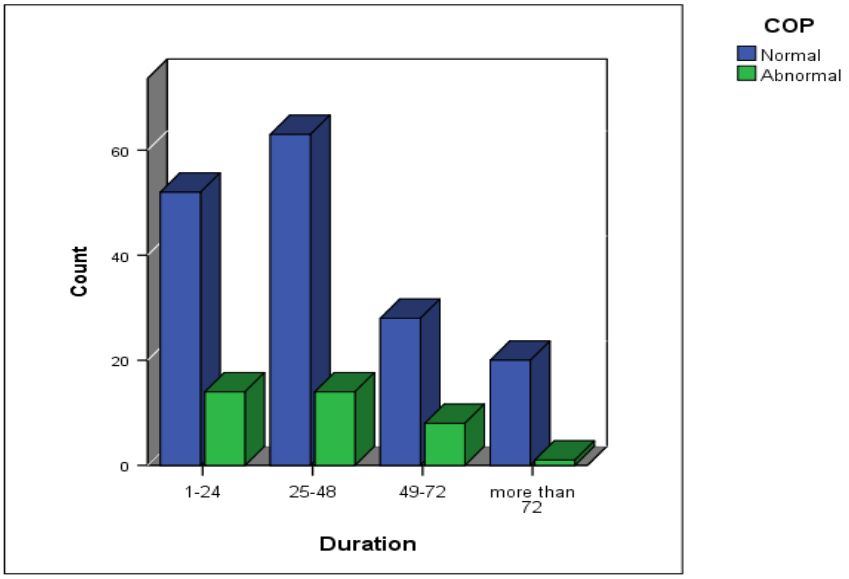

Figure4-17: Chi-Square of cystic change according Duration * COP

Table4-18: Chi-Square Tests Duration * SV

Duration		SV		Total	Pearson Chi-Square	Sig (p_value)
		Normal	Abnormal			
1-24	Count	50	16	66	5.548[a]	.136
	Expected Count	52.5	13.5	66.0		
25-48	Count	58	19	77		
	Expected Count	61.2	15.8	77.0		
49-72	Count	31	5	36		
	Expected Count	28.6	7.4	36.0		
more than 72	Count	20	1	21		
	Expected Count	16.7	4.3	21.0		
Total	Count	159	41	200		
	Expected Count	159.0	41.0	200.0		

According this table p-value= 0.13. There is no statistically significant association between duration and SV change

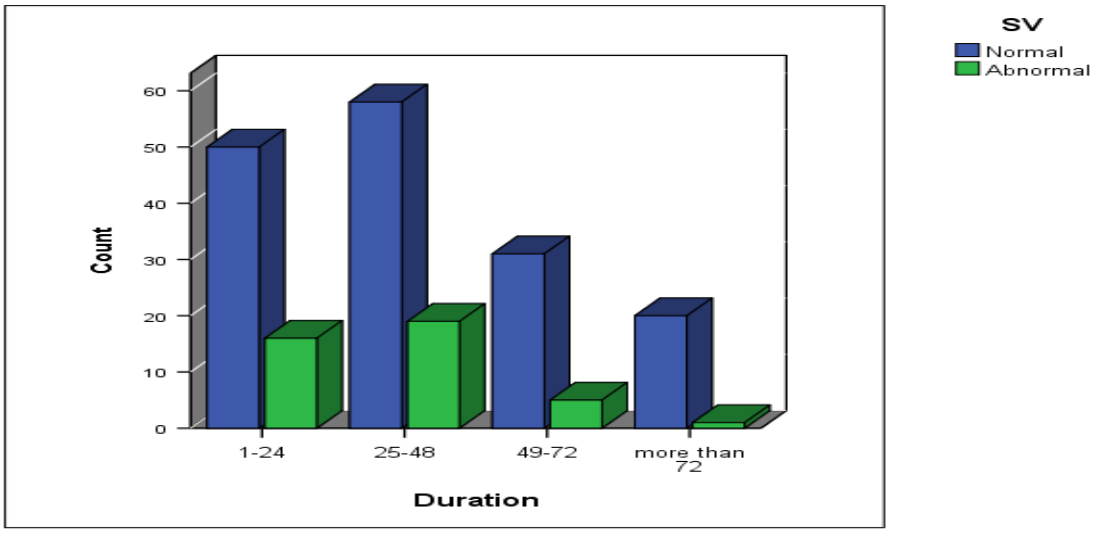

Figure4-18: Chi-Square of cystic change according Duration * SV

Table4-19: Chi-Square Tests Duration * HR

Duration		EF		Total	Pearson Chi-Square	Sig (p_value)
		Normal	Abnormal			
1-24	Count	61	5	66	3.445[a]	.328
	Expected Count	57.8	8.3	66.0		
25-48	Count	66	11	77		
	Expected Count	67.4	9.6	77.0		
49-72	Count	29	7	36		
	Expected Count	31.5	4.5	36.0		
more than 72	Count	19	2	21		
	Expected Count	18.4	2.6	21.0		
Total	Count	175	25	200		
	Expected Count	175.0	25.0	200.0		

According this table p-value= 0.13. There is no statistically significant association between duration and HR change.

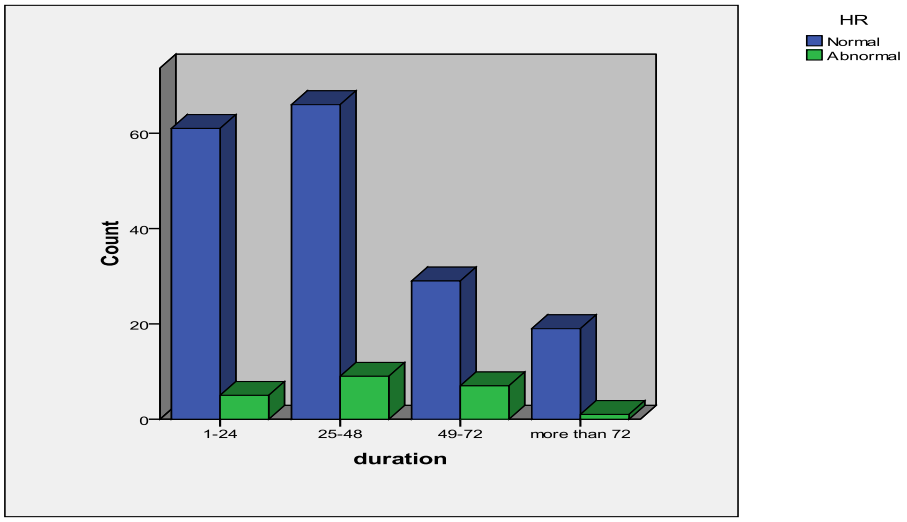

Figure4-19: Chi-Square according Duration * HR

Table4-20: Chi-Square Tests Duration * FS

Duration		EF		Total	Pearson Chi-Square	Sig (p_value)
		Normal	Abnormal			
1-24	Count	57	9	66	6.654[a]	.084
	Expected Count	52.8	13.2	66.0		
25-48	Count	55	22	77		
	Expected Count	61.6	15.4	77.0		
49-72	Count	29	7	36		
	Expected Count	28.8	7.2	36.0		
more than 72	Count	19	2	21		
	Expected Count	16.8	4.2	21.0		
Total	Count	160	40	200		
	Expected Count	160.0	40.0	200.0		

According this table p-value= 0.08. There is no statistically significant association between duration and FS change.

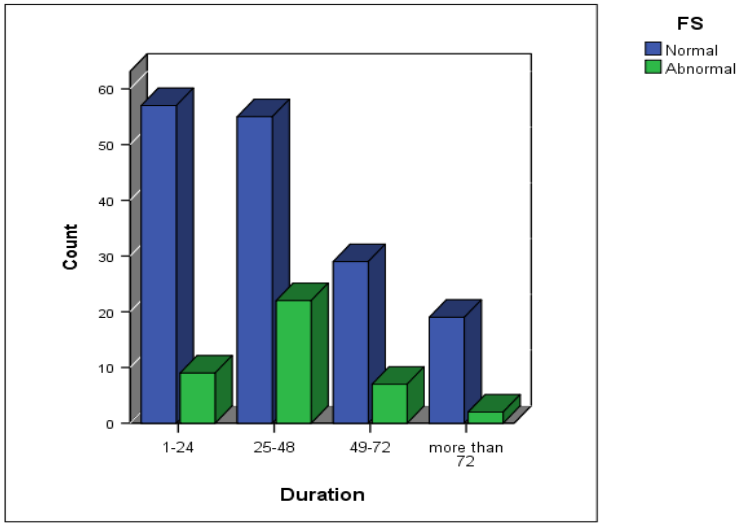

Figure4-20: Chi-Square according Duration * FS

Table (4-21): Correlations Variables Echocardiography:

Hemodialys findings	Sound waves on the Echocardiography							
	septum	post	LVD	E_F	COP	SV	HR	FS
Gender	.087	.034	-.204**	.087	-.283**	-.141*	.008	-.056
	.21	.62	.00	.21	.00	.047	.91	.43
Age	-.142*	-.101	.221**	.108	-.044	-.158*	.060	.266**
	.044	.15	.01	.129	.54	.025	.40	.00
weight	.027	-.163*	.083	-.047	.030	.111	-.264**	-.115
	.704	.02	.24	.51	.76	.18	.00	.10
Duration	.006	.083	-.084	.038	-.083	-.147*	.071	-.005
	.930	.24	.23	.59	.24	.037	.32	.94

**: Highly correlation Sig: Significant P < 0.05.

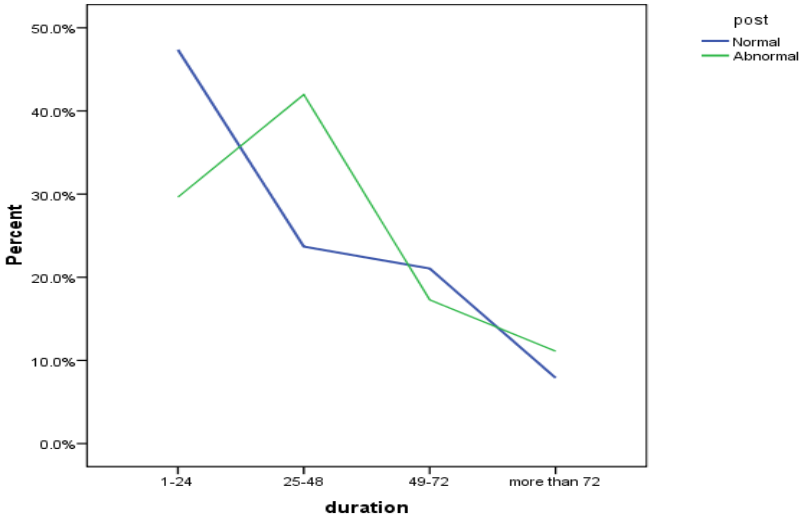

Figure4-21: hemodialysis Duration*post

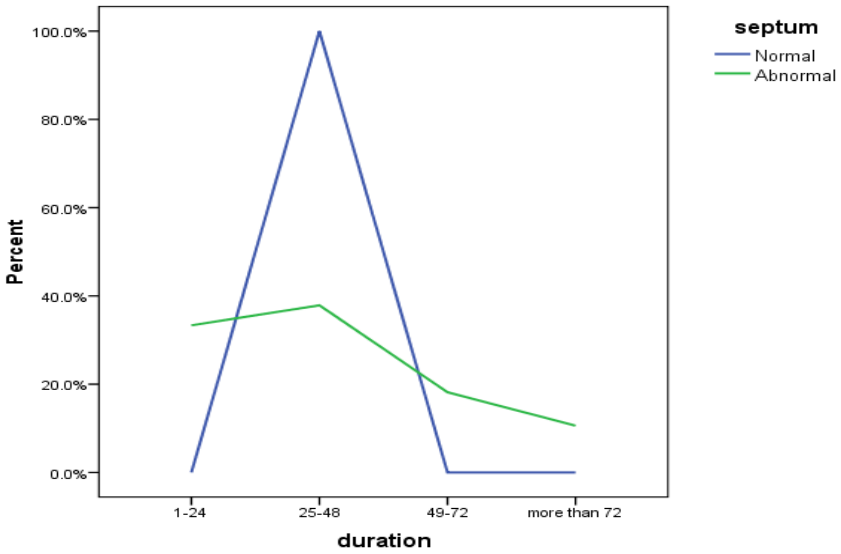

Figure4-22: hemodialysis Duration*Septum

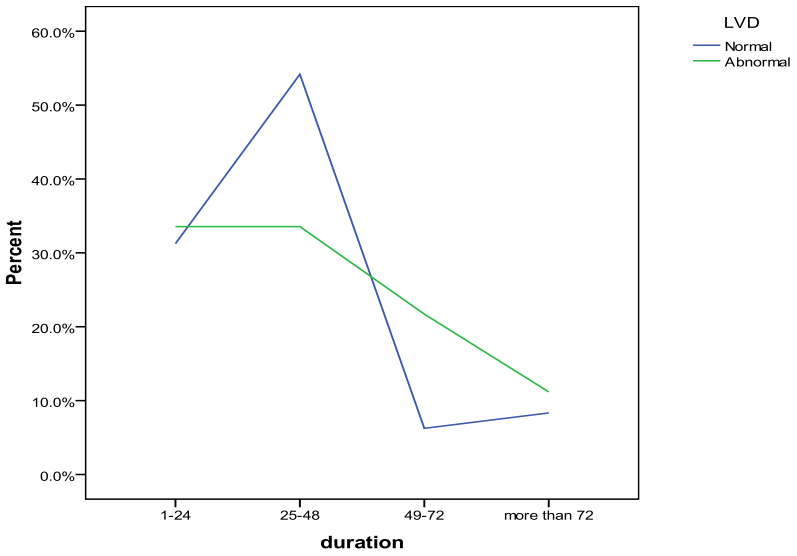

Figure4-23: hemodialysis Duration*LVD

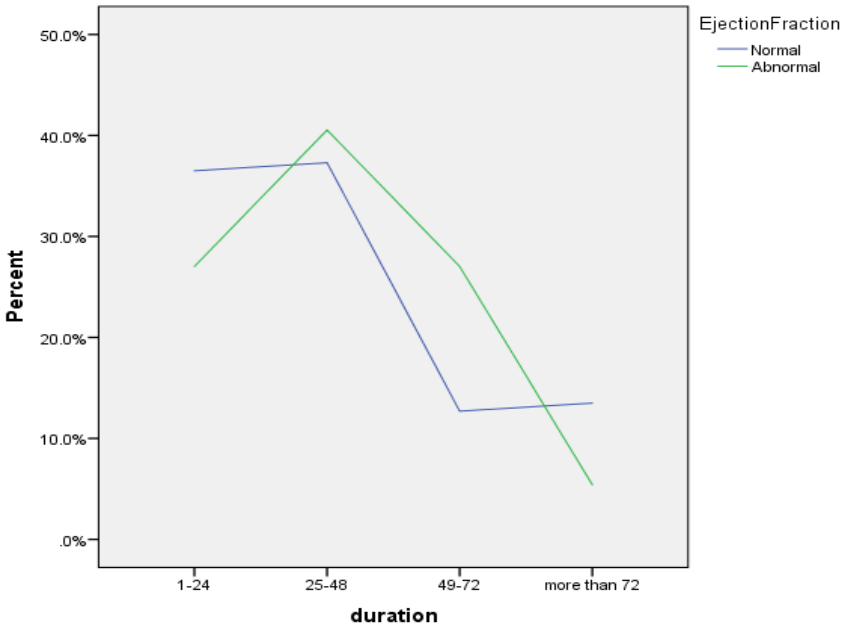

Figure4-24: hemodialysis Duration*E_F

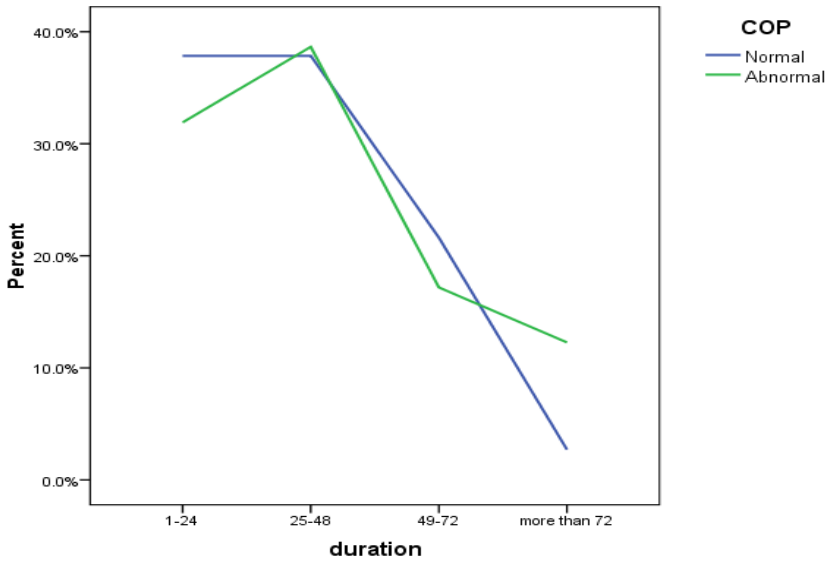

Figure4-25: hemodialysis Duration*COP

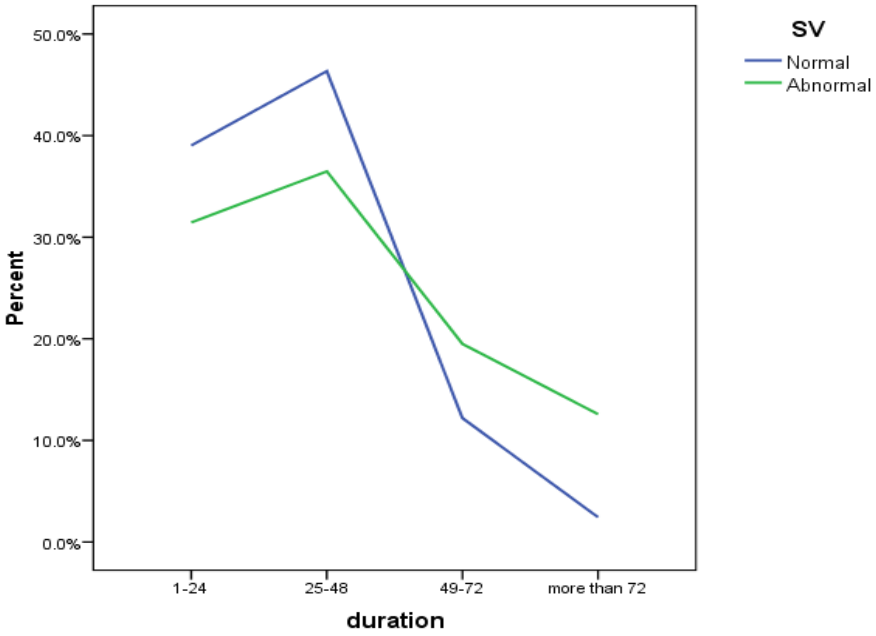

Figure4-26: hemodialysis Duration*SV

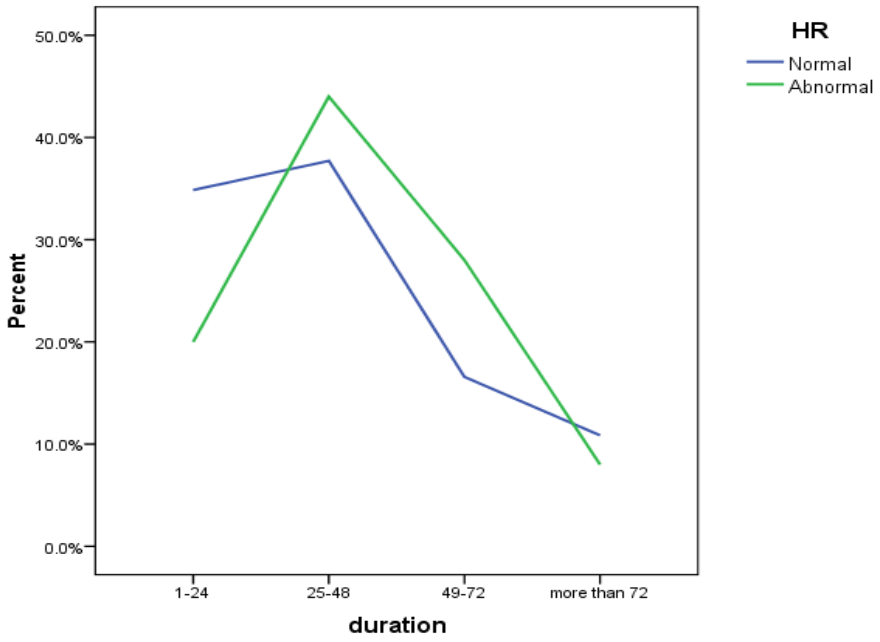

Figure4-27hemodialysis Duration*HR

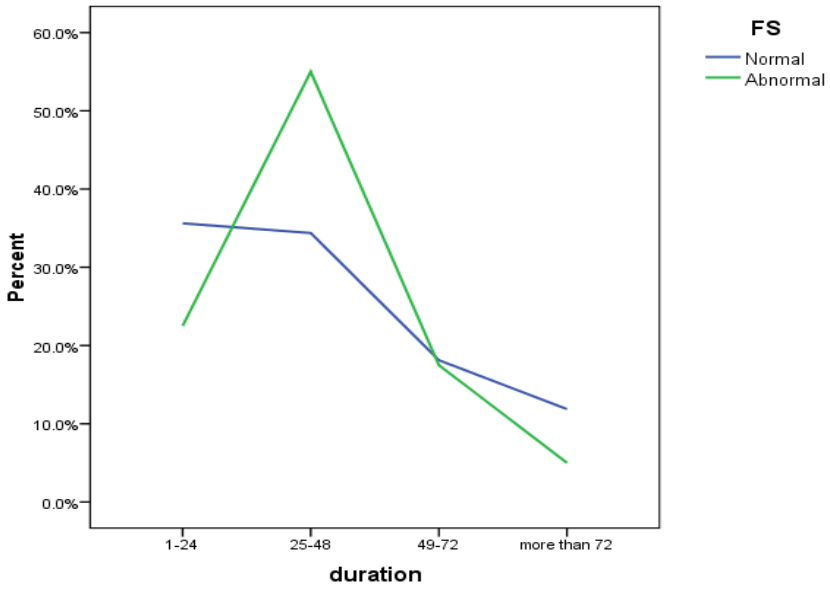

Figure4-28: hemodialysis Duration*FS

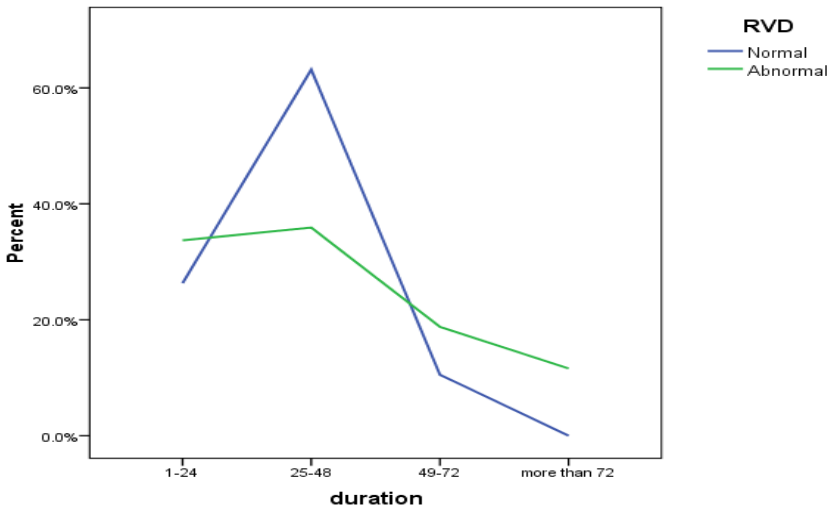

Figure4-29: hemodialysis Duration*RVD

Chapter Five

Discussion, Conclusion, and Recommendation

5.1 Discussion

This study was conducted in the renal center in Sennar Teaching Hospital on 200 patients on regular dialysis, 43% of them male, and 57% are female, this number is quietly different from Mustafa et al, who studied 42 patients, in Ankara in 2010, they found that 50% are male and 50% female and this higher number may be due to that here in Sudan women using painkillers more, and hair dye for cosmetics. (Table 4-1) and figure (4-1)

These patients categorize age wise into 4 groups, the most frequent age group ranged from 20 to 30 years old composed of 54patients which are 27%, and the next group is 30 to 40 years old composed of 44patients which are 22.0%. The third group more than 60 years old composed of 42patients which are 21.0% and the fourth group from 50 to 60 years old composed of 36patients which are 18.0% and the last group is 40 to 50 years old composed of 24patients which are 12.0%small. (Table4-2) and figure (4-2).

The body weight of 110patients was ranged from 40.0 -60.0 kg and represented 55%، 62patients (31%) more than 60.0 kg and the last index of 28patients (14%) were 20.0 -40.0 kg, respectively hemodialysis always associated with weight loss. (Because of the filtering of blood sugar). (table4-3) and figure (4-3).

This study showed that 38% of the patients stand on dialysis for 25-48 months followed by 33% on dialysis for 1-24 months (66 patients) then 18% (36 patients) stand for 49-72 months and finally 11%(22patients) stand for more than 72 months. (Table4-4) and figure (4-4).

This research showed that only 1% of patients (2patients) have normal septum thickness and 99% (198 patients) had thick septum wall. (table4-5) and figure (4-5), in considering posterior wall thickness 19% of patients (38patients)had normal

posterior wall thickness, and 81% (162 patients) had a thick posterior wall. (Table4-6) And figure (4-6). This is a little bit higher than *Foley et a*l. who's studied 432 dialysis patients prospectively in the 1980s focused on distortions of left ventricular architecture, namely LVH. They found that 75% had LVH. (*Foley RN, Parfrey Pet al 1995)*, (*Foley RN, Parfrey Pet al 1996),*. And also more than Damir et al 2015 whose studied 50 patients on regular HD, they found that LV hypertrophy was observed in 78% of patients*(Damir et al 2015).*and relatively in line with Zoccali et al. they studied 254 patients in 2008 they concluded that77% had LVH. (*Zoccali et al. 2008)* and more near to Mallamaci et al. whose studied 246 patients in 2001 and found that 79% had LVH.*(Mallamaci et al. 2001)*.

Where that 76% of patients (152patients) had dilated cardiomyopathy left ventricular diameter at end diastole (LVD) is more than 53mm, the residual 24% (48patients) had a normal left ventricular diameter. (Table4-7) and figure (4-7).

Regarding left ventricular Ejection fraction (EF) 63% of patients (126patients) had Normal EF and 37% (74 patients) suffering different grade of systolic dysfunction. (table4-8) and figure (4-8). And 80% (160 patients) of patients had normal fractioning shorting, and 20%of cases (40 patients) had reduced wall motion. (table4-9) and figure (4-9).And this proved by cardiac output where 81 %(162patients) of patients had normal cardiac output and 19 %(38 patients) had reduced cardiac output. (table4-10) and figure (4-10). This study cleared that 79 %(158patients) of patients had normal stroke volume and 21% (42patients) had small stroke volume. (Table4-11) Figure (4-11).This is a little bit higher than *Foley et a*l. whose studied 432 dialysis patients prospectively in the 1980s focus on LV function They found that 31% had systolic dysfunction. (*Foley RN, Parfrey Pet al 1995)*, (*Foley RN, Parfrey Pet al 1996)*.And relatively in line with Zoccali et al. they studied 254 patients in 2008 they concluded that 22% had systolic dysfunction. (*Zoccali et al. 2008)* and more near to Mallamaci et al. whose studied

246 patients in 2001 and found that 13% had systolic dysfunction.*(Mallamaci et al. 2001)*.And all most match with Greaves et al. whose studied 84 patients in 1994, and they found that 36% of patients have systolic dysfunction*(Greaves et al 1994*).

This research showed that: 87.0% of patients had normal heart rate and another hand 13.0% had tachycardia. (Table4-12) and figure (4-12), so there is no statistically significant association between duration and septum change (P-value= 0.34). (Table4-13) and figure (4-13).

There is no statistically significant association between duration and post change (P-value= 0.10.). (Table4-14) and figure (4-14).

There is statistically significant association between duration and LVD change (p-value= 0.02.). (Table4-15) and figure (4-15).

There is statistically significant association between duration and EF change (P-value= 0.022). (Table4-16) and figure (4-16).

P-value= 0.35. There is no statistically significant association between duration and COP change. (Table4-17) and figure (4-17).

There is no statistically significant association between duration and SV change (p-value= 0.13.) (Table3-18) and figure (4-18).

There is no statistically significant association between duration and HR change(p-value= 0.13.). (Table4-19) and figure (4-19).

There is no statistically significant association between duration and FS change (P-value= 0.08.). (Table4-20) and figure (4-20).

Compare to patients with Echocardiography studies. Age levels show negative correlations with Septum, Posterior wall, cardiac output, and a positive correlation with, LVD, Ejection fraction, heart rate and fraction shortening. Weight levels show negative correlations with a posterior wall, Ejection fraction, heart rate,

fractioning shorting and a positive correlation with Septum, LVD, cardiac output, stroke volume

Duration levels showed negative correlations with LVD, cardiac output, Stroke Volume, fraction shortening and a positive correlation with Septum, posterior wall, Ejection fraction, heart rate (Table 4-21).

The left ventricular mass index (HR) wall thickness, (FS) wall thickness, (COP) wall thickness, and (LVD) and (SV) are significantly high as compared to control. On the other hand, the (Septum), (POST) and (EF) is significantly low. (Table 4-22).

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:

- Trans thoracic echocardiography could be used as a routine checkup, follow up to help treatment hemodialysis patients
- Tran's thoracic echocardiography is very important to ESKD patients to detect the complications as an ischemic cardiac disease, systolic and diastolic left ventricular dysfunction, and cardiac dimensions.
- Advice to have Tran's thoracic echocardiography department in any renal center.
- Need further research to detect changes that may occur in the other heart structure like Valvular lesion, pericardial disease, and right side heart disease.

5-3 Conclusions:

This study has been done in sinnar teaching hospital for 200 ESKD patients on hemodialysis their age above20 years old (88male, 112 female), any patient with known history of heart disease is excluded.

The goal of the study is evaluation left the ventricular systolic function in patients with chronic renal failure on regular hemodialysis by Tran's thoracic echocardiography finding regarding ejection fraction, stroke volume, cardiac output, and left ventricular walls and cavity dimensions.

The study concluded that 99% had a thick septum, 81% thick posterior wall, 76% dilated cardiomyopathy, 63% impaired systolic dysfunction, 79% reduced stroke volume, 13% tachycardia, and 80% reduced wall motion.

The study showed that the Age had negative correlations with Septum, Posterior wall, LVD, Fraction Shortening, and a positive correlation with, LVD, stroke volume, cardiac output and heart rate. In related Weight levels show negative correlations with heart rate, posterior wall, cardiac output, fractioning shorting, stroke volume and a positive correlation with LVD, Ejection fraction, and Septum, but the Duration showed negative correlations with Ejection fraction, Septum and a positive correlation with stroke volume, posterior wall, heart rate and cardiac output, LVD, fraction shortening. (Table3-21).

The left ventricular mass index, (HR), (FS), (COP), and (LVD) and (SV) are significantly high as compared to control. On the other hand, the (SEPTUM), (POST) and (EF) is significantly low ESKD patients have hypertrophied dilated hearts, so it is easier to assess heart by echocardiography than the other normal patients.

Tran's thoracic echocardiography scanning is very important to detect any change that may occur in the heart during long-term hemodialysis; further research needs in hearts of these patients.

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.

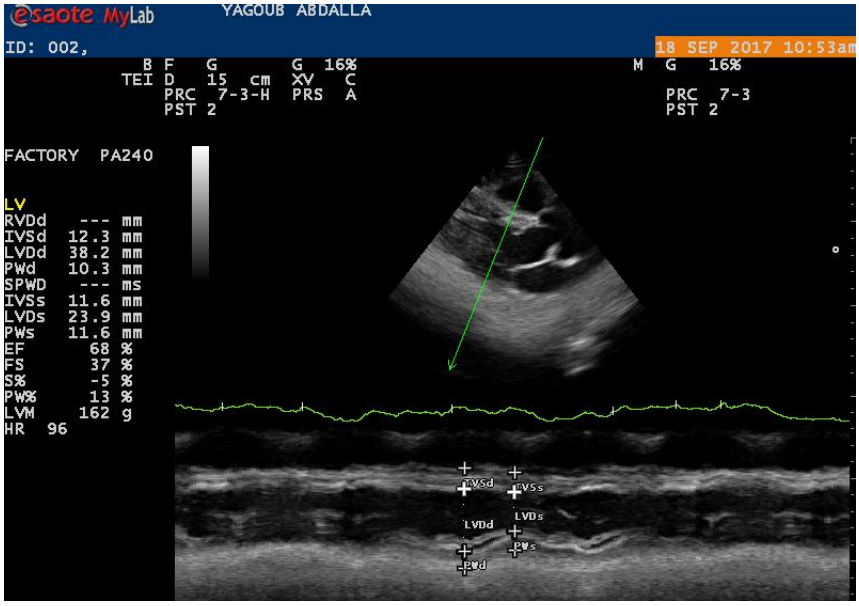
Appendices

Appendix (1)

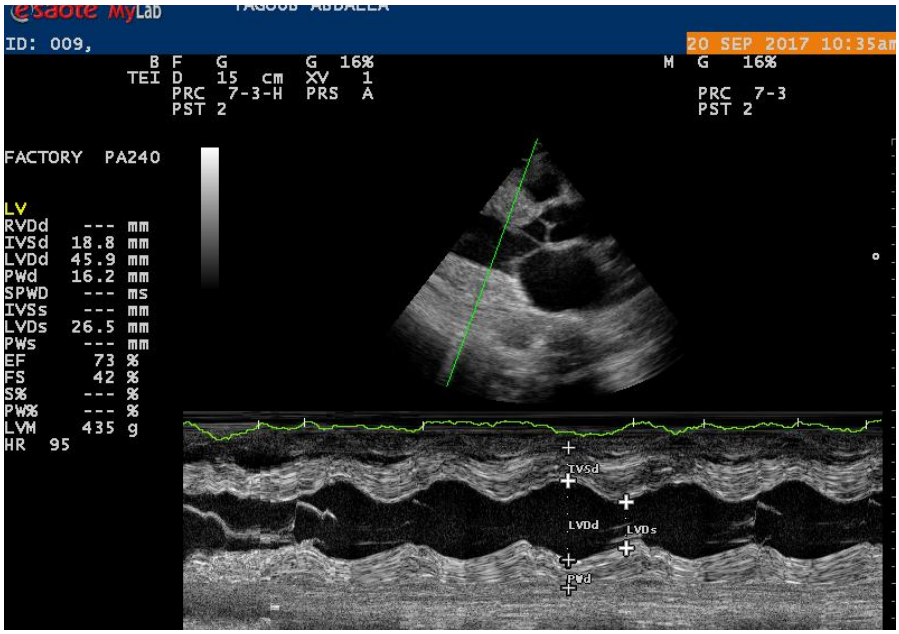
Data collection sheet

no	Gender	Age	Wight	Duration	Septum thickness	Post wall	LVD	LVS	EF	SV	HR	COP	FS
1													
2													
3													
4													
5													
6													
7													
8													
9													
10													
11													
12													
13													
14													
15													

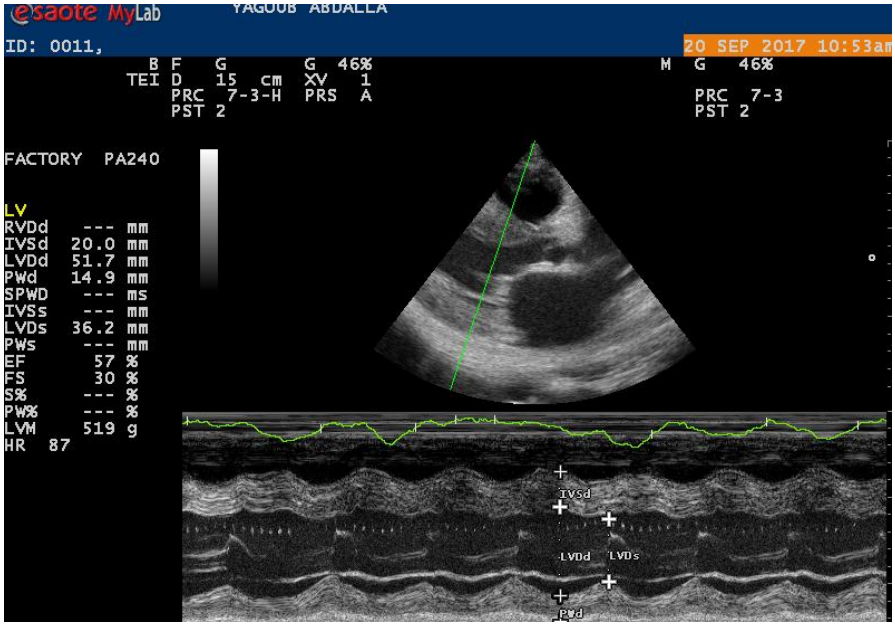
Appendix (2)



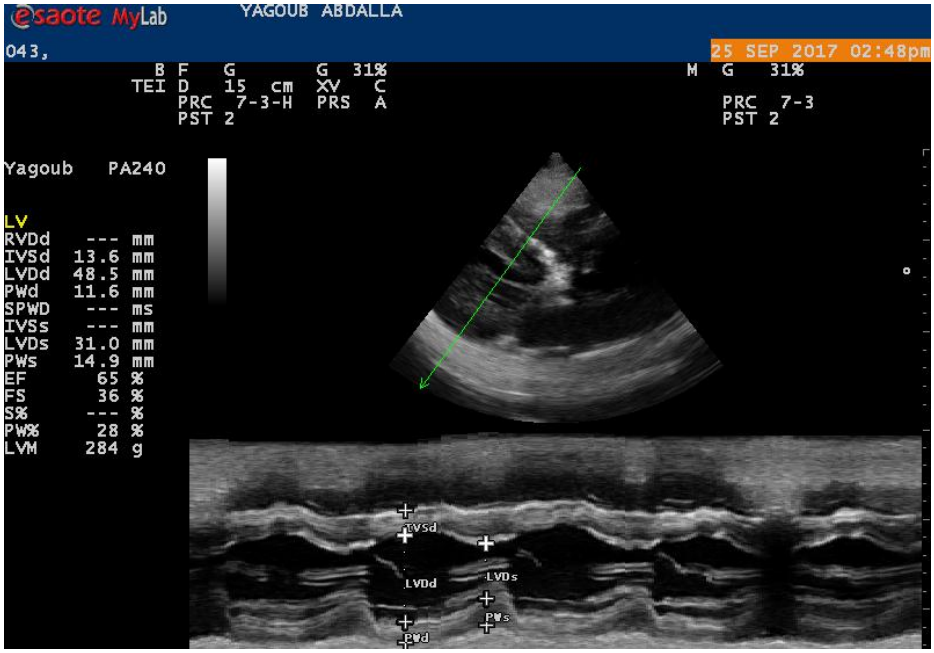
Case (1) 62years old male on dialysis for 2 years with normal systolic function EF 68%



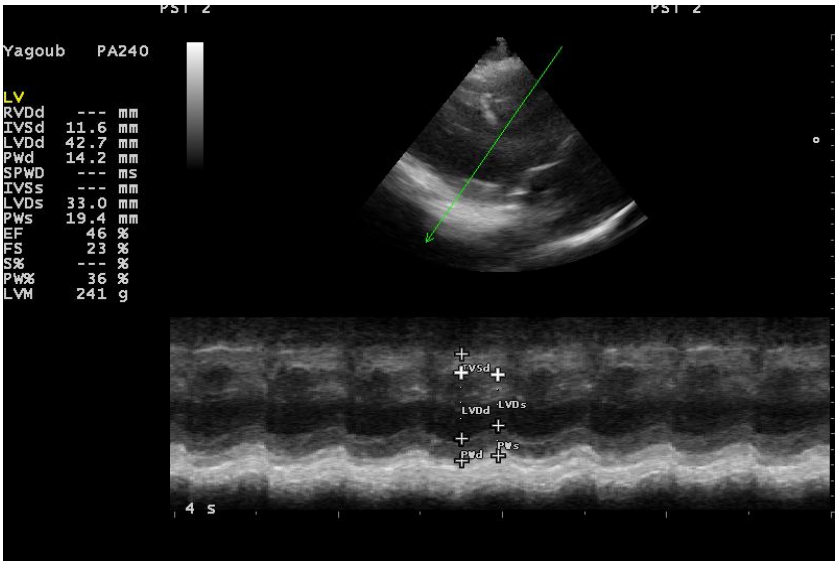
Case (2) 55years old male on dialysis for 3 years with normal systolic function EF73%



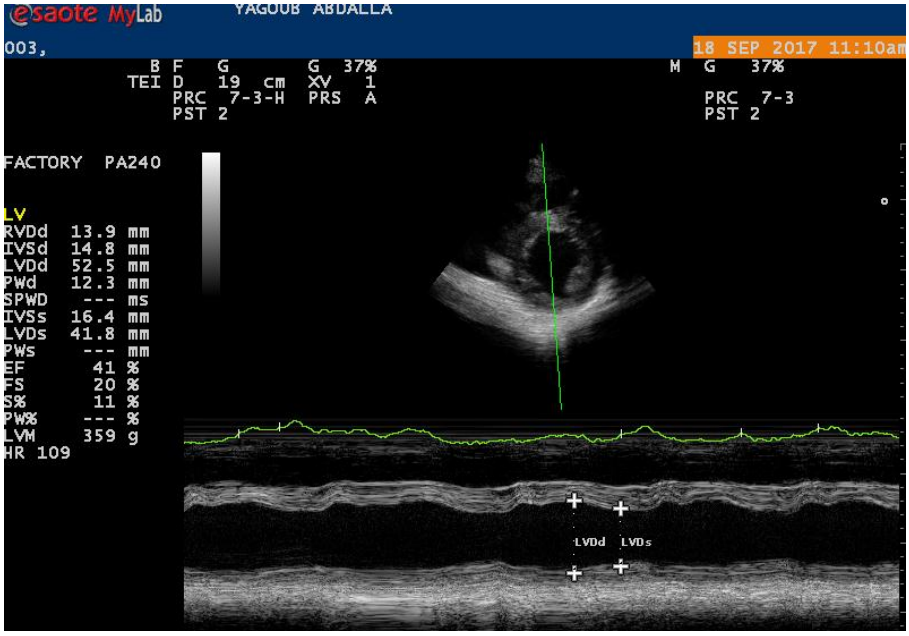
Case (3)25years old male on dialysis for 5 years with normal systolic function EF 57%



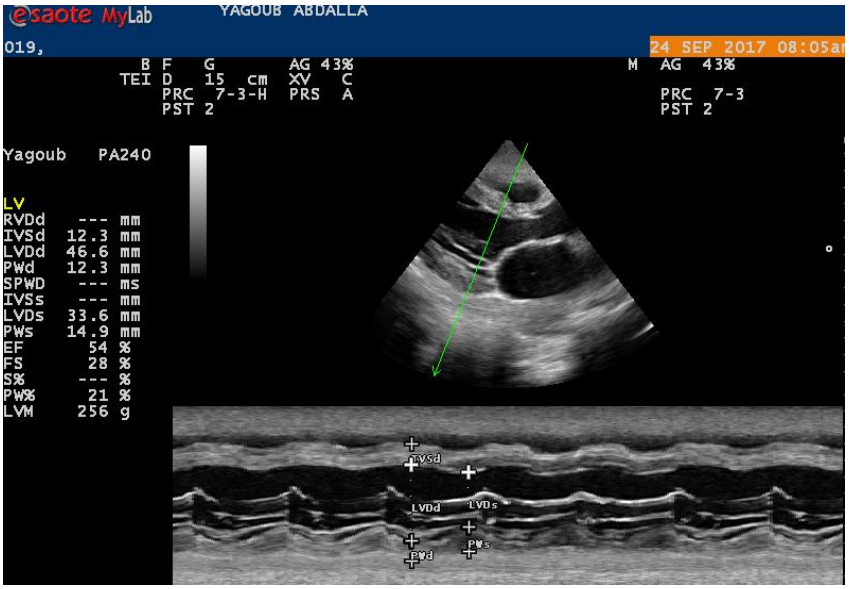
Case (4)50years old female on dialysis for 3 years with normal systolic function EF65%



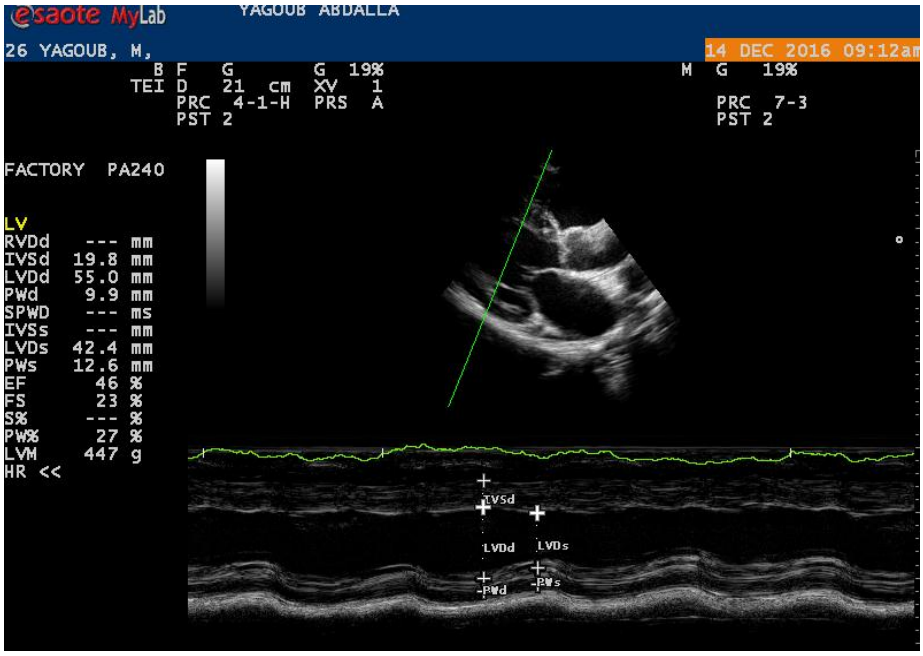
Case (5)43years old female on dialysis for 4 years with systolic dysfunction EF 46%



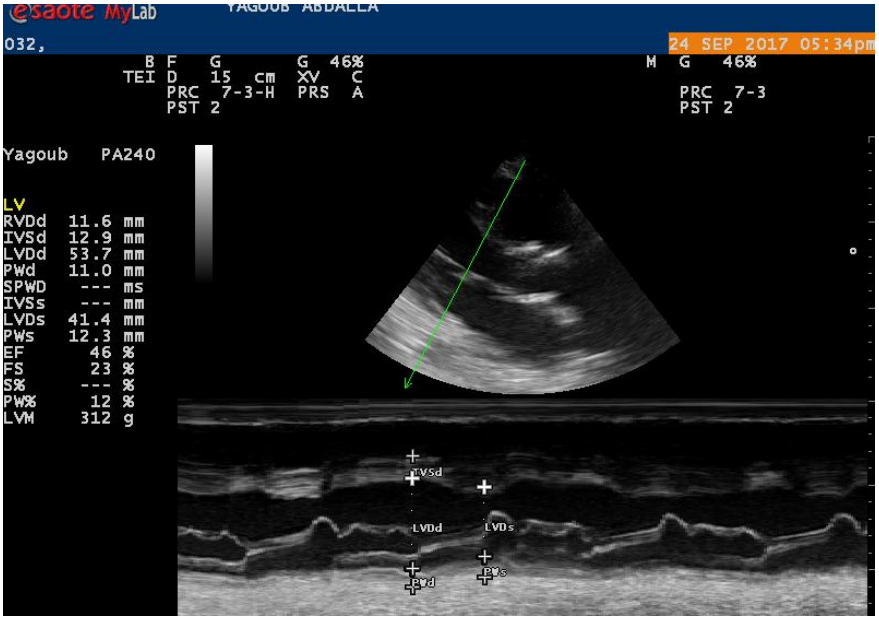
Case (6)30years old female on dialysis for 5 years with systolic dysfunction EF 41%



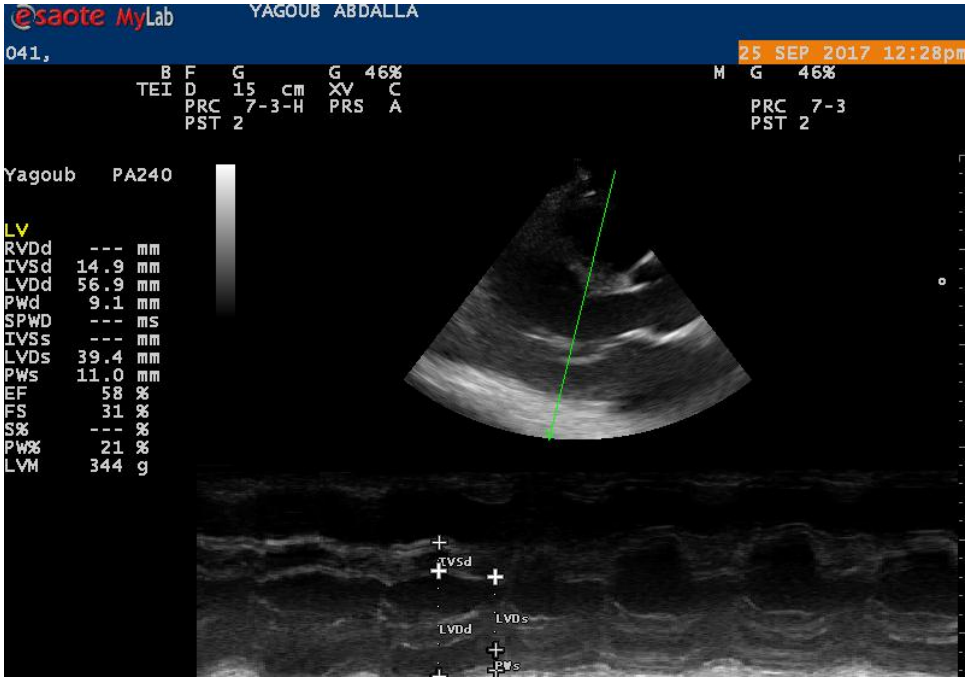
Case (7)45years old male on dialysis for 3 years with systolic dysfunction EF 54%



Case (8)42years old female on dialysis for 4 years with DCM, LVD 55mm.

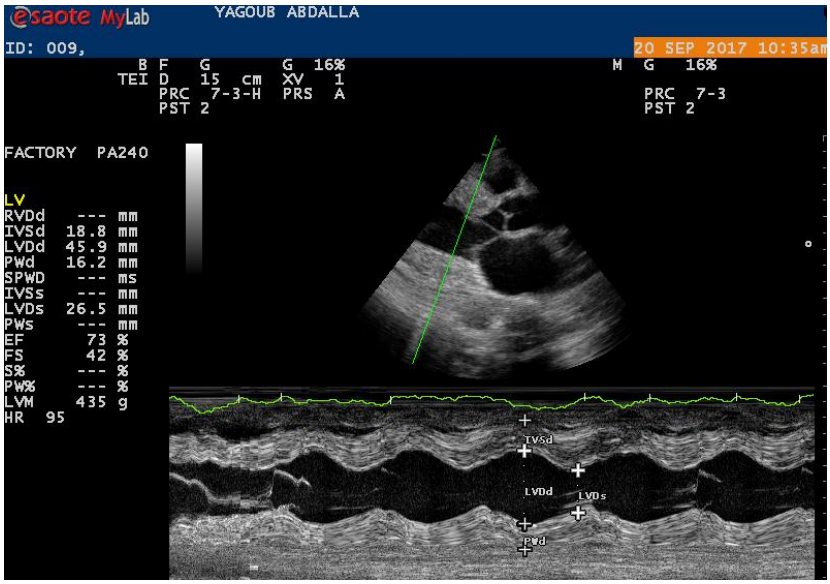


Case (9)32years old female on dialysis for 1year with DCM, LVD 54mm

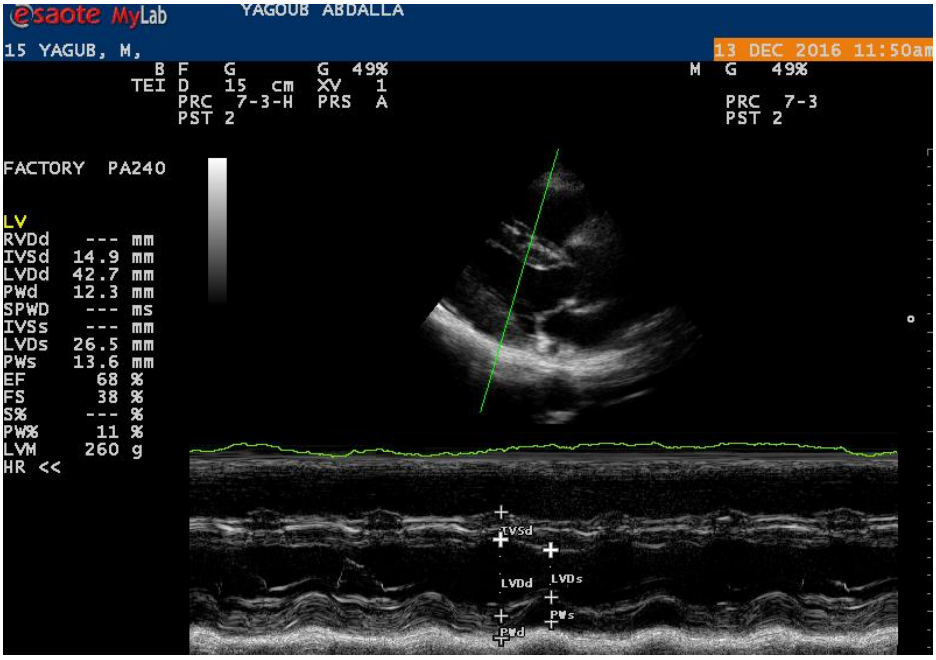


Case (10)60years old male on dialysis for 3years with DCM, LVD 57mm.

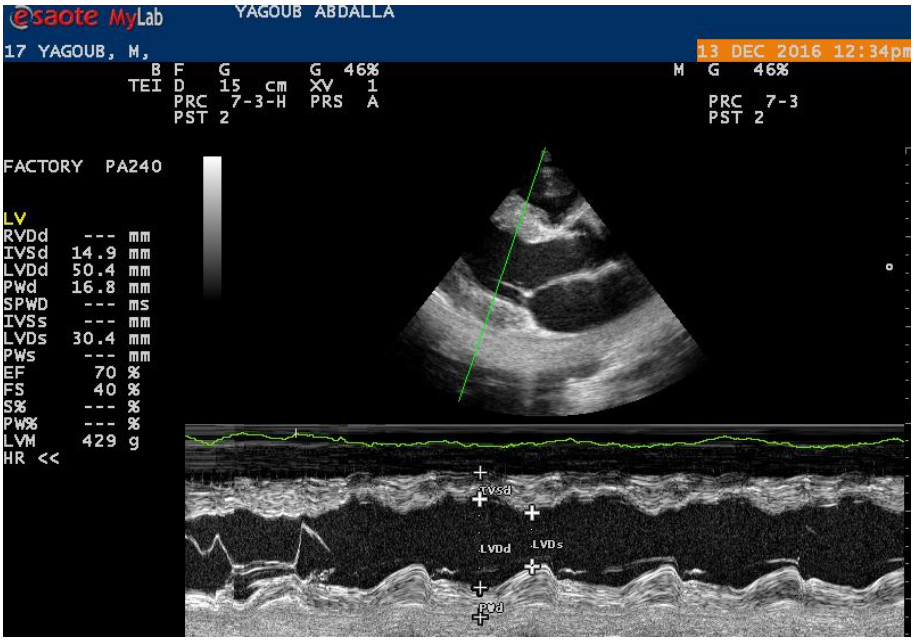
LVH



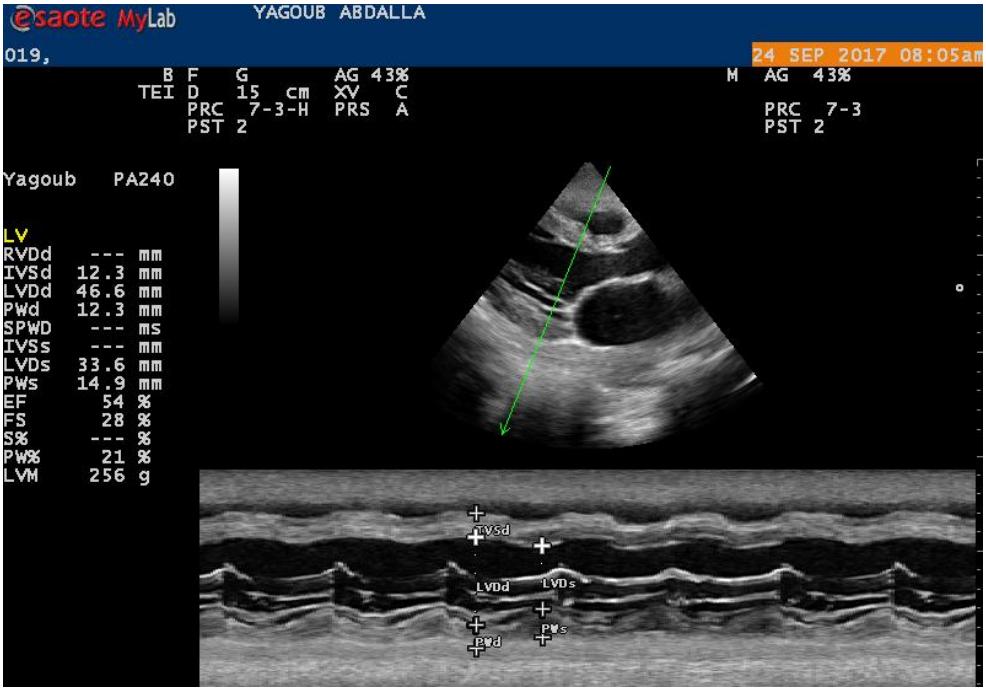
Case (11)55years old male on dialysis for 3 years with LVH, septum is 18.8mm.



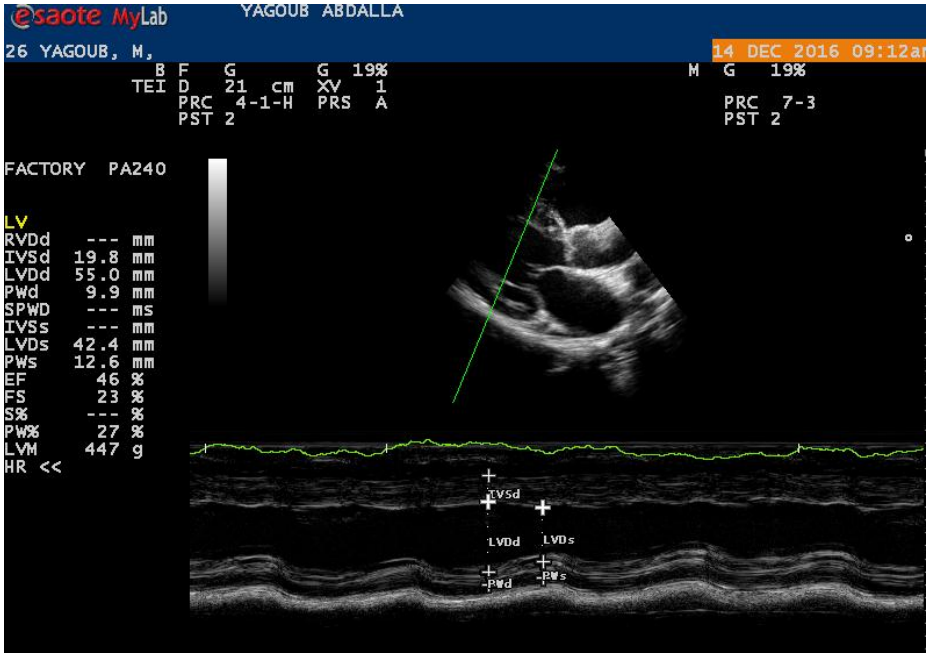
Case (12)62years old male on dialysis for 2 years with LVH septum 14.9mm.



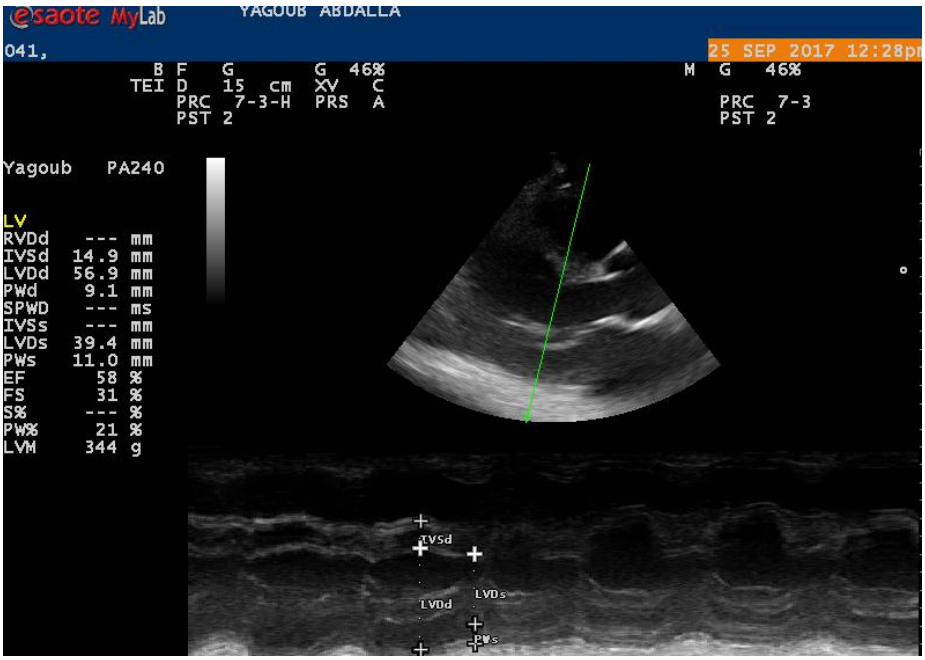
Case (13)56years old male on dialysis for 4 years with LVH, septum is 14.9mm.



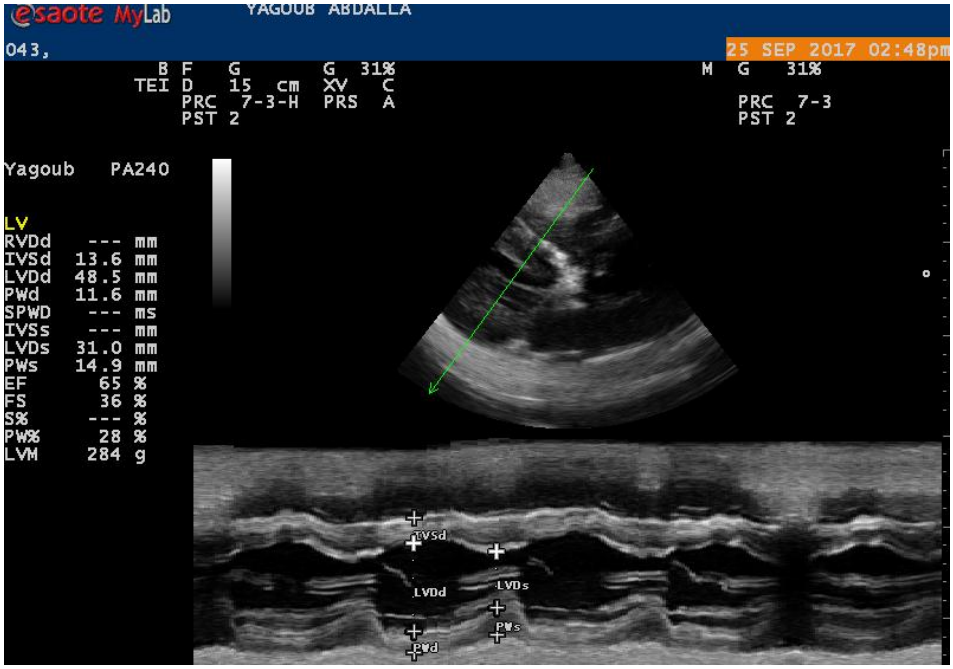
Case (14)45years old male on dialysis for 3 years with LVH, septum thickness 12.3mm.



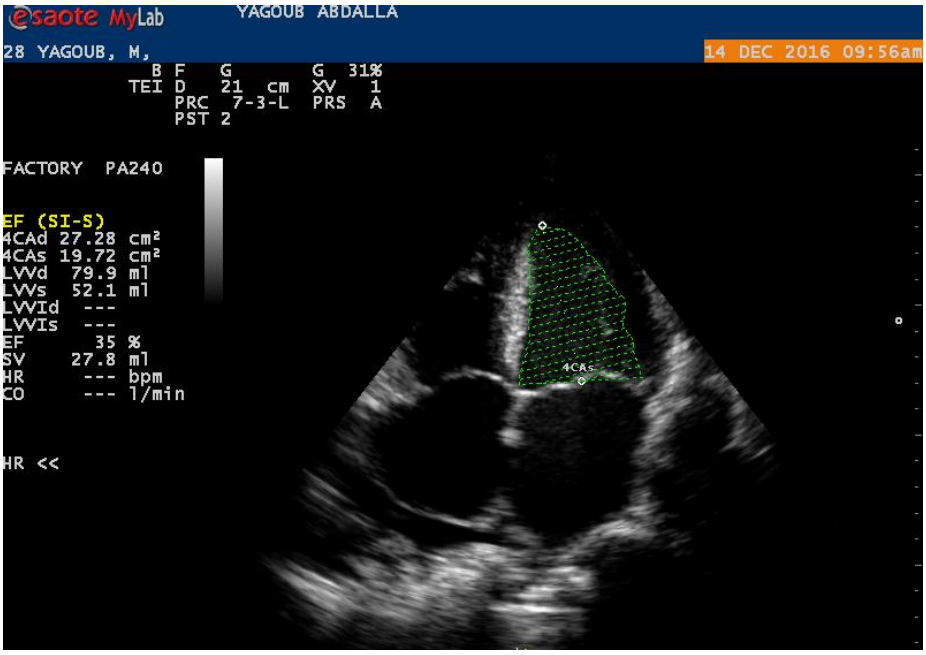
Case (15)42years old male on dialysis for 6 years with LVH, septum thickness 19.8mm



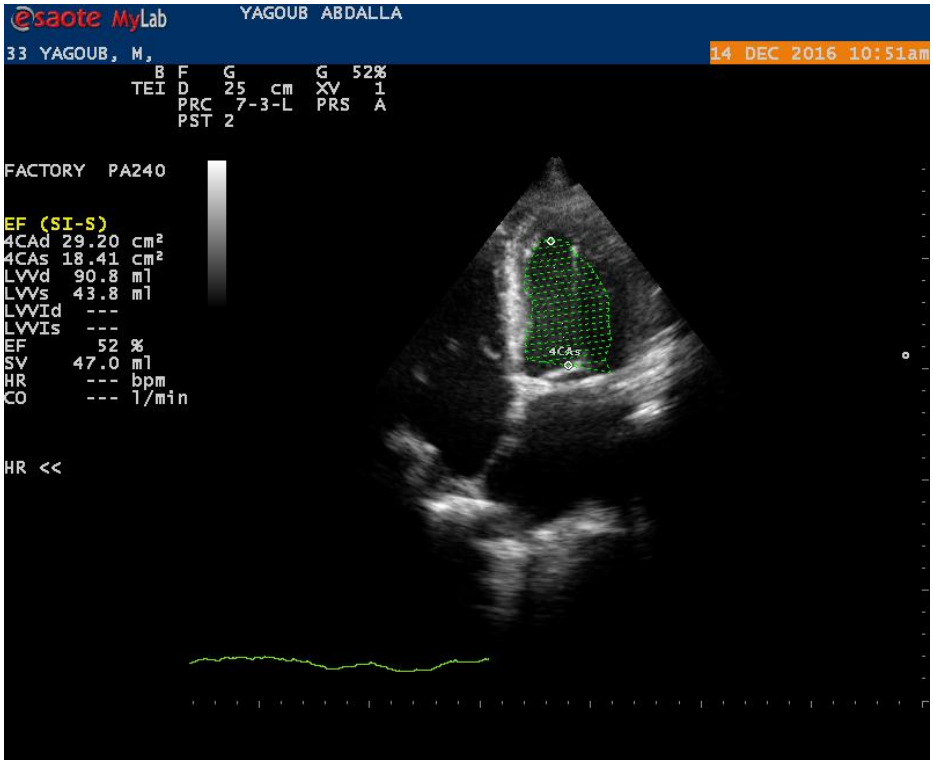
Case (16)28years old female on dialysis for 4 years with LVH septum thickness is 14.9mm



Case (17)50years old female on dialysis for 3 years with LVH, septum thickness is 13.6mm.



Case (18)68years old male on dialysis for 1 year with impaired systolic function EF 35%, stroke volume is 27.8ml



Case (19)36years old male on dialysis for 3 years with EF 52%, stroke volume is 47ml

My lab gold 30

My lab gold 30